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Early Childhood Mortality in Kenya

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Trends, Age Patterns, and Determinants of Early Childhood Mortality in Kenya

by

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INTRODUCTION

Kenya is among the most heterogeneous of African nations. Rich ethnic and linguistic diversity, and sharp climatic and ecological variation characterize this country of about 23 million people. It is true that marked regional and socioeconomic variations remain regarding the health and survival of Kenya's children (NCPD/IRD, 1989). While comparison of recent estimates from the 1989 Kenya Demographic and Health Survey (KDHS) with those of the 1977/78 Kenya Fertility Survey (KFS) suggest a significant improvement in child survival prospects over the last decade, nearly 1 in 10 Kenyan children still do not live to see their 5th birthday. Efforts to sustain the decline in childhood mortality and the policies on which those efforts hinge may be facilitated by detailed information regarding changes in mortality risk over time and the distribution of mortality risk across social, economic, environmental, and biodemographic characteristics of the population.

This study has two parts. In the first, KDHS and KFS birth history data are used to document, in-depth trends in the level and age pattern of childhood mortality during the last two decades. The quality of the KDHS mortality data is assessed using tests of internal and external consistency, and a re-evaluation of current levels of age-specific mortality is undertaken. The second part of the paper delves into the question of the socioeconomic determinants of age-specific mortality risk among Kenyan children. In this paper the focus is on the role of better maternal education and the intermediate factors that affect the education advantage in child survival. In particular, interest is centered on whether health services use and the pattern of family formation serve to mediate the maternal education effect.

1 TRENDS IN THE LEVEL AND AGE PATTERNS OF CHILDHOOD MORTALITY

1.1 The Data and Methods

This study uses data from the 1989 Kenya Demographic and Health Survey (KDHS)[1] and the 1977/78 Kenya Fertility Survey (KFS).[2] Both KDHS and KFS samples were national in coverage, with the exclusion of the North Eastern Province and four northern districts that together comprise only about 5 percent of Kenya's population. A total of 7,150 and 8,100 women age 15–49 were interviewed in the KDHS and KFS surveys, respectively. A detailed description of the sampling methodologies and actual samples of women can be found in the KDHS and KFS final reports (NCPD/DHS, 1989; Republic of Kenya/WFS, 1980).

Direct period life table estimates of mortality were calculated from the birth histories of the KDHS and KFS. The structure of these two birth histories are nearly identical, except that nonlive birth pregnancies were recorded in the KFS, but not in the KDHS. Also, infants under 1 month of age at death, can be broken down by day of death from the KDHS data, but not from the KFS data. Three 5-year period estimates were produced from each survey with the most recent estimate from each survey including exposure in the year of the survey, plus all exposure in the 5 full preceding calendar years.[3]

Mortality probabilities during the neonatal period (zero completed months), the postneonatal period (1 to 11 months), toddler period (12 to 23 months), and period between the second and fifth birthdays were calculated, as were the conventional summary measures:

Infant Mortality Rate ($_1q_0$)—probability of dying before the first birthday;

Under-two Mortality ($_2q_0$)—probability of dying before the second birthday;

Child Mortality ($_4q_1$)—probability of dying between the first and fifth birthdays; and Under-five Mortality ($_5q_0$)—probability of dying before the fifth birthday.

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[1] The KDHS was carried out jointly by the National Center for Population and Development and the Central Bureau of Statistics, Nairobi, with technical assistance provided by the Institute for Resource Development (IRD), Macro International, Columbia, Maryland.

[2] Implemented by the Central Bureau of Statistics (CBS), Nairobi, with technical assistance from the International Statistical Institute (ISI), Netherlands.

[3] This makes for well over 5 years of exposure in the most recent period estimate but maintains exactly 5 calendar years of exposure in the two preceding period estimates for both surveys.

All analytical procedures were applied uniformly across the data of both surveys in order to maximize comparability of the estimates.

1.2 Data Quality

Rates of childhood mortality are subject to both sampling and nonsampling errors. The results of some checks for various non-sampling errors are described below; namely, underreporting of early childhood deaths (which would result in underestimates of mortality), and misreporting ages at death (which would tend to distort the age pattern of under-five mortality).[4]

1.2.1 Accuracy of Age at Death Data

Rounding and gross misstatement of ages at death may distort the age pattern of mortality. This is of particular concern when misreporting shifts deaths between age segments for which rates are calculated—which is the case for the calculation of infant and child mortality when there exists significant "heaping" of ages at death at 12 months. Figure 1.1 shows the age at death distribution for deaths that occurred before the second birthday for the 5-year periods before each survey. The close similarity is striking between the reporting patterns from the two surveys: a large heap at 12 months, a small deficit of deaths at 10 and 11 months, and a small heap at 18 months. The 18-month heap will not affect rate calculation because it falls in the middle of the age segment 12–23 months. The 12-month heap (some of which is apparently drawn from the under-1 period), on the other hand, will lead to underestimates of infant mortality and overestimates of ${}_1q_1$ and ${}_4q_1$.[5] The size of the heaps are small compared to the total number of infant deaths, so that the effect of reassigning a fraction (even as many as half, which is an extreme assumption) of the heap back to the infant age segment would be minimal—about a 5 percent increase in ${}_1q_0$. More importantly, trend analysis is not affected because of the nearly identical reporting pattern between the KFS and KDHS. No adjustment was therefore made for heaping for analysis of trends.

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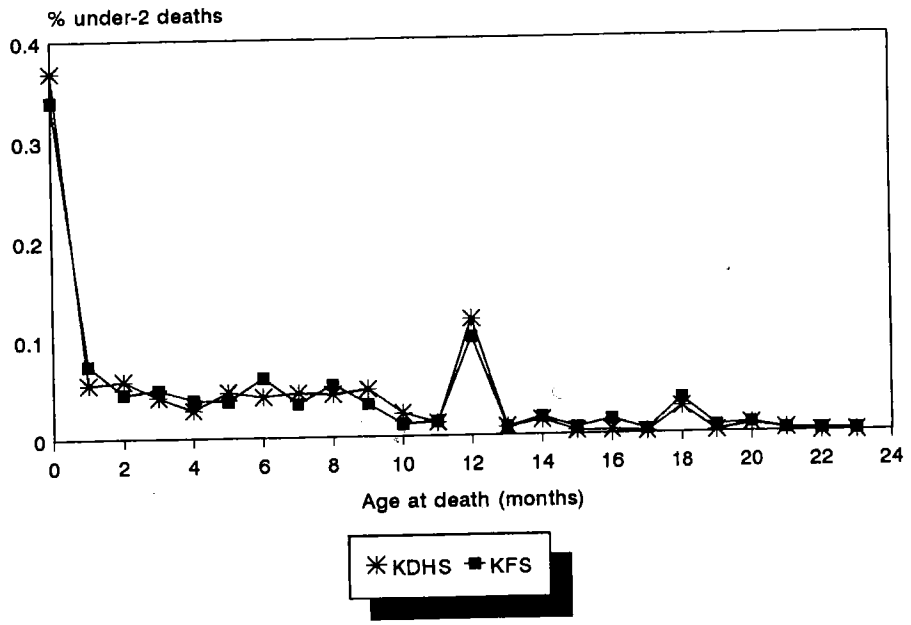
[4] The completeness of age at death reporting was quite good in both the KFS (97.5%) and the KDHS (98.0%) for the 15-year period preceding the surveys.

