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AFRICA'S LAGGING DEMOGRAPHIC TRANSITION:  
EVIDENCE FROM EXOGENOUS IMPACTS OF MALARIA ECOLOGY AND AGRICULTURAL TECHNOLOGY

Dalton Conley  
Gordon C. McCord  
Jeffrey D. Sachs

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Africa's Lagging Demographic Transition: Evidence from Exogenous Impacts of Malaria Ecology and Agricultural Technology

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**ABSTRACT**

Much of Africa has not yet gone through a "demographic transition" to reduced mortality and fertility rates. The fact that the continent's countries remain mired in a Malthusian crisis of high mortality, high fertility, and rapid population growth (with an accompanying state of chronic extreme poverty) has been attributed to many factors ranging from the status of women, pro-natalist policies, poverty itself, and social institutions. There remains, however, a large degree of uncertainty among demographers as to the relative importance of these factors on a comparative or historical basis. Moreover, econometric estimation is complicated by endogeneity among fertility and other variables of interest. We attempt to improve estimation (particularly of the effect of the child mortality variable) by deploying exogenous variation in the ecology of malaria transmission and in agricultural productivity through the staggered introduction of Green Revolution, high-yield seed varieties. Results show that child mortality (proxied by infant mortality) is by far the most important factor among those explaining aggregate total fertility rates, followed by farm productivity. Female literacy (or schooling) and aggregate income do not seem to matter as much, comparatively.

Dalton Conley  
Department of Sociology  
New York University  
295 Lafayette Street, 4th Floor  
New York, NY 10012  
and NBER  
dalton.conley@nyu.edu

Jeffrey D. Sachs  
The Earth Institute at Columbia University  
314 Low Library  
535 West 116th Street, MC 4327  
New York, NY 10027  
and NBER  
sachs@columbia.edu

Gordon C. McCord  
The Earth Institute at Columbia University  
314 Low Library  
535 West 116th Street, MC 4327  
New York, NY 10027  
gm2101@columbia.edu

## *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. And in this vein, many refinements have been made to these models over the last decade.

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.) Are Africa's high fertility rates the result of illiteracy, poverty, rural populations, discrimination against girls and women, high child mortality, lack of access of the poor to contraception? What policies would be most supportive of a rapid, voluntary fertility transition? This paper seeks to provide some new quantitative assessments 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 (1984) posits 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 ONE ABOUT HERE]

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, improved nutrition) were seen to be the basic driver to reduced fertility rates as well, albeit with a lag of one or more generations. The 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.

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. Several scholars have shown that reduced family size affects human capital investment (Angrist, Lavy and Schlosser 2004; Conley and Glauber 2006; Black, Devereux and Salvanes 2006) on the micro level as well as economic growth at the macro level (Behrman and Rosenzweig 2001; Hazan and Berdugo 2002; Joshi and Schultz 2005; Moav 2005). Likewise, the argument can be made that at least some of the powerful correlation of high child mortality and high fertility represents increased child mortality due to higher fertility thanks to increased strain on household caloric resources and decreased parental care and supervision with the addition of more children. Similarly, government “family planning efforts” could be reflecting (rather than affecting) demand for reduced family size. With these concerns in mind, in this paper we pursue a strategy of deploying some largely exogenous variation in geophysical conditions and agricultural technology to derive estimates of the causal impact of infant (as a proxy for child) mortality, rural productivity, availability of abortion, literacy and income on fertility. We then discuss the implications of these findings for the lagging demographic transition in Africa.

To preview our empirical approach, we argue that the ecology of malaria transmission is exogenous to fertility over the time period we are studying. Malaria is currently the fourth leading cause of death (after neonatal disorders, diarrhea, and pneumonia) for children under five in low income countries<sup>1</sup> and is responsible for at least one in every five child deaths in sub-Saharan Africa.<sup>2</sup> Estimates of malaria

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<sup>1</sup> See Black (2003).

<sup>2</sup> 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.

mortality in Africa range from one million to three million deaths per year. Malaria mortality, in turn, is highly sensitive to ecological conditions—as well as basic health infrastructure. (More on this below.)

Specifically, we deploy an ecological index of malaria transmission (used elsewhere as well; see, e.g., Sachs [2002]; Carstensen and Gundlach [2006]) that combines ecological factors—rainfall and temperature—with biological ones such as the human biting rate of the mosquito species that serves as the vector for the transmission of malaria to develop an index of malaria risk (see, Kiszewski (2004) and Sachs (2002) for more details on its construction). The distribution of this malaria ecology index is shown graphically 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 are exogenous.

[FIGURE TWO ABOUT HERE]

However, since there may be lingering concerns that any cross-country differences may be reflecting unobservable heterogeneity, we also pursue a longitudinal strategy, instrumenting for infant mortality (as a proxy for child mortality) within countries over time. Since the malaria ecology instrument does not vary over time – and therefore cannot be used as an instrument in a panel – we use the proportion of the population at risk of exposure to malaria. The narrowing of the range of malaria transmission during the past 50 years, while subject to human control through the application of DDT and improved malaria case management in sub-tropical regions,

should not be endogenous to the fertility rate. Some sub-tropical regions *could* use DDT to eliminate transmission – if these regions had low baseline rates of transmission to begin with – while most tropical regions, and especially sub-Saharan Africa, could not break malaria transmission through the application of existing technologies, since baseline rates of transmission were too high to be eliminated by existing technologies.<sup>3</sup> As we can see in Figure 3, below, at first blush, the geography of malaria risk over time overlaps—to a great extent—with the map of total fertility rates. Note that neither the areas of malaria transmission nor the malaria ecology index include actual malaria cases or deaths, but only the transmission risks, which we argue should be exogenous to fertility. It is important to note that 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, spontaneous abortion, and maternal mortality.<sup>4</sup> Since these 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 on fertility toward zero. However, there is some evidence that malaria may reduce lactation period (Bates et al. 2004), which might increase fertility through decreased child spacing. Likewise, malaria in pregnancy is also associated with low birthweight and increased neonatal and infant mortality—which is in line with our models.

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<sup>3</sup> In technical terms, the question is whether the suite of intervention tools is enough to drive the basic reproduction number ( $R_0$ ) of malaria to below 1. This depends strongly on the pre-intervention  $R_0$ . When the pre-intervention number is close to 1, elimination of transmission is feasible. When it is much greater than 1 (and sometimes higher than 100 in rural Africa), then elimination of transmission is not feasible. Even with continuing transmission, however, control of illness and mortality is possible. Thus, maps of malaria transmission may not change, while the burden of illness and death could change markedly.

<sup>4</sup> See for example Etard (2003), Maubert (1999) and Sartelet (1996) and Guyatt (2001). See also [http://www.rbm.who.int/cmc\\_upload/0/000/015/369/RBMInfosheet\\_4.htm](http://www.rbm.who.int/cmc_upload/0/000/015/369/RBMInfosheet_4.htm) for overview information on malaria in pregnancy.

[FIGURE THREE ABOUT HERE]

[FIGURE FOUR ABOUT HERE]

Third, we examine the impact on agricultural productivity of Green Revolution technologies. Specifically, we argue that a country's share of crops that use high-yield seed varieties has an important impact on agricultural productivity and therefore the allocation of women's time between child rearing and farm work, as well as the returns to education. High-yield seed varieties increase cereal yields per hectare. These varieties also maximize calories per bushel (sometimes at the expense of micronutrients, which has been one of the critiques of Green Revolution technologies [see, e.g., Shiva 2005]).

