Correlated Mortality Risks of Siblings in Kenya: An Examination of the Concept of Death Clustering and a Model for Analysis

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Abstract

This paper uses the 1998 Kenya DHS to examine the correlation of mortality risks across siblings. Previously, the random-effect parameter in the random model has been interpreted in terms of unmeasured and unmeasurable factors, suggesting the presence of death clustering. This interpretation is problematic because the concept of unobserved heterogeneity is not the same as death clustering. This paper attempts to clarify the concept of death clustering and demonstrates that the concept needs to be closely associated, and therefore examined, with the sequence of births and deaths in a family. Earlier approaches have been insensitive to sequencing both in the clarification of the concept and in the analytical methods used to examine the presence and extent of death clustering. Using binary sequence models that also incorporate unobserved heterogeneity we show that the parameter for death clustering is conceptually distinct from the parameter usually obtained for unobserved heterogeneity.

Key Words: Child death clustering, Correlation of child mortality risks, Determinants of death clustering, Kenya, sub-Saharan Africa.

I.   Introduction

Since Das Gupta (1990) suggested the concept of "death clustering", demographers have been preoccupied with understanding why deaths concentrate in certain families. Death clustering has been understood as a greater heterogeneity in the distribution of child deaths than would be expected if deaths were distributed randomly. Additionally, it has been viewed as what is left unexplained after the observed correlates are controlled, and is thus attributed to unobserved or unobservable genetic, behavioural and environmental factors related to mortality (Guo, 1993; Ronsmans, 1995; Sastry, 1997a; Das Gupta, 1997). It is also sometimes viewed as the correlation of survival outcomes among siblings.

Studies of childhood mortality in developing countries have largely utilised women's retrospective birth histories data from World Fertility Surveys and Demographic and Health Surveys. The main approaches to examine death clustering, therefore, essentially view it as a way of accounting for the correlated mortality risks of children in the same family (See, for example, Palloni, 1990; Zenger, 1993). In the literature, the major approach to the study of death clustering is to examine whether there remains a residual variation after accounting for observed determinants of mortality by using random effect models (e.g., Curtis et al. 1993; Madise and Diamond, 1995; Sastry, 1997b). The random-effect parameter is interpreted in terms of unmeasured and unmeasurable factors, suggesting the presence of death clustering. The problem with this interpretation is that the concept of unobserved heterogeneity is equated to the concept of death clustering, while these are by no means the same, besides the fact that the notion of unobserved heterogeneity can include any imaginable factors and conditions without identifying what exactly it is. Thus, there is a need to clarify the concept of death clustering itself. This paper attempts to do this and brings out the fact that the concept needs to be closely associated, and

therefore examined, with the sequence of births and deaths in a family. Earlier approaches have

been insensitive to this important idea, either in the clarification of the concept or in the

analytical methods used to examine the presence and extent of death clustering.

The sequences of births and deaths can be analyzed through binary sequence models that

can also incorporate unobserved heterogeneity. Using this model, we show that the parameter for

death clustering is conceptually distinct from the parameter one usually obtains for unobserved

heterogeneity. This study is different from previous studies of death clustering as the focus in

this line of analysis will be the child mortality experience of mothers. Considering death

clustering in the context of a woman's experience of a sequence of births and deaths is intuitively

appealing as the pattern indicated by women successively losing or not losing their children

suggests a non-random process. Thus, we show that binary sequence models are the most

appropriate models to study death clustering. Since the mother is the principal child carer, using

the mother as the unit of analysis could provide a better picture of the underlying socioeconomic,

environmental and behavioural factors associated with child mortality (McMurray, 1997). Also,

a mother-based analysis could provide information for health planners in a form that can be

properly interpreted in the formulation of health interventions. It is assumed that the mother

represents a family and that there was one woman selected from each household. In the case of

polygynous families, each woman and her children keep separate housekeeping arrangements.

Kenya offers an ideal setting in which to examine death clustering and the unobserved

family effect. In particular, its infant and child mortality and fertility profile is appropriate for

studying child death clustering. For instance, infant mortality in 1998 was 74 deaths per one

thousand births (NCPD et al., 1999). Although fertility has been declining over the years, the

1998 total fertility rate of 4.7 (NCPD et al., 1999) is still high. As Sastry (1997b) has observed,

our ability to measure the clustering of mortality risks is much greater in settings with high fertility and high mortality. Since it is the association among siblings' survival which permits the estimation of the family effect, a larger number of children per family and also a larger number of families will result in more precise estimates of the phenomenon of death clustering.

II.   Child death clustering: An overview

Studies that conceptualise the problem as an expression of unmeasured heterogeneity extend standard logistic regression techniques to allow for correlation by assuming that the level of mortality risk varies among families and follows a probability distribution. This assumption leads to the random intercept model which describes the probability of death, conditional on the random intercept, as a linear function of possible family- or community-level explanatory variables (Zenger, 1993). It is also possible to assume there are different levels in the data (See, for example, Curtis et al., 1993; Sastry, 1997a; 1997b; Kuate-Defo, 2001; Sear et al., 2002) or that coefficients of the explanatory variables also are random, thus producing a more complicated correlation structure (Zenger, 1993; Raudenbush and Bryk, 2002). In the case of survival analysis, models which include random effects are known as frailty models. Since the random intercept models assume an exchangeable correlation structure, all siblings are associated equally. A second approach considers child death clustering as a measure of excess observed deaths versus expected deaths (Das Gupta, 1997; Ronsmans, 1995). This approach uses count models to estimate the expected distribution of deaths from the observed child deaths in families. Count models assume that the probability of a child death is the same for all families in a given group and do not permit one to examine whether interfamily variation in mortality is related to the experience of close siblings (Zaba and David, 1996).