[5] Under-two mortality ${}_2q_0$ and under-five mortality, ${}_5q_0$ are not affected.

Figure 1.1
Percentage distribution of under-2 deaths by reported age at death. 1977/78 KFS and 1988/89 KDHS



1.2.2 Underreporting of Childhood Deaths

An approach commonly taken to evaluate the possibility of omission is to examine patterns in the data more plausibly explained by data error (omission) than by the existing demographic reality. There are certain expectations that must be dealt with before looking at the data. For instance, we expect the sex ratio at birth (M/F x 100) to fall between 102 and 107, with more boys than girls born. Table 1.1 shows that the sex ratio at birth for the 0-5 and 11-15 year periods before the KDHS and for the 11-15 year period before the KFS fall outside the expected range, and may indicate that boys have been disproportionately omitted from the birth histories.

If this is the case and omitted boys experienced higher mortality than those included in the birth histories, then our rates of mortality would be biased downward, i.e., underestimated. Also, under this scenario of omission of male deaths, one expects the relative risk of neonatal mortality associated with sex (sex risk ratio = NMR(boys)/NMR(girls)) to be biased downward. This is, however, apparently not the case. Sex risk ratios are well above 1.00 for the two KDHS periods and one KFS period with low

Table 1.1 Sex ratio at birth

Ratio of early neonatal to all neonatal mortality, ratio of neonatal to infant mortality, and ratio of infant to under-five by sex and by five-year calendar period before the 1977/78 KFS and the 1988/89 KDHS.

	Years before KFS			Years before KDHS		
	11-15	6-10	0-5	11-15	6-10	0-5
	Calendar Period			Calendar Period		
	1962-66	67-71	72-78	1973-77	78-82	83-89
Sex ratio at Birth	100.6	102.1	103.2	99.8	103.4	99.2
Sex risk ratio (Neonatal) (NMRmales/NMRfemales)	1.49	1.18	1.44	1.30	1.11	1.52
Ratio of Early Neonatal (0-6 days) Deaths to all Neonatal Deaths						
Males	NA	NA	NA	.675	.832	.728
Females	NA	NA	NA	.699	.724	.653
Both Sexes	NA	NA	NA	.686	.783	.700
Ratio of Neonatal Mortality to Infant Mortality						
Males	.476	.464	.473	.488	.383	.507
Females	.361	.445	.372	.361	.379	.393
Both Sexes	.427	.456	.423	.436	.382	.454
Ratio of Infant Mortality to Under-five Mortality						
Males	.622	.595	.639	.557	.626	.678
Females	.593	.606	.586	.637	.606	.653
Both Sexes	.608	.600	.603	.597	.616	.667

NA - Data on age at death for neonatal deaths not collected in days.

sex ratios at birth, indicating that if boys were omitted, they were those that survived. In other words, the hypothesis of omission of male deaths is not well supported.

One also expects a greater concentration of deaths shortly after birth for boys than girls; for both sexes, mortality should be highest in the neonatal period and then to fall rapidly thereafter. Conventional wisdom suggests that omission of child deaths is more common if the death occurs shortly after birth (before the child has been named, baptized, or otherwise considered a full-fledged member of the

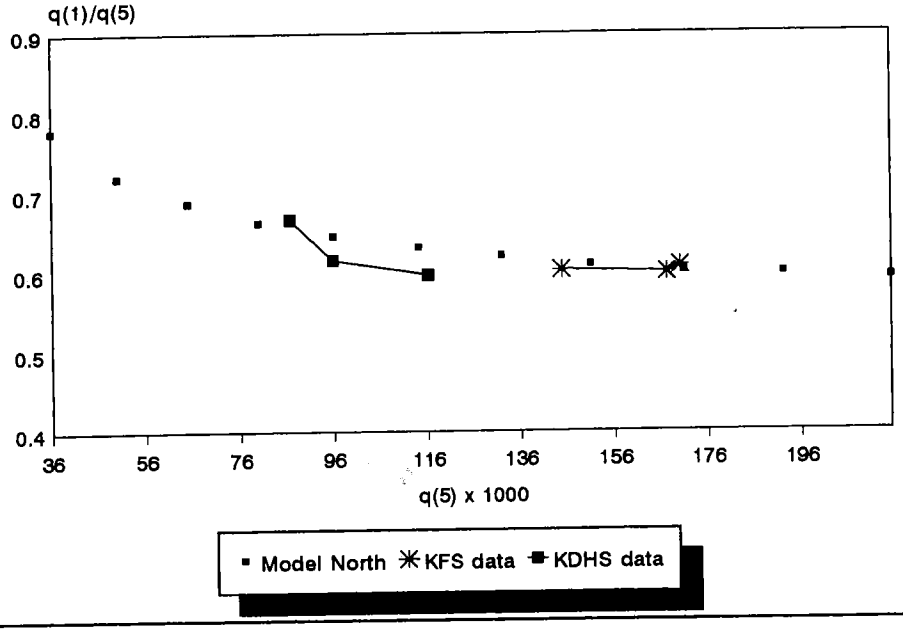
society), and that omission becomes more likely as the period of recall becomes increasingly removed from the survey date.

Noted in Figure 1.1 earlier is the substantial concentration of deaths in the first month. Given in Table 1.1 are some ratios that describe in more detail the age pattern of mortality by calendar period and by sex during the neonatal period and during the infant period. The ratios of early to all neonatal deaths roughly follows the expected pattern, whereby there exists a greater concentration of deaths for boys than girls early on. The exception to this occurs in the period 11–15 years before the KDHS, and points again to the possible omission of male deaths in that period. This is supported by the fact that while the ratio for girls does not decline with increasing time since the survey, the male ratio does. However, this evidence is not terribly compelling. The overall range of the ratios (0.65 to 0.83) is in-keeping with identically computed ratios for other KDHS surveys conducted in countries with similar levels of mortality (Sullivan et al., 1991).

Looking at the ratio of neonatal to infant mortality, no time trend or sex differential exists in the reported age pattern of infant mortality that would signal selective omission of nonsurvivors. The fraction of infant mortality occurring in the first month is remarkably stable within and across surveys, and the expected higher male ratios are observed at every period in both surveys.

Another way to examine external validity of the mortality data is to look for atypical and inconsistent patterns in the relationship between the level and the age pattern of under-five mortality. The empirical relationship between these two are embodied in model life tables. Generally speaking, these tables inform us that as mortality level goes down, the fraction of under-five mortality concentrated at younger ages (specifically, during the infant period) increases. At the bottom of Table 1.1 are the ratios of infant to under-five mortality by calendar period, sex, and for both surveys. These ratios are plotted against our calculated ${}_5q_0$ in Figure 1.2 along with the ratios calculated from the Coale-Demeny model "North" life table for values of ${}_5q_0$ between 40 and 220 per thousand. A couple of points emerge. First, the age pattern of mortality from the KDHS data is roughly consistent with that of the KFS. Both generally follow a "North" pattern. Still, while the most recent KDHS estimate (1983-89) conforms closely with the KFS estimates and model "North" pattern, the two less recent KDHS period estimates indicate low infant mortality relative to overall under-five mortality. This may again suggest some infant death omission for the KDHS periods 1973–77 and 1978–82, but not for the period 1983–89.