In 1945, the Rockefeller Foundation and the Mexican government established the Cooperative Wheat Research and Production Program (later CIMMYT, the International Maize and Wheat Improvement Center) to improve Mexico's agricultural output, and especially wheat yields.<sup>5</sup> By the middle 1960s, these technologies were developed for use in some other regions of the world, with help from many NGOs and financing from the World Bank. In Asia, the International Rice Research Institute (IRRI) developed high-yield varieties of paddy rice. By the late 1990s, almost half of East Asian crops are planted with such high yield, "Green Revolution" varieties; over two-thirds of South Asian farming is done with these seeds; but the corresponding figure for Sub-Saharan Africa is a mere 11 percent. (See Table 1 and Figure 4, below). Cereal yields—though determined by many co-factors such as fertilizer inputs, mechanization, irrigation, and natural geography and soil fertility—reflect these differences in Green Revolution diffusion. Each extension of the Green Revolution technologies, from their home base in Japan and the U.S., to Mexico, India, East Asia, Latin America, and Africa, has depended

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<sup>5</sup> Norman Borlaug, who was instrumental to this effort, won the 1970 Nobel Peace Prize for his role.

on local research undertakings to adopt high-yield technologies to local crops, pests, and farm systems (e.g. rain-fed versus agricultural). Africa has been a very late beneficiary of the Green Revolution, because of its distinctive mix of crops (so that successful breeding in other parts of the world were relatively difficult to transfer to Africa), its very high dependence on rain-fed agriculture (while the first generation of high-yield varieties depended on irrigation), and the chronically low levels of spending on research and development on African crop improvements.<sup>6</sup>

[TABLE ONE ABOUT HERE; FIGURE FOUR ABOUT HERE]

Among those countries that have experienced the benefits of Green Revolution technologies, this increased caloric production should have both income and substitution effects. That is, the increased income from better yields should allow for women to support more children; however, we argue here (and show empirically) that the substitution effect should swamp this effect, whereby women shift from the household labor of childrearing to more productive farm work and agro-processing. Moreover, there is a more subtle but very important effect stressed by Rosenzweig (1982). The high-yield varieties required higher skills from farmers, and thus raised the marginal returns to education. Therefore, with a higher return to extra schooling, the advent of the Green Revolution tilted the quality-quantity tradeoff to lesser quantity and towards greater quality.

Further, since high yield crops—a central component of the Green Revolution—were developed by and for rich countries initially, we argue that among lower income countries, the availability of such seed varieties was largely exogenous to local

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<sup>6</sup> U.S. funding for Asian crop improvements was higher than for African crop improvements in part because the U.S. Government and influential foundations viewed Asian food security as part of the U.S.'s Cold War challenge. Africa's food insecurity elicited much less geopolitical concern.

demographic conditions. In other words, the adoption of high yield grains was not demand driven. For example, the first grains for which high yield crops were engineered were wheat (developed originally by Japan and the United States) and irrigated paddy rice (developed mainly by Japan in the first instance). Maize came later, and later still came Africa's distinctive tropical crops, including sorghum, dry rice, millet, teff, and cassava. So countries like India, Taiwan, Korea, and China, which had climatic conditions appropriate for the first high yield crops enjoyed the cereal yield boons; something which we argue helped them undergo the demographic transition before those which had had to wait for the global network of agricultural research units to develop varieties later.

Indeed, Evenson and Gollin (2003) have found that the Green Revolution occurred in two phases. The first, in the late 1960s and early 1970s, consisted mainly of the diffusion of improved varieties of wheat and rice in Asia and Latin America. For crops that are not grown in the developed countries, however, modern varieties were developed later. Although the rate of development of improved crop varieties has increased since the 1960s, disparities still remain. Relatively few improved varieties of crops such as millet and barley, which are grown primarily in semiarid and dry-land areas, existed until the 1980s. Before the 1980s, only a few improved varieties had been developed specifically for regions such as the Middle East and North Africa and sub-Saharan Africa. Before we discuss our variables in more detail and our attempts to isolate the causality of these relationships, we first review the literature on the relationship between fertility and child mortality, economic growth, and the status of women.

## *Mortality*

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 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). 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 seeks to understand whether “stochastic outcomes and fertility choice are quantitatively important.” (p. 337) He differentiates between total fertility rate and net fertility rate, that 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).” (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. In contrast to Chowdhury, Zakir and Wunnava (1999) found that fertility rates impact mortality rates, and not vice-versa. Their model employs simple GLS regression on cross-sectional data (and furthermore fails to distinguish between low- and high-income countries). The simplicity of their equations (and the fact that they do not distinguish between low- and high-income countries, which probably experience very

different dynamics) fails to acknowledge the endogeneity of fertility and mortality, leading to a model that is notably mis-specified.

Some strategies for isolating the child mortality-fertility causal pathway have relied on using adult (male) mortality as an instrument. For example, Galloway et al. (1998) use adult male mortality to instrument infant mortality in a historical analysis of Prussian data from 1875 to 1910. They claim that this measure of mortality may reflect other societal influences on child mortality (i.e. standard of living, nutrition, etc.), but should not reflect particular influences on breastfeeding associated with fertility. However, the common factors affecting both adult male mortality and child mortality are most likely correlated with fertility; furthermore, Lorentzen, McMillan, and Wacziarg (2005) have shown that adult mortality should have an independent effect on fertility (and child human capital investment) by changing the discount rate of mothers and fathers.

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. In another attempt to deal with endogeneity concerns, Benefo and Schwartz (1996) instrument child mortality using variables for community health services and environment. (These include: living farther from a market; living close to a clinic; amount of rainfall; 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, Dreze and Murthi (2001)

instrument mortality using the variable access to safe drinking water, claiming that the later variable should be unrelated to fertility except through its effect on mortality—again a questionable assumption since safe drinking water is related to economic development, which should have an independent effect on fertility. Finally, a recent paper by 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.

#### *Economic Development and Total Fertility Rates*

In the Standard Growth Model (SGM), increased population growth lowers income because of capital dilution (Ahituv 2001: 51-52). However, the Endogenous Fertility Growth Model (EFGM) links fertility to growth by endogenizing the fertility variables. Ahituv (2001) seeks to compare the EFGM and the SGM to understand the interplay between economic development and fertility. He “investigates the empirical consistency of the endogenous fertility growth model” and finds “strong evidence that the negative relations between fertility and GDP growth are a result of a bi-directional causality.” (68) Although the author recognizes the importance of finding a suitable instrument to address such endogeneity, his dataset offers no suitable instrument. Others, however, have gone further in empirically specifying the bi-directionality of this relationship. For example, Chen and Fang (2000) use 1980 fertility rates to instrument subsequent fertility rates and show that lower fertility rates spur increased economic growth.

However, high levels of economic growth simultaneously cause declines in fertility rates, of course. Back in 1982, Rosenzweig found that households exposed to the technologies of the Green Revolution experienced a significant reduction in fertility. His estimation approach relied on *experiments* in which households were the randomly exposed to programs introducing new high-yield technologies and thus present powerful evidence of the effect of (agriculturally-based) growth on fertility.

Demonstrating this relationship on a macro, cross country level has been more difficult, of course.<sup>7</sup> Dreze and Murthi (2001), for instance, found “no significant relations between fertility levels and general indicators of development and modernization such as the poverty index, male literacy, and urbanization” (2001: 46).<sup>8</sup> This should not, however, be the end of the story: While finding suitable instruments for overall income levels may be an impossible feat to accomplish, finding a plausible source of exogenous variation in rural incomes, and more specifically, farm productivity, should not be impossible (as argued above).<sup>9</sup> In fact, it is among rural farmers where fertility is generally the highest and where the returns to female labor (the substitution effect)

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<sup>7</sup> Further complicating the story is evidence that the distribution of economic (and human capital) resources within a society matters. For instance, de la Croix and Doepke (2002) argue that it is essential to account for a country’s fertility differential because it effects the formation of human capital (education). The authors use an “endogenous fertility differential” in their models of economic development. They find that when the variance of human capital rises (in other words, inequality is high), fertility of low-skilled households rises and fertility of high-skilled households falls, leaving the total effect in doubt.

