HIV and Increases in Childhood Mortality in Kenya in the Late 1980s to the Mid-1990s

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EXECUTIVE SUMMARY

After its independence in the early 1960s, Kenya's child mortality declined rapidly. Until about 1980, the under-five mortality rate (U5MR), the probability of dying by age five, fell at an annual rate of about 4 percent per annum. This rate of decline slowed in the early 1980s to about 2 percent per annum. Data from the 1998 Kenya Demographic and Health Survey (KDHS) show that, far from declining, the U5MR increased by about 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 enrollment, and the emergence of an HIV/AIDS epidemic. One potentially positive trend was that 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 KDHSs have been merged into a single data set, and multivariate analysis has been 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 use, plus controls for reproductive dynamics (such as age of mother at the birth, birth order, sex, and preceding birth interval) were introduced into the model. In addition, an indicator of the HIV epidemic, the estimated prevalence of HIV in the district of birth at the time of each child's birth, was incorporated. With no controls, the analysis confirmed an increase in mortality of about 20 percent from 1984-86 to the highest risk period, 1994-96. Including socioeconomic and biodemographic controls tended to strengthen the upward trend in mortality, indicating that the net effect of changes in these factors was toward reducing child mortality, primarily because of a major favorable shift in the distribution of births by education of mother. Introducing controls for health variables—immunization, pregnancy and delivery care, and maternal and child malnutrition—barely changed the underlying trend between 1990-92 and 1996-98, suggesting that although use of preventive health services declined somewhat, it did not have a major effect on child mortality risks. The effect of district-level prevalence of HIV, however, is significantly positive, suggesting an increase in risk of about 5 percent per 1 percent increase in HIV prevalence. Including the HIV prevalence also changed the underlying trends fundamentally, to a sharp and monotonic decline. Although models of this sort can only demonstrate association, the HIV epidemic appears to have played a major role in the recent increases in child mortality in Kenya.

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 that may threaten child survival improvements. Better policy requires a better understanding of how important these different factors may be.

For much of its existence as an 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 its independence, Kenya's economic growth was strong and operated to mitigate the potentially harmful influence of very high fertility and rapid population growth on living standards. During this time, educational levels increased rapidly. After this period of growing prosperity, a number of internal and external factors, including less liberal and open government policies 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 from 1965 to 1980, Kenya enjoyed a rather impressive and sustained decline in under 5 mortality rate (U5MR) of 3 percent to 4 percent per annum (Hill et al., 1998), twice the rate of the average country of sub-Saharan Africa during this period. During the 1980s, child mortality decline slowed in Kenya, but it 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 Inc., 1999). Direct measures of child mortality from birth histories included in the 1998 KDHS showed a 24 percent rise in the U5MR 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. During the 10 years before the survey, the chance of dying before age five varied from 34 per 1,000 (Central Province) to 199 per 1,000 (Nyanza Province), a sixfold 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 long-term 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, and 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 threat. 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. While not able to arrive at a definitive conclusion, since association can never prove causation, we were able to identify a plausible explanation that is consistent with the information available.

The conceptual framework underlying this analysis is based on the proximate determinants model developed by Mosley and Chen (1984). 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 the following: 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; and maternal factors such as age, parity, and birth intervals. These important child survival inputs are conditioned upon a number of social and economic factors that include parental education and household economic profile (disposable income and wealth).

In this conceptual framework, the HIV epidemic can be involved in several proximate variables: as a pathogenic agent to which a child is exposed, as a factor reducing access to health services, as a nutritional factor if household food production or purchasing is reduced by sickness in the household, and perhaps as a maternal factor if the mother dies. However, the epidemic has additional indirect effects on child survival, with economic effects at both the family and the community level. Mother-to-child transmission of HIV can occur during pregnancy, childbirth, or breastfeeding. Available evidence indicates that, in the absence of antiretroviral therapy, about 30 percent of children born to HIV-positive mothers will be infected and that 60 percent of infected children 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 Demographic and Health Survey (DHS) and HIV prevalence data found that most countries in sub-Saharan Africa with high HIV prevalence (5 percent or more of adults seropositive) also experienced increased U5MRs (Adetunji, 2000).

Evidence concerning changes in the 1990s in both background and proximate variables is summarized in Table 1 and reviewed below.

Child Mortality

The probability of dying by five, or the U5MR, increased by 16 percent between the five-year period before the 1993 KDHS and the three-year period before the 1998 KDHS.