Studies that examine death clustering, especially in a multilevel framework, do not clearly demonstrate how unobserved heterogeneity measured by the random intercept is related to the concentration of deaths in certain families. Zenger (1993) has also demonstrated that death clustering may not be captured by random effects models in part because of the restrictive assumptions of the model. In particular, the number of children in many families is relatively small to yield significant variance which previous studies use as a measure of death clustering. A third approach which is used in this study accounts for the correlation of mortality risks by considering a woman's births and deaths as a sequence of events. This approach conditions the survival of younger children on the survival status of elder children (e.g., Zenger, 1993). In the subsequent pages of this section we present an overview of factors thought to be behind death clustering.

Death clustering is inherently implied in the survival status of preceding children, that is, the survival of subsequent children in the family depends on whether the family has previously experienced the death of a child. For instance, in her Bangladeshi study, Zenger (1993) found that familial association in the risk of neonatal death was strongest for siblings of adjacent birth orders. The effects of survival status of the previous child have been explained through the truncation of the interval to the subsequent birth, which is in turn associated with the maternal depletion syndrome that can lead to preterm and low birthweight births and pregnancy complications. Parents may also make deliberate efforts to replace the dead child, what has been called the replacement hypothesis (See, for instance, LeGrand et al., 2003; Gyimah and Rajulton, 2004). The death of a child could also lead to maternal depression thereby, compromising the health of the child both in the womb and in early infancy (See, for instance, Steer et al., 1993). It is therefore possible to isolate maternal depression from the other mechanisms because it is

independent of the birth interval.

Previous studies have found that death clustering is also associated with certain risk factors including parity, socioeconomic status, cultural factors, and child characteristics (See, for instance, Zaba and David, 1996; Das Gupta, 1997). In India, Das Gupta (1997) found that the variation between the observed and expected child deaths in the lower economic-status groups was greater than in the higher economic-status groups. Similarly, significant clustering was found among uneducated women than among educated women. The study suggested that unobserved factors associated with death clustering are more positively associated with socioeconomic conditions and education level.

Certain households may suffer from unusual adverse conditions such as insufficient economic resources, health conditions, or access to medical care. Siblings share the same household environmental conditions and hence, any risks associated with these conditions, such as lack of sanitation and poor water supply. Also, risks associated with family behaviour and child care practices including infant feeding, use of health facilities, and general standards of hygiene could be shared by all siblings (Curtis et al., 1993; Sastry, 1997b). Cultural practices in certain population sub-groups could also lead to a concentration of deaths in those sub-groups. For instance, in highly male-oriented and patriarchal societies, death clustering could be due to an attempt to remove girls through differential childcare (Das Gupta, 1997).

Death clustering is also likely to be more pronounced among women with higher parities (Zaba and David, 1996). However, besides shared genetic characteristic that could be operating in such situations, children in big families may face competition for resources and be more likely to suffer infectious diseases due to crowding in the household (Gribble, 1993; Desai, 1995). As family size increases, not only are family resources stretched increasing the risk of malnutrition,

but overcrowding makes contagious disease to spread faster (Aaby, 1992). Illnesses are also more likely to be fatal in the presence of malnutrition.

Death clustering has also been attributed to lack of 'maternal competence' in childcare. For instance, Das Gupta (1990) concluded from participant observation results that women who experienced multiple child deaths were often less resourceful and less organized in caring for their surviving children and running the household. In relation to child care, such women were poor at making effective home diagnoses of their children's symptoms and taking active steps to help them. Again, since the large majority of illnesses are handled within the home, less resourceful mothers remain at a disadvantage. However, the qualitative information related to childcare practices is often unavailable in the standard questionnaire used in surveys conducted in the developing world.

The concentration of deaths in certain families could also result from biological factors such as genetically determined frailty. However, previous research suggests that their role is limited (See, for instance, Guo, 1993; Sastry, 1997b). This is because genetic factors inimical to child survival are kept low by natural selection, which ensures that those who die young have no chance of passing on their unfavourable genes (Guo, 1993). However, since in social surveys genetic factors and other familial effects are not measured, it is difficult to uniquely identify which of the factors are responsible for death clustering. Other important biological factors include the tendency of certain mothers to have babies of low birthweight, or to suffer difficult deliveries, or lactational failure (Das Gupta, 1990; Curtis et al., 1993; Knodel and Hermalin, 1984).

III   Binary sequence models

The models presented here use a woman's births and deaths in a sequence to assess whether the survival status of elder children is related to that of the younger children. Almost all previous studies, excepting a few (See, for instance, Zenger, 1993) ignore the sequencing of births and deaths. However, examining mortality through sequence data could help us understand the mechanisms underlying death clustering. Since births and deaths are recurrent events, we make use of binary sequence models (Abbot and Tsay, 2000; Billari, 2001; Rajulton and Ravanera, 2001) to examine the presence of death clustering in families and at the same time control for unobserved heterogeneity.

The binary sequence logistic regression model arises from the standard binary logistic model, but is enhanced by incorporating random effects as well as lag effects. The analysis is undertaken using the freely available package SABRE (Software for the Analysis of Binary Recurrent Events) (Stott, 1999). For ease of interpretation, the resulting logit coefficients can be transformed by exponentiation to odds ratios. In a binary situation, the standard logistic model is of the form:

$$[1] \qquad P(y=0 \mid X_i) = \frac{1}{1+e^{\alpha+\beta x_i}} \; ; \qquad P(y=1 \mid X_i) = \frac{e^{\alpha+\beta x_i}}{1+e^{\alpha+\beta x_i}}$$

where $P(y=0 \mid X_i)$ and $P(y=1 \mid X_i)$ is the probability of not experiencing or experiencing an event as a function of observed covariates respectively, χ_i is a vector of covariates, while β is a vector of coefficients associated with. This is a fixed model which takes into account neither the sequence nor unobserved heterogeneity; the α also captures the influence of unobserved

variables and is constant for all individuals. Unobserved heterogeneity is introduced in the model by using a case-specific error term z_i. Including a case-specific error in Equation [1] yields a random effects model:

$$[2] \qquad P(y=0 \mid X_i, z_i) = \frac{1}{1+e^{z_i + \beta x_i}}; \quad P(y=1 \mid X_i, z_i) = \frac{e^{z_i + \beta x_i}}{1+e^{z_i + \beta x_i}}$$

