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**Family Level Clustering of  
Childhood Mortality in Kenya**

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**Abstract**

Recent research on child mortality suggests that despite overall declines in mortality levels, mortality could remain relatively high due to a clustering of deaths in certain pockets of the population. Using data from the 1998 demographic and health survey (DHS) of Kenya this study examines child death clustering within families. The analysis first examines how known distant determinants of child mortality including socioeconomic and socio-cultural factors affect child survival, and how their effect is modified by proximate determinants of mortality including biodemographic and household environmental conditions. This is then followed by a control for family random effects to establish whether mortality risks in families are correlated net of the measured factors. The results show that biodemographic factors are more important in infancy while socioeconomic factors and household environmental conditions have a greater effect in childhood. Additionally, there is significant familial variation in child mortality risks even after controlling for measured determinants of mortality. This correlation of mortality risks suggests that there exists unobserved or unobservable familial factors related to mortality which lead to a concentration of deaths in certain families.

Key words: Infant mortality, child mortality, death clustering, unobserved heterogeneity, Kenya

## 1.0 Introduction

There have been several analyses of child mortality in Kenya, mainly utilising census and survey data (see, for instance, Kibet, 1981; Ewbank et al., 1986; Kichamu, 1986; Omariba, 1993; Obungu et al., 1994; Ikamari, 2000). These studies have mainly focused on establishing the determinants and differentials of child mortality. However, an important yet neglected area of research is the correlation of survival outcomes among siblings which causes deaths to cluster in families. Death clustering means a greater heterogeneity in the distribution of child deaths than would be expected if deaths were distributed randomly. Additionally, it is the remaining correlation after the observed correlates are controlled, and is due to unobserved or unobservable genetic, behavioural and environmental factors related to mortality (Ronsmans, 1995; Sastry, 1997a; Das Gupta, 1997).

Child deaths could cluster in families for many reasons. Das Gupta (1990) attributes the clustering of deaths in certain families due to lack of 'parental competence'; women who lose more than one child are, for example, less unskilled and resourceful in childcare including feeding, disease diagnosis and use of health services. Curtis and his colleagues (1993) suggest that for biological reasons some mothers experience more problems with pregnancy than others, including preterm deliveries and pregnancy complications which could also be repeated over several pregnancies. Additionally, children of the same family share the same home environment and would therefore suffer the same risks associated with that environment, such as poor sanitation.

The major assumption of the hazard model and other standard statistical techniques is

that the observations are independent. However, in demographic and health surveys (DHS) the information on children is collected from mothers in sampled households, hence children are not independent observations. This suggests that children survival risks are correlated. In the presence of correlated observations, the information provided on children from the same household is less than would have been the case if the same number of children were from different households. This leads to small standard errors and hence falsely significant estimates, while in the case of hazard models it yields small parameter estimates that are inconsistent and biased (e.g., Trussell and Rodríguez, 1990; Guo and Zhao, 2000). Kuate-Defo (2001) has also noted that in the case of survival models, the baseline hazard will be biased downwards which is similar to saying that the hazard is constant. As Guo and Zhao (2000) argue, adjusting for the correlation structure in the data corrects for the biases in parameter estimates and also provides correct standard errors and hence correct confidence intervals and significance tests. It is, therefore, necessary to account for the correlation of observations so as to derive 'true' estimates of standard errors and other associated values including valid p-values and confidence intervals.

Besides the methodological issue of correlated observations, there are also substantive considerations associated with unobserved heterogeneity. Generally, models with unobserved heterogeneity or frailty models (Vaupel, et al., 1979) capture the total effect of all unmeasured factors that influence the individual's risk of death. Unless one is conducting a biological survey, genetic factors are unobserved in social surveys such as DHS. They are therefore important in the interpretation of frailty effects (Vaupel, 1989), particularly at the family level among children born to the same mother. Other unobserved factors could

include childcare practices, cultural practices, and household environmental factors such as personal hygiene and general cleanliness. This suggests that there is need to control for possible significant influence of unmeasured heterogeneity.

There are several approaches to addressing the problem of correlation of sibling survival risks (e.g., Palloni, 1990; Zenger, 1993). Palloni (1990) has suggested, for instance, that it can be viewed as a matter of disentangling effects operating at different levels, that is, the child and the family (e.g., Curtis et al., 1993; Guo; 1993). Others have also included another level, the community level (e.g., Madise and Diamond, 1995; Sastry, 1997a; Siyam, 2001). In this paper we utilize the 1998 Demographic and Health survey for Kenya and control for correlation between siblings' survival risks at family level.

The Mosley and Chen (1984) framework, which guides the analysis in this study, suggests that variation in mortality between households within a given community is explained by differentials in socio-economic, biodemographic and socio-cultural factors and household environmental conditions. However, as Sastry (1997a) has observed, by exploiting the information that children in the same cluster share the same unmeasured factors at family or community level, one can estimate parameters that describe the distribution of the unmeasurable factors in the population. This study, therefore, estimates the presence of this correlation by including a term for unobserved heterogeneity in the estimation models. The presence of unobserved heterogeneity net of observed heterogeneity is an indication of the amount of variation across families and extent of death clustering in families.

## 2.0 Data and methods

The focus the focus of this analysis is death clustering in Kenyan families net of measured covariates of infant and child mortality. Since the response variable is child mortality, we utilise survival analysis procedures to examine the effect of various factors on the risk of death. The main advantage of survival models is that they account for the problem of censoring and selectivity in reproductive histories data, which if not corrected, would yield biased estimates. Additionally, they allow one to introduce a number of covariates to examine heterogeneity (Sastry, 1997a). A fundamental discriminating criteria between various event history models, is the distribution that the timing function is assumed to follow (Cleves et al., 2004). In this analysis we utilise the Weibull proportional hazards model of the form:

$$[1] \quad H_i(t) = \exp(\beta' x_i) \lambda \gamma^{\gamma-1}$$

where  $\gamma$  is an ancillary shape parameter estimated from the data, while scale parameter is parameterized as  $\lambda \exp(\beta' x_i)$ . The Weibull proportional hazards model was estimated by the STATA software (StataCorp, 2003) which constructs the likelihood function of the observations in the study and maximizing the resulting function with respect to the unknown parameters,  $\beta$ ,  $\lambda$ , and  $\gamma$ . The shape parameter  $\gamma$  can also be used to test for the non-constancy of the hazard; it is less than unity when the hazard is decreasing and more than unity when it rising. If it equals unity, then the hazard is constant and will be better

represented by an exponential distribution.

In the standard hazard regression model above, it is assumed that all the variation in the individual risk of death can be explained by the covariates. However, the random-effect model used to estimate the family-level death clustering assumes that individual risk of death is function of measured factors (Equation 1) and a random perturbation on the baseline hazard due the unobserved family effect. The random-effect model is of the form:

$$[2] \quad h(t_{ij} / x_{ij}, \alpha_i) = \alpha_i h(t_{ij} / x_{ij})$$

for data consisting of  $n$  groups with  $i$ th group comprised of  $n_i$  observations. The index  $i$  denotes the group ( $i = 1, \dots, n$ ), while  $j$  denotes the observation within group, ( $j = 1, \dots, n_i$ ).

In this study, index  $i$  and  $j$  correspond to mothers and children respectively. The frailty,  $\alpha$ , is a random positive quantity shared within the families and which for purposes of model identifiability is assumed to have a mean of one and variance  $\theta$ .

Just as in the distribution of the timing function in hazard models, one can either use a parametric approach or a non-parametric approach for the distribution of frailty. In this study the frailty is assumed to follow a gamma distribution. Not only has previous research made use of the gamma distribution (see, for instance, Vaupel et al., 1979; Oakes, 1982; Sastry, 1996), but there is evidence showing that the results of the hazard models are less likely to be sensitive to the choice of the frailty distribution<sup>1</sup> (Guo and Rodriguez, 1992;

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<sup>1</sup> Initial justifications for the use of nonparametric distribution were premised on the some

Sastry, 1996; 1997a, Gyimah, 2001). Moreover, there is a practical argument for using gamma distribution; its flexible shape and analytical tractability (Sastry, 1996). The variance of frailty,  $\theta$ , is estimated from the data and measures the variability of the frailty across groups.