HIV/AIDS

During the five-year period between 1993 and 1998, the National AIDS and STD Control Programme (NASCOP), responsible for the national HIV/AIDS surveillance system in Kenya, estimates that HIV prevalence rose from 9 to 13 percent (NASCOP, 1999).

Table 1. Levels and trends of various health and development indicators, Kenya 1993-98

Indicator	1993	1998	% change
Childhood mortality (5 years before KDHS)			
Under-5 mortality rate (per 1,000 live births)	96.1	111.5	16
Infant mortality rate (per 1,000 live births)	61.7	73.7	19
Child mortality rate (per 1,000 survivors to age 1)	36.7	40.8	11
Maternal and child health indicators			
Percentage of children 6-35 months with fever in last 2 weeks	48.1	44.4	-8
Percentage of children 6-35 months with diarrhea in last 2 weeks	19.8	18.2	-8
Percentage of children <12 months with cough/rapid breathing	20.8	18.5	-11
Percentage of children under age 5 stunted	32.7	33.0	1
Percentage of births in last 5 years to women with BMI < 18.5	9.2	11.9	29
Use of maternal and child health services			
Percentage of children 12-23 months received DPT-1 vaccination	95.8	95.8	0
Percentage of children 12-23 months received DPT-3 vaccination	86.9	79.2	-9
Percentage of children 12-23 months received measles vaccination	83.8	79.2	-5
Percentage of children 12-23 months fully vaccinated*	78.7	65.4	-17
Percentage of births in past 5 (or 3) years with antenatal care	93.1	92.2	-1
Percentage of births in past 5 (or 3) years delivered by skilled attendant	45.4	44.3	-2
Percentage of births in past 5 (or 3) years delivered in medical facility	44.1	42.1	-5
Socioeconomic context			
Percentage of births in preceding 3 years to mothers with no education	18.0	10.8	-67
Percentage of births in preceding 3 years to mothers with primary	59.9	64.0	6
Percentage of births in preceding 3 years to mothers with secondary+	22.1	25.2	12
Gross domestic product per capita (1995 US$)	328	334	2
Percentage of births in past 5 years to mothers in low-income H/H**	22.7	16.5	-38
Percentage of births in past 5 years to mothers in high-income H/H**	20.2	26.5	24
Biodemographic context			
Percentage of first births in preceding 5 years	20.0	25.3	27
Percentage of sixth and higher births in preceding 5 years	26.3	20.7	-21
Percentage of births following interval < 18 months in preceding 5 years	9.3	8.7	-6
Estimated HIV prevalence			
HIV prevalence (women 15 to 49) from surveillance data	8.8	13.3	34

Notes: Data are from 1993 KDHS and 1998 KDHS (National Council for Population and Development, Central Bureau of Statistics, and Macro International Inc., 1994 and 1999), or tabulations from basic data for this report.

* Includes BCG, measles, and three doses of polio and DPT vaccines.

** Income categories defined on basis of reported household (H/H) assets.

Childhood Illness

Although accurate information on cause of death is lacking, the cause of death structure of under-five mortality in Kenya is probably, like most countries in sub-Saharan Africa, dominated by pneumonia, malaria, measles, and diarrheal disease, which are estimated to have been responsible for some 60 percent of the disease burden in the region around 1990 (Murray and Lopez, 1996). DHS data for Kenya, however, do not suggest an increase in childhood illness: Between 1993 and 1998, the two-week prevalence of cough with rapid breathing among children under 12 months of age, of fever among children age 6-35 months, and of diarrhea among children age 6-35 months all decreased by close to 10 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 surveys, although the proportion of births to women with low body mass index (BMI) did increase sharply.

Use of Maternal and Child Health 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 age 12-23 months also declined, especially the proportion of children who had received all recommended vaccinations (from 79 percent in 1993 to 65 percent in 1998), but it was still at high levels by African standards.

Socioeconomic Context

The 1998 KDHS produced worrying evidence that after decades of improvement, levels of schooling among girls fell during the 1990s (National Council for Population and Development, Central Bureau of Statistics, and Macro International Inc., 1999). However, it will take some time for the effects of this change to work their way into the educational status of the mothers of a cohort of children. Table 1 shows that the proportion of births to mothers with no education fell from 18 percent in 1988-93 to 11 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 gross domestic product per capita increased little, but on-average household wealth, as measured by assets owned, increased between 1993 and 1998, and the proportion of births occurring in very poor 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) the estimates for the late 1970s (8.1). Contraceptive prevalence among currently married women was 31 percent in 1998. For the births in the five years before the 1993 and 1998 KDHSs, the proportion of first births increased from 20 to 25 percent, while the proportion of sixth and higher births dropped from 26 to 21 percent. The proportion of births following a birth interval of less than 18 months also fell slightly, from 9.3 to 8.7 percent.