<sup>8</sup> They did find female literacy had a negative effect and was statistically significant, and the son-preference index had a positive and significant effect.

<sup>9</sup> Beyond explicit fertility models, other researchers have successfully employed instrumental variable techniques to determine the effects of economic development indicators on various health outcomes (which of course, may be related to fertility themselves). Pritchett and Summers (1996), for example, utilize four instruments for economic development (terms-of-trade shocks, the ratio of investment to growth, the black market premium for foreign exchange, and the deviation of the official exchange rate from its purchasing power parity level) and find a significant relationship between with measures of infant and child mortality (which, in turn, should affect fertility).

should be the most visible. Thus, in trying to understand the impact of economic development, we focus on rural development, and more specifically, land productivity, as influenced by technological innovations in seed yields (developed outside the countries of interest).

### *Status of Women*

Demographers and economists have longed searched for variables to measure the status of women in modeling fertility patterns. Female education has become a popular proxy for measuring the status of women (Schultz 1994; Al-Qudsi 1998) since female education putatively works to change the opportunity cost of childbearing for women. Positive changes in female education are likely to impact fertility decisions through several interrelated mechanisms: first, increased education, on average, raises a woman's age at marriage; second, gains in educational attainment increase the economic value of women's time; third, positive change in female education increases women's self-esteem and desire for family improvement (i.e. favoring of 'quality' children over 'quantity') (Al-Qudsi 1998). However, these negative associations between fertility and female education are not universally accepted. Some of the earliest models posited positive fertility effects of increased female education (Barro and Lee 1994; Barro and Sala-i-Martin 1995).

Several authors have reported substantial negative fertility effects of increased female education. Schultz (1994) finds that an additional year of schooling is associated with a twelve percent reduction in total fertility and Klassen (1999) reports that a one-year increase in female education reduces the fertility rate by 0.25. Despite these reports

of negative fertility effects from gains in female education, the returns are not consistent across studies. Lorgelly, Knowles and Owen (2001) find a very weak relationship between female education and fertility. Others, meanwhile, find that education is associated with reductions in fertility only after the completion of primary schooling (most likely because wage returns from primary schooling are low or the content of primary schooling is poor) (Benefo and Schwartz 1996).

The endogeneity of female education in fertility models is well-documented throughout the literature, and several authors have attempted to overcome this simultaneity problem through the use of instrumental variables. Breierova (2004) uses a school construction program in Indonesia to measure the effects of education on both fertility and mortality outcomes. Using school construction as an instrument for average parental education, Breierova (2004) reports that mother's education has a strong negative effect on fertility, while father's education does not. Dreze and Murthi (2001) make a strong claim for a causal relationship between fertility and education through fixed effects models examining female literacy. After controlling for economic development indicators (i.e. male literacy, poverty, urbanization), their variable for female literacy remains robust. Although the authors don't link literacy rates and the status of women, they claim a direct causal relationship between literacy and fertility, rather than the influence of a third variable on these outcomes.

Other changes in the status of women are also hypothesized to impact fertility by raising women's age at marriage, changing the gender preferences of child bearers, and altering the value of children vis-à-vis work (Mason 1987). Several authors have constructed variables directly measuring the status of women (Balk 1994; Morgan and

Niraula 1995; Hindin 2000). Hindin (2000) examines survey responses from Zimbabwean women to measure women's contribution to household decisions as indicators of their autonomy and status. Morgan and Niraula (1995) use a quasi-experimental comparison of two Nepali villages to demonstrate the impact of women's autonomy on their fertility choices. Although both Hindin (2000) and Morgan and Niraula (1995) demonstrate correlations between increased autonomy and declining fertility, their analyses suffer from problems of endogeneity. Balk (1994) constructs four classifications to measure the status of women: women's mobility, authority, and attitudes, and the leniency of her household towards her. She uses village-level women's status variables as instruments, claiming that these variables reflect village norms, rather than individual status, and should therefore be exogenous to fertility. In comparison to these instruments, Balk finds that traditional measures that serve as proxies for the status of women underestimate the impact of women's status on fertility.

Amin and Lloyd (2002) challenge the literature purporting a causal relationship between the status of women and fertility rates by reporting recent fertility decline in Bangladesh and Egypt. They argue that major declines in fertility in those two countries occurred in the absence of changes in the status of women, thereby questioning the latter's validity as a determinant of fertility transition. The authors claim that "low levels of women's autonomy have been no barrier to fertility decline" (Amin and Lloyd 2002: 309) in Bangladesh and Egypt.

### *Family Planning Efforts*

As explained in Bongaarts (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. The availability of family planning services, therefore, is crucial for the demographic transition to occur; 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). Thus, while contraception use and other family planning indicators cannot be included directly in the regression (usage is strongly driven by demand, thus causing endogeneity problems), the availability of contraception and others services to those who wish to plan their families is a necessary element in the demographic transition.

Some empiricists have attempted to estimate the impact of family planning efforts. Insofar as family planning programs represent the response of government and non-profit organizations to parental demands for greater fertility control, family planning measurements will be endogenous to fertility models in developing countries (Schultz 1997). One strategy to overcome this endogeneity has been to instrument family planning programs based on funding provided by the International Planned Parenthood Federation (Schultz 1997). Despite the possibility that these programs may be demand-driven, and therefore endogenous, Schultz (1997) fails to find significant results.<sup>10</sup>

Pritchett (1994) argues that the impact of family planning programs is insignificant after controlling for changes in desired fertility. The analysis demonstrates

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<sup>10</sup> Although Schultz cannot reject the null hypothesis that family planning is exogenous using the Wu-Hausman test, he runs models assuming endogeneity and fails to find significant results. He then runs two additional models one examining the price of oral contraception and the other using country fixed effects. Of course, the price of contraception is partially demand driven, so it is endogenous as well.

that family planning programs and contraceptive use has little effect on fertility after controlling for levels of desired fertility. Gertler and Molyneaux (1994) echo conclusions about the non-importance of family planning programs. They find that changes in education and economic development are responsible for observed changes in fertility.

Reporting research on a random experiment in Bangladesh, Joshi and Schultz (2005) present evidence suggesting negative fertility effects resulting from access to family planning programs. Examining two Bangladeshi villages – one in which family planning programs were established nearly two decades ago, and one in which family planning programs were not established – the authors found that access to family planning programs was associated with 0.97 fewer children for women between the ages of 40 and 50, and about 0.6 fewer children for women between the ages of 30 and 40 and 50 and 55.

### *Data*

We compiled a national-level, cross-country dataset covering the period 1960-2004. The demographic data (fertility, infant mortality and child mortality) come from the U.N. Population Division. All other data sources are described below. (We excluded high income country-years as well as those with populations less than 500,000 individuals [examples include Anguilla, Barbados, Fiji, the United Arab Emirates before 1975, and Equatorial Guinea].)

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 2004. 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 of the key demographic variables considered in our analysis, such as TFR and infant and child mortality. Whenever we have used yearly time series data, we compute five year averages for the appropriate quinquennia.<sup>11</sup>

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.

*CMR*: Child 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. This variable, the most putatively sensitive to malaria ecology, is only available for two periods, however: 1995-1999 and 2000-2004. Therefore we use it only in the cross country analysis.

*IMR*: Infant mortality rate, or number of infant deaths per thousand live births.

Data from the U.N. Population Division reported as 5 year averages. This is available for all periods, so when we shift to a country fixed-effects framework, we substitute this variable for CMR. However, this varies quite systematically with CMR, such that the correlation in the overlapping periods is .98.

*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

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<sup>11</sup> 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.

period. For a complete description of the ME variable see <http://www.earthinstitute.columbia.edu/about/director/malaria/index.html>. 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.