The gamma distributed frailty was assumed to have following probability distribution function:

$$[3] \quad g(\alpha_i) = \frac{\alpha_i^{1/\theta-1} \exp(-\alpha_i/\theta)}{\Gamma(1/\theta)\theta^{1/\theta}}.$$

If  $\theta = 0$ , there is no variation across families and hence no effect of the family and no correlation between the survival risks of siblings. In this case, the model with frailty reduces to the standard hazard model. This also suggests that it is possible to test the significance of family effects by testing the null hypothesis  $\theta = 0$ . In the model without unobserved heterogeneity, the resulting hazard ratios are interpreted as relative risks of death. On the other hand, the random-effects hazard models no longer estimate a unique risk of death for children with particular characteristics because the risk of death depends on both the measured and the unmeasured family effect. The resulting hazard ratios from this model are therefore, family specific, that is, they represent the effect of a particular variable on the risk of death within a particular family (Neuhaus et al., 1991).

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evidence that the results of hazard models were dependent on the chosen parametric representation of the distribution function of unobserved heterogeneity (See, for instance,

### 3.0 Infant and child mortality in Kenya

We now apply the Weibull model with a term for unobserved heterogeneity to examine the risk of infant and child death in Kenya using the 1998 Demographic and Health Survey. The survey interviewed 7,881 women aged 15-49 of which 5716 had ever given birth. A complete birth history of each child 23,348 live births was obtained from the mothers including date of birth, length of preceding birth interval, current survival status, current age if child is alive and age at death for those who had died. The survey also collected background information on the mothers, which is used in the analysis of the determinants of infant and child death. Unlike other studies on infant and child mortality in the developing countries we include all the births of the women to avoid the problems of selectivity bias.

Table 1 presents the distribution of the distribution of number of children and child deaths per family. The table is interpreted in two complementary ways: the percentage of children coming from families with a given number of children and the percentage of deaths occurring to families with a given number child deaths<sup>2</sup>.

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Heckman and Singer, 1982).

<sup>2</sup> The columns represent the number of families with a given number of deaths and the rows represent the number of families with a given number of children. To obtain the rows' percentages corresponding with number of families with a given number of children, the row total of number of families is multiplied with the corresponding number of children for that group of family and divided by the total number of children (23,348). For example, 1,982 children came from families with two children. Additionally, the number of families in each row cell and the corresponding column number of deaths per family are multiplied and summed and divided by the total number of deaths in the sample (2,325). For example, 208 deaths occurred to families with four children, which represent 9 percent of all deaths. In the case of the column percentages, the row number of children in the family is multiplied with the column number of family with a given number of deaths. These are then summed up and divided by the total number of children to obtain the percentage of children from families with the corresponding number of deaths. For example, 14,537 children came from families with no death and represent 62.3 percent of all the children. Similarly, the column's total number of families is multiplied with the corresponding number of deaths for that group of family and the total is divided by the total number of deaths.

Over 80 percent of the children belong to families with three or more children in the sample; only about 5 percent of the families have one child. The 2,325 child deaths in the sample occurred to 1,483 families; 4,233 families had never experienced a child death. Only 38 percent of the families have five or more children, and yet these children make up about two-thirds of total children. About 33 percent of the deaths occurred to 4 percent of the families with three or more child deaths. Additionally, slightly over 1 percent of the families contribute four or more deaths; together they account for about 18 percent of the deaths. These results, therefore, suggest that there is substantial clustering of child deaths in certain families. Further, death clustering is indicated by the fact that the deaths in the sample occurred in only a few families as a majority of families did not have a death. Although the descriptive analysis shows that deaths cluster in families, this needs to be statistically established. Again, death clustering is defined here as residual variation net of observed heterogeneity.

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For example, 333 deaths occurred in families with three deaths and this represent 14.3 percent of the total number of deaths.

Table 1: Distribution of children and child deaths per family in Kenya, DHS 1998

Children per family	Deaths in family									Total families	Percent of	
	0	1	2	3	4	5	6	7	8		total children	total deaths
1	1012	87	0	0	0	0	0	0	0	1099	4.7	3.7
2	884	99	8	0	0	0	0	0	0	991	8.5	4.9
3	632	130	16	0	0	0	0	0	0	778	10.0	7.0
4	523	131	30	3	2	0	0	0	0	689	11.8	9.0
5	366	128	36	11	1	0	0	0	0	542	11.6	10.2
6	327	115	47	15	3	2	0	0	0	509	13.1	11.9
7	193	100	42	14	9	1	0	0	0	359	10.8	11.5
8	129	81	35	19	7	4	0	0	0	275	9.4	11.0
9	105	62	29	18	9	3	0	0	1	227	8.7	10.0
10	41	40	23	18	8	6	2	1	0	139	5.9	9.5
11	14	11	12	6	5	2	3	0	0	53	2.5	4.3
12	6	6	6	3	12	2	1	2	0	38	2.0	4.5
13	1	2	1	4	2	0	2	0	0	12	0.7	1.6
14	0	1	0	0	0	1	1	0	0	3	0.2	0.5
15	0	0	1	0	0	0	0	1	0	2	0.1	0.4
Total families	4223	993	286	111	58	21	9	4	1	5716	100	100
Percent of total children	62.3	21.7	8.3	3.9	2.3	0.9	0.5	0	0	100	----	----
Percent of total deaths	0	42.7	24.6	14.3	10	4.5	2.3	1.2	0.3	100	----	----

Source: Author's tabulation for all births in Kenya from the 1998 DHS

Table 2 presents the distribution of the variable of interest in child mortality analysis. The results show that majority of the children (80 percent) were born to rural mothers who had never migrated, with each of the rest of the migration status categories comprising less than 10 percent of the children. Almost two-thirds of the children belonged to mothers with primary education, about one-fifth to mothers with secondary and higher education with the rest belonging to mothers with no education. Most of the children lived in medium socioeconomic households (58 percent) with only about 12 percent living in high

socioeconomic households. The results in Table 2 also show that about 24 percent of the children were born in the 1990-1994 period, 22 percent in the 1985-1989 period, about 20 percent before the 1980 period, while about 17 percent each were born in the 1980-1984 and 1995-1998 periods respectively.

The results on the ethnicity and religion confirm that Kenya is a culturally diverse country. The distribution shows that *Kalenjin/Masai* were the largest group (20 percent) followed by the *Luhya* and *Luo*, 15.2 percent and 12.2 percent respectively. The *Somali/others* category were the least, 2.4 percent. Although the distribution by religious affiliation confirms that Kenya is a predominantly Christian population, the majority of the children belonged to Protestant/other Christian mothers (64 percent), followed by the Catholics (27 percent) while children of Moslem mothers comprised only about 5 percent of the sample.

Regarding household environmental conditions, an equal proportion of children (24 percent) lived in households using piped or well water for drinking, while slightly over half of the children (51 percent) lived in households drawing their drinking water from unsafe sources including rivers, lakes and tapped rain water. Additionally, most of the children lived in households with a pit latrine (76.2 percent), while only about 5 percent lived in households with water closets.

A number of biodemographic factors are important determinants of child survival. The results show that most of these factors are likely to be positively associated with child survival in Kenya. Of the 17562 children who were either second or higher order births, the preceding sibling was alive for 87 percent of the children, while only 13 percent had lost a

sibling to death. The results also indicate that birth intervals are relatively long in Kenya with over 80 percent of the children being born 19 months after their preceding sibling birth. A high number of children per household is indicated by the number of children of two and higher birth order; only 25 percent of the children were first order births with the rest being second or higher births. Most of the children (68 percent) were born when their mothers were at the prime of childbearing, ages 20-34. Nonetheless, about one-fifth of the children were born when their mothers were below age 20, while about 5 percent when their mothers were either 35 or older.

