DEATH CLUSTERING IN FAMILIES IN KENYA: Implications For Child Survival and Maternal Health

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DEDICATION

I dedicate this thesis to my parents ***Eric and Clarah Khasakhala*** *for giving me the foundation that has led to this achievement; My son* ***Kwasi,*** *for his patience during my long absence at a crucial stage in his life and to my brothers and sisters for their love and support.*

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DECLARATION

The research that has culminated in this thesis entitled **"Death Clustering in Families in Kenya: Some Implications for Child Survival and Maternal Health"**, has been carried out by me under the supervision of **Dr. Arvind Pandey**, Professor, Department of Mathematical Demography and Statistics, International Institute for Population Sciences, Mumbai.

I further declare that this work is original and has not been submitted in part or in full to any other University/Institute for the award of any Degree/Diploma.

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I certify that the above mentioned declaration is true to the best of my knowledge

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ABSTRACT

A number of demographic studies in recent years have focused on the problem of death clustering or inter-family heterogeneity in child survival (Das Gupta, 1990, 1997; Guo and Rodriguez, 1992; Curtis, Diamond and McDonald, 1993; Guo, 1993, Zenger, 1993, Ronsmans, 1995; Zaba and David, 1996). Death clustering refers to the concentration of infant and child deaths among certain mothers and is a consequence of siblings sharing many of the same mortality risks. It has been noted that ignoring the effects of death clustering in the estimation of parameters of covariates of infant and child survival may bias the results. Obtaining unbiased estimates of parameters relating to various covariates is of great value to policy makers because of the need to distinguish real from spurious effects of various covariates in order to design appropriate intervention measures.

Context

This study examines factors affecting infant and child survival in families in Kenya without and with taking into consideration unmeasured and/or unobserved factors. The second aspect of the study has explored the implications of child survival on maternal health in Kenya. Death clustering can have considerable implications for reproductive health and child survival programmes. It is posited that the risk of infant and child survival is a function of known covariates and plus those due to unmeasured/unobserved factors.

Data and Methods

The data for the study is drawn from the Kenya Demographic and Health Survey (KDHS, 1993). The study has not utilized information from all the districts in which the survey was carried but rather only 15 rural districts. These are generally the larger districts in the provinces which show wide disparities in infant and child health and maternal mortality. These districts were also oversampled in the survey to allow for production of estimates for certain variables at district level.

The information used in the study are obtained from the woman's individual questionnaire schedule. The information was based on retrospective birth histories collected from woman aged 15-49 at the time of the survey. The eligible women for were identified through the household schedules. In all, 7952 women were identified as being eligible for the individual interviews and out of these, 7540 or 95% were successfully interviewed. The rural sample consisted of 6379 eligible women and out of these, 5370 women were drawn from the 15 focus districts. The quality of the data used was also assessed and was found to yield reasonable results.

A variety of statistical methods have been used in the analysis. These include frequency distribution and comparison of observed and theoretical distribution of number of deaths in families. Standard logistic and generalised logistic regression (random effects model) and loglinear models.

Results

The results of frequency and theoretical distributions indicate that there is some evidence of death clustering at every level of childhood mortality and that at any parity, there are women who contribute higher than average deaths to the mortality risk. A number of factors have been shown theoretically to influence infant and child survival at various ages. From the results of regression analysis, in the neonatal and postneonatal periods, the variables that appear to be highly associated with survival are non receipt of BCG vaccination, immunization against (polio, DPT, measles), prematurity, maternal education and length of preceding birth interval (postneonatal period). This therefore indicates that survival in these periods is influenced by both biological and behavioural factors. At infancy, biological, behavioural and ecological zone of residence factors such as, prematurity, immunization, breastfeeding and region of residence are found to be highly associated with survival in this period. Similarly in childhood, behavioural and household environmental factors as well as ecological zone of residence are highly associated with survival in this period. The effect of maternal age which was not important in the earlier periods, but becomes important at the childhood stage. This could be as a result of the inexperience of younger women in child rearing practices.

Further, it is observed that in the neonatal period, infants of women with no education have higher survival chances than those of women in the other educational categories while in the postneonatal period children of women who have not completed primary education have lower survival chances than for children of women in the other educational categories. The results do

not change when the generalised logistic regression model is used instead of the standard logistic regression.

The results of examining the relationship between child survival and maternal health indicate that most of the variables that are associated with low BMI (indicator of maternal health), are the same as those which were highly associated with infant/child survival namely high parity, region of residence, maternal education and use of health technology for intervention.

Conclusion

The indication of death clustering in families in Kenya, may be as a result of replacement and/or hoarding strategies of family building, however before controlling for other factors this may not be the sole explanation. In examining factors that have been shown to theoretically influence infant and child survival at various ages without and with inclusion of the random effects term, there is no evidence of excess variation in parameter estimates. This findings are however not consistent with those of other similar studies which used similar models but are consistent with some which used proportional hazards models.

The results of the present study does not invalidate the hypothesis of existence of death clustering in families, but calls for further research using appropriate study designs and data sets. If indeed some families experience higher than average risks of child deaths, then interventions should be more focused to target such families.

CHAPTER 1

GENERAL INTRODUCTION

1.1 Background and Rationale

One of the most intensely studied areas in population studies is infant and child mortality. The majority of these studies have concentrated mainly on assessing the major determinants of these outcomes. Analysis of the critical factors that influence child survival, protection and development is crucial for social and economic development as well as the health status of a nation (UN, 1991). Further, infant and child mortality rates have frequently been regarded as indices reflecting the degree of poverty and deprivation in a population (Hill, 1989). It is also becoming increasingly clear that child survival is intimately related to a woman's reproductive life; more specifically her health which in turn is linked with the number and timing of pregnancies she has ever had.

Studies have shown that an infant's risk of death is closely associated with birth order and spacing between two births (Trussell and Pebley, 1984; Hobcraft et al., 1985); that children with five or more older siblings and children born less than two years apart as well as those born to young and old mothers, face the greatest risk (Ashton, 1995). As a result, the improvement in the health of women and children has become an increasingly appealing rationale for family

planning programmes both for national governments and international donor communities (Demeny, 1987, cf. Pebley and Elo, 1989). For these reasons among others, reduction in infant and child mortality has long been a goal of population policy both globally and nationally.