Data

The data on child mortality and many of the socioeconomic and biodemographic variables used in this study come from the 1993 and 1998 KDHSs. Both of these surveys excluded the very low population density areas of northern and northeastern Kenya. 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 specifically by age and time period, but they 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 downward 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. Ng'weshemi et al. (2003) estimated this bias to be quite low in Magu District in Tanzania (2.1 percent relative difference), but average HIV prevalence among women in their study was less than half that observed nationally in Kenya in 1998. Artzrouni and Zaba (2003) also show that survivor bias on child mortality estimates is quite small for periods shortly before a survey, though the effects increase for time periods more remote from the survey date.

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.

Several other important pieces of data come from the KDHSs. These surveys record the following information about health variables for births of surviving children born (to interviewed, surviving women) in defined periods prior to the survey (those born since January 1988, or roughly the five years preceding the 1993 survey, and since January 1995, or roughly the three years preceding the 1998 survey): pregnancy and delivery care as well as heights and weights of the mothers 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 socioeconomic 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 HIV prevalence at the district level are obtained from the surveillance system maintained by NASCOP. HIV surveillance data are obtained through annual surveys of surveillance sites that collect data on pregnant women visiting selected public and mission hospitals in Kenya for routine antenatal services. During the first antenatal visit for a particular pregnancy, blood is obtained from mothers for tests, such as syphilis and blood sugar, for clinical purposes. A portion of the blood that remains is then tested for HIV after all identifiers of the client have been stripped to ensure anonymity. The blood samples are tested at each site, with 10 percent of serum samples being sent to NASCOP for quality control. Surveillance for HIV initially started in 1990 at 13 sites that were considered to have a high prevalence of HIV. These sites were mainly urban. In 1994, eight peri-urban sites were added. Sixteen sites were added in the year 2001, the majority of them being rural.

Data from field sites are forwarded to NASCOP's national office for data entry. Retesting (blind) of the 10 percent of the blood serum collected for quality control is conducted at the National Public Health Laboratory (NPHL) to obtain field-testing error. HIV prevalence for each site is then obtained from the field data and adjusted for errors as per the results from the NPHL. The annual prevalence for each site is then smoothed by fitting a gamma curve to prevalence for all available years. The smoothed values from the gamma curve are assumed to be representative of the urban (or rural, for peri-urban sites) portion of the district, and they are then used to extrapolate to district, provincial, and national estimates. Each district that does not have a sentinel site of its own is assumed to have the same urban HIV prevalence as that of an urban site from another district selected on the basis of cultural similarity. Prevalence for the rural population is calculated by assuming that the prevalence of the rural population is either 40, 70, or 80 percent of the prevalence of the urban population on the basis of judgments derived from prevalence estimates from peri-urban sites. Census data on the urban and rural population of the district are then used to weight the urban and rural prevalence estimates to get the total number of people infected in the district. Provincial and national estimates are then obtained by accumulating the district estimates.

Table 2 summarizes the characteristics of women included in the NASCOP HIV surveillance database (for all observations for the years 1990-98) with the characteristics of women having births in the same period from the KDHSs. The women included in the sentinel site sample are on average younger, have lower parity, are better educated, and are more urban than the average Kenyan woman having a birth in the KDHSs. Young women tend to have a higher prevalence of HIV than older women, as indicated by the surveillance data and AIDS case reports (Kenya Ministry of Health, 2001).