Table 2: Percentage distribution of children by covariates used in analysis of child mortality in Kenya

<b>Variables</b>	<b>Number</b>	<b>Percent</b>
<b>Migration status</b>		
Rural nonmigrant	18676	80.0
Urban nonmigrant	1234	5.3
Urban-rural migrant	1798	7.7
Rural-urban migrant	1640	7.0
<b>Maternal education level</b>		
None	5458	23.4
Primary	13549	58.0
Secondary or higher	4341	18.6
<b>Household socioeconomic status</b>		
Low	7048	30.2
Medium	13503	57.8
High	2797	12.0
<b>Year of birth</b>		
<1980	4555	19.5
1980-1984	4115	17.6
1985-1989	5218	22.3
1990-1994	5588	23.9
1995-1998	3872	16.6
<b>Ethnicity</b>		
Kikuyu	3124	13.4
Kamba	2333	10.0
Kalenjin/Masai	4568	19.6
Kisii	1733	7.4
Luhya	3548	15.2
Luo	3326	14.2
Meru/Embu	1380	5.9
Mijikenda/Taita/Taveta	1938	8.3
Taita/Taveta	827	3.5
Somali/Others	571	2.4
<b>Religion</b>		
Protestant/other Christian	14829	63.5
Catholic	6298	27.0
Muslim	1204	5.2
No religion	1017	4.4

Table 2 continued

Variable	Number	Percentage
<b>Source of drinking water</b>		
River, lake, rain water	11971	51.3
Well water	5697	24.4
Piped water	5680	24.3
<b>Type of toilet facility</b>		
Pit latrine	17796	76.2
Water closet	1320	5.7
No facility	4232	18.1
<b>Previous child survival status<sup>a</sup></b>		
Alive	15399	87.3
Dead	2233	12.7
<b>Preceding birth interval<sup>a</sup></b>		
19-35 months	9390	53.3
<19 months	3029	17.2
36+ months	5213	29.5
<b>Birth order</b>		
Order 1	5716	24.5
Order 2	4617	19.8
Order 3	3626	15.5
Order 4-5	5007	21.4
Order 6-7	2725	11.7
Order 8-15	1657	7.1
<b>Age of mother at birth of child</b>		
<20	5758	24.7
20-24	7697	33.0
25-19	5386	23.1
30-34	3005	12.1
>35	1502	6.4
Total (if no inapplicable cases)	23348	100

Notes: <sup>a</sup> The numbers do not add up to 23348 because some children were first births (no preceding birth or birth interval).

The analysis begins first by examining models without the random family effect.

This was then followed by an inclusion of the random effect in the models to test for the presence of significant family effects. The analytical strategy followed is first to test the relationship between child mortality and the distant factors; this is model one of the analysis. This was then followed by an inclusion of the proximate determinants, which is the full model. The hypothesis here is that the distant factors including maternal education, migration status, household economic status, ethnicity and religion will have less impact on child mortality when control is made for the proximate determinants of mortality. However, we only report the results of the final models here.

The estimated random parameters,  $\theta$ , in the models with unobserved heterogeneity are 0.40 and 0.78 for infant and child mortality respectively (Tables 5 and 6). Both values are highly significant which further confirms that there is significant familial variation in the risk of infant and child death. There is, however, little change in the standard errors of the parameter estimates between the models with and without unobserved heterogeneity. This is expected because we adjusted for the fact that children are clustered within their mothers to obtain robust standard errors in the models without a term for unobserved heterogeneity. What is important to note is that the frailty is significant in the models that control for it. The parameter estimates of the model without unobserved heterogeneity remained largely unchanged when familial correlation is controlled. These findings suggest that the substantive conclusions drawn from the standard hazard model are robust to the failure of the independence assumption even though there is a slight change in the magnitude of the estimated effects.

Although the estimates in the earlier models is not included, the results in Table 3 show that the magnitude of effect of the distant factors including maternal education, ethnicity and religion is depressed, while that of period of birth is increased. The results, therefore, attest to the importance of biodemographic factors in survival during infancy. Regarding maternal education, the results in this model show that there is no significant difference between children of mothers with no education and those of mothers with primary education. However, being children of mothers with secondary education face a reduced risk of child death of about one-fifth.

Children of urban nonmigrant mothers face better survival prospects as compared to children of rural nonmigrant mothers. There is, however, no significant difference between children of urban-rural and rural-urban migrants on one hand and that of rural nonmigrants on the other in the risk of death in infancy. Controlling for biodemographic, socio-cultural and other socioeconomic factors, the period of child birth is still significantly associated with infant death. Substantively, children born in the 1990-1994 period were 52 percent, while those born in the 1995-1998 period were 77 percent more likely to die as compared to those born before 1980. The results further suggest that mortality remained largely unchanged in the 1980s, though lower than before 1980. The pattern of association between religion and infant mortality in the preceding models is also observed in the final model. Children of Muslim mothers face poor survival prospects, with the risk of a child of Muslim mother dying in infancy being 74 percent higher than that of children Protestant mothers.

Table 3: A Weibull model of infant mortality in Kenya with observed heterogeneity

Variables	Model 1	Model 2	Model 3
<b>Migration status</b>			
Rural nonmigrant <sup>a</sup>	1.00	1.00	1.00
Urban nonmigrant	0.61(0.10)***	0.68(0.11)**	0.70(0.11)**
Urban-rural migrant	1.12(0.10)	1.13(0.10)	1.12(0.09)
Rural-urban migrant	0.73(0.09)**	0.80(0.12)	0.80(0.11)
<b>Maternal education level</b>			
Primary <sup>a</sup>	1.00	1.00	1.00
None	1.15(0.08)**	1.13(0.08)*	1.08(0.07)
Secondary or higher	0.71(0.07)***	0.73(0.07)***	0.78(0.07)***
<b>Household economic status</b>			
Medium <sup>a</sup>	1.00	1.00	1.00
Low	1.08(0.07)	1.04(0.09)	1.03(0.06)
High	0.78(0.10)*	0.83(0.13)	0.85(0.11)
<b>Year of birth</b>			
<1980 <sup>a</sup>	1.00	1.00	1.00
1980-1984	0.89(0.08)	0.89(0.08)	0.97(0.09)
1985-1989	0.87(0.08)	0.86(0.08)	0.96(0.09)
1990-1994	1.37(0.11)***	1.36(0.11)***	1.52(0.13)***
1995-1998	1.56(0.15)***	1.54(0.14)**	1.77(0.17)***
<b>Ethnicity</b>			
Kikuyu <sup>a</sup>	1.00	1.00	1.00
Kamba	1.99(0.28)***	1.89(0.27)***	1.81(0.25)***
Kalenjin/Masai	1.63(0.23)***	1.56(0.22)***	1.46(0.20)***
Kisii	1.18(0.20)	1.17(0.20)	1.10(0.19)
Luhya	2.39(0.31)***	2.38(0.31)***	2.10(0.26)***
Luo	5.03(0.62)***	4.71(0.60)***	3.71(0.46)***
Meru/Embu	1.10(0.21)	1.10(0.21)	1.11(0.20)
Mijikenda/Swahili	1.71(0.29)***	1.56(0.27)***	1.49(0.24)***
Taita/Taveta	1.63(0.35)**	1.58(0.34)**	1.55(0.32)**
Somali/Others	1.98(0.51)***	2.10(0.52)***	1.89(0.44)***
<b>Religion</b>			
Protestant/Other Christian <sup>a</sup>	1.00	1.00	1.00
Catholic	1.08(0.07)	1.07(0.07)	1.08(0.07)
Muslim	1.85(0.28)***	1.83(0.26)***	1.74(0.23)***
No religion	1.12(0.17)	1.09(0.16)	1.11(0.15)

Table 3 continued

Variables	Model 1	Model 2	Model 3
<b>Source of drinking water</b>			
River, Lake, Rain water <sup>a</sup>		1.00	1.00
Well water		0.94(0.07)	0.95(0.06)
Piped water		1.03(0.09)	1.04(0.09)
<b>Type of toilet facility</b>			
Pit latrine <sup>a</sup>		1.00	1.00
Water closet		0.71(0.15)	0.76(0.15)*
No facility		1.26(0.09)***	1.20(0.08)***
<b>Previous child survival status</b>			
Alive <sup>a</sup>			1.00
Dead			2.33(0.17)***
<b>Preceding birth interval</b>			
19-35 <sup>a</sup>			1.00
<19 months			1.47(0.11)***
36 or more months			0.71(0.06)***
<b>Birth order</b>			
			1.07(0.02)***
<b>Age of mother at birth of child</b>			
20-24 <sup>a</sup>			1.00
Below 20			1.35(0.10)****
25-29			0.88(0.07)
30-34			0.98(0.11)
35 or more			0.97(0.16)
Total (If no inapplicable values)	23348	23348	23348
Negative Log Likelihood	9578	9569	9402
Likelihood Ratio Chi-Square	481	511	1136
Degrees of Freedom	23	27	35

Notes: <sup>a</sup>= Reference category; Standard errors are in parenthesis; Factor significance levels: \*\*\*=1% or better; \*\*=5%; \*= 10%.