Figure 1.2
Ratio of Infant to Under five mortality (q(1)/q(5)) by q(5) - Coale-Demeny Model North, KFS, and KDHS.



1.3 Results

Age-specific estimates of childhood mortality are given in Table 1.2 and summarized graphically in Figure 1.3. Several points emerge from this presentation. First, within both surveys a fairly pronounced mortality decline is suggested at all ages except the neonatal period. Second, a comparison of the mortality level estimates from the most recent periods preceding each survey indicates a substantial mortality decline over the last decade—$_5q_0$ fell 40 percent, $_4q_1$ fell 51 percent, $_2q_0$ fell 38 percent, $_1q_0$ fell 34 percent, and neonatal mortality fell 29 percent. Third, a marked discrepancy exists between the estimates of the KFS and KDHS for the "common" calendar period centered around 1975. That the discrepancy is largest for postneonatal and toddler mortality (between ages 1 month and 2 years) is rather inconsistent with the hypothesis that increasing omission with time since the survey has caused the lower KDHS rates for the period centered around 1975. These particular findings, while raising questions about the KDHS estimates for the less recent periods, speaks little to the validity of the KDHS estimates for the most recent period, 1983–89. The most plausible explanation of these results (in light of the preceding data quality analysis) is that there has been a substantial drop in mortality of magnitude represented by the change between level estimates for the calendar periods immediately preceding the KFS and KDHS, and that the less recent estimates from the KDHS are biased downward by underreporting of infant deaths.

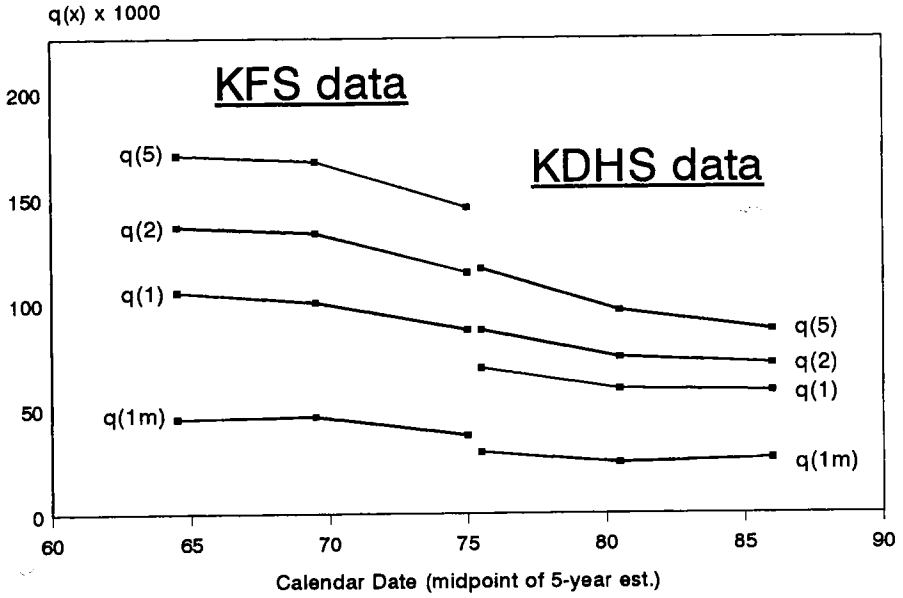
Table 1.2 Period life table estimates

Various childhood mortality parameters, by five-year calendar period before the surveys, 1977/78 KFS and 1988/89 KDHS.

		1977/78 KFS			1988/89 KDHS				
	Ages at exposure to risk (completed	Years before survey			Years before survey			Discrepancy between rate of KFS (0-5) & KDHS (11-15)	
Age specific rates (/1000)	months)	11-15 1962-66	6-10 67-71	0-5 72-78	11-15 1973-77	6-10 78-82	0-5 83-89	abs.	%
Neonatal Mortality	0	44.9	45.7	36.9	29.3	22.5	26.3	7.6	20.6
Postneonatal Mortality	1-11	63.1	57.3	52.4	41.1	37.4	32.4	11.3	21.6
Toddler Mortality ($_1q_1$)	12-23	34.9	36.6	29.0	18.8	15.4	14.0	10.2	35.2
$_3q_2$	24-59	38.9	39.3	34.9	32.1	24.1	16.9	2.8	8.2
Summary Measures (/1000)									
Infant Mortality ($_1q_0$)	0-11	105.2	100.3	87.3	69.3	59.1	58.0	18.0	20.6
Under-two Mortality ($_2q_0$)	0-23	136.4	133.2	113.8	86.8	73.6	71.1	27.0	23.7
Child Mortality ($_4q_1$)	12-59	72.5	74.4	63.2	50.3	39.2	30.7	12.9	20.4
Under-five Mortality ($_5q_0$)	0-59	170.1	167.2	144.8	116.1	95.9	86.8	28.7	19.8

Note: Discrepancy: Absolute (abs.) difference = KFS (0-5) rate - KDHS (11-15) rate
: % difference = (KFS (0-4) rate - KDS (11-15) rate) / KFS (0-4) rate

Figure 1.3
Trend in the level and age-pattern of under-five mortality, using data of the 1977/78 KFS and 1988/89 KDHS



If this reading of the data is correct, a decline in under-five mortality between 1972–78 and 1983–89 from 145 to 87 per thousand is, by any measure, a remarkable accomplishment. This corresponds to an annual drop of 4.7 percent, compared to a 2.0 percent fall for all countries of sub-Saharan Africa, 3.1 for all countries of Asia, 3.6 percent for all countries of the Middle East, and 4.0 percent for all countries of Latin America covered in a recent analysis of global trends in childhood mortality over the 1980–85 period (Hill and Pebley, 1989). Given the significant improvements in women's education, use of child health services, and fertility levels, these results, while striking, are by no means implausible. The results of this analysis indicate that increasing and concerted effort needs to be focused on the more intractable problems related to survival during the first 2 years of life and, in particular, during the infant period.

2 DETERMINANTS OF CHILDHOOD MORTALITY IN KENYA

2.1 Background and Rationale

Several previous studies have attempted to link early childhood mortality with socioeconomic factors in Kenya. These fall under two broad headings: (1) macrolevel (ecological) studies that relate regional estimates of mortality with other infrastructural and social parameters of the regions, and (2) microlevel studies that use either the women or the child as the unit of analysis and attempt to link individual-level characteristics with survival of the "index child" or proportions of women's ever-born children who have died. Parental education, especially that of the mother, has been the central variable in most of these investigations.

