Childhood Mortality in Kenya:

An Examination of Trends and Determinants in the Late 1980s to Mid 1990s

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Executive Summary

After Independence in the early 1960s, child mortality in Kenya fell rapidly. Until around 1980, the under 5 mortality rate (U5MR), the probability of dying by age 5, fell at an annual rate of about 4 percent per annum. This rate of decline slowed in the early 1980s, to about 2 per cent per annum. Recent data from the 1998 Kenya Demographic and Health Survey showed that, far from declining, the U5MR increased by as much as 25 percent from the late 1980s to the mid 1990s. This adverse trend coincided with a number of other adverse trends: stagnation in growth of per capita income, declining levels of immunization, falling school enrolment, and the emergence of an HIV/AIDS epidemic. On a more positive note, fertility fell by about 30 percent from the mid 1980s to the mid 1990s.

Controversy surrounds the factors responsible for the increase in child mortality in the 1990s, and the objective of this paper is to clarify the situation. Data from the 1993 and 1998 DHSs have been merged into a single data set, and multivariate analysis used to examine the factors associated with mortality risks in childhood. Dummy variables were used to represent different three-year time periods, from 1984-86 to 1996-98. Socioeconomic controls, including mother's education, an indicator of household wealth, urban/rural residence, and indicators of health service utilization, plus controls for reproductive dynamics such as age of mother at the birth, birth order, sex and preceding birth interval, were developed. In addition, an indicator of the HIV epidemic, the prevalence of HIV in the district of birth at the time of each child's birth, was developed. With no controls, the models confirmed an increase in mortality of about 25 percent. Including socioeconomic and biodemographic controls tended to strengthen the upward trend in mortality; in other words, had there been no changes in these factors, child mortality would have been expected to decline. Introducing controls for health variables – immunization, pregnancy and delivery care, prevalence of childhood diseases and maternal and child malnutrition – also did not alter the underlying trends substantially. Thus rising child mortality could not be explained by socioeconomic, biodemographic or health status factors. Including the prevalence of HIV in the models, however, changed the underlying trends fundamentally, from sharp increase to monotonic decline. Although models of this sort cannot demonstrate causation, only association, the HIV epidemic appears to be the most probable cause of the recent increases in child mortality in Kenya. Of the health variables, the only one found to be significantly protective was immunization coverage.

Introduction

The HIV/AIDS epidemic threatens to reverse 30 years of child mortality reductions in sub-Saharan Africa (Nicoll et al., 1994; Adetunji, 2000). However, Africa also faces a number of other economic and social problems, which may also be threatening child survival improvements. Better policy requires a better understanding of how important these different factors may be.

For much of its existence as a independent nation, Kenya has been widely viewed as a "success story" in terms of its socioeconomic development and political stability. In the 15 to 20 year period following independence, economic growth was strong and operated to mitigate the potentially harmful influence of very high fertility and rapid population growth on living standards. After this period of growing prosperity, a number of internal and external factors including sharp increases in government expenditures and an adverse movement in terms of trade started to reverse the trend from one of improving to one of declining living standards that continued at least into the early 1990s (Brass and Jolly, 1993). Trends in mortality levels among Kenya's young child population have largely mirrored these changes in the macroeconomic health of the country. During the period 1965-1980, Kenya enjoyed a rather impressive and sustained decline in under-five mortality (U5MR) of 3-4 percent per annum (Hill et al, 1999), twice the rate of the average country of sub-Saharan Africa during this period. During the 1980s, child mortality decline slowed in Kenya, but was still nearly 2 percent per annum during the first half of the decade.

The 1998 Kenya Demographic and Health Survey (KDHS) provided the first clear evidence that the decline had not only slowed but had been reversed during the 1990s (National Council for Population and Development, Central Bureau of Statistics and Macro International, 1999). Direct measures of child mortality from birth histories included in the KDHS showed a 25% rise in under-five mortality from the mid-1980s to the mid-1990s. One of the most striking mortality-related findings of the 1998 KDHS was the existence of enormous differentials by province of residence. The chance of dying before age 5 varies from 34 per 1000 (Central Province) to 199 per 1000 (Nyanza Province), a 6-fold difference. Presumably, the underlying causes of geographic differentials of this magnitude involve the uneven distribution of important epidemiological, biomedical, social and behavioral "risk factors" across the national landscape.