*Area weighted Green Revolution modern variety crops*, aggregate for all crops. The data gives Green Revolution modern variety crops planted as a percentage of all crops planted, weighted by area planted to those crops. The data covers observations from 1960 to 2000, taken in five-year averages. These estimates were provided by Robert E. Evenson at Yale. We include this variable in a reduced form equation, rather than use it as an instrument, since the pathways by which higher crop yields should affect fertility are manifold. Specifically, there are efficiency gains for women's labor and some evidence that Green Revolution technologies such as modern crops raise female returns to schooling. There are also, however, income effects as well, since these crops increase total caloric availability to the household. That said, the relationships may be contingent on other factors of production such as fertilizer inputs, farm size, irrigation and mechanization. Given these multiple determinants of the effect of modern crops, we chose to use it in a reduced form model rather than trying to explicitly test various pathways with poorly measured variables.

*Percent of the population at risk of malaria*: We overlay maps of the geographic extent of malaria risk in 1965, 1975, 1994 and 2002 from Hay (2004) with a high-resolution (2.5 arc-minutes) map of 2005 human population from CIESIN (2005). We then calculate the percentage of each country's population at risk of malaria in 1965,

1975, 1994 and 2002, and interpolate to arrive at values for each period in our dataset. Note that these percentages are only approximations, since high-resolution maps of human population are not available as far back as 1965. For this reason we decided to overlay all of the malaria maps on the 2005 population map. The variable nevertheless captures the changing geographic extent of malaria risk in each country over time.

*Abortion upon request:* Abortion is considered legal for our purposes only if the UN deems abortion to be legal and to be available upon request. Our data for this variable was taken from a series of country profiles from the United Nations Population Division “Abortion Policies: A Review” (2002), available online at <http://www.un.org/esa/population/publications/abortion/>.

The variable has a value of 0 unless abortion is legal upon request for every single year in a given five year period, in which case it is assigned a value of 1.

While obviously “abortion upon request” is acting as a very imperfect proxy for a more contraceptive-friendly climate, we believe that using explicit policy changes within countries over time mitigates the concern that it is merely acting as a proxy for general cultural attitudes toward birth control. We can be less certain that policy changes are not demand-driven. However, to mitigate the possibility that contraceptive (and thus fertility) changes within countries are driving policy changes with respect to abortion, we score the abortion variable prior to the period for which we are measuring fertility. Further assuaging endogeneity concerns within a fixed effects framework is the fact that the direction of change is not monotonic. Four countries (Bulgaria, Hungary, Poland, and Romania) ban and then re-legalize abortion in the sample; such bi-directional policy shifts suggest that ideology and politics at the state level are driving legal changes rather

than some “bottom up” demand-driven dynamic. Figure Six, below, shows the distribution of abortion upon request legality over our time series.

Finally, since “abortion upon request” is a strict definition of the legality of abortion, we also tried alternatives: whether abortion was legal if the requester provided a social or economic reason, whether abortion was legal for health reasons or whether abortion was allowed at all. The problem with these more refined specifications is that this multi-category variable was only available for the latest wave of data and hence was tested only in the cross-sectional analysis. However, readers should take comfort in the fact that alternative specifications were equally robust within this framework, and, more importantly, the parameter estimates of our other variables of interest did not change substantially when we altered the abortion variable specification.

[FIGURE SIX ABOUT HERE]

*Log of GDP per capita*, measure at purchasing-power-parity, in 2000 international dollars. Yearly data are taken from the World Bank’s World Development Indicators database (2005). We calculate our own averages for each 5 year period.

*Female literacy (ages 15-24)*: Yearly data taken from the World Bank’s World Development Indicators database (2005). We calculate our own averages for each five-year period. We chose this age band to measure female literacy since it was presumed that post-puberty adolescence and young adulthood were the critical time periods during which “fertility engines” would ignite (Wu 2005) and thus the correct population to measure. Alternate measures for this population such as secondary schooling enrollment rates did not change the results for our main variables of interest and truncated the number of valid country-years, so we chose to use the literacy rate instead.

*Region:* We use the World Bank regional classifications, with a dummy variable for Sub-Saharan African countries. Please see the World Bank, *World Development Indicators* (2005) for more information regarding how specific countries are classified.

*Population:* Total population by country in thousands. Five year averages calculated from U.N. Population Division yearly data. We use this variable as a weight for weighted least squares sensitivity analysis.

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 more than one child per woman, and reduces the mean infant mortality rate by almost 8 deaths per 1,000 live births. Mean modern variety crop diffusion is also significantly higher (roughly 46% versus 17%), driven by high diffusion in populous countries such as India and China. Abortion upon request also increases significantly (from 0.06 to 0.34), again because of China. For obvious reasons, mean values of region dummies also change considerably when we weight by population. Analysis is robust to weighting by population size; however, we present un-weighted regressions.

[TABLE TWO ABOUT HERE]

### *Methods and Findings*

In order to evaluate the effects of child mortality, income, female literacy, abortion policy, and agricultural technology 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

variables framework (instrumenting for child mortality). First we use a cross-section, using the malaria ecology instrument that does not vary over time. Next we move to a fixed-effects framework by inserting country dummies and replacing the time-invariant instrument with one that varies over time (percentage of the population at risk of malaria). In both instrumental variable frameworks, we run formal tests of the presence of endogeneity and the strength of the instrument.

First, it is worth discussing why we use the infant (under age 1) mortality rate instead of the child (under-5) mortality rate in some of our analysis. Conceptually, the effect of child mortality on fertility choices is probably better captured by an under-5 mortality rate than by an infant mortality rate, since children dying at ages 1-5 would impact their parents' fertility choices. Further, the impact of malaria on child morbidity and mortality is greatest in years 1 to 5 of life. However, data for IMR are much more widely available than for CMR, which means that using CMR would have led us to discard a lot of observations which had data on all other variables and IMR. Thus we chose to proceed with IMR as a proxy for CMR for the within-country analysis and stick with the conceptually superior measure of CMR for cross-section analysis (though substituting IMR into these models does not significantly alter our results).

[TABLE 3 ABOUT HERE]

We begin the reduced form OLS analysis looking simply at regional deviations from a baseline time trend (regression (i) in Table 3). Since we are interested 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. From regression (i) we see that sub-Saharan Africa has a TFR that is roughly 2.1 births

above the sample average. Regression (ii) then looks at the controls we are interested in. The regression surely suffers from endogeneity (we have discussed above how high fertility can impact some of the right hand side variables, especially income and child mortality), so while it is not a perfect model and does not allow us to deduce causality or interpret coefficients, it is enlightening to see the strength of the correlations between these variables and fertility. In this simplified framework, all of the variables come out as significant and with predicted signs (high infant mortality are associated with higher fertility, while increasing income, increasing women's literacy and better agricultural technology, and availability of abortion all are associated with lower fertility). 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 all regressions except (vi), since it's fixed-effects. Regression (iii) uses the same specification but with IMR instead of CMR, in order to use the full time series. IMR is in fact stronger than CMR in this framework, but female literacy is no longer significant. All the other variables still maintain their significance and sign. Regressions (iv and v) look at the sub-Saharan Africa dummy with the controls in these "naïve" models, and finds it to have come down to 0.4 or 0.6, respectively, depending of whether we are controlling for CMR or IMR. [The high African fertility rates, according to this model, are caused mainly by the high CMR]. Regression (vi) then looks at a fixed-effects framework by inserting country dummies (this now discards all cross-country variation and uses the controls only to explain within-country variation in TFR). All the variables, including female literacy, are now significant, although interestingly the income variable has switched sign, so that increasing income is now associated with increased fertility (as predicted by the Barro-

Becker model). A Hausman test to compare the two specifications (OLS in regression (iii) and fixed-effects in regression (vi)) rejected the null that the OLS model in (iii) is valid, leaving us with the fixed effects specification in (vi) as preferred among the reduced form models.