Kibet (1981), using the macrolevel approach with 1979 census data, found a strong association between district levels of maternal education and district probabilities of mortality before the second birthday, ${}_2q_0$, estimated via "Brass-type" indirect methods. While a very consistent pattern of higher early childhood mortality among uneducated mothers was shown *within* districts, other factors appear to be at play in district mortality differentials. This conclusion is based on the observation that uneducated women in low mortality districts (e.g., Nyeri) experience lower early childhood mortality than do educated women from high mortality districts (South Nyanza). Mosley's analysis (1985) of provincial differentials in ${}_2q_0$ convincingly shows that most of the Kenya's regional variation in child survival can be explained by differences in income and education.

Ewbank et al. (1986) reported on unpublished microlevel analyses of Khata (1983) and Mwaniki (1983). The results of the Khata study, based on data from the 1977 National Demographic Survey (NDS1), are noteworthy in that they appear to refute the results of the Kibet study; within 4 of 5 Kenyan ecological zones, no effect of education on childhood mortality was observed. Mwaniki, analyzing data from the 1977/78 Kenya Fertility Survey (KFS) and the 1977 National Demographic Survey, found that mother's and father's education has nearly the same effect on childhood mortality. Further, his results indicate that for both parents, 1 to 3 years of education is not sufficient to affect mortality significantly.

A study by Anker and Knowles (1980), using data from the 1974 Household Survey, supports the view that maternal education affects childhood mortality independent of its clear link with household economic condition.

Very little has been done to examine the pathways through which socioeconomic factors may operate to diminish mortality among young children in Kenya. A study of treatment patterns among the Akamba children shows that, while educated mothers in this area of Kenya are no more likely than uneducated mothers to seek modern medical assistance in the case of acute diarrhea or measles, educated mothers more commonly avoid the traditional withholding of water and milk from children with measles (Maina-Ahlberg, 1979). These results lead to the hypothesis that at least part of the education effect is through acceptance of new ideas that may reduce case-fatality from measles.[6]

Other factors reported to be important correlates of early childhood mortality include the pattern of family formation (Mott, 1982), fathers's occupation, ethnicity and religious affiliation (Ewbank et al., 1986), and the availability of sewage disposal (in the coastal and middle dry zones) (Khat, 1983).

While all these studies point to the importance of social and economic factors as covariates of childhood mortality in Kenya, no study has attempted to evaluate how such factors may operate to condition behaviors (e.g., the pattern of family formation, use of health services, etc.) that enhance survival prospects, nor whether such effects might vary with age of the child. Two recent developments enhance the prospects for a more detailed and meaningful assessment of socioeconomic determinants of early childhood death in Kenya. First, conceptualization of the process leading to illness and death is now better understood and formalized (as will be discussed in subsequent sections).

Second, previous studies relied on data that were severely limited in terms of both the number and types of covariate data that were collected, as well the structure of the survival data. For instance, the KFS data included little information on household "exposure" and economic indicators and no data on child health services, while the 1979 Census data did not include birth history data that might allow individual-level (birth-based) analysis of mortality risk at specific ages of early childhood.

The 1989 KDHS represents the first national-level survey (or census) in which birth history data can be linked at the individual level with an extensive array of variables associated with child health and survival. This provides a unique opportunity to delineate more completely a profile of high-risk households, mothers, and children. Furthermore, these data allow investigation of the nature of the

[6] Although it is difficult to estimate precisely the demographic/epidemiologic importance of measles in Kenya today, it has in the past been a severe public health problem in association with serious respiratory illness and may still be associated with 20 percent of deaths between 6 and 48 months of age (Ewbank et al., 1986).

interrelationships among socioeconomic factors, proximate determinants, and age-specific childhood mortality.

2.2 Conceptual Framework

Since the seminal work of Mosley and Chen (1984), conceptual models of childhood mortality determinants have developed in various directions depending on the substantive interests of the investigator. All, however, conserve the central paradigm of the original work; there exists a certain finite number of variables (proximate determinants) through which all socioeconomic or "background" factors must operate. Mosley and Chen posited five such variables (or sets of variables) that are causally and temporally proximate to health endpoints:

(1) Maternal factors (e.g., pattern of family formation, biodemographic variables, or birth order, maternal age, and the pace of childbearing)

(2) Environmental contamination, (e.g., exposure to disease)

(3) Personal illness control—may operate to diminish morbidity through behaviors that limit exposure and susceptibility (i.e., preventive measures) or to diminish case-fatality through curative measures

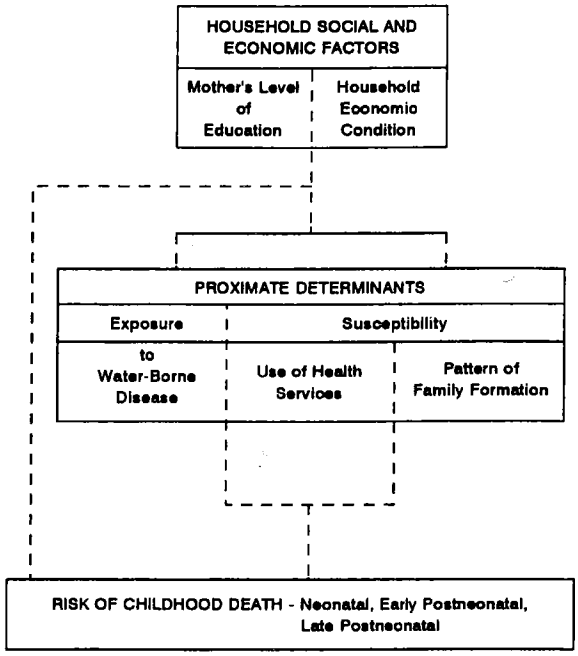
(4) Nutritional inputs

(5) Accidental injury

The particular conceptual framework that we propose (Figure 2.1) is the result of careful examination of (a) previous work (in Kenya and elsewhere) on the determinants of childhood mortality, (b) the current state of knowledge regarding measurement and statistical problems encountered in evaluating causal models with cross-sectional and retrospective data, and (c) the structure and limitations of the KDHS data. Due to the latter, the study cannot accommodate the last two variables in the Mosley and Chen model (nutritional inputs and accidental injury).

While the broad aim of the study is to delineate age-specific risk factors for early childhood mortality, a particular focus of the study was in seeking to identify whether there exists within the Kenyan context, changes in the effect of socioeconomic factors (maternal education and household economic condition) with increasing age of the child. We allude here to the oft-held notion suggested in previous studies including the comparative work of Rutstein (1984), that constraints on decisionmaking imposed by (lack of) personal assets grow more important as the child gets older. Furthermore, it is key to the formulation of relevant health/social policy to determine the mechanism by which such personal assets may translate effectively to enhanced child survival at the individual level. In this vein and to the extent

Figure 2.1
Conceptual Framework for the Analysis of Determinants of Early Childhood Mortality in Kenya. Dashed Lines Represent Effects Unmediated by More Proximately Situated Factors.



feasible with the KDHS data, we look at the three sets of proximate determinants (shown in Figure 1) from the standpoint of their role in mediating or mitigating the worst consequences of poverty, namely, childhood death.

The data, the approach to variable construction, and the statistical methods used to evaluate the conceptual framework are described in the following section.