Though the childhood mortality differentials are of interest, the main purpose of this analysis is to attempt to elucidate the reasons for the reversal in the late 1980s and early 1990s of the steady downward trend in childhood mortality. As noted above, there are a number of potentially adverse developments that have coincided with the upturn in mortality. After several decades of growth, per capita income stagnated in the 1980s. Impressive gains in levels of education were not sustained. Improvements in public health measures, particularly immunization levels, also leveled out or were reversed. The late 1980s and early 1990s saw the emergence of HIV/AIDS as a major public health factor. Our objective in this analysis is to try to identify the factors associated with the recent increase in childhood mortality. Our strategy is to examine to the extent possible the association, both temporal and geographic, of child mortality changes with changes in potential explanatory variables. We will not be able to arrive at a definitive conclusion, since association can never prove causation, but we hope to arrive at a plausible story that is consistent with the information available.

The conceptual framework underlying this analysis is based on the Mosley-Chen (1986) proximate determinants model. Child mortality may have increased because of changes in background variables operating through proximate determinants, or because of changes in proximate determinants themselves. Important proximate variables of concern include: level of access to and use of important maternal and child health services, exposure and susceptibility to varying pathogenic agents, food security and nutritional status. These important child survival inputs are conditioned upon a number of important social and economic factors which include parental education and household economic profile (disposable income and wealth). Today, child survival prospects are further compromised by both direct and indirect effects of the HIV/AIDS epidemic. Evidence concerning changes in the 1990s in relevant factors is summarized in Table 1 and reviewed below.

Child Mortality. Under 5 mortality increased by 11 percent from 1988-93 to 1993-98, and mortality between the ages of one and five increased still faster, by 18 percent.

HIV/AIDS. The health and survival chances of young children are affected by the AIDS epidemic in both direct and indirect ways. First, mother-to-child transmission of HIV can occur during pregnancy, childbirth or breastfeeding. Available evidence indicates that, in the absence of antiretroviral therapy, about 60% of children infected with HIV around birth or through breastfeeding will die before their fifth birthday (Spira et al., 1999). Indirect effects are

many and varied, but most prominently involve illness and death of parents, adverse economic consequences or dissolution of households. A recent cross-national review of DHS and HIV prevalence data found that most countries in sub-Saharan Africa with high HIV prevalence (five percent or more of adults seropositive) also experienced increased U5MR (Adetunji 2000). During the 5-year period preceding the 1998 KDHS, the national HIV/AIDS surveillance system in Kenya estimates that HIV prevalence rose from 9 to 13 percent, putting Kenya in the high prevalence group of countries (NASCOP, 1999).

Childhood illness. Although accurate information on cause of death is lacking, the cause of death structure of under five mortality in Kenya is, like most countries in sub-Saharan Africa, probably dominated by pneumonia, malaria, measles and diarrheal disease, which are estimated to have been responsible for some 60 percent of disease burden in the region around 1990 (Murray and Lopez 1996). Such evidence for Kenya as there is, however, does not suggest an increase in childhood disease. KDHS data indicate that between 1993 and 1998, the two-week prevalence of fever among children under age 3 in Kenya fell by about 10 percent and diarrheal prevalence fell by about 11 percent.

Malnutrition. The link between poor nutritional status and child mortality risk is well established, especially with regard to wasting or acute malnutrition (Pelletier et al, 1993). However, the prevalence of stunting did not change in Kenya between the 1993 and 1998 KDHS's.

Use of MCH services. Both preventive and curative child health services affect child survival. In this analysis, we can control for care of the mother during pregnancy and during delivery, in terms of quantity if not quality, and preventive health services in the form of immunization levels of young children. Access, quality and use of health services vary sharply by area of the country. On average, however, the proportion of pregnancies receiving antenatal care and of deliveries occurring in health facilities both declined slightly from 1993 to 1998. Immunization coverage of children aged 12 -23 months worsened slightly, for example from 84 percent to 79 percent for measles, but was still at high levels.

Socio-economic Context. The 1998 KDHS produced worrying evidence that after decades of improvement, levels of schooling among girls have fallen recently. However, it is likely to take some time for the effects of this change to work their way through into the educational status of the mothers of a cohort of children (Table 1 shows the proportion of births to mothers with no education fell from 16 percent in 1988-93 to 10 percent in 1993-98). A decade and a half of economic stagnation may also have had negative impacts, particularly if accompanied by increasing inequality of resource distribution. Real GDP per capita increased hardly at all, but on average household wealth as measured by assets owned increased between 1993 and 1998, and the proportion of births occurring in low-income households fell sharply.

Biodemographic Context. Fundamental changes have occurred in reproductive behaviors in Kenya since the early 1980s. Total fertility in the period 1995-98 was little more than half (4.7) what it was estimated to be in the late 1970s (8.1). Contraceptive prevalence among currently married women was 31 percent in 1998. Reduced fertility is in general expected to be associated with reductions in child mortality risk, but it is possible that an increasing proportion of high risk first births and a change from long to short birth intervals even as fertility declines could work in the opposite direction.