The large disparity between the *Luo* and other tribes on one hand and the *Kikuyu* substantially decreases when control is made for biodemographic factors according to Model 3. As compared to Models 1 and 2 when children of *Luo* mothers were 5 times more likely to die compared to those of the reference category, they were now only about 4 times more likely to die. Children of *Luhya* mothers also face poorer survival prospects; children of

mothers group are more than twice as likely to die compared to children of *Kikuyu* mothers. Additionally, being a child of either a *Kamba*, *Taita/Taveta* or *Somali* mother increased the hazard of death by over 50 percent. Again, there is no significant difference between children of *Kisii* and *Meru/Embu* mothers on one hand and those of *Kikuyu* mothers in the risk of death in infancy.

Household environmental conditions particularly the type of toilet facility in the household, significantly associate with child mortality. Living in a household with no toilet facility increases the risk of child death by 20 percent as compared to living in household with a pit latrine. On the other hand, living a household with water closet reduces the risk of child death by about 25 percent. In addition to the socioeconomic, socio-cultural and household environmental conditions discussed above, significant differences in child survival by biodemographic factors are visible. The results show that for each additional birth order, the risk of in infancy increased by about 7 percent. The risks of death for children born to mothers aged over 35 were not significantly different from those of mothers aged between 20-24 years. However, children born to mother aged below age 20 at the time of birth were 35 percent more likely to die as compared to those of mothers aged between 20-24 years.

As compared to children whose previous sibling was alive, the risk of death was 133 percent higher for children whose previous sibling had died. There are also significant differences by the length of the preceding birth interval. Children who were born less than 19 months after their preceding sibling were 47 percent more likely to die compared to those born between 19 and 35 months after their previous sibling. On the other hand, being born

36 months after the previous sibling reduced the risk of death during infancy by about 30 percent.

Table 4 presents child mortality parameter estimates and standard errors for the standard hazard model that adds biodemographic factors to socioeconomic, socio-cultural and household environmental factors in preceding models. Except for the period of child birth, the direction of effect of the covariates in the preceding models remains largely unchanged. Generally, the pattern of effect of biodemographic factors is consistent with the hypothesized expectations. However, the magnitude of the effects clearly demonstrates that biodemographic factors are less important in child mortality relative to infant. The effect of survival of the immediately preceding sibling on survival of the index child are evident; children whose previous sibling had died were 47 percent more likely to die compared to those their sibling was alive. There are also significant differences by length of the preceding birth interval. Relative to children born between 19 and 35 months after their preceding sibling, children born less than 19 months after their preceding sibling were 25 percent more likely to die in childhood. On the other hand, children born 36 months after the preceding sibling were about 25 percent less likely to die compared to children born 19-35 months after the previous sibling. Again, the pattern effect of maternal age at birth of the child is similar to that observed for infant mortality. The effects of young maternal age at birth also continue during childhood; children born when their mothers were aged below 20 were 46 percent more likely to die compared to those born when their mothers were aged 20-24 years.

Table 4: A Weibull model of child mortality in Kenya with observed heterogeneity

Variables	Model 1	Model 2	Model 3
<b>Migration status</b>			
Rural nonmigrant <sup>a</sup>	1.00	1.00	1.00
Urban nonmigrant	0.86(0.20)	0.95(0.22)	0.95(0.22)
Urban-rural migrant	1.07(0.15)	1.06(0.15)	1.05(0.14)
Rural-urban migrant	1.10(0.09)	1.22(0.26)	1.19(0.25)
<b>Maternal education level</b>			
Primary <sup>a</sup>	1.00	1.00	1.00
None	1.31(0.13)***	1.26(0.13)**	1.26(0.13)**
Secondary or higher	0.53(0.08)***	0.54(0.08)***	0.58(0.08)***
<b>Household economic status</b>			
Medium <sup>a</sup>	1.00	1.00	1.00
Low	1.25(0.11)***	1.19(0.11)*	1.19(0.11)*
High	0.71(0.14)***	0.79(0.16)	0.80(0.16)
<b>Year of birth</b>			
<1980 <sup>a</sup>	1.00	1.00	1.00
1980-1984	1.11(0.13)	1.10(0.13)	1.20(0.14)
1985-1989	1.36(0.15)***	1.35(0.15)***	1.49(0.17)***
1990-1994	1.98(0.23)***	1.94(0.22)***	2.20(0.27)***
1995-1998	1.62(0.29)***	1.58(0.30)***	1.84(0.34)***
<b>Ethnicity</b>			
Kikuyu <sup>a</sup>	1.00	1.00	1.00
Kamba	3.10(0.83)***	2.90(0.78)***	2.85(0.77)***
Kalenjin/Masai	2.52(0.63)***	2.36(0.59)***	2.22(0.57)***
Kisii	5.27(1.40)***	5.22(1.38)***	5.01(1.31)***
Luhya	7.10(1.66)***	7.10(1.67)***	6.62(1.56)***
Luo	10.60(2.49)***	9.72(2.33)***	8.56(2.05)***
Meru/Embu	1.81(0.57)	1.78(0.56)*	1.79(0.57)*
Mijikenda/Swahili	5.04(1.51)***	4.41(1.34)***	4.21(1.24)***
Taita/Taveta	3.20(1.08)***	3.05(1.04)***	3.04(1.02)***
Somali/Others	1.52(0.78)	1.60(0.82)	1.52(0.77)
<b>Religion</b>			
Protestant/Other Christian <sup>a</sup>	1.00	1.00	1.00
Catholic	1.03(0.10)	1.02(0.10)	1.03(0.10)
Muslim	0.88(0.25)	0.88(0.24)	0.87(0.23)
No religion	0.80(0.19)	0.76(0.18)	0.77(0.18)

Table 4 continued

Variables	Model 1	Model 2	Model 3
<b>Source of drinking water</b>			
River, Lake, Rain water <sup>a</sup>		1.00	1.00
Well water		0.92(0.10)	0.93(0.10)
Piped water		1.08(0.13)	1.10(0.13)
<b>Type of toilet facility</b>			
Pit latrine <sup>a</sup>		1.00	1.00
Water closet		0.56(0.17)*	0.60(0.18)*
No facility		1.38(0.15)***	1.34(0.14)***
<b>Previous child survival status</b>			
Alive <sup>a</sup>			1.00
Dead			1.47(0.17)***
<b>Preceding birth interval</b>			
19-35 <sup>a</sup>			1.00
<19 months			1.25(0.14)**
36 or more months			0.77(0.09)***
<b>Birth order</b>			1.05(0.35)
<b>Age of mother at birth of child</b>			
20-24 <sup>a</sup>			1.00
Below 20			1.46(0.16)***
25-29			1.01(0.12)
30-34			0.83(0.15)
35 or more			0.90(0.23)
Total	21727	21727	21727
Negative Log Likelihood	3977	3969	3943
Likelihood Ratio Chi-Square	326	359	472
Degrees of Freedom	23	27	35

Notes: <sup>a</sup>= Reference category; Standard errors are in parenthesis; Factor significance levels: \*\*\*=1% or better; \*\*=5%; \*= 10%.