For the purpose of identifiability, the z_i are assumed to be identically distributed with density function *f(z)* and independent of x_i, the observed variables included in the model. The density function *f(z)* describes the distribution of the unobserved heterogeneity and its distribution must be specified. For obvious reasons, there is no theory to guide the selection of an appropriate distribution for the density function *f(z)*. There is also evidence that results are unlikely to be sensitive to the distribution assumed for unobserved heterogeneity (See, for example, Rodríguez, 1994; Sastry, 1997a). In the specific case of binary outcome, one of the most useful distributions is a mixture distribution called logistic-normal. The package SABRE uses the logistic-normal distribution to measure unobserved heterogeneity (Drakos, 1997).

In addition to random effects, the binary sequence models can also incorporate lagged effects and Markovian effects (Drakos, 1997). Both are transitional models based on the Markov assumption that the probability of child death depends on the survival status of previous children. The number of previous births and deaths determining the transition probability is called the *order* of the Markov chain model. For simplicity, in this study we estimate a joint lag parameter which reflects the fact that immediate pairs of siblings are associated more closely than those farther apart.

The lagged binary model is of the form:

$$[3] \qquad g(y_{it}/.) = \frac{[\exp(\beta' x_{it} + v y_{i,t-1} + z_i + \varepsilon_i)]^{y_{it}}}{1 + \exp(\beta' x_{it} + v y_{i,t-1} + z_i + \varepsilon_i)} \quad .$$

Apart from the usual notations used above, the parameter v denotes the lag parameter. In practice, the lagged response is treated as another explanatory variable and its associated parameter v is regarded as an extra element of the β vector (Drakos, 1997). It measures whether the survival status of an elder child has an effect on the survival of the younger child. Thus, the lag parameter is a more meaningful and precise measure of the presence of death clustering. The scale parameter, z_i, is a measure of the effect of unobserved factors, capturing the heterogeneity in the sequence of deaths experienced by mothers.

Once the presence of death clustering has been established by the lag parameter, the Markov model can be used to examine the process that creates it. The Markov model estimates two simultaneous equations corresponding to the two survival statuses of the preceding child, that is, when the preceding child is dead or alive. The Markovian structure can be built into the standard binary logistic-normal model by specifying:

$$[4] \qquad p_0 = P(y_{it} = 1 / y_{i,t-0} = 0) = \frac{\exp(\beta_0' x_{it} + z_0 \varepsilon_i)}{1 + \exp(\beta_0' x_{it} + z_0 \varepsilon_i)} \quad \text{and}$$

$$[5] \qquad p_1 = P(y_{it} = 1 / y_{i,t-0} = 1) = \frac{\exp(\beta_1' x_{it} + z_1 \varepsilon_i)}{1 + \exp(\beta_1' x_{it} + z_1 \varepsilon_i)} \quad .$$

The parameters p_0 correspond to the probability that a woman's child survives on the condition that the preceding child is alive, while and p_1 is the probability that a women's child dies on the condition that the preceding child is dead. As before, β_0 and β_1 are vectors of parameter estimates, while z_0 and z_1 are additional scale parameters measuring the unobserved heterogeneity arising from the sequence of a woman's births and deaths net of observed explanatory variables.

IV.   Data and covariates

The 1998 Demographic and Health Survey (DHS) for Kenya used in this study was the third in the series of similar surveys undertaken in the country. The 1998 survey was similar to the 1989 and 1993 surveys carried out by the National Council for Population and Development and Macro International of USA within the framework of demographic and health surveys for developing countries. A key objective of the 1998 Demographic and Health Survey was to provide current information on fertility and childhood mortality levels, nuptiality, fertility preferences, awareness and use of contraception, use of health services, and knowledge and behaviours related to HIV/AIDS and other sexually transmitted diseases (NCPD et al., 1999).

The survey was based on individual interviews of women in the reproductive ages, 15-49 years and their partners in the sampled households. The survey successfully interviewed 7,881 of 8,233 eligible women from 8,380 sampled households yielding a response rate of 96 percent. Although the DHS has been undertaken periodically every five years since 1989, they are cross-sectional in nature because every time a new sample is drawn. Further details about the survey are available in the final report (NCPD et al., 1999).

Of the 7,881 women who were successfully interviewed in the 1998 DHS, 5,716 had

given birth to at least one child with the total number of children ever born at least one month

before the survey being 23,348. We use a cut-off of one month before the survey to ensure that

such children were exposed to at least the risk of neonatal death. An important issue that is not

well addressed in the literature is the distribution of number of children and deaths in families

which can be used as a benchmark for the existence of death clustering. For instance, families

with one child death, but five surviving children cannot be said to experience death clustering.

Also, families with one child cannot contribute to the understanding of death clustering.

Consequently, the analysis here is restricted to families with two or more siblings. The

descriptive results in Table 1 serve as a basis for determining whether there is need to undertake

a statistical analysis of death clustering. The table is interpreted in two complementary ways: the

percentage of children who belong to families with a given number of children and the

percentage of deaths occurring to families with a given number of child deaths.

Over 80 percent of the children belong to families with two or more children in the

sample; only about 5 percent of the children came from families with 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. 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; a

majority of families did not have a death.

[Table 1 about here]

The survival status of the preceding siblings is important in child mortality studies because it indicates higher risks in families where succeeding children die. Previous research shows that in the situations where a preceding child was dead, the risks of the succeeding child dying are heightened (See, for instance, Sastry, 1997b; Ikamari, 2000; Hinderaker et al., 2003). Additionally, this pattern of association has been suggested to be indicative of child death clustering (e.g., Gubhaju, 1985; Curtis et al., 1993). In this analysis an estimated statistically significant lag parameter will indicate that there is death clustering in families.