Data

The data on child mortality and many other of the socio-economic and biodemographic variables used in this study come from the Kenya Demographic and Health Surveys conducted in 1993 and 1998. For the multivariate analysis reported below, data from the 1993 and 1998 surveys were combined into a single data set. Births before 1982 were excluded. The resulting data set contains records on some 31,000 children. Both surveys included full birth histories, providing information about the date of birth, survival status and, if dead, date of death of each child reported by a woman included in the sample. These data provide a basis for estimating child mortality specific by age and time period, but do not include information on the survival of children of women who have died. An association between mortality risks of mother and child, a likely result of the HIV/AIDS epidemic, will bias estimates of child mortality, downwards if the association is positive. Thus estimates of child mortality risks based on the surveys are likely to be too low for periods seriously affected by the HIV/AIDS epidemic.

Several other important pieces of data come from the DHSs. These surveys record information about health variables for births or surviving children born (to interviewed, surviving women) in defined periods prior to the survey (those born since January 1988, or roughly the preceding 5 years, for the 1993 survey, and since January 1995, or roughly the preceding 3 years, for the 1998 survey): pregnancy and delivery care and height and weight of the mother for all such births, and immunization status, heights and weights, and two week prevalence of diarrhea, fever, and cough, with additional information on treatment for these illnesses for all surviving children,. The data also provide socio-economic information about the mother and her household, such as her level of education, the household's ownership of selected material assets, and whether the household is located in an urban or rural area. The birth history provides information about biodemographic characteristics of the child, such as sex, age of mother at the time of the birth, birth order of the child, and preceding birth interval.

Estimates of annual HIV prevalence at the district level (the KDHS sampling frame includes 34 districts in all) have been developed by the National AIDS and STDs Control Programme (1999). These estimates were developed as follows (NASCOP 1999). Prevalence data are collected from 13 urban antenatal care sentinel sites (an average of 400 samples per site per year). Annual observations from each sentinel site are first smoothed by fitting a gamma curve to them to obtain a smooth annual sequence. Next, the urban part of each district is assigned the prevalence estimate from one of these sentinel sites (either from the sentinel site in that district or, if there is no site in the district, from a site in the most "similar" district) for each year over the period 1990-98. Next, each district is categorized as having high, medium, or low urban-rural mobility (based on research in rural and peri-urban settings). In high mobility districts, rural prevalence is assigned a value of 80% of the assigned urban value; rural areas of medium mobility districts get 70 percent of the urban prevalence, and rural areas of low mobility districts get 50 percent of urban prevalence. Finally, district-level urban-rural distributions from the 1990 Population Census are used as weights for the urban and rural prevalence estimates to get overall district estimates. District-level estimates of HIV prevalence have been used in this analysis by assigning to each child record the district-level prevalence in the year of the child's birth.

Outcome Variable

The outcome variable is the log odds of dying in a particular age range of childhood. The age ranges used are neonatal (first month of life), early postneonatal (one to five months), late postneonatal (six to 11 months), early second year (12 to 17 months), late second year (18 to 23 months), third year (24 to 35 months), fourth year (36 to 47 months), and fifth year (48 to 59 months). Each child is included in as many age ranges as he or she completed (or could have completed, if the child died in the interval) before interview date. Thus a child born 65 months before the 1993 Survey who was still alive at the time of the Survey would be included in all eight age ranges. On the other hand, a child born 65 months before the 1993 Survey who died at age 27 months would only be included in the six age ranges up to 35 months. A child born 27 months before the 1993 Survey and still alive at the Survey would only be included for five age ranges (since the sixth was not completed), and a child born 27 months before the survey who died at age 25 months would also only be included for five age ranges (since the child could not have completed the two year age range had he or she survived). In total, the combined data set contains some 199,000 child/age range records.

Independent Variables

Independent variables are in general self-explanatory. Age ranges are defined by dummy variables. In order to

study trends, three year time periods (1984-86, 1987-89, 1990-92, 1993-95 and 1996-98) are defined. An age range for a given child is assigned to a time period on the basis of when the mid-point of the age range is reached. Thus a child born in August 1989 would contribute both its neonatal and early postneonatal exposure to the period 1987-89, since the mid-point of these ranges would be reached in 1989. The late postneonatal through the two completed years age ranges fall in the period 1990-92, and the three and four completed year age ranges fall in the period 1993-95 (even though a few months of the age three period falls in the period 1990-92). The period of an age range for a child that dies in the age range is determined by the mid-point of the range, not by the mid-point of the time survived by the child.