Recent trends show that the infant and child mortality rates have declined considerably during the second half of this century throughout the world. According to United Nations' World Population Prospects (1994), the average infant mortality rate in the world was 156 infant deaths per 1000 live births in 1950 while it was 63 per 1000 live births in 1994. These global figures however tend to mask wide disparities between and within countries. In 1993, the average infant mortality rate for developing countries was 69 deaths per 1000 live births compared to 9 deaths per 1000 live births in developed nations (UNICEF, 1995). Similarly, within countries these disparities exist among different regions and sub-groups of the same population. For example, in Kenya, the geographical variations in the levels of child mortality between the different districts show a range of the order of 4 to 1 between the highest and the lowest Blacker et al., (1987). Thus, the understanding of the causes of infant and child mortality differentials between and within countries and sub-groups of populations has been desirable.

The International Conference on Population and Development (ICPD, Cairo,1994) program of Action noted that " Poverty, Malnutrition, decline in breastfeeding, inadequacy or lack of sanitation and health facilities were the important factors associated with high infant and child mortality" and need to be addressed with great concern (UN Programme of Action, Section 8.12). A number of studies have identified these factors as being crucial to the survival chances

of children. It has also been observed in some of these studies that, even after taking into consideration a variety of socio-economic, behavioural and biological factors, there still exists a residual variation within and between households and families which are not accounted for when routinely used statistical techniques are employed (Guo, 1993; Zenger, 1993). It is this residual variation in the risk of infant and child deaths between and within sub-groups and families in the same population that has accelerated the interest of population scientists in recent times. This has led to the questioning of the accuracy of estimates derived without taking into consideration the interrelationship of siblings' observations in their risks of mortality when studying determinants of infant and child deaths.

Recent studies have in fact shown that deaths tend to cluster in families which implies that siblings' observations to mortality risks are dependent on one another. The term "death clustering" has been used to examine whether the residual variation observed in the risks of child survival in families continue to persist once known determinants of mortality have been accounted for (Ronsmans, 1995). According to Ronsmans, the residual variation or unmeasured heterogeneity, can then point towards sources of heterogeneity which are not easily observable such as the genetic make-up of the family, its behavioural characteristics or the different ecological settings in which children of different families are socialized.

One of the earliest studies to demonstrate that siblings' survival experiences were likely to be clustered, was carried out by Das Gupta (1990) in Rural Punjab. In this study, it was found that child deaths tend to strongly cluster in families and that 12.6% of the families in the study

community bore the burden of 62.2% of all child deaths. However, an even earlier study by Meegama (1980) observed that 33 percent of reported child deaths in Sri Lanka Fertility Survey occurred to the 1.5 percent of mothers with multiple child deaths and 43 percent of neonatal deaths occurred to 3 percent of mothers who had experienced multiple neonatal deaths. He concluded that "it would seem.....child mortality rates are high because of the conditions in a small number of households, i.e, those in which a mother had lost two or more children.

Other recent studies have also examined the possible bias in the estimate of infant and child mortality determinants that assume that sibling data are independent. Guo (1993) has examined the possible bias in estimates produced by questionable assumption that sibling data are independent and using the multivariate life table model, estimated the unmeasured familial effects shared by siblings in the population of Guatemala. Similarly, Curtis et al. (1993) have used the random-effects logistic regression as another multivariate life table model to analyse the effects of the preceding birth interval on post-neonatal mortality in Brazil, after controlling for the correlates of survival outcomes between siblings; while Zenger (1993) has examined familial association of neonatal mortality and its relationship to birth spacing effects on mortality using transitional (Markov) random effects and marginal models for correlated data. In a more recent study, Ronsmans (1995) has conducted a detailed simulations exercise to identify the role of within and between family heterogeneity in the risk of child death in a rural area of Senegal, while Curtis and Steele (1996) have examined variations in the strength and structure of familial association in neonatal mortality risks in four populations using standard methodology for all the countries (random effects model and pairwise odds ratio).

In a study by Madise and Diamond (1995), the socio-economic and demographic factors influencing infant and child mortality in Malawi were examined using the logistic binomial analysis. The main purpose of this study was to identify the socio-economic factors which predict infant mortality and in particular to assess whether there was evidence for clustering of mortality risks between siblings. The findings reveal a strong familial correlation of mortality risks during both the neonatal and post-neonatal periods but the effect of geographical area of residence was stronger in the post-neonatal period.

The proliferation of recent studies on causes of familial association in their risks of child survival in different societies and the use of more refined statistical techniques to elucidate the findings, indicate the importance of these aspects on studies of the determinants of infant and child mortality differentials. And it re-emphasises the view that risks of dying are never equally spread in populations or families. There are two main issues in these studies: one is why families differ in their risks of mortality experiences; and the other which is methodological in nature is how to detect and account for the observed differences. Available studies point out that the results of their research should not be generalised to other populations but have suggested that the causes of familial association in the risks of child survival in different societies require further investigation. Consequently, the need to build dependence between family observations induced by familial clustering of deaths into models, was identified by Trussell and Menken (1984) as a research priority.

1.2 The Study Problem

Kenya as a country located in Sub-Saharan Africa, has recorded tremendous declines in overall infant and child mortality levels since 1948 (i.e, from a rate of 262 deaths per 1000 live births in 1948 to 105 deaths per 1000 live births in 1989; the infant mortality rate currently stands at 62 deaths per 1000 live births). There are wide geographical variations in the risk of child survival, with some provinces showing very worrisome trends. This implies that certain regions and sub-groups of the population are subject to higher risk of infant and child mortality than others. The regions which display high infant and child mortality rates are also those with high maternal mortality rates in the country (UNICEF/PSRI, 1994).

Thus, if Kenya is not to lose its recent gains in reducing infant and child mortality rates, it is imperative that the causes of regional and sub-group familial disparities be identified. With such focus, appropriate interventions at regional and family levels will be easier to devise, promote and implement. Further, heterogeneity in child survival risks can have considerable implications for reproductive health and child survival programmes. For example, in many developing countries health services are made available largely in response to demand. Hence, if child deaths are heavily concentrated in families, this would suggest that substantial improvements in child mortality could be achieved by adopting the more cost-effective techniques of focusing on the sub-group of families with a high risk of child deaths (Gupta, 1997).

This study is therefore an attempt to examine the extent of familial association in the risks of

infant and child deaths and their implications on the determinants of infant and child survival. The second aspect of the study is to explore the implications of child survival on maternal health in Kenya. It has been argued that the health of women and children are intimately interlinked. Healthy mothers produce healthy babies and if a mother is healthy, she can effectively look after her child with vigour and competence.