2.3 Data and Methods

2.3.1 Survival Data

This investigation examines mortality risk through the first 2 years of life. Three age-segment models are evaluated: the neonatal period (first 30 days of life), the early postneonatal period (1–6 months), and the late postneonatal period (7–23 months). There are both substantive and methodological reasons for this approach. Substantively, it is argued that neonatal risk is influenced heavily by pregnancy outcome—low birth weight, obstetrical complications, etc.—as well as being the period during which tetanus neonatorum takes its toll. The late postneonatal period (7–23 months) is the period during which the vast majority of Kenyan mothers will wean their children, and thus a period during which

nutritional inputs and infectious disease associated with increasing exposure to household and community pathogens and decreasing maternal protection (i.e., increased susceptibility, ceteris paribus) is likely to be reflected in differential survival. Interpretation of results is more directed within a framework of knowledge concerning accepted age patterns to potentially fatal morbidities.

Methodologically, it makes sense to roughly apportion deaths equally across age segments, which is what this breakdown accomplishes (i.e., about a third of all under-two deaths occur during each segment with adequate numbers in each). Perhaps more importantly, this age breakdown avoids biases in estimation associated with covariate-related differences in the tendency to heap reported age at death at 12 months. We look only at mortality under age 2 simply because the number of deaths in the 2- to 5-year period is prohibitively small, when considering only births in the last 5 years.[7] Under-two deaths account for 84 percent of under-five deaths recorded during the 1983 to survey-date period. Of all births under observation in this study, 222 died in the neonatal period, 163 died in the 1–6 month period, and 217 died in the 7–23 month period.

2.3.2 Explanatory Variables

The construction of the explanatory variables was guided by two principal considerations: existing theory and the structure and distribution of the data. For instance, while it is of theoretical and practical interest to examine the effects of university education of mothers on childhood mortality risk, the number of such cases precluded this level of analysis. Sex of the child and whether the child was a twin are considered control variables and are included in all baseline models.[8]

Household socioeconomic factors (1) *Education of mother.* A tremendous amount of literature supports the idea that educational attainment of the mother is positively related to child survival chances. These data were collected by asking the woman about the highest level of education she ever attended: none, primary, secondary, or university. Secondary and university levels are collapsed and form the analytical reference category. (2) *Economic condition of the household.* No data on actual household income was obtained in the KDHS. As a proxy for household wealth, data on household possessions (radio and television), vehicles owned by the household (motorcycle, car, tractor), and material used in

[7] Only births occurring since the cut-off date for collection of health service use information (January 1983) are included in the analysis, excluding those born in the month of survey.

[8] This was done simply to protect against possible imbalance in the sample regarding these high-risk factors across our study variables. This, under the reasonable assumption that sex of the child and twinning play no causal roles in our hypothetical framework.

the construction of the dwelling's floor (dirt or not) were used to construct an index that can take the values zero through six.[9] The index was then collapsed into three categories: 0,1 = LOW; 2,3 = MID; and 4-6 = HIGH, which form the reference group.

Proximate Determinants the following variables form the tier of proximate determinants:
(1) *Exposure to disease (environmental contamination).* Heightened exposure to water-borne pathogens is thought to increase the risk of potentially fatal, dehydrating gastrointestinal disease. We proxy such exposure with two variables: nonpiped (open) drinking water source and primitive excreta disposal reported in the household information section of the questionnaire. However, such measures can only capture the level of actual exposure to pathogenic agents with an *unknown* degree of precision and reliability. Also bear in mind that these measures can be causally related to economic condition of the household but are related to mother's education only in their relationship (observed or unobserved, at the community or household level) to economic development. Actual behaviors affected by maternal education that lead to variation in exposure to her children are unobserved.

The exposure variables are used only in the models looking at mortality risk after the first month of life.

(2) Susceptibility. *(a) Family Formation Pattern.* Birth order, maternal age at birth, and preceding birth interval length are all known to be associated with mortality risk. These variables are constructed from the birth history information. First births and births of high order; young age at birth (< 20 years) and advanced age at birth (35+ years); and short birth interval (< 18 months) and long intervals (48+ months) are posited a priori as high-risk factors.
(b) Use of child health services. The scope of data collection on use of health services potentially related to child survival was limited in the DHS. Information on service use that can be applied to both living and dead children is restricted to reported antenatal care, reported delivery services, and reported use of tetanus toxoid. In the analysis of neonatal mortality risk, we look at: (1) whether the mother reported to have received at least one dose of tetanus (TET)—"When you were pregnant with (name of child), did you receive any injection to prevent the baby from getting tetanus?" and (2) at least one reported antenatal visit to a medically trained person (ANC)—"When you were pregnant with (name of child), did you see

[9] One point is given for possession of a radio but no television, and two points for a television (irrespective of radio possession). One point is given for a motorcycle but no car or tractor, and two points for a car or tractor (irrespective of motorcycle possession). Two points are given for a nondirt floor.

anyone for advice on this pregnancy?... Who?" For (2), a response of doctor or trained nurse/midwife satisfied this definition.

In the analysis of postneonatal mortality risk, a health service use index (HSUI) was produced using (2) from above and (3) whether the mother reported to have received medically trained assistance at delivery of the index child—"Who assisted you with the delivery of (name of child)?" The definition of "medically trained" for (3) was the same as for (2) above.[10] Three levels of the index are defined: neither (2) nor (3) was true = LOW USE; either (2) or (3) was true = MEDIUM USE; both (2) and (3) were true = HIGH USE. While this index does not measure use of any particular health service that would be expected to influence significantly the risk of dying in the 1-23 month age segment, it almost certainly reflects a mother's propensity to use modern health services. Nevertheless, while interpretation of results for the neonatal model may be specific (e.g., tetanus toxoid's effect on diminution of neonatal risk through protection against tetanus), interpretation of the results for the early and late postneonatal models should be interpreted only with a great deal of caution regarding possible causal effects.

2.3.3 Analytical Methods

Basic statistical model. The structure of data indicated use of a statistical model that would allow use of censored observations. Without such procedures, analysis of mortality up to the second birthday would have required exclusion of about 40 percent of the births in the last 5 years, so as to avoid biases in estimation. Use of hazards regression methods permit inclusion of all births for the analysis of agesegments extending beyond the neonatal period.[11]

For the neonatal period, we use the common logistic model of the form:

$$\ln (q/1\text{-}q) = b_0 + B_iX_i,$$

where q_i is the probability of neonatal mortality for the ith individual given the array of independent measures, X_i; and where b_0 is the baseline constant and b_i represents a series of unknown coefficients estimated via the maximum likelihood procedure (Hosmer and Lemeshow, 1989) using the SPSS-PC program LOGIST.

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[10] The variable, use of delivery services, was not used in the neonatal model because potentially serious bias may be introduced related to reverse causality (i.e., identification of problem pregnancies may well lead to greater use of modern delivery services).

[11] Neonatal risk analysis does not involve censoring because only births born at least 1 month before the survey date were included.

Dummy variable construction is employed in the case of all variables. The estimated coefficient, b_i, exponentiated, is interpreted as the odds of mortality (q/1-q) for individuals with certain characteristics relative to the odds of mortality in a reference baseline group of individuals (relative odds or odds ratio). In the present case where q is very small, the odds ratio translates effectively to a relative risk.