Socio-economic variables include mother's education (categorized as zero, incomplete primary, complete primary, incomplete secondary, complete secondary and higher); household wealth quintile (an index based on household ownership of material possessions, such as radio, television, telephone, refrigerator, bicycle, motorbike and car, and on housing quality, whether the house has electricity, a finished floor and a "permanent" roof, corrugated iron or tiles) categorized as very poor (bottom quintile), poor (20-39%), "middle class" (40-79%) or rich (top 20%)[1]; and usual type of place of residence of the mother (urban or rural).

Biodemographic variables were constructed to control for possible changes in reproductive dynamics. The variables used were age of mother at the time of the birth (under 20, 20 to 34, and 35 or over), the child's birth order (categorized as first, second to fifth, or sixth and over), the child's sex and whether the preceding birth interval was very short (less than 18 months).

Four variables were developed to measure health status or the impact of the health system at the local level. These variables were based on information collected by the two surveys about children born in the five (1993) or three (1998) years before each survey. Although the surveys were carried out in the same sample clusters in 1993 and 1998, separate estimates of the health variables were made for the period before each survey. To ensure comparability between 1993 and 1998 data, only information on births in the three years before the 1993 survey was used, except for heights and weights, for which data were available for both surveys for children under age five.

For each health variable, indicators were developed for the community (the sample cluster) rather than for the individual child, since for many indicators, such as immunization history, information was only collected for surviving children. Use of community estimates also tends to minimize possible endogeneity. However, for many clusters, the number of births or of surviving children with information was too small to support a stable estimate. An arbitrary system was used to stabilize the values by using a weighted average of the cluster value and the district value. If a cluster had five or fewer observations, the district value was used. If the cluster had 50 or more observations, the cluster value was used. In between, a weighted average of the cluster and district value was used, with the weight being determined by the square root of the distance of the number of observations between five and 50.

A variable reflecting access to health care was created by combining information on at least one antenatal visit and delivery care by a health professional. A variable reflecting immunization was created by combining proportions of eligible children aged 12 to 35 months with BCG, DPT1, DPT3 and measles immunizations. A variable reflecting community levels of illness was created by combining proportions of children under age 3 with diarrhea, fever or cough with rapid breathing in the two weeks before the survey. Finally, a variable reflecting nutrition was obtained by combining the proportion stunted of children aged 12 to 35 months and the proportion of recent mothers (not currently pregnant and at least three months post-partum) with a Body Mass Index (BMI) below 18.5.

The distributions of weights that were used to create the stabilized cluster indicator varied by type of indicator. For antenatal care and delivery care, the average weight for the cluster value was about 0.3, indicating a weight for the district value of about 0.7. The district value was used for rather less than 25 percent of clusters, and for a few clusters the cluster value itself was used. For immunizations, on the other hand, the average cluster weight was only 0.10, for over half the clusters the district value had to be used, and the largest weight was 0.68. Two week prevalence of diarrhea, cough with difficult breathing and fever had intermediate weights, with the district value being used in over 25 percent of cases, and the largest weight being about 0.8. Weights for mothers' body mass indexes ranged from zero to one, with a median of 0.63, whereas weights on stunting were zero for over 25 percent of cases, averaged about 26 percent and peaked at 88 percent. For all these variables, therefore, the cluster value is

[1] Principal Components Analysis was applied to this set of items to estimate appropriate weights for pooling them into a single index (Filmer and Pritchett, 1996))

really a blend of the cluster and district values.

Methods

Logistic regression has been used to estimate the effects of the independent variables on the log odds of dying in a particular age range. All age ranges are included simultaneously, so it is assumed that the proportionate effects of all variables are constant across all age ranges. We subsequently relax this assumption by analyzing age ranges independently. For all the regressions, standard errors were adjusted for clustering on the sample cluster variable. The modeling strategy used was to start only with age range and time period variables. The interpretation of the exponentiated coefficients on the time period variables in these models is the average mortality risk across age ranges relative to the baseline period, 1984-86. In other words, they are indicators of trend. Control variables are then introduced, first for socio-economic variables alone, then for biodemographic variables alone, then for socio-economic and biodemographic variables, and finally for all these variables plus the district-level prevalence of HIV in the year the child was born. The primary interest in these models for the purpose of this paper is the effect of the controls on the time period (trend) variables. If a set of control variables changes the time period odds ratios significantly, then those control variables are related, possibly causally, to the trend. If, on the other hand, introducing a set of control variables has no effect on the trend variables, the trends are essentially independent of the controls.

The health variables described earlier only refer to fairly short periods before each survey. Models in which the health variables are used are therefore time-limited to only the 1990-92 period using 1993 KDHS data and 1996-98 period, using 1998 KDHS data, and the only time period variable used is for 1996-98 relative to 1990-92. This limitation has an additional effect, of limiting analysis very largely to one child per woman for each period.