1.2.1 Study Objectives

The present study addresses the problem of clustering of infant and child deaths in families in rural Kenya and their implications on child survival and maternal health. The specific objectives are to:

(a) assess the extent of clustering of infant and child deaths in families in Kenya at neonatal, postneonatal and child mortality levels.

(b) identify factors affecting infant and child survival in Kenya without and with accounting for excess variation in child deaths.

(c) examine the implications of child survival on maternal health.

1.2.2 Hypotheses

The study intends to test the following hypotheses:

1. Deaths are clustered in families;
2. When deaths are clustered, there are significant differences in estimates of

determinants of infant and child mortality if routinely statistical techniques are used.

1.3 Review of Literature and Conceptualization of Study

1.3.1 Introduction

There is no general theory concerning the determinants of mortality and the mechanisms through which these determinants operate (UN, 1991). Yet a number of viewpoints have been expounded by researchers in an attempt to explain the determinants of mortality decline and its effect on population growth in the twentieth century both in the developed and developing countries. The most comprehensive exposition was by Mckeown (1976) who argued that the majority of the acceleration in the world population resulted more from mortality decline than a rise in fertility. The decline in mortality is said to have been occasioned by a reduction of deaths from infectious diseases, which in turn was brought about by introduction of hygienic measures such as purification of water, efficient sewage disposal and improved food hygiene particularly with respect to milk.

Other subsidiary reasons for mortality decline was the substantial decrease in proportion of deaths from two non-infective causes i.e, infanticide and starvation. The decline in mortality from these two causes is said to have occurred as a result of improved medical technology, contraception and nutrition. McKeown therefore attributed mortality decline to improved public

health measures and improvements in medical technology as well as nutrition. Some researchers have, however, attributed the decline in mortality in the developing countries during the same period to improvements in social and economic developments resulting from social policy measures (Preston, 1980). This viewpoint stems from the argument that major technological changes that occurred (e.g, immunization against a host of infectious diseases, vector eradication, chemotherapy etc.) had to be embodied in social programs in order to affect the majority of the people in less developed countries. These approaches to mortality decline and the accompanying population growth tended to polarize the determinants in either social (Preston,1980) or medical (Mckeown, 1976) groups. As pointed out by Mosley and Chen (1984) "traditionally, social science research on child mortality focused on the association between socioeconomic status and levels and patterns of mortality in populations, while medical research focused primarily on the biological processes of disease. The differing concerns and methodologies have compartmentalized such knowledge and constrained the development of useful approaches to understanding child survival".

Mosley and Chen (1984) proposed a new analytical framework aimed at reconciling the previous approaches and which revolutionalised approaches to the study of child survival by combining the social and medical phenomenon. Consequently, studies on infant and child mortality have been advanced by development of frameworks that specify the biological and behavioural mechanisms through which social, economic, cultural factors etc, operate to produce the survival of a child to age five (Mosley and Chen, 1984; Venkatacharya, 1985, Mosley, 1985). Others have developed frameworks specifically for the analysis of infant mortality (Jain, 1984) and on

the relationship between child survival and pace of child bearing (Pebley and Elo, 1989).

Mosley and Chen's Framework is one of the earliest which identified five groups of proximate or intermediate determinants of child health at an aggregate level: factors related to the mother (age, parity, birth interval); environmental contamination; nutrient deficiency; injury and personal illness control. It posited that all these were influenced by socio-economic determinants which included (1) individual level variables (individual productivity as measured by education and occupation and traditions, norms and attitudes); (2) household level variables (income, wealth); (3) community level variables (ecological setting, political economy, health system).

This framework crystallizes the implicit assumption in the literature that variations in mortality between households within a given community (where households are subject to the same community level conditions and are influenced by the same culture) is explained by differentials in socio-economic factors such as education, occupation, income and wealth. This implies that any residual variation in the household is what statisticians refer to as 'white noise' or a nuisance (Zenger, 1993). However, this residual can no longer be dismissed as 'white noise' with evidence of death clustering in families from recent studies. On the other hand Jain (1984), distinguished between factors operating at the village level, household level and individual level. The individual level factors which are of interest to this study were further divided into six categories according to the timing of their relevance, i.e, prenatal, natal and postnatal and type of medical care, i.e, preventive and curative. Jain argued that timing and type of care were the two important ingredients of individual level factors affecting infant mortality. He further

observed that maternal factors (age, parity and birth intervals) would influence infant survival 'through' their effects on maternal health and nutrition and could affect infant mortality indirectly through another set of factors namely quality of child care. Jain's framework included the element of timing of infant deaths which was not catered for in the Mosley and Chen framework. The framework by Venkatacharya (1985) was aimed at studying the process of infant and child morbidity and mortality. The framework attempted to systematize the factors which affect the 'life process of an individual' focusing on the survival of children from birth to age five. It was based on the premise that human populations were heterogenous with respect to risk of mortality. The heterogeneity over time and space can stem from gender, nutrition, genetic endowment, socio-economic level, education level, environment etc. These factors interact with the curative and preventive interventions leading to differences in the structure of the causes of deaths as earlier discussed by Preston and Nelson (1974).

In recent times, given the availability of data to which researchers can link women's reproductive histories with the health and survival status of individual children, there is renewed focus on the association of inter-birth intervals with poor child health and survival. Pebley and Elo (1989) have developed a framework which shows the principal relations between birth spacing and child health. The relationships are based on the premise that potential linkages between fertility patterns and child health are complex and may operate in both directions, i.e, child mortality can affect the timing of the next birth but the timing of the next birth may also affect child health and survival prospects.

However, in all the frameworks, there is an implied relationship between maternal health and child survival and child survival and maternal health. McCarthy and Maine (1992) have developed a framework for analysing determinants of maternal mortality which includes most of the variables for studying child survival. The framework outlines the cultural, social, economic, behavioural and biological factors that influence maternal mortality. The main conclusion underlying this framework is that all determinants of maternal mortality (or efforts to reduce maternal mortality), must operate through a sequence of only three intermediate outcomes namely: (1) reduce the likelihood that a woman will become pregnant, (2) reduce the likelihood that a pregnant woman will experience a serious complication of pregnancy or childbirth or (3) improve the outcomes for women with complications. The following section presents a review of the literature including some results of studies undertaken using modified versions of some of these frameworks with a view to coming up with a suitable framework for this study.

1.3.2 Factors Associated with Child Survival and Maternal Health

The numerous factors influencing infant and child survival and maternal health can be classified into socio-economic, biological and behavioural, and specific studies are outlined as under.