Besides the survival status of elder children, two other biodemographic factors, birth interval and maternal age at birth of the child, are included in the analysis. The inclusion of these factors and the analysis of their effect on child death is guided by the Mosley and Chen (1984) theoretical framework and previous research on child mortality and death clustering (See, for example, Zenger, 1993; Sastry, 1997; Rutstein, 2000). In the Mosley-Chen framework the determinants of child mortality are separated into distant (socioeconomic and socio-cultural factors) and proximate factors (biodemographic factors and household environmental conditions); the distant factors of mortality necessarily operate through the proximate factors to affect child survival. Biodemographic factors are mainly maternal conditions affecting the growth and development of the foetus during pregnancy and also determine the health status of the newborn child and its probability of survival especially in infancy (Mosley and Waters, 1996). Household environmental conditions are represented by the type of toilet facility reflecting the level of environmental contamination and which determines the transmission of infectious agents to children (Mosley and Chen, 1984). The socioeconomic factors considered in

this analysis are maternal education, migration status and household socioeconomic status, while ethnicity, religion and marital status and type of marriage represent socio-cultural factors. The latter shape and modify the economic choices and health-related practices of individuals according to the cultural traditions and norms of the society (Mosley and Chen, 1984).

Studies have demonstrated that children born within a short preceding birth interval face a greater risk of death than those born after a longer preceding birth interval (See, for example, Curtis and McDonald, 1991; LeGrand and Philips, 1996; Muhuri and Menken, 1997). In this analysis we make a distinction between children born less than 19 months, 19-35 months and 36 or more months after their immediate preceding sibling.

The age of the mother at birth of the child represents biological and physiological effects associated with childbirth. For instance, the immature reproductive systems of young mothers and primaparity and the depleted physiological system of older mothers due to repeated pregnancies makes them susceptible to pregnancy complications and bearing low birthweight babies (Trussell and Hammerslough, 1983; Miller, 1993; Alam, 2000). Young and old maternal age at birth is therefore associated with deleterious effect on child survival. To reflect the risk associated with early and late childbearing, maternal age at birth is categorised into five groups: below 20, 20-24, 25-29, 30-34 and 35 or more years.

Previous research in Kenya has found that toilet facilities are an important determinant of child survival (See, for example, Anker and Knowles, 1983; U.N., 1991, Omariba, 2004). However, toilet facilities on its own may not capture the complexity that constitutes household environmental conditions which could include treatment of water, bodily hygiene, food preparation and garbage disposal. This suggests that an indicator scale with several measures of environmental conditions may be more informative; this information, however, is not collected

in the DHS. There are three types of toilet facilities in this study; flush toilet, pit latrine and no

facilities.

A wealth of existing research shows that maternal education is the most influential

socioeconomic factor of 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). Education also

changes the traditional familial relationships regarding decisionmaking giving mothers a greater

say in childcare issues (See, for example, O'toole and Wright, 1991; Caldwell, 1986). In this

analysis we make a distinction between women with no education, those with primary education

and women with secondary and above education.

Studies that have examined the relationship between migration and child health show that

there is nothing intrinsic about living in urban areas that favours higher child survival. This

suggests that if safe water and adequate sanitation and health facilities are available in rural

areas, place of residence mortality disparities will be substantially reduced. The place of

residence reflects environmental influences that expose parents and women in particular, to

social, economic and cultural conditions that affect child care and rearing. This analysis

considers a woman's current place of residence and her childhood place of residence to

differentiate between rural- and urban- migrants and nonmigrants. Although a woman could have

moved several times before settling in her current place of residence, the focus in this study is on

lifetime migration because it is the best indicator of change of social status (e.g., Sastry, 1996).

In this analysis, we include ownership of household items, itself a proxy of amount of

disposable income, as a measure of household socioeconomic status. Wealthier households, as

indicated by the amount of disposable income they possess, are likely to access and afford

healthcare and provide better living conditions for the child (Mosley and Chen, 1984). The items

used to construct this variable are ownership of radio, television, refrigerator, bicycle,

motorcycle and car. Households are categorised into three socioeconomic status groups: 'low' if

the household does not own any of the items, 'medium' if the household owns either a radio or

bicycle, and 'high' if the household owns a television, refrigerator, motorcycle or car.

A major finding in mortality research in Kenya is the contrast between the *Kikuyu* and the

Luo, representing the extremes of low and high mortality respectively (Tabutin and Akoto, 1992;

Brockerhoff and Hewett, 2000; Omariba, 2004). The *Kikuyu* are not only considered to be more

educated and urbanized, they reside in Central province which enjoys higher socioeconomic

development. From a cultural perspective, ethnic mortality differences reflect differential

childcare practices including feeding, breastfeeding, birth spacing behaviour and use of health

services. There are about 40 ethnic groups in Kenya, but the DHS collected ethnic information

for only a few. Although the languages for some groups are somewhat distinct, the groups are

culturally similar and therefore, to achieve parsimony they are grouped together. The groups

included in the analysis are *Kamba, Kalenjin, Kikuyu, Kisii, Luhya, Luo, Meru, Mijikenda* and

Taita.

Religious groups propound certain values, ideals and desirable behaviour which could

impact demographic behaviour. As regards mortality, religious affiliation is an indicator of

people's beliefs and practices about the nature, cause and response to disease and death (Tabutin

and Akoto, 1992; Gregson et al., 1999; Ogunjuyigbe, 2004). Religious affiliation is included in

this analysis as a dummy with four categories; Catholics, Muslims, Protestants and No religion.