We tested the robustness of the FE regression (vi) by running some alternatives. As a first test, we omit the income variable, since the purported endogeneity of the income variable might be biasing other coefficients. All variables except literacy remain significant, and the coefficient on IMR remains at 0.1. As a second test, we reran regression (vi) using an alternative definition of the abortion variable (defining abortion as being legal in the 5 years only if it is legal in all 5 years). The results do not change.

[TABLE 4 ABOUT HERE]

We next move to the instrumental variables framework in a cross-section (with time invariant instruments, as explained above). Table 4 shows the regression results of both the first stage and the second stage least squares. As before, we estimate Huber-White standard errors robust to clustering within countries. The results show CMR (instrumented by Malaria Ecology), legality of abortion, and modern variety crops to be significant, while literacy and income are not. We tested the strength of the instrument with an F-test in the first stage, and following the convention that an F value greater than 5 is necessary for the instrument to be strong, we concluded from our F of 11.5 that the instrument is strong enough.

[TABLE 5 ABOUT HERE]

We test robustness of this instrumental variables model by running some alternatives, as shown in Table 5. First, in regression (ii), as with the FE robustness tests,

we omit the income variable in regression (ii) to get a range for the point estimates of the other coefficients. The CMR coefficient doubles in magnitude (to 0.02), suggesting that low income and high CMR are both correlated with, and perhaps caused by, malaria. Next, regression (iii) we limit the sample to low-income countries, and the results are roughly consistent—again here the coefficient on CMR is double that of the base model (i), and the coefficient on the abortion variable rises in magnitude; however, the modern variety crops variable is no longer significant (though the point estimate is the same and the standard error is higher thanks to the smaller sample size). The problem is likely that the cross-country variation in the crop variable is too small in the restricted sample. Most importantly, the effect of CMR on TFR is robust across all specifications, and the effect holds if CMR is replaced with IMR (not shown). The legality is abortion variable is similarly robust and, in fact, appears to be stronger for low income countries. Finally, it is notable that income and literacy do not play a statistically significant role in any of these models.

[TABLE 6 ABOUT HERE]

Our last set of models use a fixed-effects framework by adding country dummies, and an instrument that varies over time (the percentage of population at risk of malaria). (We now shift to IMR, since that allows for longer panels rather than just the two periods for which CMR is available.) Results of the first stage and 2SLS are shown in Table 6, and show that IMR, income and crops planted to modern varieties have a statistically significant effect on determining within-country variation of TFR (abortion legality and literacy are now not significant). Note that the coefficient on income is positive and significant in the structural model. We test the endogeneity of the IMR variable (the null

of exogeneity can be rejected at a one percent alpha level) and for the strength of the instrument (the F-test yields a value of 17.24, so we can conclude that the instrument is strong).

Looking across the various models employed, we can summarize by saying that the effect of these variables on TFR have shown various degrees of significance. The most important effect has come from the child survival variable. The coefficients on IMR have ranged between 0.01 in the non-IV fixed-effects specification to 0.06 in the IV fixed-effects specification. This range corresponds to having a decrease of TFR of 0.7 to 4.4 if IMR is to decrease by 66% from its 1990 value in sub-Saharan Africa (the Millennium Development Goal for child mortality). The coefficient on CMR of 0.01 from the IV specification (without fixed effects) passed all robustness tests, and this value would correspond with a decrease of TFR of 1.2 if CMR is reduced by 66%. Even this conservative value is a powerful effect; a more plausible point estimate based on the range of results is that TFR would decrease by 2 with a 66% decrease in child mortality. The coefficient on the modern variety crops stays at around -0.01 for all specifications, which would indicate that a 50% higher use of modern variety crops would be associated with a TFR value lower by 0.5. Finally, the coefficient on the abortion legality variable hovers at around -0.9 across models, which would mean that legalizing abortion on request is associated with a 0.9 lower TFR. Literacy, on the whole, did not have significant explanatory power on TFR, and the income variable switched signs between across specifications (negative in the cross-country, positive in the fixed-effects).

*Discussion: Babies Rule?*

While we find some evidence for direct government policy (“abortion”) and for quality-quantity tradeoff via agricultural productivity improvement (Green revolution), the biggest driver quantitatively remains child survival. This is where the theory of the demographic transition started: save the children and families will choose to have fewer children. GDP per capita, urbanization, female literacy, and so on all seem to be less important. This is surprising, on the one hand, but not in contradiction 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 2002). Since the child mortality transition has also proceeded in a wide range of settings, it is plausible that fertility has been driven more by child mortality than by socioeconomic status.

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 (Lancet, 2005; UN 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 adoption of high-yield crop varieties 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 2004 was 5.3. If the Millennium Development Goals are achieved and child mortality is decreased by two-thirds, our results would predict a decrease in TFR of 2 babies. A 50% increase in use of modern variety crops would decrease TFR by .5, and a less pro-natalist environment (proxied by legalized abortion upon request) would decrease TFR by 0.9. These reductions would correspond to a new

TFR in sub-Saharan Africa of just below 2 (replacement rate). While these predictions are very crude, nevertheless the prospect for a rapid, policy-supported transition to lower fertility in Africa, say a TFR of less than 3.0 by 2015, looks reasonable. This will be the subject of a follow-up policy analysis.

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 understand the lag structure in changes to the independent variables. A future paper will look intensively at case studies of rapid fertility declines and attempt to better model lagged effects of the determinants of TFR.

The discussion here may also shed light on the role of ecology versus institutions in economic development. To the extent that malaria ecology affects the demographic transition as well as mortality rates, the observed high settler mortality rates used by Acemoglu, Johnson, and Robinson (2001) as an instrumental variable for current economic institutions would not be appropriate. The high mortality rates would be correlated with an underlying disease ecology which in turn is a direct contributor to economic performance through its depressing effect on the demographic transition. There are other reasons, too, why malaria can cripple economic development, beyond the effects on demography, though the demographic effects are likely to be among the most powerful. In a similar way, settler mortality may be correlated with timing and pathways of agricultural transformation as well. The Green Revolution came first to the temperate and sub-tropical regions, and latest to the tropics, the places where settler mortality was the highest. In short, settler mortality is correlated with ecological variables that have a direct reach into modern economic development, so that settler mortality can not be

deemed to be an appropriate instrument. This topic is worthy of a longer discussion, which we will take up on another occasion.<sup>12</sup>

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<sup>12</sup> Indeed, their measures (and those of Easterly and Levine, 2002 & Rodrik, Subramanian and Trebb 2002, who also do not control for child or infant mortality in their models) are strongly correlated with the prevalence of high-yield seed variety usage, with total fertility rates, with infant mortality, with infant mortality net of adult mortality (which is key to the AJR argument), and with infant mortality as instrumented by malaria ecology. We are not the first to hint at a direct effect of climatically determined health variables on economic growth. Lorentzen, McMillan and Wacziarg (2005) use a similar strategy to show that adult mortality has a direct effect on economic growth, net of institutional context. However, institutional context remains significant in their model; and the adult mortality story is plausibly orthogonal to settler mortality given the survivorship story that AJR told. The same cannot be putatively assumed for infant or child mortality.