Socio-Economic Factors:

One of the most studied socio-economic aspect that has shown influence on child survival, is

maternal education. The influence of maternal education as a key factor affecting child survival has been discussed by a number of authors beginning with Caldwell (1979). The majority of these studies have tried to find pathways through which maternal education operates to influence child survival. As a result, there is considerable evidence of strong inverse association between level of maternal education and child survival but some studies have found no association at all (Amankwaa, 1996). Maternal education has therefore been seen as a crucial factor in behaviour change that may in turn influence survival chances of infants and children. However, there has been no consensus on the pathways through which maternal education exerts its influence.

Caldwell (1979) and other researchers who have studied the influence of maternal education on child survival, argue that it acts through a number of pathways to enhance child survival. These include a shift from fatalistic acceptance of health outcomes, to the implementation of simple health knowledge (Caldwell, 1979; Farah and Preston, 1982; Pebley and Stupp, 1987; Bicego and Boerma, 1991); an increased ability to manipulate the modern world, including interaction with medical personnel. This may however not be true in all communities. In a recent study by Sodemann et al., (1997), it was found that, despite timely health seeking behaviour by mothers of sick children, infant and child mortality was still high in the study community due to poor health care management in health institutions. In such instances maternal education may not influence survival chances, but rather it may act to enhance the ability to pay for the services and the interaction with health care providers in the community.

Another pathway suggested by Caldwell (1979) is the shift in familial power structures which

allows educated women to exert greater control over health choices for her children. Hobcraft (1984) and Mensch (1985) suggested that the husbands socioeconomic characteristics (especially education) were more strongly associated with improved child survival in Sub-Saharan African countries. Lindenbaum (1990) stressed the apparent role of cleanliness among educated women in explaining differentials in child mortality in Bangladesh, while Cleland (1990) argued for the role of education in ensuring utilization of health care services for children. Some studies have argued that maternal education is associated with greater emphasis on child quality implying that fewer children are more likely to survive, have more food and human capital investments, and thus evolve as higher quality citizens (i.e. being healthier, better educated, more affluent and emotionally better developed (LeVine et al., 1991). One thing that has not been determined is the threshold level of education which is required to be reached before the benefits for child survival begin to accrue. Hobcraft et al., (1984) and Mensch, (1985) have noted that even a small amount of education is usually associated with improved chances of child survival and that gains generally increased with increasing levels of education.

Results from a follow-up study of Filipino children in the Cebu area indicated that one-year increment in maternal education increased the chance of utilizing preventive health service use by about 4% during any month in the first year of life (Cebu Study Team, 1991). Similarly data from a small scale study in Indonesia indicated that educated women had greater awareness of correct immunization schedules (Streatfield et al. 1990).

Some studies on the maternal education-mortality nexus have shown, however, that the education

advantage does not influence child survival at all ages. Hobcraft et al., (1984) using direct estimation procedures that allow for age-specific analysis, found that net education effect was much stronger during the 1 to 5 years of age segment than during infancy. Out of 24 countries used in the analysis, education effect remained statistically significant for the neonatal and postneonatal mortality periods after inclusion of economic controls in only 9 countries. Similarly, Rutstein (1984), using WFS data of 41 countries showed that the education advantage (with few exceptions) was more pronounced after childhood as opposed to during infancy. Also Bicego and Boerma (1991) using data of 17 DHS surveys (conducted during 1987-90), looked at the maternal education effect on age pattern of mortality in childhood. They found that there were significantly elevated risks of dying throughout the first two years of life associated with low levels of mother's education. This association was more pronounced during the postneonatal period where the risk of death was more than twice as sensitive to the education effect than to the neonatal period. This could perhaps be explained by the fact that neonatal mortality risk is more linked (than postneonatal risk) to adverse effects of pregnancy outcomes such as intra-uterine growth retardation, prematurity and obstetrical trauma as well as exposure and susceptibility to the tetanus pathogen, i.e, outcomes that are modified by medical technology (Bicego and Boerma,1991).

With regard to maternal ill-health, it has been postulated that education (through its association with late age at marriage or increased use of contraceptives within marriage) is likely to be associated with lower fertility and hence with fewer pregnancies. Education can also be associated with the development of fewer complications among pregnant women if better-

educated women in general are in better health than others before and during pregnancy. And that education could be associated with a greater likelihood of receiving appropriate care for complications that may arise given that better educated women might also be better informed about the symptoms of complications and could therefore be more likely to make a timely decision to seek care (McCarthy and Maine, 1992). Unfortunately, no empirical evidence exists to support these views. Further, maternal education-child survival relationship has been found to be weak in sub-saharan African countries (Hobcraft, 1993) but the reasons for this weak relationship has so far been mere speculation.

A major theory on linking increased education of the mother and child survival is that education gives women the power and the confidence to take decision into their own hands (Ware, 1984). Pebley and Stupp (1987) argued that mother's with some education may be able to avoid some of the potential risks associated with short birth intervals. Considerable evidence also exists on the education-conditioned use of health services. It has been noted that the better is the education of mothers, the more common would be the use of maternal and child care services. This could influence both child survival and maternal health.

Bio-Demographic Factors:

Studies have documented increased health risks for mothers and children associated with family formation or bio-demographic factors, i.e, maternal age, birth order or parity and birth interval (Pebley and Stupp, 1987). The evidence pertaining to the relationship between these factors and

family health is reflected in the outcome of pregnancy, in the health and development of children and in the health of mothers. It has been argued that maternal ill-health in pregnancy is responsible for a substantial proportion of infant deaths in developing countries and is considered to produce three overlapping ill-effects namely: foetal malnutrition, premature birth and foetal infections (Jelliffe and Stanfield, 1978, WHO, 1994). Similarly, too early, too many and too late births have been associated with maternal ill-health and subsequently death (Maine, 1981). Thus, many of the reproductive variables that affect infant and child survival (e.g. maternal age, birth order or parity and birth interval), also influence the outcome of maternal well-being.

The World Health Organisation noted that around 8.1 million infants die each year, one third of them within the first month of life and a large proportion a few days of birth. The majority of the deaths occur as a result of the complications during pregnancy and child birth. It has also been observed that since shorter birth intervals and higher risk of repeat infant death occur in families that have experienced one early infant death, the death of a second child after a short interval may not be due to the interval but other factors that cause the first death and the shortened interval (Winikoff, 1983; Pebley and Elo, 1989; Miller et al., 1992). This implies familial association in the risk of infant and child mortality which may be as a result of maternal ill-health.