Results

In the multivariate analysis, primary interest focuses on the relative odds of mortality in the various time periods. These periods are 1984-86, 1987-89, 1990-92, 1993-95 and 1996-98. The first model, using only age range and time period, is shown as the first line of Table 2. Risks are very similar in 1984-86 and 1987-89, rise somewhat (about 13% in the odds across all age groups) in 1990-92, rise further in 1993-95 (nearly 25% higher than in 1984-86) but decline in 1996-98 (while still remaining above levels in the mid 1980s).

A variety of explanations could account for the apparent increase in child mortality risks from 1984-86 to 1996-98. The 1998 survey could have done a better job of collecting information about child deaths than the 1993 survey, giving the impression of rising mortality risks. This hypothesis is tested by introducing a dummy variable for survey, estimated for the overlapping time periods. The dummy has no effect on the time period coefficients. Socio-economic conditions could have worsened over time. Line 4 of Table 2 controls for maternal education, household wealth quintile as estimated from assets owned and housing quality, and type of place of residence. The increase in mortality risk in 1993-95 and 1996-98 relative to 1984-86 is marginally increased by including these controls. Changes in reproductive dynamics, for example a shortening of birth intervals or an increased proportion of high risk first births, could account for the trend, in view of the sharp fertility changes that have occurred in Kenya over the last 15 years. Line 5 of Table 2 shows the period odds ratios when controls are added for age of mother at the child's birth, child's birth order, child's sex and whether the preceding birth interval was shorter than 18 months. Relative to the no controls model, period effects are once again marginally strengthened. Controls for both socio-economic and biodemographic factors (line 6) also makes no difference.

What does make a difference to the time period coefficients is estimated district HIV prevalence in the year of the child's birth. Controlling for prevalence alone (line 2) or prevalence in combination with all the socio-economic and biodemographic variables (line 7) changes the odds ratios completely, from rising sharply from the late 1980s to the mid 1990s to declining monotonically across all time periods, to such an extent that by 1996-98 the odds of dying are less than half what they had been in 1984-86. The results strongly suggest that HIV prevalence is largely responsible, directly or indirectly, for the increasing mortality in the 1990s.

Table 3 explores the possibility that an erosion of the use or the quality of health services accounted for the mortality increase. As described above, indices of health status and health service have been constructed from the data from each survey. Since these variables reflect the status of the cluster in the period shortly before or at each survey, analysis has been limited to two time periods, 1990-92, based only on the 1993 survey, and 1996-98, based only on

the 1998 survey. Unlike the results in Table 2, no observations from the 1998 survey were used for the 1990-92 period, so results differ slightly. These models also include very few cases in which more than one child of the same mother is included in the analysis.

The same modeling strategy is followed. A first model controls only for time period. Mortality risks across all ages of childhood are approximately the same in the period 1996-98 as in the period 1990-92. Introducing the four health system indices (row 2) does not change this significantly, though changes in health indicators alone would actually have been associated with a decline in child mortality from 1990-92 to 1996-98. The only two health indices that are significant, however, are the summary vaccination measure, associated with a reduction in risk of about 15 percent, and the sickness index (based on proportions with diarrhea, fever or cough with rapid breathing), associated with an elevated risk of about one-third. The access variable (antenatal care and delivery care), and the nutrition variable (stunting among children and low BMI among recent mothers) are not significantly associated with child mortality risks. The third, fourth and fifth models introduce socio-economic, biodemographic, and then both socio-economic and biodemographic controls. The effects on excess mortality in 1996-98 or on the impacts of the health indices are negligible. In the final model, HIV prevalence in the district at the time of the birth of each child is introduced. Mortality risks in 1996-98 are significantly lower, by about 25 percent, once HIV prevalence is controlled. The effect of immunization ceases to be significantly protective (suggesting a negative association between immunization and HIV prevalence), and the effect of sickness is reduced (though it retains significance). It is clear that mortality in 1996-98 relative to 1990-92 is strongly associated with HIV prevalence at the district level, to such an extent that when this prevalence is controlled for, mortality risks are significantly lower in 1996-98 than in the base 1990-92 period, even controlling for changes in immunization levels. The HIV prevalence variable in the final model is highly significant, with an odds ratio of 1.05 (since prevalence is measured in percentage terms, a one percent increase in prevalence would be associated with a five percent increase in mortality risk).