Table 5 presents the results of frailty models of infant mortality. In models with frailty, the effects of migration status, education, source of water, preceding birth interval, birth order and maternal age at birth remain unchanged. The magnitude of effect of period of birth and previous child survival status decreased in the models with frailty. On the other hand, the magnitude of effect of religion increased in the models with frailty, while for some categories of ethnicity it increased, remained unchanged or declined. For example, in the models with all the covariates of interest, the hazard ratio associated with *Luo* increased from

3.71 in the model without frailty to 4.00 in the model with frailty and from 2.10 to 2.15 for *Luhya*. However, it declined for the *Kalenjin/Masai*, *Taita/Taveta* and the *Somali/Others*. Similarly, the hazard ration for survival status of previous child significantly declines from 2.33 in the model without frailty to 1.90 in the one with frailty. This translates into a decrease of 18 percent difference in the hazard ratio associated with the death of the previous child between the models without frailty and with frailty. This means that conditional on the estimated level of frailty, compared to children whose previous sibling was alive, children whose previous sibling had died were 90 percent more likely to die.

The significance of the frailty term suggests that despite controlling for factors usually associated with mortality in infancy, significant differences between children in the risk of death persist in Kenya. The results generally show that in the model without frailty the effect of important infant death factors such as the survival status of the previous child are likely to be overstated. Additionally, the effect of previous child survival status reflects an important dimension of familial mortality risk (Sastry 1997b), hence the weakened effect of the variable in the presence of frailty.

Table 5: A Weibull model of infant mortality in Kenya with observed and unobserved heterogeneity

Variables	Model 1	Model 2	Model 3
<b>Migration status</b>			
Rural nonmigrant <sup>a</sup>	1.00	1.00	1.00
Urban nonmigrant	0.63(0.10)***	0.71(0.12)**	0.71(0.12)**
Urban-rural migrant	1.16(0.12)	1.17(0.12)	1.15(0.11)
Rural-urban migrant	0.75(0.10)	0.82(1.12)	0.82(0.11)
<b>Maternal education level</b>			
Primary <sup>a</sup>	1.00	1.00	1.00
None	1.18(0.09)**	1.16(0.09)**	1.12(0.08)
Secondary or higher	0.69(0.06)***	0.72(0.07)***	0.77(0.07)***
<b>Household economic status</b>			
Medium <sup>a</sup>	1.00	1.00	1.00
Low	1.08(0.07)	1.04(0.07)	1.04(0.07)
High	0.77(0.09)**	0.82(0.10)	0.85(0.10)
<b>Year of birth</b>			
<1980 <sup>a</sup>	1.00	1.00	1.00
1980-1984	0.89(0.07)	0.89(0.07)	0.95(0.08)
1985-1989	0.86(0.07)*	0.85(0.07)*	0.92(0.08)
1990-1994	1.35(0.10)***	1.33(0.10)***	1.46(0.12)***
1995-1998	1.51(0.13)***	1.49(0.13)***	1.69(0.16)***
<b>Ethnicity</b>			
Kikuyu <sup>a</sup>	1.00	1.00	1.00
Kamba	2.02(0.29)***	1.92(0.28)***	1.83(0.25)***
Kalenjin/Masai	1.62(0.21)***	1.55(0.21)***	1.45(0.18)***
Kisii	1.17(0.21)	1.17(0.20)	1.12(0.19)
Luhya	2.41(0.32)***	2.40(0.32)***	2.15(0.28)***
Luo	5.28(0.67)***	4.94(0.65)***	4.00(0.51)***
Meru/Embu	1.09(0.21)	1.09(0.20)	1.11(0.20)
Mijikenda/Swahili	1.66(0.30)**	1.51(0.27)**	1.46(0.25)***
Taita/Taveta	1.62(0.33)**	1.58(0.32)**	1.56(0.30)**
Somali/Others	1.93(0.44)***	1.99(0.56)***	1.85(0.41)***
<b>Religion</b>			
Protestant/Other Christian <sup>a</sup>	1.00	1.00	1.00
Catholic	1.12(0.08)	1.12(0.08)	1.11(0.07)
Muslim	1.90(0.31)***	1.86(0.30)***	1.77(0.27)***
No religion	1.17(0.20)	1.13(0.19)	1.13(0.18)

Table 5 continued

Variables	Model 1	Model 2	Model 3
<b>Source of drinking water</b>			
River, Lake, Rain water <sup>a</sup>		1.00	1.00
Well water		0.95(0.07)	0.97(0.07)
Piped water		1.03(0.09)	1.04(0.09)
<b>Type of toilet facility</b>			
Pit latrine <sup>a</sup>		1.00	1.00
Water closet		0.69(0.13)*	0.74(0.14)*
No facility		1.25(0.10)***	1.22(0.09)***
<b>Previous child survival status</b>			
Alive <sup>a</sup>			1.00
Dead			1.90(0.14)***
<b>Preceding birth interval</b>			
19-35 <sup>a</sup>			1.00
<19 months			1.45(0.10)***
36 or more months			0.71(0.06)***
<b>Birth order</b>			1.06(0.021)***
<b>Age of mother at birth of child</b>			
20-24 <sup>a</sup>			1.00
Below 20			1.34(0.10)***
25-29			0.89(0.07)
30-34			1.02(0.11)
35 or more			1.04(0.17)
THETA	0.84(0.10)***	0.82(0.09)***	0.40(0.08)***
Total (if no missing values)	23348	23348	23348
Negative Log Likelihood	9495	9489	9380
Likelihood Ratio Chi-Square	470	483	700
Likelihood Ratio Chi-Square of THETA=0	165	159	44
Degrees of Freedom	23	27	35

Notes: <sup>a</sup>= Reference category; Standard errors are in parenthesis; Factor significance levels: \*\*\*=1% or better; \*\*=5%; \*= 10%.

Table 6 presents the results of the frailty models of child mortality. The direction of the effect of the covariates in models with frailty is also consistent with our hypothesized expectations. The results show that children of mothers with primary education, those born in the late 1980s and 1990s; children of non-*Kikuyu* mothers, those living in a household

with no toilet facility, children born less than 19 months after the preceding sibling and those born when their mothers were aged below 20 have significantly higher risk of death in childhood. For example, in the final model, children of *Luo* mothers were nine times, those of *Luhya* mothers seven times while those of *Kisii* mothers were five times more likely to die as compared to children of *Kikuyu* mothers. The results further show that being a child of a mother with secondary or higher education, being born 36 or more months after the preceding sibling and living in a household with water closet is significantly associated with lesser hazards of death in childhood. The main surprising finding relates to the survival status of the preceding child; unlike in the model without frailty the death of the preceding child is insignificant in the presence of frailty. Nonetheless, children whose preceding sibling had died were 13 percent more likely to die as compared to those whom their sibling was alive.

The pattern of change in the magnitude of effect of the covariates also largely mirrors that observed for infant mortality. Generally, for most of the covariates, the models without frailty tend to overestimate their true effect because of unmeasured factors. This is particularly true of the survival status of the preceding child which could be tied to unmeasurable biological factors that could cause the death of several children of the same mother.