In the study by Miller et al. (1992), it is noted that in both Bangladesh and the Philippines, women who reach high parities tend to be those who have experienced greater than average rates of infant and child loss. In Bangladesh, for example, the mean proportion of previous children

dead increased from 0.16 among women of parity 1 to more than 0.32 among women of parity 9 and above. The corresponding proportions for Philippines (Cebu) were 0.05 and 0.14 respectively.

The importance of family formation or bio-demographic variables and their association with infant and child mortality and maternal ill-health has continued to be a subject of interest to demographers and related fields. The relationship has been explained through the maternal depletion hypothesis, sibling competition, impaired lactation as a result of poor health and nutritional status of the mother and transmission of infections as well as lack of time to attend maternal health care services. The above pathways operate through the socio-economic status of the family, region of residence, behavioural factors and bio-demographic factors to influence child survival and maternal health. It has been shown that changes in reproductive patterns can influence child and maternal health through a number of different mechanisms. The most notable, being changes in maternal age at childbearing, birth order or parity and/or the interval between births (Pebley and Stupp, 1987; Koenig et al., 1990; Boerma and Bicego, 1991; Miller et al., 1992). There is abundant literature on the effects of these variables on child survival and maternal health (Hobcraft et al. 1983; 1985; Cleland and Sathar, 1983; Bongaarts, 1987; Trussell and Pebley, 1984; Potter, 1988; Trussell, 1988).

The focus on differentials in child survival prospects by mother's age, parity or birth order and spacing of pregnancies is based on what Potter (1988) calls simple physiological mechanisms, i.e., age and parity are supposed to be related systematically to fitness to reproduce, and birth

intervals are thought to influence child survival in part through their effects on mother's nutritional status. The apparent J- or U- shaped association of maternal age and parity with the risk of child mortality has long been recognised. The risks are highest among very young and older mothers and at the first and highest parities. Pebley and Stupp (1987) noted that higher mortality among children born to older mothers may result from a decline in the efficacy of the reproductive system with age. Other indications of this decline were lower fecundability and higher rates of foetal loss (associated with a higher frequency of chromosomal abnormalities (Leridon, 1977). Conversely, high mortality at very young ages may occur because the reproductive systems have not matured sufficiently to manage a birth. One of the consequences of young age at birth associated with maternal ill-health or disability is vesico-vaginal fistulae. This is more common among very young mothers who are more likely to experience prolonged labour as a result of premature pelvises, while high parities influences uterine prolapse as a result of frequent births (McCarthy and Maine 1992).

Results from studies carried out in the eighties using mainly the World Fertility Survey (WFS) data and multivariate analyses, suggest that in developing countries the apparent effect of high parity on child mortality may actually be due to the correlation between parity and other factors such as maternal age, child spacing and socio-economic status (Hobcraft et al. 1984; Cleland and Sathar, 1984). However, results from WFS did not show very sharp disadvantages for child survival associated with higher parities or with births to mothers aged over 35 (Hobcraft et al., 1985).

Trussell and Pebley (1984) drew results from five studies which used mainly WFS data, to explore the association between changes in reproductive behaviour if effective family planning program in developing countries were initiated and the effects they would have on child survival and maternal mortality. The changes considered were the alteration of ages at which women have their children, reduction of the proportion of births that occur at higher parities and the lengthening of the interval between births. The results indicated that if child bearing was limited to the 'Prime' reproductive ages of 20-34, infant and child mortality rates would fall by 5 percent and if limited to the age range 20-39, it woould also reduce maternal death ratio by 11 percent. The elimination of fourth to higher order births would reduce infant and child mortality by 8 percent and the elimination of fifth and higher order births would reduce the maternal ratio by 4 percent. Universal adoption of an 'ideal' birth spacing pattern in which all births subsequent to the first, were spaced at least two years apart, might reduce infant mortality by 10 percent and child mortality by 21 percent. Although Bongaarts (1987) and Potter (1988) have questioned these findings, this does not however nullify the effects of these factors on infant and child survival and maternal health.

Birth Spacing

Recent research has focused on the issue of the pace of childbearing more than on maternal age, birth order or parity. Analyses based on WFS data and more recently on Demographic and Health Survey (DHS) data have made a strong case for the increased mortality risks for children with short birth intervals (Hobcraft et al, 1985; Pebley and Millman, 1986, Boerma and Bicego,

1991). However, the actual biological or behavioural mechanism through which the birth spacing-child survival operate are still a subject of research. Some of the possible pathways of influence which have been cited as being responsible for the adverse effects of short birth intervals on child survival, relate to the maternal depletion syndrome, mentioned earlier. It has been argued that women with short birth intervals between two pregnancies, have insufficient time to restore their nutritional reserves and thereby affect foetal growth. The effect of birth interval on child survival, varies with age. The effects of intra-uterine growth retardation and prematurity which lead to low birth weight babies, are felt more in the prenatal period. In the case of maternal depletion syndrome hypothesis, Pebley and Elo (1989) have observed that short intervals since previous pregnancy as well as short intervals since previous live birth may be important because women may require time to recover from non-live births especially those of long gestation.

In the study by Palloni and Millman (1986) for 39 countries of WFS, it was found that the median relative risk of dying was higher for children with preceding birth intervals of less than two years compared to children born after longer intervals in all ages, i.e, 58 percent higher in the neonatal period, 96 percent in the postneonatal period, 45 percent during the second year of life and 30 percent higher during 2-5 years of age. Similarly Boerma and Bicego (1991) using data for 17 DHS countries and using the logistic regression model observed that short preceding birth intervals (less than 24 months) increased the mortality risk during the neonatal period by 98 percent. They however noted that part of the excess risk could be due to prematurity. Thus, controlling for the survival status of the previous birth reduced the excess risk from 98 percent

to 88 percent. The observed decline suggested that part of the association between short birth intervals and neonatal mortality was due to increased intra-familial mortality risks.

Recent studies have begun to look more closely at the relationship between birth interval and infant and child mortality and how they can be affected by familial clustering of deaths (Curtis et al. 1993; Zenger, 1993) using statistical models that incorporate familial correlation. The assumption here is that the survival outcomes of siblings are correlated. Biologically, some mothers experience more problems in pregnancy than others. These problems are likely to be repeated in other pregnancies (Majumder, 1990; Curtis et al., 1993). Further, siblings share the same environment and consequently any risks associated with that environment. They also share risks associated with family behaviour and child care practices relating to infant feeding, use of health facilities and general standards of hygiene. Thus, with regard to birth intervals-infant mortality relationship, Curtis et al. (1993) have argued that it is possible that some aspects of family behaviour affect both child survival and length of birth-interval which consequently leads to the selection of children from high risk families to short birth-interval categories. Similarly, Zenger (1993) found that after a death, the odds of the next child dying, are greater at all interval lengths than the odds of dying after a survivor, indicating that sibling's deaths are correlated. This was in reference to neonatal mortality risks in Bangladesh.