Table 2. Characteristics of mothers surveyed in sentinel surveillance and Demographic and Health Surveys in Kenya between 1990 and 1998

Variable	Category	Surveillance subjects 1990-98, % (n=53,748)	DHS subjects 1990-98 % (n=66,763)
Age groups	15-24	58.4	48.2
	25-34	35.3	40.8
	35+	5.8	11.0
Parity	0	39.2	22.3
	1-4	51.6	54.1
	5+	9.2	23.6
Education	None	0.0	15.2
	Primary	68.2	61.0
	Secondary	29.9	22.3
	College	1.9	1.5
Residence	Urban	28.6	16.3
	Rural	71.4	83.7

Variables

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 (12-17 months), late second year (18-23 months), third year (24-35 months), fourth year (36-47 months), and fifth year (48-59 months). Each child is included in as many age ranges as he or she completed before the interview date. If a child died during an interval completed prior to the interview date, the child was included within that interval. For example:

- A child born 65 months before the 1993 survey and still alive at the time of the survey would be included in all eight age ranges.
- 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.
- However, a child born 27 months before the survey who died at age 25 months would only be included for five age ranges (since the sixth interval could not have been completed, even had the child survived).
- And a child born 27 months before the 1993 survey and still alive at the survey would also only be included for five age ranges (since the sixth interval was not completed).

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. For studying 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 the period in which the midpoint 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 midpoint of these ranges would be reached in 1989. Age ranges for the late postneonatal through two completed years fall in the period 1990-92, and the age ranges for three and four completed years fall in the period 1993-95 (even though a few months of the age three period fall in the period 1990-92). The period of an age range for a child who dies in the age range is determined in the same manner.

Socioeconomic variables include mother's education (categorized as none, 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, as well as on housing quality, whether the house has electricity, a finished floor, and a "permanent" roof of corrugated iron or tiles), categorized as very poor (bottom quintile), poor (20 to 39 percent), middle class (40 to 79 percent), or rich (top 20 percent)[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, 20-34, 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).

Three 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 KDHS) or three (1998 KDHS) 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. So that 1993 and 1998 data could be compared, 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 district rather than for the individual child, since for many indicators, such as immunization history, information was only collected for surviving children. An attempt was made to use health indicators at the sample cluster level, roughly interpretable as a community. However, for most indicators, the number of observations was small, so the results reported here are based on the district average.[2]

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[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, 1998).

[2] To stabilize values, we tried 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. However, using the stabilized cluster estimate made no substantive difference to the models relative to using the district average.

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 averaging proportions of eligible children age 12 to 35 months with each of the four immunizations (BCG, DPT1, DPT3, and measles). Finally, a variable reflecting nutrition was obtained by combining the proportion of stunted children age 12 to 35 months and the proportion of recent mothers (not currently pregnant and at least three months postpartum) with a BMI below 18.5. Information on proportions of children recently sick with fever, diarrhea, or cough was not used, on the grounds that it might be influenced by HIV prevalence.

Methods

Logistic regression has been used to estimate the effects of the independent variables on the log odds of dying, controlling for age range. Since all age ranges are included in the models simultaneously, it is assumed that the proportionate effects of all variables are constant across age, though we subsequently test this assumption. Throughout, the models allowed for random effects at the observation level of the cluster. 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 mortality trend. District-level estimates of HIV prevalence during the year in which a given child was born were then included. Control variables were then introduced, initially without HIV prevalence, first for socioeconomic variables alone, then for biodemographic variables alone, then for socioeconomic and biodemographic variables, and finally for HIV prevalence. 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 with 1993 KDHS data and the 1996-98 period with 1998 KDHS data. The only time period variable used is for 1996-98 relative to 1990-92. This limitation has the additional effect of limiting analysis largely to one child per woman for each period.

The estimates of HIV prevalence used in the analysis are for districts, but these estimates are smoothed by the gamma function even for those districts with sentinel sites, and for the majority of districts, the estimates are based on extrapolation from estimates for a similar district. We tested the sensitivity of our results to this extrapolation in three ways: limiting analysis only to districts with surveillance sites, using only observed (unsmoothed) prevalence estimates, and including a dummy variable in the analysis of all districts to indicate whether a district had a surveillance site or not.

Results

HIV and Trends in Childhood Mortality

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 3. Risks are similar in 1984-

86 and 1987-89, rise somewhat (about 9 percent in the odds across all age groups) in 1990-92, rise further in 1993-95 (21 percent higher than in 1984-86, and strongly significant), but decline in 1996-98 (while still remaining above levels in the mid-1980s). A quite different pattern emerges when HIV prevalence is controlled (row 2 of the table): Instead of increasing, child mortality risks decline strongly and significantly, to only half of their 1984-86 level by 1996-98. The third model (without HIV) introduces a control for survey (whether an observation was from the 1993 or 1998 KDHS); the results are almost identical to those of the first model, indicating that the trends are not the result of differential data quality in 1993 and 1998. The fourth, fifth, and sixth models introduce socioeconomic controls, biodemographic controls, and both together, respectively. These controls have no significant effect on the trend variables, though they tend to make the upward trend slightly stronger, suggesting a small net protective effect for socioeconomic and biodemographic changes during the period. The last model reintroduces HIV prevalence into the full model. Once again, the effect on trend is unmistakable: A steady rise is replaced by a sharp and significant decline. The results strongly suggest that HIV prevalence is largely responsible, directly or indirectly, for the increasing mortality in the 1990s.