Previous research on the relationship between marital status or type of marriage and child mortality shows that the risk of death is higher among children of unmarried mothers (e.g., Ewbank et al., 1986; U.N., 1991; Kuate-Defo, 1996) and those of mothers in polygynous unions compared to children in monogamous unions (e.g. Mulder, 1990; 1992; Strassman, 1997). In sub-Saharan Africa, marriage continues to provide a sense of security and social support for women, which is likely to be associated with better child health (Kuate-Defo, 1996; Oni, 1996). For instance, widowhood and divorce have been suggested to have a negative effect on women's financial status because it removes the primary provider of the financial and other resources for raising healthy children (See, for example, Kuate-Defo, 1996; Basu, 2000).

Although mortality differences by type of marriage can be expected, previous research on the direction of effect of polygyny on mortality among children is inconclusive. Whereas some of the research shows that polygyny has a negative effect on child survival (e.g., Amankwaa, 1996; Mulder, 1990; Kuate-Defo, 1996; Strassman; 1997; Omariba, 2004), other research shows that polygyny has either a positive effect (e.g., Ukwuani, 2002), little or no effect on child survival (e.g., U.N., 1985; Ukwuani, 2002). The negative effects of polygyny operate through dilution of household resources due to the need to support a greater number of women and children (e.g., Desai, 1995; Strassman, 1997); through young and old ages at marriage and birth (See, for example, Oni, 1996); and favouritism by husbands in the allocation of resources. In contrast, polygyny positively impacts child survival through longer inter-birth intervals and breastfeeding (e.g., Amankwaa, 1996) and co-wife cooperation in childcare (Isaac and Feinberg, 1982; Omorodion, 1993). Marital status and type of marriage measures whether a woman was in a monogamous union, polygynous union or was single; the latter category includes the never married, widowed and divorced or separated women.

The description of the variables selected for this analysis is presented in Table 2. The birth interval and maternal age at birth of the child are not included because they are specific to children. However, the analysis showed that the mean interval between births was 34 months with a standard deviation of 15, while the mean maternal age at birth for all children was 23 with a standard deviation of 4.

[Table 2 about here]

V.   Results

Binary sequence models are now employed to examine child mortality and death clustering in Kenya. Two binary logistic models that incorporate unobserved heterogeneity are estimated; the first includes all the variables except maternal age at birth of child and the preceding birth interval, while the second adds these two as well. Next, three lagged response models are estimated; the first only estimates the lag parameter to show that there is death clustering. The second model includes all the variables except maternal age at birth of child and preceding birth interval, while the third includes these two as well. This analytical strategy is adopted because birth interval and maternal age at birth of the child are more closely associated with the survival status of the previous child, and could also indicate differential survival risks for children closer in age than for those farther apart. Finally, one Markovian model is estimated with all the variables of interest for each of the two previous child survival statuses, that is, when the preceding child is dead and when it is alive.

Table 3 presents the results expressed as odds ratios of the binary logistic models with a case-specific random error term and the lagged response models. Except for ethnicity, there are

no apparent changes in the effects of the other covariates between the random logit model and

lagged response model. The results in the lagged response model show that ethnicity retains a

significant effect on mortality risk, although the magnitude of effect is attenuated compared to

the random logit model. With the presence of the lag parameter ethnicity can be seen to be

related to death clustering.

[Table 3 about here]

The results of our models largely correspond to the pattern of relationships between the

risk of child death and the familial covariates observed in previous studies. Substantively, the

odds of child death are higher for women with no education compared to those with primary

education, for all ethnic groups compared with *Kikuyu*, for Muslims compared to Protestant

Christians and for women living in households without a toilet facility compared to those living

in households with a pit latrine. In contrast, the odds of child death are lower for women with

secondary and higher education compared to those with primary education, for urban

nonmigrants compared with rural nonmigrants and for women living in households with water

closets relative to those living in households with a pit latrine. The effect of migration status and

household socioeconomic status in the lagged response model, however, is statistically

insignificant.

The scale parameters in the random logit models of 0.78 and 0.74 are significant and

mean that even after controlling for the familial variables there are still unmeasured factors

which are likely to lead to differences in the risk of child death between families. The scale

parameter is a case-specific random error term and it measures the heterogeneity in the

experience of sequence of deaths and therefore suggests that there is a possibility of death

clustering. Since the analysis is at "family" level, it is also a measure of the effect of unobserved

family factors on child mortality. An important observation from these results is the change in

the magnitude of unobserved heterogeneity between the models in Table 3. There is little change

in the unobserved heterogeneity parameter between the first and second random logit model.

However, when a lag parameter is introduced as in the lagged response models, the scale

parameter is substantially attenuated, which suggests that in models which do not include a lag

effect, unobserved heterogeneity is overstated.

The results from the lagged binary models clearly demonstrate and corroborate the

descriptive analysis (Table 1) that there is significant death clustering experienced by families.

The lag parameters show that the mechanism underlying death clustering is related to the

survival status of the preceding child; that is, death clustering exists principally because survival

risks of immediate pairs of siblings are associated. These results demonstrate that the parameter

for death clustering is conceptually distinct from the parameter one usually obtains for

unobserved heterogeneity.

The lag can also be interpreted as an odds ratio since these are logistic models. The

results of the first model that includes no covariates show that families which have experienced a

child death are 2.6 (exp(0.95)) times more likely to experience another child death. Further,

controlling for socioeconomic and socio-cultural factors, such families are 2.2 (exp(0.79)) times

more likely to experience a child death. The lag parameter reduces from 0.95 in Model 1 to 0.79

in Model 2, which does not include maternal age at child's birth and preceding birth interval.

Since the lag parameter is regarded as an extra element of the β vector, its attenuation suggests

that maternal education, ethnicity, religion, marital status and type of marriage and type of toilet

facility are also significantly associated with death clustering. In the third model, which

includes preceding birth interval and maternal age at birth, the lag parameter reduces further, but

there is little change in unobserved heterogeneity. Controlling for all covariates of interest,

families which have experienced a child death are 1.9 times more likely to experience another

child death.