Our primary focus in this paper is on trends, or period effects in mortality. It is also interesting, however, to examine the effects of socio-economic and biodemographic control variables, particularly in the absence or presence of a control for HIV prevalence. Table 4 shows the odds ratios for education, wealth, type of place of residence, age of mother at birth of child, child's birth order, child's sex, and whether the preceding birth interval was short. Odds ratios are shown for four models: socioeconomic variables only, biodemographic variables only, socioeconoic and biodemographic controls, and finally all these controls plus HIV prevalence in the district in the year of the child's birth. The results are in line with expectations. Children born to mothers with no or incomplete primary education have the highest mortality risks, and the risks thereafter decline monotonically with increasing maternal education. Completed primary reduces mortality risks by 25 percent, and post-secondary education is associated with mortality risks nearly 70 percent lower than for mothers with no education. Household wealth is also strongly (and monotonically) associated with mortality risk: children born into the most affluent households have mortality risks over 40 percent lower than those born into the poorest, even after controlling for all the other variables in the model. The association of these variables with mortality is essentially independent of the HIV variable. In contrast, the type of place of residence variable is affected by the HIV variable. In models without the HIV control, urban areas have somewhat higher mortality risks than rural, but once HIV prevalence is controlled for, child mortality is lower in urban areas.

Biodemographic variables also behave much as expected. Being born to a mother under age 20 is a risk, but being born to a mother over 35 is no riskier than being born to one who is 20 to 34. High parity births are at higher risk than lower birth orders, but first births are no riskier than second to fifth order births. Girls have somewhat lower mortality risks than boys, though the effect is not significant. Being born after a very short birth interval, less than 18 months, is associated with greatly elevated risk.

The models presented in Tables 2 through 4 assume proportionality – that the effect of each variable is proportionately the same in each age range. Serological studies suggest rate ratios for HIV+ children that rise with age: for example, in Rwanda, the rate ratio for HIV-infected children was about 4 in infancy, but about 9 in the second year of life (Spira et al., 1999). Results in Table 5 show models for each age range separately. Note that, for this analysis, the age ranges two, three and four completed years have been combined because of small numbers of events. The first set of models controls only for time period (results shown in Panel (a) of Table 5), the second set of models controls for time period and socioeconomic and biodemographic factors, and the third model adds HIV prevalence. Differences between the time period only and the time period plus socioeconomic and biodemographic control models are small. Excess risks in the more recent periods are greatest for children aged from one month to two years, whereas neonatal mortality shows no trend over the period. Once HIV is controlled, however, all the age ranges show strong downward trends.

It is interesting to compare the coefficients on the HIV variable across age groups (Table 6). HIV is associated with higher risks in all age ranges, but the effect is smallest in the neonatal period. The risk is high across all the remaining age ranges, however. The age-specific effects may be distorted by survival bias: the older a deceased child would have been, the less likely it is that the mother will still be surviving to report the death.

Discussion

In both the analysis of all periods (but not controlling for health indicators) and the analysis of only the periods 1990-92 and 1996-98 (controlling for health indicators), the prevalence of HIV at the district level at the time of the birth of the child is found to be strongly associated with the increased mortality risks of children in the 1990s in Kenya. The association does not prove causation, but the strength and consistency of the association across a variety of analyses would require a remarkable degree of coincidence if the effect is not causal. If it is causal, there are a number of possible mechanisms. It may be a direct effect, whereby seropositive children are dying at higher rates than seronegative children. There are also a number of possible indirect effects. HIV positive parents may be less well able to look after their children than those who are not infected. Economic conditions may just be worse in badly hit areas than in less affected areas. However, the household asset/wealth variable should control for this to some extent. Areas of high HIV prevalence may have higher levels of opportunistic infections, particularly tuberculosis, that increase the risks of all children, not just those that are seropositive. However, the sickness variable would be expected to control for this effect if it reflects anything real. Another possibility is that parents in high HIV prevalence areas interpret any sickness in a child as a sign of AIDS, and thus give less care and attention to a child that is sick, regardless of the child's serostatus. Lacking serological data, we have no definitive way of differentiating the effects of these various mechanisms. However, since several of the indirect effects are potentially controlled by other variables in the model, it seems plausible that it is the direct effect which is operating most strongly.

The effect of HIV is almost certainly underestimated, because of the retrospective nature of the reports. Vertical transmission of HIV to the child implies that the mother is seropositive, and although the child will probably die more quickly than the mother, in a setting such as a Kenya with virtually no antiretroviral treatment the mother will surely die. There is thus a relatively short period in the epidemic during which child mortality rates will appear to be elevated (through a direct effect of the epidemic) on the basis of retrospective reports of women. Once the women themselves die, the higher risk children will not be reported. Mortality rates of children will then appear to stabilize. It is possible that this effect is observed in the period 1996-98, which has somewhat lower apparent child mortality than the period 1993-95 in this analysis.