However, the above studies did not control for the effects of prematurity on interval length and child mortality. Miller et al. (1992) using data from Bangladesh and Philippines, controlled for the prematurity effect and observed that children born shortly after a preceding birth interval of

less than 15 months, are 60-80 percent at greater risk of dying during the first two years of life in both countries. They concluded that the effects of gestation are substantial and as such they should be taken into consideration in adjusting estimates of the 'spacing effects' from models in which birth interval is used as the measure of spacing. However, information on gestational age is usually not available in most survey data.

Behavioural Factors

Apart from the biological mechanisms mentioned above which operate mainly in the pre-natal period, behavioural factors may also affect the outcome of pregnancy. The assumption here is that pregnant mothers with short birth spacing, still have very young children requiring attention and as such may not attend antenatal care at all or will attend later in pregnancy.

On the other hand, women with longer birth intervals, may be motivated to monitor the pregnancy and have higher levels of attendance of antenatal care (and be more likely to deliver in health institutions). In the analysis by Boerma and Bicego (1991), this was more pronounced in five out of seven DHS countries in Sub-Saharan Africa where it was found that short birth interval was a hinderance to attending antenatal care. Another line of argument is that poor health and nutritional status of the mother may also have postnatal consequences, such as impaired lactation (Pebley and Millman, 1986; Retherford, 1989). It has been further argued that sibling competition is another mechanism in the birth interval/child spacing association especially in the postneonatal period. The argument here is that the newborn child has to compete with another young sibling for household resources and mother's care. The obvious

effects of this is that the nutritional status of the youngest child, i.e., child with the short birth interval will be impaired. Lack of adequate care for the child may result in higher incidence of illness due to unclean environment exposure and higher infectious pathogens etc.

Majumder (1990), using logit linear model found that irrespective of mother's age at birth and birth order, children whose immediately preceding sibling died in infancy, have considerably higher risks of dying also in infancy than children whose preceding sibling survived the whole period of infancy. However, in the case of child mortality the risk was greater for children whose preceding sibling was alive at age one. This could be due to sibling competition. An additional biomedical mechanism which has been cited in this nexus, regards the transmission of infectious diseases. The argument states that a short birth interval implies that the index child has an older sibling aged less than 24 months, when infectious diseases such as diarrhoea and respiratory infections are at its highest level in childhood, there are high chances of transmission from one sibling to the other. Aaby (1988) has shown that measles mortality in populations is high because of the relatively high proportions of secondary case in an outbreak. This is more because of over-crowding rather than short birth intervals.

Breastfeeding

Increasing survival chances of children in developing countries is one of the primary health care goals. To this end, one of the cornerstone of the child survival strategy promoted by the World

Health Organisation (WHO) and UNICEF is breastfeeding. Thus, breastfeeding has received a lot of attention and has been shown to be negatively associated with child survival. The relationship between breastfeeding and childhood mortality (especially in early childhood), has been well documented with varying results from study to study.

The literature generally indicate that breastfed children are less susceptible to the risk of infant and child death as compared with artificially fed children and that the duration as well as the intensity of breastfeeding are positively associated with child survival. This implies that wholly breastfed children tend to have a lower risk of dying than partially breastfed ones (Da Vanzo, 1986; Palloni and Tienda, 1986). A part from the widely reported significant negative effects of breastfeeding on child survival, another aspect of the relationship is the age pattern of mortality. Studies have found that the strong effects of breastfeeding on mortality in childhood, gradually declines as the child grows older.

In the study by Palloni and Tienda (1986), a negative relationship between breastfeeding and child mortality was found for ages between 1 and 23 months. Similar results were found by Da Vanzo (1986) and Holland (1989). Some studies have also come up with findings which are contrary to the relationship stated above (Palloni and Millman, 1986). The mechanisms through which breastfeeding operates to influence child survival are not very well understood. Palloni and Tienda (1986) noted that what is known about the relationship has been derived from clinical and epidemiological evidence which suggest that breast milk has some properties that relate to child survival. More specifically, breast milk is said to meet the nutritional requirements for the

normal growth of a child for the first six months and that breast milk has substances that give the infant natural immunity from bacterial, intestinal and respiratory infections (Winikoff, 1980). Recent studies have thereby concentrated more on the relationship between breastfeeding and birth interval (Retherford, et al., 1989). It has been hypothesised that in societies where breastfeeding is longer, mortality is higher and use of contraceptives is rare, breastfeeding has strong effects on child survival and postpartum amenorrhea. However, breastfeeding does not explain the effects of previous birth interval on early childhood mortality but contributes substantially to explaining the effects of the following birth interval on early childhood mortality through the early weaning mechanism (Palloni and Millman, 1986; Retherford et al., 1989).

Household Exposure Factors

These factors are expected to increase the risk of potentially fatal diarrhoeal diseases. Bicego and Boerma have noted that the level of household exposure will, in part, be related to the pathogenicity of household drinking water and whether or not the household has adequate waste disposal facilities. In a study using Nepal Fertility and Family Planning survey data, Gubhaju et al., (1991) have argued that in the Nepalese population in general, socio-economic and environmental factors were not important determinants of infant mortality especially in rural areas where a large proportion of the population are illiterate, small proportion have access to electricity, portable water and toilet facility. However, they hypothesised that socio-economic and environmental factors may be important for child survival in the urban area of Nepal, which have a higher proportion of literate women, many households with access to electricity, portable

water supply and toilet facility; but due to overcrowding, improper sanitation, drainage and sewage disposal facilities, environmental factors may adversely affect the child survival prospects in Urban Nepal. In another study by Pant (1991) using the same data as Gubhaju et al., (1991) but for Urban Nepal only, concluded that analysis of effects of household characteristics showed lower infant and child mortality in households which had access to a toilet or electricity. Lower child mortality was found in households which had tube-well or piped water.