Table 3. 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
Age only	Reference	0.98	1.09	1.21**	1.08
HIV prevalence	Reference	0.81**	0.74**	0.64**	0.48**
First survey dummy	Reference	0.98	1.10	1.22**	1.09
Socioeconomic factors	Reference	0.99	1.10	1.24**	1.11
Biodemographic factors	Reference	0.98	1.11*	1.25**	1.13
Socioeconomic and biodemographic factors	Reference	0.99	1.12*	1.28**	1.16
Socioeconomic and biodemographic factors and HIV prevalence	Reference	0.81**	0.75**	0.66**	0.50**

Socioeconomic controls: Education of mother (categorized as none, incomplete primary, complete primary, incomplete secondary, complete secondary, higher), household wealth quintile (very poor, poor, middle class, 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, second to fifth, sixth and higher), sex of child (male, female), preceding birth interval (<18 months, 18+ months).

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

Significance levels: * 5 percent
** 1 percent

Table 4 explores the possibility that an erosion of the use or the quality of health services accounted for the mortality increase, using the indices of health status and health service described above. Unlike the results in Table 3, no observations from the 1998 survey were used for the 1990-92 period, so results differ slightly. Table 4 follows the same modeling strategy as Table 3. The 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, but once HIV prevalence is included (row 2 of Table 4), the underlying child mortality decrease (in the absence of HIV) is around 40 percent, and the coefficient on the HIV prevalence is 0.08, indicating approximately an 8 percent increase in

underlying risk (odds) for every 1 percentage point increase in HIV prevalence. Introducing the three health system indices without HIV (row 3 of Table 4) does not change this significantly, though the summary vaccination measure is highly significant and indicative of lower mortality risk. 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. Adding socioeconomic, biodemographic, and then both socioeconomic and biodemographic controls in models four to six, respectively, does not affect the basic outcome. Once again, however, introducing HIV makes a huge difference, with a mortality decline between 1990-92 and 1996-98 of about 30 percent. The coefficient on HIV is slightly smaller than in row 2, but it is still 0.06 and highly significant. Immunization remains significantly protective, though not quite as strongly as in the earlier models (suggesting a negative association between immunization coverage and HIV prevalence).

Table 4. Odds ratios for 1996-98 relative to 1990-92, controlling for health and health system variables

Controls	1990-92 (1993 KDHS)	1996-98 (1998 KDHS)	HIV prevalence[4]
Age only	Reference	0.98	N/A
Age and HIV	Reference	0.61**	1.083**
Health indices (access,[1] vaccination,[2] and nutrition[3])	Reference	1.00	N/A
Socioeconomic and health	Reference	1.04	N/A
Biodemographic and health	Reference	1.00	N/A
Socioeconomic, biodemographic, and health	Reference	1.03	N/A
Socioeconomic, biodemographic, health, and HIV	Reference	0.69**	1.062**

N/A=not applicable.
** Significance level 1 percent.
[1] Access is a district estimate of use of antenatal care and delivery by a medically trained person.
[2] Vaccination is a district estimate of immunization prevalence, based on BCG, DPT1, DPT3, and measles immunizations among 12 to 35 month olds.
[3] Nutrition is a district estimate of nutrition, calculated as the average of the proportion of children 12-35 months stunted and the proportion of women with low BMI. A high value indicates high malnutrition.
[4] HIV prevalence is a district estimate of prevalence in the year of an individual child's birth.