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**Table 1: Regional Averages of Area Weighted Aggregates of Modern Variety Crop Adoption (Percentages of Crop Coverage)**

<b>Regional Averages of Area Weighted Aggregates of % Green Revolution Modern Variety Crop Adoption</b>									
	1960	1965	1970	1975	1980	1985	1990	1995	2000
East Asia and Pacific	0	1	7	15	21	26	33	40	48
Europe (Turkey only)	0	0	7	21	28	29	29	39	45
Latin America and Caribbean	0	1	3	6	11	14	19	22	27
Middle East and North Africa	0	0	2	8	10	13	16	24	32
South Asia	0	0	13	29	41	45	54	62	68
Sub-Saharan Africa	0	0	0	0	1	3	5	8	11

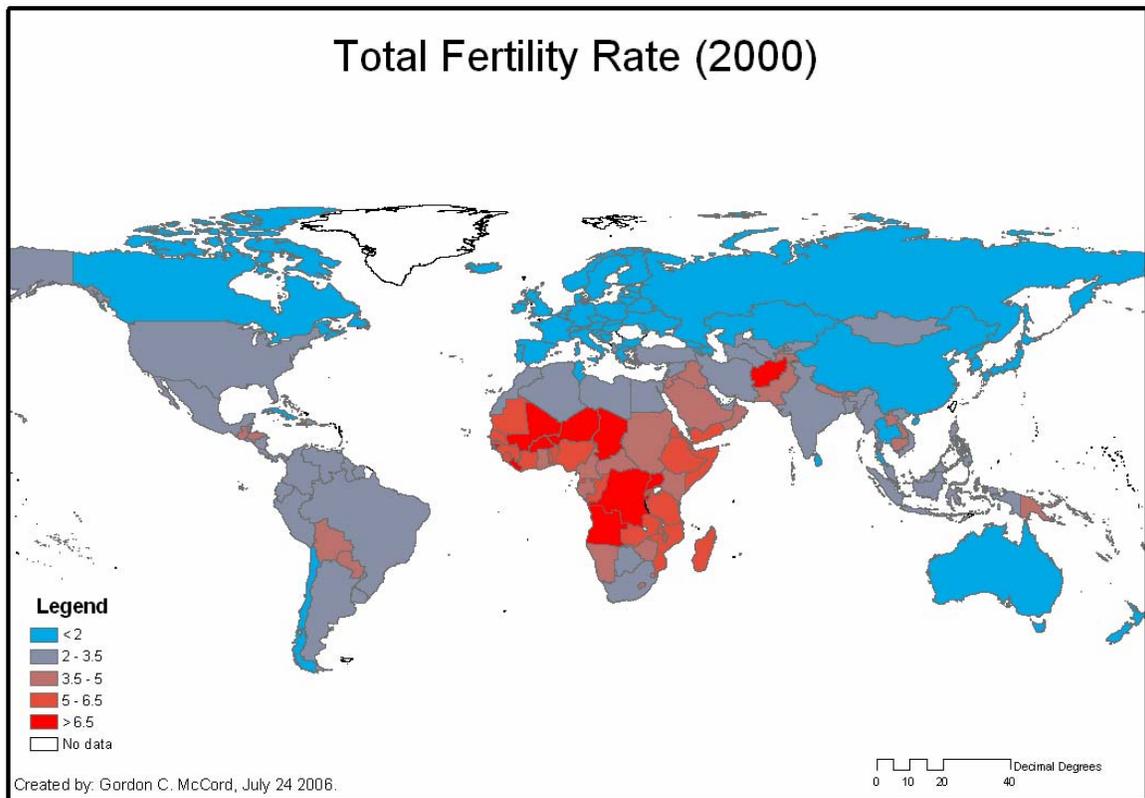
Source: Robert E. Evenson

**Table 2: Means and Standard Deviations of Variables Used in Analysis**

<u>Variable</u>	<u>Mean</u>		<u>Standard Deviation</u>	
	Un-weighted	Population weighted	Un-weighted	Population weighted
<i>TFR</i>	4.89	3.99	1.96	1.73
<i>Infant mortality rate</i>	84.2	74.93	50.3	40.15
<i>Female Literacy (Ages 15-24)</i>	73.04	75.21	28.3	24.07
<i>Log of GDP per capita, PPP</i>	7.9	7.72	0.84	0.75
<i>Share of crops planted to modern varieties (MVs)</i>	10.64	35.26	17.9	29.24
<i>Abortion on request</i>	0.21	0.38	0.41	0.49
<i>Malaria Ecology Index</i>	4.6	2.2	7.14	4.9
<i>Percentage of population at risk of malaria</i>	0.58	0.66	0.41	0.31
<i>Sub-Saharan Africa dummy</i>	0.33	0.11	0.47	0.32
<i>Year</i>	1980.2	1983.13	12.89	12.58

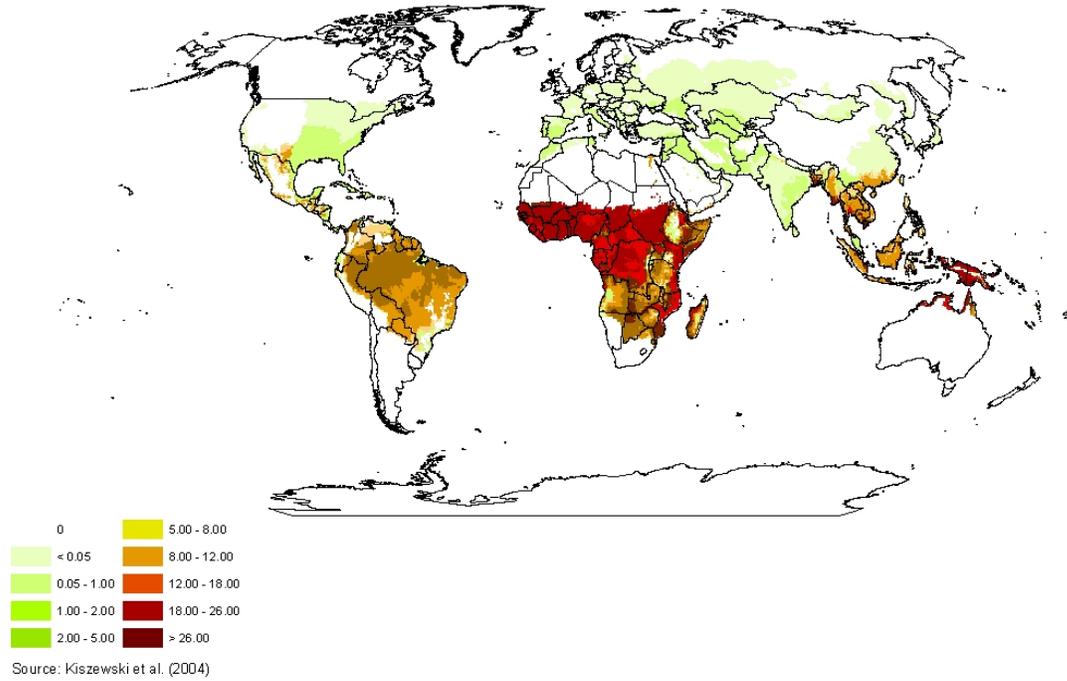
\*Means and standard deviations computed for entire sample, excluding high income countries and countries with populations smaller than 500,000 individuals