The Cox Hazards regression is used for analysis of early (1–6 months) and late (7–23 month) postneonatal risk. The form of the model is:

$$\ln [h(t)_i/h_0(t)] = b_iX_i,$$

where $h(t)_i$ is the predicted hazard or mortality risk at age(month) t for an individual with an array of covariate values, X_i; where $h_0(t)$ is the baseline and arbitrary hazard at age t, and b represents the regression coefficients estimated by the partial-likelihood method of Cox (1972). All covariates are fixed within the 1–6 month age period and within the 7–23 month period (i.e., no time-varying covariates). The estimated coefficients, when exponentiated, are interpreted in the same way as in the logistic model (i.e., as a relative risk or higher risk category relative to low-risk reference category of individuals). However, this estimate of relative risk should be viewed (as it is estimated) as a scalar that raises or lowers (depending on the sign of b) the underlying, baseline hazard uniformly (or proportionately) within each of the early and late postneonatal age segments.[12] The STATA procedure COX was used to estimate the hazards models.

Model estimation and inference. First, the gross effect of each of the study variables on mortality risk is estimated and presented to allow the reader to assess uncontrolled effects. These estimates are produced by including only single variables (with n levels) in the logistic and hazards regressions; the exception being for the family formation variables (maternal age at birth, birth order, and birth interval) that were entered together as a block.

Net effects are estimated in-line with the causal/temporal ordering of factors implied in the analytical framework (Figure 2.1) and represented in this generalized sequence of equations:

Equation 1 $\quad \text{logit } q_1 = b_0 + b_1X_1$

[12] While the proportionality assumption is never fully satisfied with real data, sharp departures or changes in the direction of covariate effects with increasing age will tend to mask or attenuate true effects. The scope for this type of mispecification bias is quite narrow within the 1–6 month age-segment model. Checks on the proportionality of risk within the 7–23 month age-segment model were accomplished by inspecting covariate-specific survival curves and plots of ln(-ln(s(t))) versus ln(t), where s(t) equals the survival function at age t. In no case were age-varying effects pronounced and monotonic so as to alter substantially the conclusions drawn from the results reported here.

Equation 2 $\text{logit } q_{1,2} = b_0 + b_1X_1 + b_2X_2$

Equation 3 $\text{logit } q_{1,2,3} = b_0 + b_1X_1 + b_2X_2 + b_3X_3$

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Equation k $\text{logit } q_{1,2,3,k} = b_0 + b_1X_1 + b_2X_2 + b_3X_3 + \ldots b_kX_k$

where equation 1 includes only household socioeconomic factors; equations 2 and 3 add proximate determinants other than health service use (e.g., exposure variables, family formation variables), and equation k adds the variable(s) related to health service use. The importance of individual elements of the model is assessed by a change in the model log-likelihood associated with the addition of that variable(s):

$$-2\,(MLL_k - MLL_{k-1}) \qquad X^2 \text{ (No. degrees of freedom)}$$

For instance, the change in the log-likelihood upon adding the health service use index (2 dummy variables for 3 levels of use), when compared against a X^2 distribution (with 2 degrees of freedom) indicates the probability of observing the parameter estimates under the null hypothesis that $b = 0$.

Effect mediation is assessed by observing changes in the estimated effect of household variables on mortality risk upon addition into the series of equations of proximate variables. For example, with addition of proximate variables related to health service use, the coefficient for maternal education drops towards zero; it is inferred that part of the effect of maternal education on mortality risk operates through better health service use.

2.4 Results

2.4.1 Gross Effects

Table 2.1 gives estimated gross effects and the percentage of births falling into each of the covariate levels (excluding the reference group). Bear in mind that the gross effects for the proximate variables, especially those that are clearly "choice" variables (i.e., preconditioned by socioeconomic conditions, e.g., use of health services), are likely to be biased upward in this first crude look at relationships.

The expected effects of gender and twin status were observed; males and twins were much more likely to die in the neonatal period than female and singleton births. These effects wane with increasing age of the child.

Table 2.1 Gross effects of model variables on neonatal
Early postneonatal (1-6 mos) and late postneonatal (7-23 mos.) mortality risk. Effects expressed as relative risks (RR). KDHS, 1983-89

	Neonatal RR	1-6 months RR	7-23 months RR	Percentage of Births[1]
Baseline variables				
Twin (Yes)	5.79 ***	3.49 ***	1.30	2.9
Sex (Male)	1.57 ***	0.88	0.96	50.0
Socioeconomic factors				
Maternal education				
- None	1.87 **	2.11 **	2.38 ***	25.5
- Primary	1.28	1.64 *	2.00 **	55.8
Economic Status				
- Low	2.13 **	2.32 *	2.52 **	23.5
- Middle	1.31	2.04	1.44	68.5
Exposure factors				
Drinking Water Source				
- not piped	N/A	1.02	1.19	64.1
Toilet facilities				
- no facilities	N/A	1.72 *	3.17 ***	15.2
- pit latrine, bucket	N/A	0.91	1.71	75.4
Family formation factors				
First Birth Order	1.99 ***	0.98	1.08	18.3
Six+ Birth Order	1.39 *	1.40	0.72	30.2
Mother < 20 yrs old	1.40	2.08 ***	2.21 ***	16.5
Mother > 34 yrs old	1.20	0.83	1.19	13.3
B. Interval < 18 mos.	2.64 ***	1.87 **	1.48	9.0
B. Interval > 48 mos.	1.50 *	0.70	0.50 **	12.4
Health services use				
no tetanus toxoid	1.89 ***			
no antenatal care	1.34 *			
Health Service Use Index: (HSUI)				
- Low		1.83 **	1.59 **	13.2
- Mid		1.07	1.49 **	43.6

[1] 6,913 total births, 6,631 survivors of neonatal period, 5,784 survivors of early postneonatal period.
* variable significant at $p = .10$; ** at $p = .05$; *** at $p = .01$

Maternal education and household economic status are strong correlates of mortality risk during all three age segments, with a slightly greater effect associated with increasing age. Children of uneducated women experience nearly twice the risk of neonatal death and over twice the risk of postneonatal mortality as children of women who attended secondary school. Children born into the poorest homes had twice the risk of neonatal mortality and about 2.5 times the risk of postneonatal mortality, as did children from the most advantaged households.

Of the exposure variables, type of toilet facility is more strongly associated with postneonatal mortality risk than the source of drinking water. Surprisingly, the drinking water source has virtually no effect on risk; type of toilet facility has a marked effect, especially during the weaning period (7–23 months).

In looking at variables representing the pattern of family formation, several points emerge. First, the only pronounced birth order effects occur in the neonatal period, during which time first births experience twice the mortality risk as children of birth orders 2–5. Second, older maternal age at birth appears weakly or unrelated to under-two mortality risk, while young age at birth (< 20 years) significantly elevates risk after the neonatal period. Short birth intervals increase the chance of dying by 160 percent during the neonatal period, 87 percent during the early postneonatal period, and by 48 percent during the late postneonatal period.