Table 6: A Weibull model of child mortality in Kenya with observed and unobserved heterogeneity

Variables	Model 1	Model 2	Model 3
<b>Migration status</b>			
Rural nonmigrant <sup>a</sup>	1.00	1.00	1.00
Urban nonmigrant	0.86(0.20)	0.97(0.24)	0.95(0.23)
Urban-rural migrant	1.10(0.16)	1.11(0.16)	1.10(0.16)
Rural-urban migrant	1.11(0.20)	1.23(0.24)	1.19(0.23)
<b>Maternal education level</b>			
Primary <sup>a</sup>	1.00	1.00	1.00
None	1.31(0.13)***	1.28(0.13)**	1.28(0.13)**
Secondary or higher	0.52(0.0)***	0.55(0.08)***	0.56(0.09)***
<b>Household economic status</b>			
Medium <sup>a</sup>	1.00	1.00	1.00
Low	1.26(0.12)***	1.20(0.11)*	1.20(0.11)*
High	0.69(0.13)**	0.75(0.15)	0.77(0.15)
<b>Year of birth</b>			
<1980 <sup>a</sup>	1.00	1.00	1.00
1980-1984	1.10(0.13)	1.10(0.13)	1.18(0.14)
1985-1989	1.33(0.15)***	1.32(0.15)**	1.45(0.18)***
1990-1994	1.92(0.22)***	1.90(0.22)***	2.15(0.28)***
1995-1998	1.59(0.28)***	1.56(0.28)***	1.81(0.34)***
<b>Ethnicity</b>			
Kikuyu <sup>a</sup>	1.00	1.00	1.00
Kamba	3.11(0.86)	2.86(0.80)***	2.86(0.79)***
Kalenjin/Masai	2.52(0.65)***	2.32(0.60)***	2.21(0.57)***
Kisii	5.30(1.42)***	5.19(1.40)***	5.04(1.35)***
Luhya	7.19(1.77)***	7.20(1.79)***	6.90(1.70)***
Luo	10.8(2.66)***	9.80(2.43)***	8.98(2.23)***
Meru/Embu	1.78(0.59)*	1.76(0.59)*	1.77(0.59)*
Mijikenda/Swahili	4.94(1.49)***	4.28(1.31)***	4.08(1.24)***
Taita/Taveta	3.20(1.08)***	3.04(1.03)***	3.06(1.03)***
Somali/Others	1.52(0.79)	1.61(0.84)	1.53(0.80)
<b>Religion</b>			
Protestant/Other Christian <sup>a</sup>	1.00	1.00	1.00
Catholic	1.06(0.11)	1.05(0.11)	1.05(0.10)
Muslim	0.90(0.23)	0.89(0.22)	0.90(0.22)
No religion	0.80(0.20)	0.74(0.19)	0.77(0.19)

Table 6 continued

Variables	Model 1	Model 2	Model 3
<b>Source of drinking water</b>			
River, Lake, Rain water <sup>a</sup>		1.00	1.00
Well water		0.89(0.10)	0.90(0.10)
Piped water		1.07(0.14)	1.09(0.14)
<b>Type of toilet facility</b>			
Pit latrine <sup>a</sup>		1.00	1.00
Water closet		0.58(0.18)*	0.61(0.19)*
No facility		1.44(0.16)***	1.42(0.16)***
<b>Previous child survival status</b>			
Alive <sup>a</sup>			1.00
Dead			1.13(0.13)
<b>Preceding birth interval</b>			
19-35 <sup>a</sup>			1.00
<19 months			1.29(0.14)**
36 or more months			0.77(0.09)**
<b>Birth order</b>			1.04(0.03)
<b>Age of mother at birth of child</b>			
20-24 <sup>a</sup>			1.00
Below 20			1.46(0.17)***
25-29			1.01(0.13)
30-34			0.86(0.15)
35 or more			0.92(0.23)
THETA	0.94(0.17)****	0.92(0.18)***	0.78(0.18)***
Total (if no missing values)	21728	21728	21728
Negative Log Likelihood	3949	3941	3924
Likelihood Ratio Chi-Square	349	364	399
Likelihood Ratio Chi-Square of THETA=0	56	55	37
Degrees of Freedom	23	27	35

Notes: <sup>a</sup>= Reference category; Standard errors are in parenthesis; Factor significance levels: \*\*\*=1% or better; \*\*=5%; \*= 10%.

#### 4.0 Discussion and conclusions

This analysis of infant and child mortality in Kenya has shown that the magnitude of effect of biodemographic factors including the survival status of previous child, length of preceding birth interval, birth order and maternal age at birth of child was greater for infant mortality. On the other hand, socioeconomic, socio-cultural and environmental factors were more important in childhood. There was no much change in the estimates from the models with observed heterogeneity and the ones with unobserved heterogeneity. This is because we adjusted for the non-independence of observations using the Huber-White sandwich estimator to produce robust standard errors in the former models. Nonetheless, although the effects of most factors on mortality remained largely robust in the frailty models, there were significant changes in the estimated relative risks.

The findings confirm previous research on the effects of the survival status of the previous child particularly on infant death (e.g., Das Gupta, 1990; Sastry 1996; 1997b; Ikamari, 2000). However, the effect of the survival status of the preceding child on infant mortality is attenuated while its relationship with child mortality seems spurious once we introduced a control for frailty. This suggests that it is acting primarily as a control for familial mortality risks in the standard Weibull survival model.

The findings of this study are consistent with the pathways through which the death of a preceding child affects the survival of the index child. First, the death of the previous child could affect the survival of the subsequent child through the truncation of the interval to the subsequent birth. When the death of the child occurs at infancy, the mother stops breastfeeding leading to the return of menses, thereby, exposing her to the risk of conceiving

(see, for instance, Bongaarts and Potter, 1983; Gyimah, 2001). In addition, the mother's body requires about 24 months to replenish the vital nutrients such as calcium and iron that are needed to support fetal development (Scrimshaw, 1996). This truncation of the inter-birth interval, therefore, leads to the *maternal depletion syndrome*. On the other hand, parents may make a deliberate effort to replace the dead child, what has been called the *replacement hypothesis* (see, for instance, Lantz et al., 1992; Gyimah, 2001; LeGrand, 2003). There is yet a third mechanism through which the death of preceding child affects the survival of the succeeding one, *maternal depression*. According to this hypothesis, the death of child leaves the mother depressed, thereby, compromising the health of the child both in the womb and in early infancy (Steers et al., 1992 [cited in Arulampalam and Bhalotra, 2004]).

Although the effect of the death of the previous child shortens the time to conception of the next birth, birth interval may operate independently among certain mothers to affect child survival (See, for instance, Lindstrom and Berhanu, 2001; Sear et al., 2002). Significant differences by length of preceding birth interval were observed in this study with the results confirming the negative effects of short birth interval on child survival. As was discussed in relation to the death of the preceding sibling, short inter-birth intervals lead to physiological depletion of maternal energy and nutrition. This in turn leads to pre-term and low birthweight babies who are more prone to illness and pregnancy-related complications which increase the child's risk of death. In addition, short birth intervals also mean that the mother is adding the number of children of the almost similar age in the household, thereby increasing the susceptibility of these children to infectious diseases due to their likely physical proximity during the day or due to household congestion (Aaby, 1992; Alam, 1995;

Woldemicael, 1999). The other theoretical through which short birth interval affects child survival is sibling competition. As Gribble (1993) has noted, with the birth of each additional child, the average allocation decreases and so closely spaced children face higher competition among each other for familial resources.

Significant mortality differentials by maternal age at birth of child were observed. These results also confirm previous research findings that have found a higher risk of death for children of teenage mothers (e.g., Obungu et al., 1994; Alam, 2000; Sear et al., 2002) and for those of older mothers (e.g., McMurray, 1997; Lindstrom and Berhanu, 2001). There are two theoretical mechanisms through which maternal age at birth is thought to influence child survival; the physiological and the social and psychological perspectives. The physiological hypothesis attributes the higher risk of death among younger women to their immature reproductive systems which are not yet ready to handle the complexities of child birth (Trussell and Hammerslough, 1983). The social and psychological hypothesis, on the other hand, argues that younger mothers are not yet ready to take on parental responsibilities (Alam, 2000). Such mothers are less likely to have decision-making authority within the household and more likely to lack financial resources to seek medical attention for their children. Additionally, due to social stigma associated with being young mothers, they may not seek prenatal care and therefore, any complications related to the pregnancy may go undetected until it is too late.

Although the proximate determinants were significantly associated with both infant and child mortality, the effect of distant factors was not negligible. Even after controlling for other observed factors and frailty, there were significant differences in the risk of infant and

child death by socioeconomic, socio-cultural and environmental factors. Maternal education is significantly associated with child survival both in infancy and childhood. These results reaffirm prior research that has demonstrated that for any significant change in child survival to occur, mothers need to at least attain secondary education (Caldwell, 1979; Cleland and Van Ginneken, 1988; Cleland, 1989; O'toole and Wright, 1991; Rutstein, 2000; Uchudi, 2001).