**Figure 1: Total Fertility in 2000, by Country**

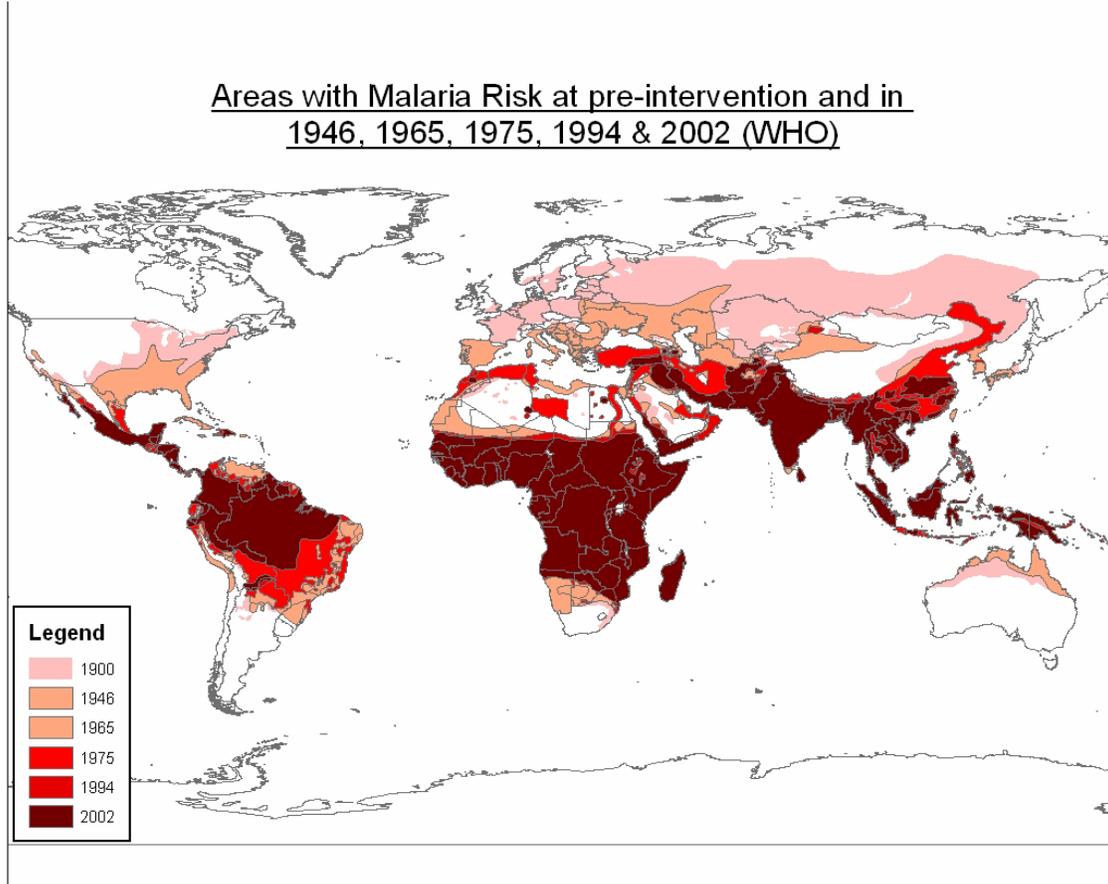


**Figure 2: Malaria Ecology, by Country**

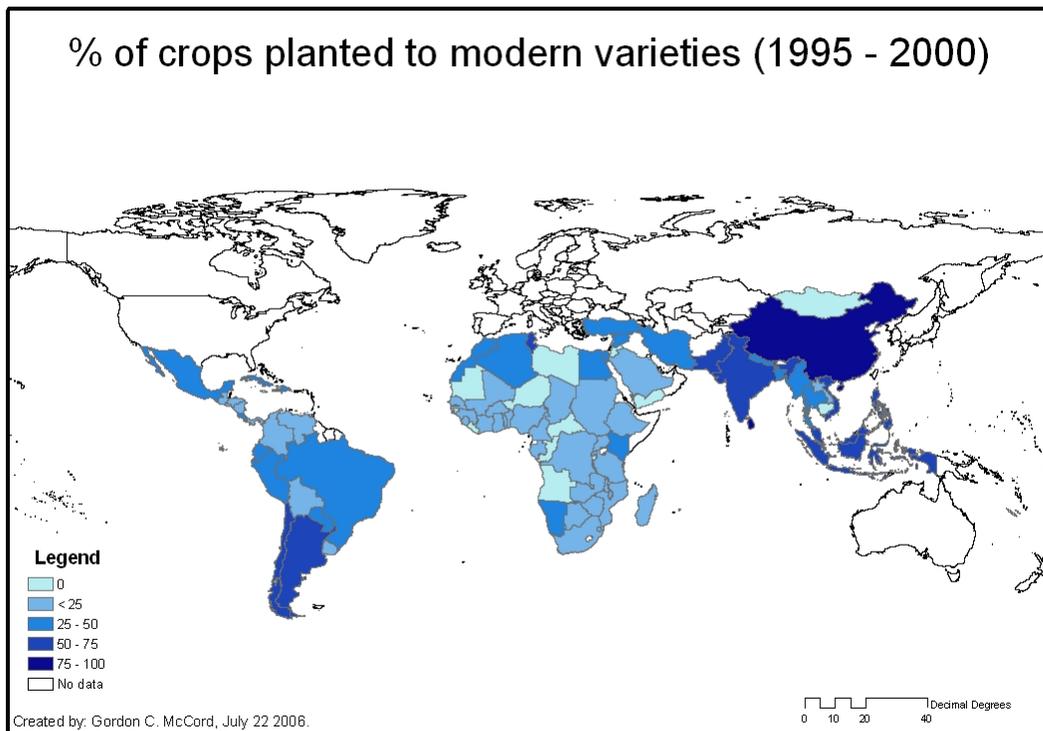
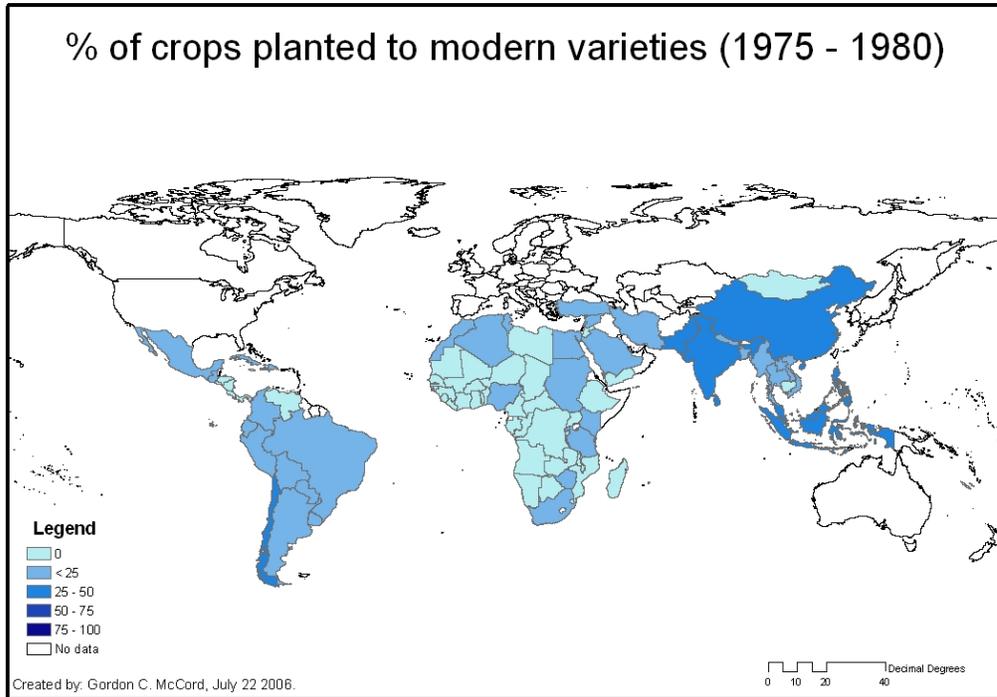
Malaria Ecology Index