Having had at least one dose of tetanus toxoid during the index pregnancy is strongly associated with decreased neonatal risk, while the effect of prenatal advice is less pronounced. After the neonatal period, nonuse of health services (as proxied by HSUI) shows moderate risk-elevating effects. Recall, however, that insofar as reported, use of these services is conditioned by personal assets, the estimated gross effects give little or no indication of the causal role played by health services use in determining mortality risk.

2.4.2 The Neonatal Period

Nearly half of infant deaths and about one-third of all under-five childhood deaths in Kenya occur during the neonatal period. Table 2.2 gives the estimated relative risks of neonatal death related to the model variables described earlier. The effects of socioeconomic status of the household during this period are weaker than at later ages but are still significant. Children of the poorest households are 77 percent more likely to die than the children from most advantaged households, and children of uneducated mothers are 47 percent more likely to die than children of well-educated mothers during the first month of life. These effects are slightly amplified upon addition of the family formation variables, then diminished upon addition of health service use variables. (These results, while by no means compelling, suggest that higher socioeconomic level is associated with both risk-diminishing [better use of health services) and risk-elevating behaviors (shorter birth intervals].) More importantly, the results indicate that much of the effect of maternal education and economic condition on neonatal risk remains unexplained.

First birth order, young maternal age at birth, and especially short preceding birth interval significantly increase risk of neonatal death. Estimated effects indicate 101 percent higher risk for first births, irrespective of maternal age at birth; 58 percent higher risk for children born to women under 20 years of age; and nearly 3 times higher risk for children with preceding intervals less than 18 months, compared to the reference group risk. The short interval effect is, however, likely to be biased slightly upward by a disproportionate number of premature births in the short interval category (Miller et al., 1991). There exists a small effect of very long intervals on neonatal risk, but this may be explained by the fact that long intervals often represent cases of repeated pregnancy loss, which, at once, biases measurement of pregnancy spacing (i.e., interbirth interval length overestimates interpregnancy interval length) and is related to subsequent poor survival chances for the eventual live birth.

Table 2.2 Relative risk of neonatal mortality Associated with model variables
Results of logistic regression. KDHS 1983-89. (N = 6913)

Variable	Equation 1	Equation 2	Equation 3
Socioeconomic factors			
Maternal Education			
None	1.47 *	1.58 **	1.48 *
Primary	1.05	1.04	1.01
Economic Index			
Low	1.77 *	1.82 *	1.81 *
Mid	1.26	1.24	1.26
Family formation factors			
Birth Order			
First		2.01 ***	2.00 ***
Six +		1.15	1.16
Maternal Age at Birth			
Less than 20 yrs.		1.58 *	1.58 *
35+ yrs.		1.05	1.04
Birth Interval Length			
Less than 18 mos.		2.82 ***	2.81 ***
48+ mos.		1.48 *	1.48 *
Use of health services			
No Tetanus Toxoid			1.75 **
No Antenatal Care			1.10
Degrees of Freedom	4	6	2
p (change in model X)	0.010	0.000	0.183

Note: N = 6913
* two-tailed significance at p = .10;
** p = .05;
*** p = .01.

Among the health services use variables, only reported nonuse of tetanus toxoid has an important effect on neonatal mortality; children of women who had not received a tetanus injection reported 75 percent higher mortality after controlling for socioeconomic condition of the household and the pattern of family formation. Part of this may indicate a genuine protective effect of tetanus toxoid against death due to tetanus neonatorum, but also probably includes the effect of unmeasured factors that condition the choice to either use or report use of the service. The small gross effect of antenatal assistance (Table 2.1) is nearly erased with inclusion in the regression of the other antecedent variables.

2.4.3 The Early Postneonatal Period (EPN, 1–6 months)

Recall that modelling procedures for analysis of risk after the first month are different from neonatal risk analysis in three ways: first, hazards regression is used; second, variables that proxy household environmental contamination are included as an additional step in the estimation process; and third, the indicator of health ser-vices use is of a different construction.

Table 2.3 Relative risk of early postneonatal mortality associated with model variables
Results of hazards regression. KDHS 1983-89. (N = 6631)

Variable	Equation 1	Equation 2	Equation 3	Equation 4
Socioeconomic factors				
Maternal Education				
None	1.89 **	1.79 *	1.72	1.63
Primary	1.48	1.57	1.42	1.38
Economic Index				
Low	1.84 *	2.14 *	2.13 *	2.12 *
Mid	1.81 *	2.14 *	2.15 *	2.06 *
Exposure factors				
Source of Drinking				
Water is Nonpiped		0.93	0.94	0.94
Toilet Facilities				
None		1.06	1.00	0.97
Pit Latrine, Bucket		0.62	0.59	0.58
Family formation factors				
Birth Order				
First			1.27	1.26
Six +			1.08	1.08
Maternal Age at Birth				
Less than 20 yrs.			2.05 ***	2.08 ***
35+ yrs.			0.78	0.75
Birth Interval Length				
Less than 18 mos.			1.97 ***	1.96 ***
48+ mos.			0.68	0.68
Use of health services				
LOW score on HSUI				1.61 **
MID score on HSUI				0.99
Degrees of Freedom	4	3	6	2
p (change in model X)	0.069	0.062	0.006	0.082

Note: N = 6631.
* two-tailed significance at p = .10;
** p = .05;
*** p = .01.

Table 2.3 shows that maternal education and household economics exert a more powerful influence on EPN mortality than during the neonatal period. While the expected "stepped" increase in risk with decreasing education level was observed (48 percent excess risk for primary school, 89 percent for no school), note that only among the most advantaged group of children is the effect of economic condition observed (i.e., at about half of LOW/MID risk). The education effects fall by about 30 percent for no education and about 20 percent for primary education categories, upon addition of proximate variables into the model, indicating their mediating role.

The exposure variables, after controlling for economic condition of the household, show no significant effects.

The importance of the family formation variables, while not quite as striking as during the neonatal period, remains remarkable. Also, during the neonatal period, first birth order has a dominant effect on risk relative to the maternal age effect; during the subsequent 6 months, maternal age becomes more important in determining risk. One interpretation of this finding is that the first birth order effect specifically affects pregnancy outcome and obstetrical complications, while young age at birth affects outcome largely through social forces—teenage women may be ill-prepared to deal with children.

The short birth interval effect is still very pronounced during the EPN period—children in this group experience nearly twice the mortality risk of children in the 18–47 month interval group. The fact that the coefficient for long intervals (48+ months) reverses direction gives support for the notion that the long interval effect observed during the neonatal period is indeed an artifact of how childspacing is measured in this study.

The children of nonusers of health services (i.e., LOW) experience 60 percent higher EPN mortality risk than children of service users (MID and the reference HIGH group can be collapsed at relative risk 1.00).