There are several mechanisms through which education affects child survival. Higher child survival among educated mothers has been attributed to higher socioeconomic status resulting from education, its improvement of maternal basic childcare skills, her domestic management of child illness, efforts at preventive care and effective use of modern health services (Das Gupta, 1990; Caldwell, 1979; 1994). Additionally, education changes the traditional familial relationships regarding decisionmaking giving mothers a greater say in childcare issues (See, for instance, O'toole and Wright, 1991; Caldwell, 1986). For example, it has been argued that because educated mothers are more likely to seek medical attention for their children and to enjoy favourable responses from the medical personnel, survival chances of their children are likely to be higher.

There are also significant differences in the risk of infant and child mortality by ethnicity; children of all other ethnic groups have significantly higher risk of death compared to Kikuyu children. However, the magnitude of the effect of ethnicity was larger in childhood as compared to infancy. These findings are also consistent with previous studies on childhood mortality in Kenya (See, for instance, Omariba, 1993; Tabutin and Akoto, 1992; Brockerhoff and Hewitt, 1998). Ethnic variation in child survival is usually explained

in terms of differences in socioeconomic development between groups and childcare and reproductive behaviors which are underpinned by ethnic cultural and traditional practices. The socioeconomic explanation holds that in areas of comparable socioeconomic development indicated by education, urbanization, good living conditions and health facilities, there will be little differences in mortality levels (UN, 1988). Again, the magnitude of effect of ethnicity was greater in childhood than in infancy. This is consistent with previous research that has shown that child deaths are more affected by levels of household resources and socioeconomic status (see, for instance, Ahonsi, 1995; Woldemicael, 2000). For example, the *Kikuyu* are the most westernized in Kenya and also inhabit the Central province which is more developed the country in terms of economic production, education and health facilities.

There is no doubt that disparity in levels of socioeconomic development account for mortality differentials between tribes. However, what is not usually examined is why certain groups are more economically advantaged. Brockerhoff and Hewitt (1998), for instance, have attributed the good mortality conditions of certain ethnic groups their dominance in the national political economy. They argue that mortality is lower among children from ethnic groups that have held national political authority since political independence or possess historical economic advantage over other groups. This is true in both countries that have enjoyed political stability such as Kenya and in those that have experienced internecine wars. Kenya has not only enjoyed political stability in its forty years of independence, but it has only had three presidents during this period. The first president, Jomo Kenyatta, was *Kikuyu*, the second, Daniel Moi was *Kalenjin* while the current president Mwai Kibaki, is a *Kikuyu*.

According to Brockerhoff and Hewitt (1998) the survival chances for member of potent groups are enhanced through favourable economic conditions in the households and communities. This comparative advantage, they argue, results from an uneven distribution of state-controlled resources and publicly conferred entitlements including health resources. The results of this analysis suggest that the Kikuyu and Kalenjin enjoy favourable conditions such as shorter distance to health facilities even outside urban areas due to their nonrandom location or uneven economic development including employment creation. For instance, the median distance to the nearest public health facility was 15 kilometres for the *Kikuyu* compared to 20 kilometres for the other ethnic groups (Brockerhoff and Hewitt, 1998). In his twenty-four years rule, Daniel Moi transformed the part of Rift Valley province where is from into a modern area building all-weather roads, supplying electricity hospitals, schools, a university and an international airport.

Nonetheless, ethnic disparity in mortality among children could persist even in areas of comparable levels of development and physical environment. The cultural explanations of differences in mortality risks, therefore, places emphasis on childcare practices and reproductive behavior which are largely conditioned by cultural and traditional dictates. As Hill (1985) has observed the very different lifestyles of the different ethnic groups produces characteristic patterns of mortality and fertility even where the physical environment is roughly comparable between the groups. For instance, differences in pace of childbearing as indicated by length of inter-birth intervals; length of breastfeeding and in the practice of family planning especially use of modern contraceptives could account for ethnic differences in mortality. Therefore, differences in child survival between ethnic groups are likely to

mirror the regimen of reproductive behavior operating in each group. For instance, the *Kisii* have for long had one of the highest fertility rates in the country, which is attributed to inter-birth intervals averaging less than two years (Levine et al., 1996).

Other cultural practices, such as polygyny could also have a negative effect on child survival. Polygyny could negatively affect child survival through several mechanisms including little care about and less investment by men in the health of their wives and children and depletion of resources and income due to the need to support a greater number of women and children (Borgehoff, 1990, Oni, 1996; Strassman, 1997). Consequently, the health and survival prospects of children in polygynous unions are likely to be poorer. For instance, Borgehoff (1990) found that among the *Kipsigis* which is a *Kalenjin* sub-tribe, the presence of many co-wives had a negative effect on the surviving children in polygamous families.

Another possible explanation for ethnic mortality differentials is the pattern of disease related to sexual behavior. For example, in a Cameroonian study, (Kuate-Defo, 1992) found that Hauossa-Foulbe children had higher neonatal mortality than others due to a high incidence of venereal disease among their parents. On the other hand, mortality was lower in the rest of childhood due to reduced exposure to acute diseases and proper dietary practices. In Kenya, ethnic differences in the prevalence of HIV/AIDS could account for the large differences between *Luo* children on one hand and that of other ethnic groups. For instance, a surprising finding from the results relates to infant mortality differences between the *Kisii* and *Luo* who are the predominant ethnic groups in Nyanza province. Although the results could be pointing to differential child care practices, the part of Nyanza province

inhabited by the latter group suffers the greatest brunt of the HIV/AIDS epidemic (Kenya Ministry of Health, 2001; NCPD and MI, 2004). Among the *Luo* the widespread practice of levirate marriages has largely been attributed to the higher prevalence of HIV/AIDS. Additionally, this is also tied to cultural explanations for the cause of disease and death. The belief that HIV/AIDS is caused by witchcraft or breaking of certain traditional taboo could have been responsible for marrying of women whose husband have died of AIDS.

Also, mortality was higher in childhood among the *Luo* but the difference is much less compared to that of children *Kisii*, *Luhya* mothers. In childhood, the effects of AIDS in an area of high prevalence are largely indirect being reflected through the negative effects orphanhood. Studies in Kenya and elsewhere in sub-Saharan Africa indicate that AIDS orphans are malnourished and lack access to healthcare due to the inability of the extended family to cope with the high rate of orphanhood. Many of these orphans are also likely to be homeless, live in child-headed households and live in the streets where they face many dangers to their health (Nyambedha et al, 2003; Ainsworth and Semali, 2002). Besides differences in health behaviors regional climatic and ecological affect the distribution of diseases. For instance, the epidemiological and etiological setting of Rift Valley province home to the *Kalenjin* is less conducive to the spread of measles and malaria. On the other hand, areas in Nyanza province around Lake Victoria and in Coast province along the Indian Ocean are known to be conducive for malaria (Brass and Jolly, 1993; Mott, 1982; Mosley, 1983).

The risk of infant and child mortality was also found to be significantly associated with the period of child birth. Substantively, the results show that as compared to children

born before 1980, children born in the late 1980s and in the 1990s faced higher risk of death. For example, the risk of a child dying in childhood was 45 percent higher for children born in the 1985-1989 period and about twice as high for those born in the 1990-1994 and 1995-1998 periods. These results generally confirm what was observed in other studies in the country (e.g., National Research Council, 1993; Brass and Jolly, 1993; Hill et al., 2001). The higher risk of death in the 1990s coincided with the political changes that saw Kenya revert to multiparty democracy and the suspension of bilateral and multilateral aid. The withdrawal of multilateral and bilateral aid may have affected programmes such as immunization and maternal health which are largely dependent on donor funding. Additionally, declining government expenditure on health and introduction of user fees in healthcare due to implementation of structural adjustment programs may have further limited access to healthcare (e.g., Mwabu, 1995; Isaksson, 2001).