As it is, the lagged response model does not conclusively demonstrate how the survival

status of the preceding child is related to death clustering; only the Markov model can reveal this

process. Table 4 presents the results expressed as odds ratios of the Markov model corresponding

to the two survival statuses of preceding children. The scale parameter corresponding to the

Markov model for when the preceding child is dead is not significant, but the one for when the

child is alive is substantial and significant. These results, therefore, show that when the

preceding child is dead, death clustering is clearly evident and no more covariates are needed to

explain the risks of death.

[Table 4 about here]

The results of the Markov model show that when the preceding child is dead, maternal

education, ethnicity, marital status and type of marriage, birth interval and maternal age at birth

are significantly associated with the risk of child death. The pattern of relationship is also

consistent with the theoretical expectations. An important observation to make is that when the

preceding child is dead, the effect of short birth interval (less than 19 months) is insignificant,

while that of longer birth interval is highly significant. A longer birth interval, even when the

preceding child is dead, is associated with higher child survival. The results of this study are

consistent with previous research which shows that the death of the previous child removes the potential effects of sibling competition, disease transmission and physiological demands of lactation (Boerma and Bicego, 1992; Zenger, 1993; Manda, 1999).

Another important pattern is that revealed by maternal age at birth of child; the magnitude of effect of maternal age at birth is larger when the preceding child is dead. For instance, children born when their mother was aged 30-34 are about one and half-times more likely, while those born when their mother was aged 35 and above are more than twice as likely to die compared to those born when the mother was aged 20-24. In particular, maternal age at birth is different for each child, which suggests that the mortality risks of immediate pairs of siblings born under similar conditions are more likely to be correlated than for siblings farther apart in age. For instance, some children may be born when the mother is suffering some critical illness, or when the family is going through some economic stress.

Also, unlike in the other binary sequence models, the effect of ethnicity is not significant for most of the ethnic groups. Since the model corresponds to the specific case where the preceding child was dead, the results indicate that deaths are more likely to cluster in *Luo* and *Somali* families. Further, death clustering is more likely to occur in polygynous families and those in which the mother is not educated.

Significant associations are also noticeable in the model corresponding to when the preceding child is alive. Except for household socioeconomic status, all the other variables are significantly associated with the odds of child death. Also, except for migration status, the pattern of association is also consistent with the theoretical expectations. An important pattern of relationship is that revealed by ethnicity; the magnitude of effect of ethnicity is quite substantial when the preceding child is alive. For example, *Luo* mothers are about six times, while *Luhya*

mothers are three times more likely to experience a child death compared to *Kikuyu* mothers. These results suggest that the correlation of mortality risks among siblings is more likely to be associated with ethnicity.

An important finding from this model is that the effect of unobserved factors, as indicated by the scale parameter of 0.70, is still significant. As was discussed earlier, in addition to measuring the effect of unobserved factors, the unobserved heterogeneity parameter could possibly indicate the presence of death clustering in situations where immediate pairs of siblings are alive. This is because, although the immediately preceding child is alive, the previous-but-one sibling in the sequence could be dead. In order to capture the effect of the survival status of the previous-but-one sibling and to confirm positively that there is death clustering, would require one to estimate the second order Markov model or as many orders as the pairs of siblings in the data.

VI.   Discussion and Conclusion

In the current literature, the frailty parameter obtained in multilevel random models has been interpreted in terms of unobserved factors suggesting the presence of death clustering. Essentially, deaths are assumed to cluster in families due to unobserved factors captured by the heterogeneity term at different levels of data. This is however problematic because death clustering is conceptually distinct from unobserved heterogeneity. Further, employing a multilevel framework may not be useful because the models assume that children of the same family are equally correlated and the risks remain constant across time. This assumption may, however, not hold in situations where familial and maternal characteristics change over time

(Zenger, 1993). This paper proposed that binary sequence models which consider the births and deaths experienced by mothers are better suited to analyse death clustering because they reflect the fact that risk factors for siblings close in age are more alike than for those farther apart. Since they are logistic models, they capture the probabilistic mechanisms that give rise to possible death clustering.

The descriptive analysis showed that there is substantial death clustering in Kenya. For instance, 33 percent of the deaths occurred to 4 percent of the families with three or more child deaths. The binary sequence analysis not only confirmed these results, but it showed that death clustering arises because of the correlation in the risks of deaths between immediate pairs of siblings in a family's sequence of births and deaths. The results also show that in models that do not include the lag parameter, the effect of unobserved factors is likely to be overstated. In addition, they also include familial correlation of child mortality risks and are therefore not easily interpretable.

The analysis of factors that could be associated with death clustering shows that the effect of the other covariates remained unchanged from the model without the lag parameter (random logit model), but the effect of ethnicity was substantially attenuated. This attenuation indicates that ethnicity is acting as a proxy for familial correlation of mortality risks in the random logit model. But, the effect of ethnicity in the Markov model corresponding to when the preceding child was alive was quite substantial, suggesting again that ethnicity is related to the clustering of child deaths in families. In contrast, the results from the Markov model corresponding to when the preceding child is dead explicitly show that the deaths of immediate pairs of siblings are correlated and that since death clustering is captured, the unobserved heterogeneity term for this model becomes insignificant. This result suggests that familial factors associated with child

mortality change over time. This model particularly points to the possibility of death clustering in *Luo* and *Somali* families, in those with no toilet facilities, in polygynous families and those in which the mother has no education.