Review of Infant and Child Mortality Studies in Kenya

A number of studies have been conducted in Kenya which have looked at infant and child mortality differentials using various approaches. These studies have shown that there exists wide regional, socio-economic and demographic variations in levels of infant and child mortality (Anker and Knowles, 1977; Kibet, 1982; Mott, 1982; Mosley,1983; UN, 1986; Blacker et al.,1987, Brass, 1993, Brass and Jolly, 1993; Koyugi, 1994; Ikamari, 1996). The following section outlines some of these studies and their main findings.

Brass (1993) and Brass and Jolly (1993) investigated changes and differentials in child mortality using the 1977/78 Kenya Fertility Survey (KFS) data and 1988/89 Kenya Demographic and Health Survey (KDHS). In this study, it was found that infant and child mortality declined from 184 per 1000 live births in 1948 to 60 per 1000 live births in 1989, while child mortality declined from 262 per 1000 live births in 1948 to about 100 per 1000 live births in 1989; life expectancy increased from 35 years in 1948 to 59 years in 1989. Some of the factors which have

been shown to be responsible for these declines are maternal education, paternal education, region of residence and household income (Anker and Knowles, 1980; Kibet, 1982; UN, 1985; Mosley, 1989). Maternal education has been identified as a very important predictor of infant and child mortality. Infants and children of educated mothers have been found to lower risks of death than those of uneducated mothers (Kibet, 1982; Mosley, 1989).

Mosley (1989) has noted that improvements in maternal education accounted for 86 percent of the decline in child mortality between 1962 and 1979 and between provinces of Kenya and that the rest of the decline was explained by improvements in household incomes. Studies have also shown that substantial regional variations exist in Kenya (Kibet,1982; Ewbank et al., 1986; Blacker et al., 1987; NCPD, 1989; Venkatacharya, 1991; NCPD, 1993). Infant and child mortality has been found to be higher in Nyanza, Western and Coast Province than in the other Province. Regional differences in access and utilisation of health services, levels of social and economic development, nutritional status and ecological conditions have been mentioned as the possible explanatory factors. Anker and Knowles (1980) indicated that the endemic presence of malaria, a proxy for disease prevalence, was significantly and negatively associated with expectation of life at birth and child survival to age three. They found that children born to families residing in malarial areas had a lower (by about 7 years) life expectancy at birth than those born to families residing in non-malarial areas or where malaria was less common.

Rural/Urban residence differentials in infant and child mortality have also been documented in several studies in Kenya. Rural residence has been associated with slightly higher infant and

child mortality than urban residence (Ewbank et al., 1986; Venkatacharya, 1991). The analysis carried out by Ewbank et al., showed that the rural-urban differentials in infant and child mortality, disappeared in the presence of controls for various socio-economic and demographic factors. In venkatacharya's study however it was found that there were significant under-five mortality differentials by place of residence after controlling for maternal and paternal education, ethnicity and religion. The data used in this study was derived from the KFS 1977/78 and the method used was that of Trussell and Preston (1982).

The effects of maternal factors such as age, marital status and type of marriage have also been found to influence infant and child mortality in Kenya (Mott, 1982; Muganzi, 1984). Infant and child mortality have further been found to be negatively associated with the length of breastfeeding (Muganzi, 1984; Bankole and Olaleye, 1991). Birth order and mother's age at birth, have also been indicated to exert some influence on infant and child mortality (Rutstein, 1984). Birth intervals have also been associated with infant and child mortality (Muganzi, 1984). Using data from the KFS 1977/78, Muganzi (1984) found a positive association between inter-pregnancy interval and child survival during infancy. However, a small scale study carried out in Machakos district in Kenya found that birth interval had no effect on child health and mortality. The results obtained indicated that children with shorter and succeeding birth intervals did not experience a higher risk of mortality or growth retardation during the first two years of life than the children with long birth intervals (Boerma and Van Vinnen, 1984).

Using the 1979 census results, Ewbank et al., (1986) found a small mortality differential by sex

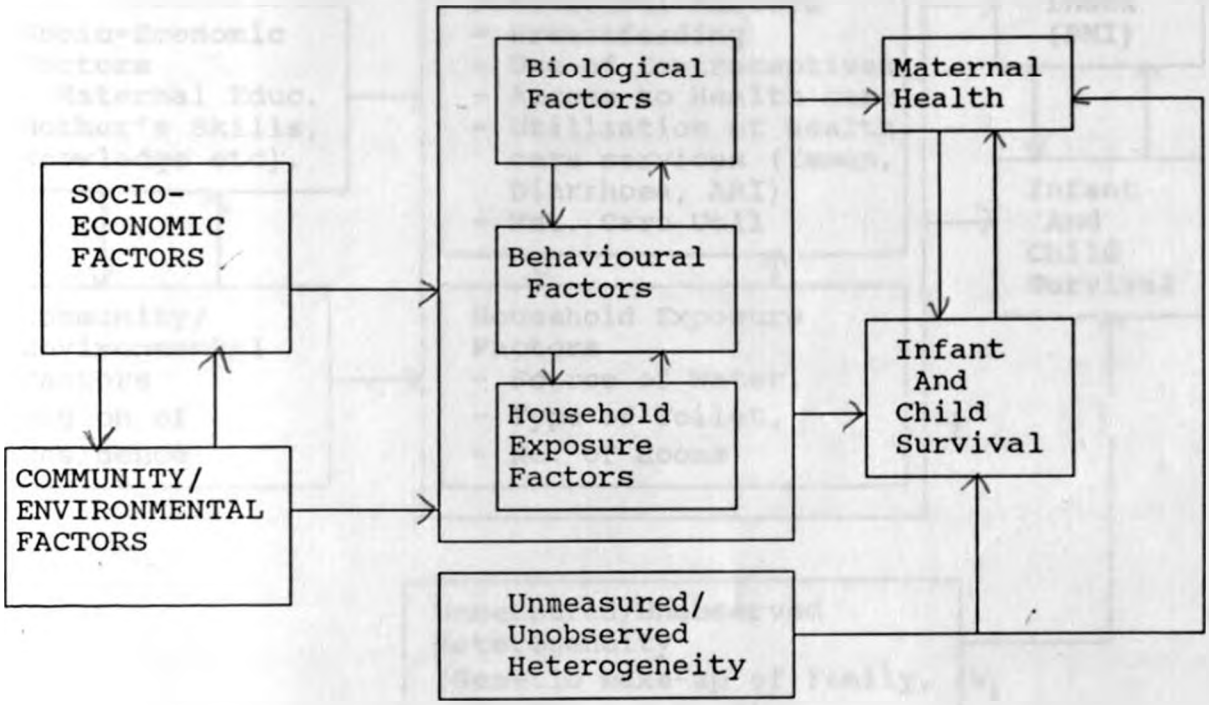
of the child, male children were found to have a slightly higher infant and child mortality than female children. Similarly in the study by Muganzi (1984) and Mott (1982) independently found a weak association between sex of the child and infant and child mortality.