The results show that although there are significant associations between the risk of death and toilet facilities, the effect of source of drinking water was only significant in the bivariate models but not in the multivariate models. Household sanitation and source of water are indicators of unmeasured familial behaviours including standards of hygiene and cleanliness which have an impact on the health of children (e.g., Sastry, 1996; Jalan and Ravallion, 2003). As was discussed in chapter two, clean water supply improves the survival prospects of children by preventing the spread of water-borne diseases, improving personal through washing of hands and food preparation tools and reduction of water collection time which in increases time spent in food procurement and preparation which ensures the

consumption of less contaminated food (see, for instance, Esrey, 1996; Poppel and Heijden, 1997; UNICEF, 1997; Jalan and Ravallion, 2003). Nonetheless, since young children do not use toilets, the hygienic disposal of waste is equally important especially in breaking the faecal-oral transmission of pathogens (Esrey and Habicht, 1986). Moreover, previous research has suggested that excreta disposal is perhaps a much more important determinant of child health in developing countries than water quality, especially where the prevalence of diarrhea is high (see, Gubhaju et al., 1991; Mock et al., 1993; Esrey, 1996). The results of this study are, therefore, consistent with past research that has shown that the effect of toilet facility is more pronounced in childhood (e.g., Obungu et al., 1994; Woldemicael, 2000). In particular, mortality is likely to be higher when supplementary foods are being introduced to the child.

On the other hand, although the lack of significant association between the source of water and mortality were unexpected, the results are not entirely surprising. Previous research has also shown that the effect source of water may not be as important as sanitation (e.g., Esrey, 1996), while other research shows an opposite effect (e.g., Kanaiaupuni and Donato, 1999). In Mexico, for instance, Kanaiaupuni and Donato (1999) found that the effect of running water on child survival was negative, approximately 20 percent lower than the average. They argued that due to regular water shortages, families store water in various containers, some of which may be contaminated, to avoid high charges for household water delivery. Ultimately, in this kind of environment the effect of water on child health largely depends on conditions of use, such as whether families boil the water or undertake in

treatment of the water before using it. Additionally, what DHS and other similar surveys do not to ask and which could be important in understanding the water-health relationship are standards of hygiene and cleanliness maintained in the households because availability of water is directly linked these practices.

Although the bivariate analysis showed that the risk of death differed by the mother's migration status, no significant and consistent associations were found in the multivariate analysis. However, previous research has shown that a woman's migration has a strong negative association with child's risk of death. This relationship is usually explained through several pathways. For instance, differences in healthcare resources, community characteristics and the disease environment between origin and destination areas could also improve the survival prospects of migrant children (e.g., Brockerhoff; 1990; Sastry, 1996). Brockerhoff (1990) for instance attributed the improved survival prospects of children of rural-to-urban migrant women in Senegal to access to health services in urban areas. Migration, however, is not always beneficial to child health (e.g., Brockerhoff, 1994; 1995; Ssengonzi et al., 2002). The difficulties associated to adapting to a new environment and the disruption caused by the move itself are two factors that have been identified as being positively associated with child death among migrants (Brockerhoff, 1994; 1995; Tam, 1994; Brockerhoff and Brennan, 1998). The direction of effect of migration status in this study especially in relation to urban-rural and rural-urban migration tends to support the latter explanation. In addition, although movement from the rural to urban areas is associated with improved living majority of the rural-urban migrants live in poor, crowded and dilapidated dwellings in the slum areas of the towns and cities (Brockerhoff and Brennan, 1998).

However, children of rural-urban migrants are somewhat less likely to die in infancy as compared to those rural nonmigrants which could be attributed to better delivery care in urban areas.

The results have also shown that the risk of death varies by household socioeconomic status, but the effects are more important in childhood than infancy. Although the results of infant mortality are not surprising, they are unexpected because higher household socioeconomic status is associated with conditions that favour child survival even in infancy. For instance, higher socioeconomic status household will be able to provide expectant women with nutritious food, afford prenatal care and better delivery care (see, for instance, Wong et al., 1987). Again, despite attempts to link child mortality with household socioeconomic status, the results are largely inconclusive (see, for instance, Rahman, 1985; Pelletier et al, 1995). Pelletier and his colleagues (1995) found that in Ethiopia, the level of resources *per se* is not a reliable marker of child welfare and survival and observed that maternal use of time particularly the allocation of time on child feeding and quality care were more important. Where the overall health of the child is good resistance to disease and infection is high. However, the overall health of a child is a product of both good childcare and where they are available, a proper utilization of household resources including finances.

Although its effect was insignificant on child mortality, there were religious differences in the risk of childhood mortality. As compared to children of Protestant Christians, Muslim children were 77 percent more likely to die in infancy. Mortality differentials by religion may be related to differences in attitudes and beliefs on nature and causation of disease and death and childcare practices between religious groups (e.g.,

Gregson, 1999; Ogunjuyigbe, 2004). Another possible explanation relates to the low women status among Muslims (see, for example, Caldwell and Caldwell, 1993; Jejeebhoy and Sathar, 2001). Jejeebhoy and Sathar (2001), for instance, found that Moslem women in northern parts of the Indian subcontinent have less autonomy in relation to decision-making, mobility, freedom from abusive relations with husbands and access to and control over economic resources. Caldwell and Caldwell (1993) have also demonstrated that in Muslim areas child mortality is higher than would be predicted from income levels and health resources. They explain this as being partly a direct consequence of relative lack of mothers' autonomy which also leads to low maternal education. Therefore, in cultures where women have less autonomy the inability to make decisions regarding childcare and treatment, in particular, will have negative consequences on the survival of their children. This is particularly true in Coast province where the larger majority of Kenyan Muslim reside and an area known for low maternal education (NCPD and MI, 1999). Studies on child health have also found that Muslim children are likely to be more disadvantaged compared to those of other religions due to lack of knowledge and use oral rehydration therapy to treat diarrhea (e.g., Costello et al., 1996), which is a major cause of child deaths in developing countries (Kirkwood, 1991; WHO, 2002).

The findings on the relationship between birth order and mortality are consistent with expectation, its effect is more important in infancy. The results of our analysis are contrary to what has been observed in Bangladesh and Eritrea (See, for instance, Zenger 1993; Woldemicael, 1999), but consistent with O'toole and Wright (1991) findings in Rwanda. O'toole and Wright (1991) used birth order as a dummy, linear and nonlinear variable in

turns to assess its relationship with child mortality, but in all these analyses, birth order was found to be a relatively unimportant determinant of child mortality. Generally, however, the results of birth order show the adverse effects associated with increasing parity and would be consistent with the mechanisms discussed in relation with survival status of previous child and preceding birth interval.

Finally, there is significant variation in the risks of death between families. There are many reasons why deaths could cluster in families. For instance, Das Gupta (1990) argues that mothers differ in their basic childcare abilities which means that behavioral factors including feeding and may play an important role. Although the DHS contains information on healthcare and infant feeding behaviour including breastfeeding, this information is available only for children born within three years of the survey. Therefore, including these variables in the analysis will introduce significant bias in the results because this information is not available for a majority of the children. However, previous research on familial effects on infant and child death is inconclusive. For instance, Curtis and his colleagues (1993) found strong variation between families in the risk of death in Brazil, while Guo (1993) and Sear and her colleagues (2002) did not find strong family effects in Guatemala and Gambia respectively. On the other hand, Sastry (1997b) found that when controls were made for unobserved community effects, familial effects were insignificant, while Zenger did not find any familial effects in Bangladesh. As Sastry (1997b) has observed, where data used for analysis comes from relatively homogeneous populations the variation in unmeasured community characteristics across families is likely to be small. Additionally, the studies are done in countries and communities with diverse cultural and

socioeconomic conditions and the manner of effect of these factors will vary.

Consequently, in some countries there will be greater variation between families in the risk of death while in another the variation will be smaller. Evidently, child death clustering and its causes in different societies is an area that deserves further research.

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