Although ethnic mortality differentials could be due to differential levels of socioeconomic developments between areas inhabited by the various groups, cultural differences in childcare practices and beliefs on disease causation and patterns of diseases could be more important. Regarding beliefs on disease causation and the pattern of diseases, the results on the higher mortality among the *Luo* compared to other tribes are particularly noteworthy. For example, although they both inhabit Nyanza province, the risk of child death in all models was consistently higher among the *Luo* than the *Kisii*. The high mortality among children of *Luo* mothers could be attributed to the high incidence of HIV/AIDS in this group (Kenya Ministry of Health, 2001) due to the prevalence of levirate marriage and the belief that HIV/AIDS is caused by witchcraft and/or breaking of certain traditional taboos (Ocholla, 1991). This is consistent with Hill's (1985) observation that the diverse lifestyles of different ethnic groups produce characteristic patterns of mortality and fertility even among groups living in similar physical environments. Also, among the *Mijikenda*, the first line of treatment for pregnancy related illness involves consulting traditional medicine men and witchdoctors, because such illnesses are attributed to spiritual causes (Boerma and Mati, 1989). Potential pregnancy complications may therefore go undetected until it is too late to save the life of the child and the mother. Nonetheless, the relationship between ethnicity and child mortality experience of families would benefit from further research especially examining ethno-cultural differences in childcare and health related behaviour.

Short birth intervals are consistently associated with higher odds of child death. In addition to the maternal depletion syndrome, short birth intervals increase the number of children of almost similar ages in the household. Consequently, this heightens susceptibility of these children to infectious diseases due to their physical proximity (Zenger, 1993; Alam, 1995) and leads to siblings' competition for household resources including individualized maternal care (Gribble, 1993). The effects of birth interval cannot be separated from those of the previous child survival status. Short birth intervals increase the risk of death and the death of an elder child also shortens the interval to the next birth. If the effects of preceding child death reflect birth spacing mechanisms, improving availability and use of contraception could reduce death clustering and overall risk of death. However, if the preceding child's death operates through the depression and replacement mechanisms, then the policy options are less certain.

Das Gupta (1997) has argued that if health workers use the occurrence of the first child death in a family as marker for targeting intervention and are successful in preventing subsequent deaths, child mortality could be reduced substantially. In Kenya, based on the number of families with two or more deaths, if such targeting was completely successful it could have resulted in a reduction in child mortality of almost three-fifths (see Table 1). In less developed countries it would not be politically feasible to target families for interventions purely on the basis that they lost a child. However, it would be easier to justify targeting uneducated women with health information and programs to improve childcare as a means to secure child survival and reduce death clustering.

The significant unobserved heterogeneity in the model corresponding to the when the preceding child was alive suggests that death clustering among such families is not adequately captured by the first-order Markovian model estimated here. A further area of research, which is

however limited in the currently available statistical software, would involve estimating the

second order Markov model or as many orders as the pairs of siblings in the data. As the results

suggest the lag effect is likely to diminish the farther back we move from a particular index child

(Zenger, 1993), because the mechanisms through which the survival status of the previous child

affects the survival of other children are stronger for children who immediately follow each other

For example, the effect of death of the first order birth on the death of the fourth order birth

could be smaller than its effect on the second-order birth. The results in this paper also suggest

that estimating all the lags in the data would reduce unobserved heterogeneity further.

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Table 1: Distribution of children and child deaths per family in Kenya, Demographic and Health Survey 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	----	----

Table 2: Percent Distribution of Covariates Used in the Binary Sequence Models, Kenya Demographic and Health Survey 1998

Variables	Percent	Number
Migration status		
Rural nonmigrant	75.1	4292
Urban nonmigrant	7.6	432
Urban-rural migrant	7.6	435
Rural-urban migrant	9.7	557
Maternal education level		
Primary	59.0	3373
None	16.0	917
Secondary or higher	24.9	1426
Household socioeconomic status		
Medium	56.7	3240
Low	28.7	1641
High	14.6	835
Ethnicity		
Kikuyu	17.9	937
Kamba	10.6	606
Kalenjin	16.4	1024
Kisii	7.4	422
Luhya	14.3	816
Luo	12.9	735
Meru	6.1	351
Mijikenda	8.1	463
Taita	3.5	201
Somali/Others	2.8	161
Religion		
Protestant	63.2	3612
Catholic	27.0	1542
Muslim	5.8	332
No religion	4.0	230
Marital status		
Monogamous marriage	67.2	3839
Polygynous marriage	12.7	725
Single	20.2	1152
Type of toilet facility		
Pit latrine	75.7	4329
Water closet	8.1	463
No facility	16.2	924

Table 3: Binary Sequence Models of Under-five Mortality in Kenya, Demographic Health Survey 1998

Variables	Random logit model		Lagged response model		
	Model 1	Model 2	Model 1	Model 2	Model 3
Migration status					
Rural nonmigrants [a]	1.00	1.00		1.00	1.00
Urban nonmigrants	0.76(0.16)*	0.75(0.16)*		0.86(0.17)	0.85(0.17)
Urban-rural migrants	1.15(0.10)	1.15(0.10)		1.16(0.10)	1.17(0.10)
Rural-urban migrants	0.96(0.13)	0.94		1.03(0.14)	1.03(0.14)
Maternal education					
Primary [a]	1.00	1.00		1.00	1.00
None	1.26(0.07)**	1.24(0.07)**		1.23(0.07)**	1.19(0.07)**
Secondary and higher	0.64(0.10)***	0.66(0.09)***		0.66(0.10)***	0.66(0.10)***
Socioeconomic status					
Middle [a]	1.00	1.00		1.00	1.00
Low	1.08(0.06)	1.08(0.06)		1.08(0.07)	1.08(0.07)
High	0.82(0.12)	0.83(0.12)		0.87(0.13)	0.87(0.13)
Ethnicity					
Kikuyu [a]	1.00	1.00		1.00	1.00
Kamba	2.16(0.14)***	2.16(0.14)***		1.74(0.15)***	1.75(0.15)***
Kalenjin	1.64(0.13)**	1.60(0.13)**		1.50(0.14)**	1.45(0.14)**
Kisii	1.92(0.16)***	1.91(0.15)***		1.77(0.16)***	1.78(0.16)***
Luhya	3.47(0.13)***	3.40(0.13)***		2.80(0.14)***	2.78(0.14)***
Luo	6.39(0.13)***	6.18(0.13)***		4.96(0.14)***	5.00(0.14)***
Meru	1.19(0.18)	1.22(0.18)		1.11(0.19)	1.13(0.19)
Mijikenda	1.86(0.18)***	1.86(0.18)***		1.57(0.18)**	1.57(0.20)**
Taita	2.01(0.19)***	2.03(0.19)***		1.77(0.20)**	1.78(0.20)***
Somali and Others	2.01(0.23)***	1.98(0.23)***		1.84(0.24)**	1.79(0.24)**
Religion					
Protestant [a]	1.00	1.00		1.00	1.00
Catholic	1.11(0.06)	1.11(0.06)		1.09(0.07)	1.09(0.07)
Muslim	1.49(0.16)**	1.48(0.16)**		1.38(0.16)*	1.40(0.17)*
No religion	1.01(0.16)	1.01(0.16)		0.93(0.17)	0.93(0.17)
Marital status					
Monogamous union [a]	1.00	1.00		1.00	1.00
Polygynous union	1.31(0.07)***	1.30(0.07)***		1.26(0.07)**	1.25(0.08)**
Single	1.25(0.08)**	1.26(0.08)**		1.21(0.08)**	1.24(0.08)**