Biodemographic and Socioeconomic Determinants

Our primary focus in this paper is on trends, in the form of period effects on mortality. It is also interesting, however, to examine the effects of biodemographic and socioeconomic control variables, particularly in the absence or presence of a control for HIV prevalence. Table 5 shows the odds ratios for variables representing mother's education, household wealth, type of place of residence, age of mother at the birth of the child, the child's birth order, the child's sex, and whether the preceding birth interval was short. Odds ratios are shown for four models: socioeconomic variables only, biodemographic variables only, socioeconomic and biodemographic variables, and both of these variables plus estimated HIV prevalence in the district during 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. Complete primary education reduces the odds of death by 20 percent, and postsecondary education is associated with mortality risks nearly 60 percent lower than those of mothers with no education. Household wealth is also strongly (and monotonically) associated with mortality risk: Children born into the most affluent households are 40 percent less

likely to die than those born into the poorest, even after controlling for all 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, although not significant, changes when the HIV variable is introduced: Without the HIV control, urban areas appear to have somewhat higher mortality risks than rural areas, but when HIV prevalence is controlled for, child mortality is lower in urban areas than in rural areas. In general, urban areas have higher HIV prevalence than do rural areas, so this pattern is in line with expectations.

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 20-34. Controlling for socioeconomic factors reduces the estimated excess risk of being born to a young mother relative to the model controlling for biodemographic factors alone, which suggests that part of the risk is accounted for by poor or less educated women being more likely to have births at early ages. High parity births are at higher risk than are lower birth orders in the biodemographic model, but the effect disappears once socioeconomic factors are controlled. Girls have somewhat lower mortality risks than boys. Being born after a very short birth interval, less than 18 months, is associated with greatly elevated risk that is not attenuated by socioeconomic controls. The association of these variables with mortality is essentially independent of the HIV variable.

Testing Proportionality Assumptions

The models presented in Tables 3 through 5 assume proportionality—that the effect of each variable, including HIV prevalence, is proportionately the same in each age range. Serological studies suggest rate ratios for HIV-positive children relative to HIV-negative children that rise with age: For example, in Rwanda, the rate ratio for HIV-infected children was about 4 in infancy (that is, the mortality hazard in infancy for HIV-positive children was four times that for HIV-negative children), but about 9 in the second year of life (Spira et al., 1999). Table 6 shows the coefficient of HIV prevalence in models for separate age ranges; in this case, because of small numbers of events, the ranges used are neonatal, one to five months, six to 11 months, 12 to 23 months, and two to four years. HIV is associated with significantly higher risks in all age ranges, but the effect is smallest in the neonatal period. The risk is high across all remaining age ranges, although there is a hint that risks may begin to decline after age two. 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.

Sensitivity to Extrapolation in the Sentinel Surveillance Data

The above analysis is based on KDHS data from 34 districts, but the HIV prevalence data are derived from urban sentinel sites in only 13 districts from 1990 and eight peri-urban sites from 1994. For a majority of districts, therefore, HIV prevalence was inferred from a district regarded as "similar," rather than based on an in-district sentinel site. Three analyses were conducted to assess the possible bias resulting from this extrapolation. The first analysis consisted of a model that included all observations, but added an additional variable (whether the district was a surveillance district or not), interactions between that variable and each time period variable, and interactions between that variable and the HIV prevalence variable. The second analysis restricted the model to data only for the surveillance districts. For the third analysis, an additional variation of the health model was applied, limited to surveillance districts only and using the observed annual values, not the smoothed HIV prevalence estimates.

Table 5. Odds ratios for control variables in proportional hazards models

Variable	Socioeconomic model	Biodemographic model	Socioeconomic and biodemographic models	Socioeconomic, biodemographic, and HIV models
Mother's education				
None	Reference	N/A	Reference	Reference
Incomplete primary	1.11	N/A	1.10	1.08
Complete primary	0.81**	N/A	0.82**	0.81**
Incomplete secondary	0.67**	N/A	0.69**	0.68**
Complete secondary	0.65**	N/A	0.69**	0.69**
Higher	0.37**	N/A	0.42**	0.43*
Household wealth				
Very poor	Reference	N/A	Reference	Reference
Poor	0.81**	N/A	0.82**	0.83**
Middle class	0.75**	N/A	0.75**	0.76**
Rich	0.61**	N/A	0.61**	0.60**
Residence				
Rural	Reference	N/A	Reference	Reference
Urban	1.14	N/A	1.12	0.93
Age of mother at birth of child				
Under 20	N/A	Reference	Reference	Reference
20 to 34	N/A	0.57**	0.73**	0.73**
35 or over	N/A	0.56**	0.72**	0.71**
Birth order of child				
First	N/A	Reference	Reference	Reference
Second to fifth	N/A	1.20**	0.93	0.91
Sixth or higher	N/A	1.66**	1.17	1.13
Sex of child				
Male	N/A	Reference	Reference	Reference
Female	N/A	0.90*	0.91*	0.92*
Preceding birth interval				
<18 months	N/A	Reference	Reference	Reference
18+ months	N/A	0.57**	0.56**	0.56**
HIV prevalence in district at child's birth	N/A	N/A	N/A	1.07**

* Significance level 5 percent.
** Significance level 1 percent.