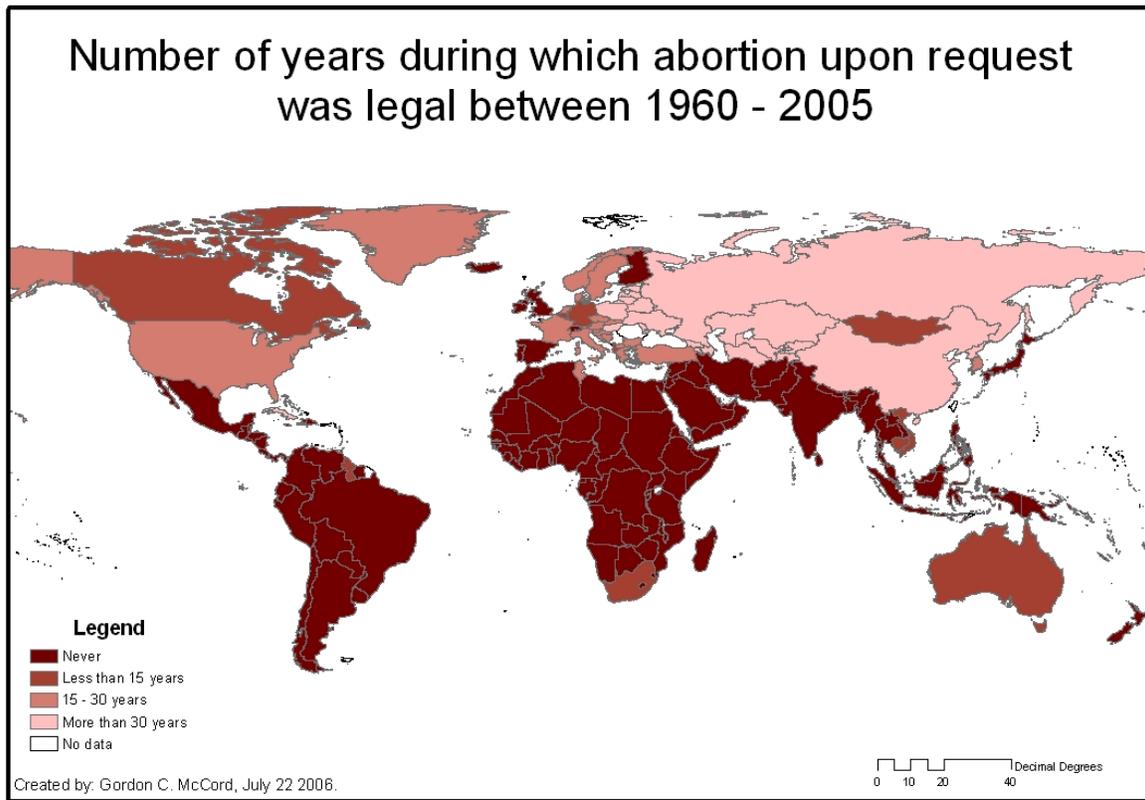
**Figure 3: Malaria Risk by Year**



**Figure 4: High Yield Crop Variety Diffusion, 1975 – 80 and 1995 - 2000**



**Figure 5: Abortion on Demand by Country**



**Table 3: Reduced Form OLS and Fixed-Effects Regressions of Total Fertility Rates**

Dependent Variable:	(i)	(ii)	(iii)	(iv)	(v)	(vi)
<u>Independent Variables</u>	TFR	TFR	TFR	TFR	TFR	TFR
Sub-Saharan Africa dummy	2.08 (8.27)			0.38 (1.44)	0.56 (2.31)	
Literacy of females ages 15-24		-0.01 (-1.33)	-0.01 (-1.10)	-0.01 (-1.85)	-0.01 (-1.43)	-0.01 (-2.71)
Log GDP per capita, at PPP prices		-0.54 (-2.99)	-0.37 (-2.43)	-0.6 (-3.29)	-0.32 (-2.16)	0.36 (2.64)
Child Mortality Rate		0.01 (4.89)		0.01 (2.86)		
Infant Mortality Rate			0.02 (4.41)		0.02 (3.42)	0.01 (5.05)
Legality of Abortion on demand		-0.85 (-3.14)	-0.89 (-2.79)	-0.79 (3.28)	-0.82 (-2.72)	-0.61 (-3.06)
% crops planted to modern varieties		-0.01 (-2.49)	-0.02 (-3.40)	-0.01 (-2.14)	-0.01 (-2.54)	-0.02 (-4.53)
Year	-0.08 (-12.84)	-0.03 (-3.70)	-0.03 (-3.67)	-0.03 (-4.11)	-0.04 (-4.57)	-0.04 (-5.87)
constant	165.98 (13.13)	65.18 (4.26)	62.42 (3.93)	72.64 (4.69)	78.24 (4.83)	79.86 (6.19)
	N =	415	140	415	140	415
	R-squared =	0.54	0.86	0.79	0.86	0.80
				0.86	0.80	0.95

t-statistics in parentheses

Sample excludes high-income countries and countries with populations below 500,000

Regressions (i) through (v) have robust standard errors through clustering by country, while regression (vi) includes country dummies

**Table 4: IV-Regressions (no Fixed-Effects) of Total Fertility Rate – First and Second Stages**

Dependent Variable:	(i) CMR	(ii) TFR
<u>Independent Variables</u>		
Literacy of females ages 15-24	-0.54 (-3.48)	-0.01 (-0.72)
Log GDP per capita, at PPP prices	-42.40 (-10.13)	-0.4 (-1.47)
Child Mortality Rate (instrumented)		0.01 (2.70)
Legality of Abortion on demand	4.02 (0.43)	-0.86 (-3.37)
% crops planted to modern varieties	-0.28 (-2.56)	-0.01 (-2.03)
Malaria Ecology	2.16 (5.39)	
Year	-0.03 (-0.03)	-0.03 (-3.66)
constant	517.82 (0.26)	65.30 (4.21)
N =	138	138
R-squared =	0.82	0.86

Regression (i) is the first-stage regression, (ii) is the 2SLS

Instrument for CMR is Malaria Ecology

F-test of the null of the instruments' effects on CMR yields an F of 11.47.

Instrument is therefore found to be strong.

Sample excludes high-income countries and countries with populations below 500,000

Regression has robust standard errors through clustering by country

**Table 5: Alternate and Sub-sample Specifications of the IV Model**

Dependent Variable:	(i)	(ii)	(iii)
<u>Independent Variables</u>	TFR	TFR	TFR
Literacy of females ages 15-24	-0.01 (-0.72)	-0.01 (-0.89)	0.00 (-0.34)
Log GDP per capita, at PPP prices	-0.4 (-1.47)		-0.56 (-1.08)
Child (under-5 Mortality Rate) (instrumented)	0.01 (2.70)	0.02 (4.13)	0.02 (2.12)
Legality of Abortion on demand	-0.86 (-3.37)	-0.83 (-3.90)	-1.20 (-2.59)
% crops planted to modern varieties	-0.01 (-2.03)	-0.01 (-2.04)	-0.01 (-1.53)
Year	-0.03 (-3.66)	-0.03 (-3.27)	-0.03 (-1.88)
constant	65.30 (4.21)	59.51 (3.56)	63.48 (2.10)
	N = 138	138	67
	R-squared = 0.86	0.84	0.75

t-statistics in parentheses

Instrument for CMR is Malaria Ecology

Sample excludes high-income countries and countries with populations below 500,000

Regression has robust standard errors through clustering by country

(i) is the original specification; (ii) excludes the income variable; (iii) limits the sample to low-income countries

**Table 6: First Stage and Second Stage IV Regressions with Country Fixed Effects**

Dependent Variable:	(i)	(ii)
<u>Independent Variables</u>	IMR	TFR
Infant Mortality Rate		0.06 (3.67)
Log GDP per capita, at PPP prices	-0.82 (-0.30)	0.40 (2.03)
Literacy of females ages 15-24	-0.26 (-2.88)	0.00 (0.28)
Legality of Abortion on demand	-5.39 (-1.36)	-0.31 (-1.03)
% crops planted to modern varieties	-0.07 (-0.96)	-0.01 (-2.01)
% population at risk of malaria	15.04 (4.15)	
Year	-1.19 (-10.32)	0.02 (0.95)
constant	2432.62 (11.00)	-45.01 (-0.99)
	N = 415	415
	R-squared = 0.96	0.90

t-statistics in parentheses

Regression (i) is the first-stage regression, (ii) is the 2SLS

IMR instrumented with % population at risk of malaria

F-test of the null of the instrument's effects on IMR yields an F of 17.24.

Instrument is therefore found to be strong.

Regression includes country dummies, not reported

Sample excludes high-income countries and countries with populations below 500,000