Although indicators of health services use (immunizations, births in health facilities) and maternal nutrition worsened during the 1990s, reproductive dynamics (short birth intervals, high parity births) and socio-economic condition (proportion of births to educated women, household wealth) changed in ways that would have reduced child mortality. Unobserved factors also appear to have changed in favorable ways, such that mortality risks of children would have fallen by more than half from the mid 1980s to the late 1990s had it not been for the HIV epidemic. Our analysis did not identify ways in which policy could mitigate the adverse effects of the HIV epidemic. Interactions of the HIV prevalence variable with health sector variables, with urban versus rural residence, or with household wealth did not produce significant effects. Interactions with education of mother—specifically with secondary or higher education—suggested that the children of better educated women in high HIV prevalence areas were at lower risk.

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Table 1 : Levels and Trends of Various Health and development Indicators: Kenya, 1993 - 1998

Indicator	1993	1998	% Change
Childhood Mortality (5 Years before KDHS)			
Under-5 Mortality Rate (per 1,000 live births)	98.9	111.5	11.3
Infant Mortality Rate (per 1,000 live births)	67.7	73.7	8.1
Child Mortality Rate (per 1,000 survivors to age 1)	33.5	40.8	17.9
Maternal and Child Health Indicators			
Percent of children under age 3 with fever in last 2 weeks	46.5	42.3	-9.9
Percent of children under age 3 with diarrhea in last 2 weeks	19.0	17.1	-11.1
Percent of children under age 5 stunted	32.7	33.0	0.9
Percent of children under age 5 underweight	22.3	22.1	-0.9
Percent of children under age 5 wasted	5.7	6.1	6.6
Percent of births in last 5 years to women with BMI < 18.5	9.2	10.3	10.7
Use of Maternal and Child Health Services			
Percent of children 12-23 months received DPT-1 vaccination	95.8	95.8	0.0
Percent of children 12-23 months received DPT-3 vaccination	86.9	79.2	-9.7
Percent of children 12-23 months received Measles vaccination	83.8	79.2	-5.8
Percent of children 12-23 months fully vaccinated*	78.7	65.4	-20.3
Percent of births in past 5 (or 3) years delivered in medical facility	44.1	42.1	-4.8
Percent of births in past 5 (or 3) years with antenatal care	95.2	94.2	-1.1
Socio-Economic Context			
Percent of births in preceding 3 years to mothers with no educ.	18.0	10.8	-66.7
Percent of births in preceding 3 years to mothers with primary	59.9	64.0	6.4
Percent of births in preceding 3 years to mothers with second+	22.1	25.2	12.3
Gross domestic product per capita (US(1995)$)	328	334	2.0
Percent of births in past 5 years to mothers in low income H/H**	22.7	16.5	-37.6
Percent of births in past 5 years to mothers in high income H/H**	20.2	26.5	23.8

· Includes BCG, measles, and 3 doses of polio and DPT vaccines
** Income categories defined on basis of reported household assets

TABLE 2 : Odds Ratios for Time Periods Relative to 1984-86 Adjusted for Various Controls (Proportional Hazard assumption across age groups)

Controls	1984-86	1987-89	1990-92	1993-95	1996-98
None	Reference	1	1.14*	1.26**	1.13
HIV Prevalence	Reference	0.82**	0.71**	0.56**	0.40**
First Survey Dummy	Reference	1	1.14*	1.26**	1.13
Socio-economic	Reference	1	1.14*	1.32**	1.17
Biodemographic	Reference	1.02	1.16*	1.33**	1.22*
Socio-economic & Biodemographic	Reference	1.01	1.16*	1.36**	1.23**
Socio-econ. & Biodemog & HIV	Reference	0.83**	0.73**	0.61**	0.44**

Socio-Economic Controls: Education of mother (categorized as None, Incomplete Primary, Completed Primary, Incomplete Secondary, Completed Secondary, Higher)

Household wealth quintile (Very poor, poor, middle class(40%-80%), rich)

Type of place of residence (Rural or urban)

Biodemographic Controls: Age of mother at child's birth (<20, 20-34, 35+), child's birth order (first, 2-5, 6+), sex of child (male, female), preceding birth interval < 18months, 18+ months.

HIV Prevalence: Estimated HIV prevalence of district in year when child was born.