Table 3 continued

Variables	Random logit model		Lagged binary model		
	Model 1	Model 2	Model 1	Model 2	Model 3
Type of toilet facility					
Pit latrine [a]	1.00	1.00		1.00	1.00
Water closet	0.71(0.18)*	0.72(0.18)*		0.73(0.19)*	0.74(0.20)*
No facility	1.39(0.07)***	1.39(0.07)***		1.23(0.07)**	1.24(0.08)**
Birth interval					
19-35 [a]		1.00			1.00
< 19 months		1.62(0.07)***			1.51(0.08)***
36 or more months		0.68(0.07)***			0.68(0.07)***
Maternal age					
20-24 [a]		1.00			1.00
< 20		1.42(0.07)***			1.32(0.08)***
25-29		0.98(0.07)			0.97(0.07)
30-34		1.12(0.08)			1.12(0.08)
35 or more		1.28(0.10)**			1.27(0.08)***
Lag			**0.95(0.08)***	**0.79(0.08)***	**0.64(0.08)***
Scale	**0.78(0.04)***	**0.74(0.04)***	**0.49(0.11)***	**0.54(0.07)***	**0.56(0.07)***
Deviance	13444	13279	10994	10527	10409
Sample size [b]	4617(22249)	4617(22249)	4617(22249)	4617(22249)	4617(22249)
Degrees of Freedom	24	31	1	24	30

Notes: [a]= Reference category; Standard errors are in parenthesis; Factor significance levels: ***=1% or better; **=5%; *= 10%; [b]= Number of children in parentheses.

Table 4: Markov Model of Under-Five Mortality by Survival Status of Previous Child, Kenya
Demographic and Health Survey 1998

Variables	Alive	Dead
Migration status		
Rural nonmigrants [a]	1.00	1.00
Urban nonmigrants	0.92(0.19)	0.64(0.39)
Urban-rural migrants	1.26(0.11)*	0.88(0.17)
Rural-urban migrants	1.11(0.15)	0.77(0.31)
Maternal education		
Primary [a]	1.00	1.00
None	1.15(0.08)*	1.24(0.12)*
Secondary and higher	0.66(0.11)***	0.75(0.21)
Socioeconomic status		
Middle [a]	1.00	1.00
Low	1.12(0.08)	0.93(0.12)
High	0.88(0.14)	0.90(0.29)
Ethnicity		
Kikuyu [a]	1.00	1.00
Kamba	1.81(0.17)**	1.15(0.38)
Kalenjin	1.41(0.15)*	1.39(0.34)
Kisii	1.86(0.18)**	1.27(0.40)
Luhya	3.13(0.15)***	1.41(0.34)
Luo	5.87(0.15)***	2.31(0.32)*
Meru	1.16(0.21)	0.84(0.50)
Mijikenda	1.44(0.21)*	1.81(0.42)
Taita	1.94(0.22)**	1.11(0.47)
Somali and Others	1.60(0.28)*	2.50(0.51)*
Religion		
Protestant [a]	1.00	1.00
Catholic	1.10(0.08)	1.09(0.13)
Muslim	1.54(0.19)**	0.87(0.31)
No religion	1.04(0.19)	0.68(0.33)
Marital status		
Monogamous union [a]	1.00	1.00
Polygynous union	1.23(0.09)*	1.33(0.13)*
Single	1.27(0.10)*	1.11(0.16)

Table 4 continued

Variables	Alive	Dead
Type of toilet facility		
Pit latrine [a]	1.00	1.00
Water closet	0.67(0.22)*	1.07(0.44)
No facility	1.26(0.09)*	1.07(0.14)
Preceding birth interval		
19-35 [a]	1.00	1.00
< 19 months	1.66(0.08)***	1.17(0.12)
36 or more months	0.74(0.08)***	0.52(0.16)***
Age of mother at birth of child		
20-24 [a]	1.00	1.00
< 20	1.23(0.09)*	1.41(0.16)*
25-29	0.94(0.08)	1.20(0.16)
30-34	1.05(0.09)	1.49(0.18)*
35 or more	1.04(0.12)	2.24(0.20)***
Scale	**0.70(0.07)***	**0.001(0.21)**
Deviance	10342	
Sample size [b]	4617(22249)	
Degrees of Freedom	60	

Notes: [a]= Reference category; Standard errors are in parenthesis; Factor significance levels: ***=1% or better; **=5%; *= 10%; [b]= Number of children in parentheses.