Table 6. Coefficient on HIV prevalence variable by age range of child

Controls for socioeconomic and biodemographic characteristics.

Age group	All periods	Periods 3 and 5 only
Neonatal	0.039**	0.043**
1-5 months	0.115**	0.115**
6-11 months	0.107**	0.101**
12-23 months	0.119**	0.085**
2-4 years	0.066**	0.094**

** Significance level 1 percent.

Table 7 shows the results of these simulations, in terms of the odds ratios for the time period variables, the HIV prevalence variable, and appropriate interactions, as well as the odds ratios from comparable models without control for surveillance districts. In the basic model with all districts and a dummy variable to identify surveillance districts, the odds ratio for the surveillance dummy is 0.77, indicating much lower mortality risks in surveillance than in nonsurveillance districts after controlling for other factors. The time trends are also quite different: Period dummies show period risks dropping rapidly in nonsurveillance districts but being approximately flat in surveillance districts. The HIV prevalence variable suggests almost a 10 percent increase in the risk of dying per 1 percent increase in HIV prevalence in the nonsurveillance districts, but only about a 4 percent increase in surveillance districts. The interaction of HIV prevalence and surveillance district has a significant odds ratio of 0.95, further suggesting not just lower mortality in surveillance districts but, within such districts, relatively lower mortality in those with higher HIV prevalence. These findings are supported by the second model, limiting analysis to surveillance districts only, and by both of the health models. It appears that the surveillance districts are systematically different from nonsurveillance districts, which likely reflects the nonrandom selection of surveillance sites or, possibly, systematic errors in the extrapolations from sentinel to nonsentinel sites. It is worth noting, however, that the HIV prevalence variable remains associated with significantly higher odds of child death, except for the model with unsmoothed values, in which it is still positive but not quite significant, perhaps because of noise in the observed values of HIV prevalence.

Table 7. Odds ratios for time period, HIV from sensitivity tests

Variable	Basic model[1]	Basic model, surveillance dummy	Basic model, surveillance districts only	Health model[2]	Health model, surveillance districts	Health model, surveillance districts, observed previously
Period 2	0.81**	0.74**	0.93	N/A	N/A	N/A
Period 3	0.75**	0.61**	1.02	Reference	Reference	Reference
Period 4	0.66**	0.51**	1.00	N/A	N/A	N/A
Period 5	0.50**	0.34**	0.96	0.69**	0.75	0.84
Surveillance dummy (SD)	N/A	0.77**	N/A	N/A	N/A	N/A
SD period 2	N/A	1.31*	N/A	N/A	N/A	N/A
SD period 3	N/A	1.74**	N/A	N/A	N/A	N/A
SD period 4	N/A	2.04**	N/A	N/A	N/A	N/A
SD period 5	N/A	2.90**	N/A	N/A	N/A	N/A
HIV prevalence	1.073**	1.094**	1.038**	1.062**	1.036*	1.021
HIV prev. * S.D.	N/A	0.95**	N/A	N/A	N/A	N/A
Number of spells	197,037	197,037	78,277	45,319	18,230	22,282

* Significance level 5 percent.
** Significance level 1 percent.
[1] The basic model controls for age of child, socioeconomic and biodemographic variables, and district-level estimated HIV prevalence in the year of the child's birth for all districts and time periods. It is the same model as reported in the last row of Table 3.
[2] The health model controls for age of child, socioeconomic and biodemographic variables, district-level estimated HIV prevalence in the year of the child's birth, and district-level indices of access to health care, immunization coverage, and nutrition for all districts and the two time periods preceding each DHS survey. It is the same model as reported in the last row of Table 4.

Discussion

In all of the analysis conducted, the prevalence of HIV at the district level at the time of the birth of the child is found to be associated with increased mortality risk for 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 were 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 are. There are also a number of possible indirect effects. HIV-positive parents may be less able to look after their children, as compared with those who are not infected. Economic conditions may be worse in HIV-prevalent areas than in less affected areas, although 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 who are seropositive. Health services in high HIV prevalence areas may be overwhelmed by AIDS cases and thus less able to give necessary care and attention to a child that is sick, regardless of the child's serostatus, though again the health variables we include should control for such differences to some extent. Lacking serological data, we have no definitive way of differentiating the

effects of these various mechanisms, but it seems plausible, given controls for socioeconomic, biodemographic, and health sector factors, that the direct effect is important.