Significance levels:

* 5 percent

** 1 percent

TABLE 3: Log Odds of Death 1996-98 Relative to 1990-92 Controlling for Health and Health System Variables

Controls	1990-92 (1993 DHS)	1996-98 (1998 DHS)	Access	Vaccination	Sickness	Nutrition
None	Ref	1.03	N/A	N/A	N/A	N/A
Health Indices	Ref	0.99	0.96	0.86**	1.33**	0.95
Socio-Econ & Health	Ref.	1.03	0.97	0.88**	1.33**	0.93
Bio-Demog & Health	Ref.	0.99	0.96	0.86**	1.32**	0.95
Socio-Bio & Health	Ref.	1.03	0.97	0.88**	1.33**	0.93
Sosio, Bio,Health & HIV	Ref.	0.75*	0.9	0.97	1.21**	0.96

Health System Variables: Access is a cluster estimate of use of antenatal care and delivery by a medically-trained person.

Vaccination is a cluster estimate of immunization prevalence in cluster, based on DPT1, DPT3 and Measles among 12 to 35 month olds.

Sickness is a cluster estimate of prevalence of diarrhea, cough with difficult breathing, and fever

Nutrition is a cluster estimate of nutrition, based on proportions of children 12-35 months stunted and on proportions of women with low BMI

Odds Ratio for HIV
Prevalence in final model: 1.05**

TABLE 4 : Log Odds for Control Variables in Proportional Hazards Models

Variable	Socioeconomic Model	Biodemographic Model	Socio-Biodem Model	Socio-Biodem + HIV Model
Mother's Education				
None	Reference	N/A	Reference	Reference
Incomplete Primary	1.1	N/A	1.11	1.08
Complete Primary	0.76**	N/A	0.78**	0.79**
Incomplete Secondary	0.63**	N/A	0.67**	0.66**
Complete Secondary	0.58**	N/A	0.63**	0.65**
Higher	0.31**	N/A	0.36**	0.39*
Household Wealth				
Bottom Quintile	Reference	N/A	Reference	Reference
2nd Quintile	0.75**	N/A	0.76**	0.81**
3rd and 4th Quintiles	0.70**	N/A	0.71**	0.75**
5th Quintile	0.55**	N/A	0.56**	0.56**
Residence				
Rural	Reference	N/A	Reference	Reference
Urban	1.08	N/A	1.07	0.82*
Age of Mother at Birth of Child				
Under 20	N/A	Reference	Reference	Reference
20 to 34	N/A	0.60**	0.70**	0.73**
35 and over	N/A	0.59**	0.67**	0.71**
Birth Order of Child				
First	N/A	Reference	Reference	Reference
2nd to 5th	N/A	1.1	0.98	0.94
6th and higher	N/A	1.56**	1.24*	1.15
Sex of Child				
Male	N/A	Reference	Reference	Reference
Female	N/A	0.93	0.93	0.93
Preceding Birth Interval				
Less Than 18 Months	N/A	Reference	Reference	Reference

18 or More Months	N/A	0.57**	0.55**	0.55**
HIV Prevalence in District at Child's Birth	N/A	N/A	N/A	1.09**

TABLE 5: Log Odds of Death by Age Group & Period (No Proportionality Assumption)

Age Range	1984-86	1987-89	1990-92	1993-95	1996-98
a) No Controls					
Neonatal	Reference	0.78*	1.06	1.01	0.98
1 to 5 months	Reference	1.15	1.18	1.43*	1.08
6 to 11 months	Reference	1.01	1.26	1.42*	1.31
12 to 17 months	Reference	1.24	1.06	1.2	1.46
18 to 23 months	Reference	1.22	1.13	2.10**	2.04*
Two to four years	Reference	0.85	0.86	0.82	0.74
b) Controls for Socio-econ, Biodemog					
Neonatal	Reference	0.78*	1.03	1.05	1.02
1 to 5 months	Reference	1.16	1.23	1.56**	1.15
6 to 11 months	Reference	1.01	1.29	1.56**	1.45
12 to 17 months	Reference	1.16	1.1	1.37	1.78*
18 to 23 months	Reference	1.13	1.12	2.26**	2.27**
Two to four years	Reference	0.86	0.88	0.91	0.84
c) Controls for Socio-econ, Biodemog & HIV					
Neonatal	Reference	0.72**	0.89	0.77	0.69*
1 to 5 months	Reference	0.85	0.56**	0.41**	0.21**
6 to 11 months	Reference	0.79	0.72	0.55*	0.37**
12 to 17 months	Reference	0.93	0.67	0.54*	0.52
18 to 23 months	Reference	0.79	0.46**	0.42	0.23*
Two to four years	Reference	0.73*	0.61**	0.47**	0.32**

TABLE 6: Coefficient on HIV Prevalence Variable by Age Group

Age Group	Coefficient on HIV Prevalence
Neonatal	1.04**
1 to 5 Months	1.14**
6 to 11 Months	1.13**
12 - 17 Months	1.11**
18 - 23 Months	1.18**
Two to Four Years	1.10**

Controls for: Socioeconomic, biodemographic characterisitcs