2.4.4 The Late Postneonatal Period (LPN, 7–23 months)

The magnitude of effects of household socioeconomic status on LPN mortality risk (Table 2.4) is in the same range as effects observed for the EPN period—roughly twice the risk of death is associated with no maternal education and lowest economic level compared to the respective reference group risk. An interesting pattern of effect mediation is observed regarding changes in the education effects upon addition of proximate variables. First, addition of the exposure variables causes the education effect to move toward the null value of 1.00; secondly, addition of the family formation variables pushes the education effects sharply away from the null value; and thirdly, addition of the health service use index reduces the education effect again. This pattern of effects indicates that maternal education is associated with both risk-elevating and risk-diminishing factors. While more education leads to greater use of health services and better exposure conditions (leading to lower risk), the children of more educated women tend to fall into high-risk categories for family formation (leading to higher risk, ceteris paribus). Part of the latter effect may be causal (shorter birth intervals related to shorter duration of breastfeeding) but is probably also partially related to methodological artifact. Because this is a birth-based analysis, and because fertility is lower among more educated, women a greater fraction of all births to educated women fall into the first birth category.

Looking at the effects of the exposure variables (after controlling for antecedent socioeconomic conditions), source of water has little influence in delineating mortality risk during the LPN period. Lack of any household toilet facilities, on the other hand, significantly elevates—by some 90 percent—LPN risk.

Table 2.4 Relative risk of late postneonatal mortality associated with model variables

Results of hazards regression. KDHS 1983-89. (N = 5784)

Variable	Equation 1	Equation 2	Equation 3	Equation 4
Socioeconomic factors				
Maternal Education				
None	1.83 **	1.61	1.94 **	1.83 **
Primary	1.65 *	1.61	1.57	1.52
Economic Index				
Low	1.96 *	1.84	1.86	1.79
Mid	1.26	1.21	1.24	1.22
Exposure factors				
Source of Drinking				
Water is Non-piped		0.85	0.92	0.91
Toilet Facilities				
None		1.89 *	1.63	1.57
Pit Latrine, Bucket		1.14	1.06	1.04
Family formation factors				
Birth Order				
First			1.24	1.24
Six +			0.62 **	0.62 **
Maternal Age at Birth				
Less than 20 yrs.			2.04 ***	2.05 ***
35+ yrs.			1.11	1.02
Birth Interval Length				
Less than 18 mos.			1.55 *	1.57 *
48+ mos.			0.51 **	0.52 **
Use of health services				
LOW score on HSUI				1.27
MID score on HSUI				1.27
Degrees of Freedom	4	3	6	2
p (change in model X)	0.026	0.083	0.000	0.494

* two-tailed significance at $p = .10$;
** $p = .05$;
*** $p = .01$.

The pattern of effects across birth order and maternal age at birth remain roughly the same during the LPN period as was observed for the EPN period—a large young maternal age effect and much smaller first birth order effect. Noteworthy is the continued diminution of the short birth interval effect with increasing age of the child. Also, the persistence of a "protective" effect of long intervals (48+ months) during this period further supports the idea that the effect of birth interval length on survival is cumulative ("dose-sensitive") and that the observed "harmful" effect of long birth intervals during the neonatal period is probably spurious.

Estimates of the effect of health services use (once separated from its link with socioeconomic condition and pattern of family formation) on LPN risk are small and insignificant.

3 SUMMARY AND CONCLUSIONS

In the first part of this study, it was concluded that rates of early childhood mortality have fallen sharply in Kenya over the past decade. In the second half of the study, using KDHS for a recent time period (1983–89), multivariate analysis has identified some factors related to under-two mortality risk, factors that we feel are key to the development of appropriate strategies aiming to sustain the improvement in child survival over the next decade and into next century. The results signal a continued need to alleviate socioeconomic underdevelopment in Kenya. Education of women should remain a cornerstone of Kenya's rural development efforts; it is among a population of knowledgeable and receptive women that organized efforts to improve the health of children will best be mobilized and accepted.

Economic factors inevitably play an important role in influencing human behavior; the results of this study underscore the need to narrow economic differentials, thereby removing a major constraint with which Kenyan families now wrestle in their efforts to maintain child well-being. Economic status of families, partially conditions the choice to construct and maintain adequate excreta disposal facilities, a factor shown to be related to survival of young children (7–23 months of age).

Rapid childbearing will continue to be an impediment to improving child health and survival in Kenya unless further steps are taken to insure both the information and the means to create and satisfy demand for contraception. A common feature of modernization is the diminution in the length of breastfeeding, a natural means of spacing pregnancies. This study suggests that, while maternal education is by and large associated with better survival, the pattern of family formation adopted by more educated women (specifically, shorter birth intervals) tends to partially offset this advantage.

The study also identifies teenage childbearing (controlled for birth order) and first birth order (controlled for young age at birth) as important determinants of child survival. First-time mothers represent a group whose biological as well as social apparatus may require some support. This means that health and social programs should target these women for special intervention before the birth of the child, a child who is otherwise at significantly increased risk of death in the neonatal period. Whether one considers teenage pregnancy a problem is often a question of the particular setting in which it occurs. In much of rural Kenya, marriage and childbearing before the age of 20 may not be considered socially maladaptive. On the other hand, children of young mothers in Kenya experience over twice the risk of postneonatal mortality (1–23 months) as other children. Since these effects are estimated independent of

socioeconomic effects (and birth order and interval effects), one may conclude that either (1) the inexperience inherent at young age regarding child care as well as the social stigma (in some settings) directed toward very young mothers work against the health and survival of their children, or (2) these women are self-selected and possess characteristics (unobserved in this study) that at once predispose them to early childbearing and less healthful lifestyles. In either case, effective family planning programs should include an effective component specifically designed to address the issues of teenage sexuality and contraception.

This study attempted to evaluate use of health services as an intermediate factor linking socioeconomic condition with child survival. While no clear conclusions can be drawn from the results, two important points emerge from the analysis. First, greatly elevated risk of neonatal death is associated with reported nonuse of tetanus toxoid during the "index" pregnancy. Second, a small part of the effect of maternal education during all three age segments being examined here can be explained by use of health services. Perhaps more importantly, the bulk of the effects of the socioeconomic variables remains unexplained by our study. A potentially fruitful area of research, we believe, would involve looking at patterns of nutrition during weaning across socioeconomic levels. This, of course, requires the type of data unavailable from large surveys of the KDHS type.

Other areas of study likely to bear useful results include establishing the cause-of-death structure in different parts of the country, including a determination of whether malaria and AIDS are already important contributors to childhood mortality, and where the current geographic foci of these diseases are to be found in Kenya. Such information will undoubtedly prove useful in terms of health service resource allocation as well as in the determination of the types of messages that need to be formulated for and directed toward mothers and fathers in different parts of the country. Research examining the role of poor pregnancy outcome and low birth weight is necessary in this country where at least 80 percent of mothers live and work in agricultural areas; where fertility, while declining, is still very high; and where the bulk of childhood mortality occurs during the first year, when frailty at birth is expected to be a key causal factor leading to premature death.

Unfortunately, it seems likely that the resurgence of malaria and an insurgence of HIV-1 infection into Kenya may have the effect of reversing, or at least significantly slowing the decline in rates of early childhood mortality. It therefore becomes especially important to redouble efforts to improve the conditions under which Kenyan children may live. These efforts must include establishing the well-spaced baby norm; providing access to contraception especially for certain high-risk groups of women;

improving conditions related to household exposure to disease—especially those related to toilet facilities; and, of course, continued improvement in the social and economic development of women.

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