The effect of HIV in this analysis 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 Kenya, with virtually no antiretroviral treatment, the mother will surely die. Child mortality rates based on retrospective reports of women will thus be distorted by survivor bias. This effect may not be very large early in an HIV epidemic, and it will not have large effects for mortality estimates for periods shortly before either survey; but in a more mature epidemic, such as that in Kenya in the late 1990s, this bias will have substantial effects for period estimates a decade or more before the survey. If prevalence plateaus, after a lag of a few years, the size of the bias is likely to plateau as well, and mortality rates of children will then (other things being equal) 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.

The most puzzling finding of this analysis is the wide difference between districts that have HIV surveillance sites and those that do not. The models suggest that, in the former, childhood mortality risks were essentially constant throughout the period under study, once socioeconomic, biodemographic, health sector, and HIV prevalence rates were controlled, whereas in the latter there was a strong secular decline. Sentinel sites were initially located in urban areas; it is only quite recently that rural sites have been established. Extrapolation to district-level estimates was based on assumptions about urban/rural differences in prevalence, and extrapolation to districts without sites was based on perceptions of similarities between districts. Our results could be indicative of strong selection effects due to the nonrandom selection of sentinel sites, or they could indicate that the extrapolations were probably incorrect, both in level and in time trend, overestimating the pace of increase in prevalence in the nonsentinel districts. Further analysis of the district-level prevalence trends, such as in comparison to rates of change of proportions of children under age 15 with one or both parents dead, is necessary.

Our analysis suggests that the recent increases in childhood mortality in Kenya are most likely associated with the HIV epidemic. However, because of the differences between surveillance and nonsurveillance districts, we cannot draw conclusions about what would have happened to child mortality in Kenya in the absence of the HIV epidemic. Our best guess would be the analysis limited to, or controlling for, surveillance districts, suggesting little change in underlying risks (after controlling for observed factors) from the mid-1980s to the late 1990s. Our analysis also does 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, and 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.

RECOMMENDATIONS

The HIV epidemic in Kenya has increased mortality risks at most ages. At the time of the 1993 KDHS, 7 percent of children under the age of 15 had lost at least one parent; this number had risen to 9.4 percent by the 1998 KDHS. This paper provides evidence that HIV prevalence at the district level is associated with increased child mortality risks in Kenya in the 1990s. This finding is not surprising, considering the fact that 13 percent of women of reproductive age are HIV positive and that 30 percent of babies born to infected mothers are likely to become infected, a majority of whom will die before reaching their fifth birthdays. On the basis of our results, we make the following recommendations.

i. Further study is needed to differentiate between the direct consequences of HIV on child mortality (through reduced immune function) and the indirect consequences (through the death of one or both parents, economic costs, reduced health service effectiveness because of the burden of AIDS cases, and increased exposure to infectious diseases). Interventions can only be effectively planned if these alternative pathways are better quantified.

ii. Health policy in Kenya needs country-specific information on the pattern of excess AIDS mortality in children. A population surveillance site is needed to provide such information. Such a site could also be the setting for intervention research.

iii. Programs for the prevention of mother-to-child transmission (PMTCT) of HIV should be initiated in districts where they do not currently exist and strengthened in districts where they do exist. The government of Kenya, with assistance from international donors, is currently making efforts to expand PMTCT services (for example, Kenya is one of the U.S. President's Emergency Plan focus countries).

iv. The circumstances of children who are orphaned by HIV/AIDS and are uninfected need to be investigated to provide appropriate services for such children.

v. Survey instruments such as those used by the DHS project should be designed in high HIV prevalence settings to ensure that adequate information is collected about children whose mothers have died, including heights, weights, and immunization coverage. The most recent DHS questionnaires now include anthropometry and anemia testing in the Household Questionnaire, rather than in the Women's Questionnaire, to ensure that such data are collected on all children under age six, including those whose mothers do not live in the household. This form of the questionnaire was implemented in the 2003 KDHS.

vi. Sentinel surveillance is unlikely to provide adequate information about prevalence because of a range of selection problems. Information from surveillance sites could be strengthened by using indirect indicators (such as proportions with dead parents from census data). The 2003 KDHS includes population-based HIV testing, which will provide additional insights and analytical opportunities.

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