They concluded that sustained decline in mortality in Kenya has resulted from socio-economic development and related cultural changes, which can be seen clearly in the relationship between education and mortality.

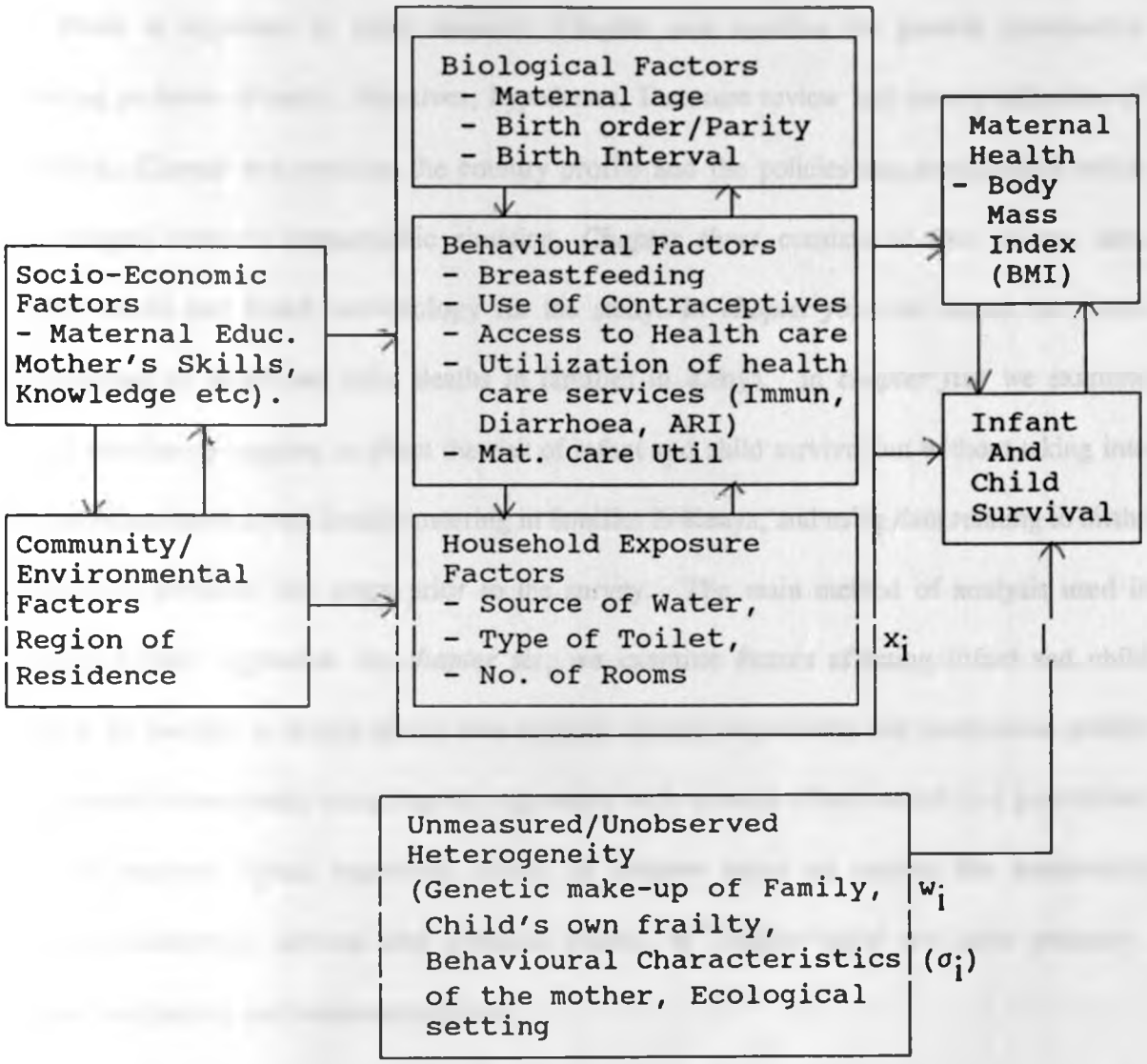
Based on previous researches in the area, the proposed study plans to achieve the objectives stated earlier using the following conceptual and operational frameworks. It is posited that the survival chances of siblings are correlated; that the correlation could be due to many characteristics/factors shared by children of the same family. These familial characteristics/factors may be categorised as socio-economic (maternal education), biological/biosocial (maternal age, birth order/parity, birth interval and breastfeeding), behavioural (access and utilization of health care services, household facilities etc), and the unobserved/unmeasured heterogeneity (genetic make-up of family, ecological setting etc). The same factors that are responsible for high infant and child mortality, may also responsible for maternal ill-health and subsequently death.

1.3.3 Conceptual Framework:

A framework for the analysis of familial association in the risks of infant and child mortality and maternal health.



1.3.4 Operational Framework



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[1]Note: $f(p_{ij})=x_{ij}\beta+\sigma_i v_i$ where $v_i = w_i - E(w_i)/(\sqrt{var(w_i)})$, σ_i is the scale parameter for the unobserved and/or the unmeasured covariates. The chances of child survival is a function of known covariates plus that due to the unmeasured/unobserved factors.

1.4 Organization of Thesis

The thesis is organised in eight chapters. ***Chapter one*** contains the general introduction including problem of study, objectives, hypotheses, literature review and conceptualization of the study. ***Chapter two*** provides the country profile and the policies and programmes which have shaped Kenya's demographic situation. ***Chapter three*** consists of data source, data quality checks and broad methodology for the study. In ***chapter four*** we assess the extent of clustering of infant and child deaths in families in Kenya. In ***chapter five*** we examine factors that theory suggests to affect the risk of infant and child survival but without taking into consideration the observed death clustering in families in Kenya, and using data relating to births which have occurred five years prior to the survey. The main method of analysis used is standard logistic regression. In ***chapter six***, we examine factors affecting infant and child survival in families in Kenya taking into account sibling dependence and unobserved and/or unmeasured heterogeneity using logistic regression with random effects which is a generalised form of standard logistic regression model. In ***chapter seven*** we explore the relationship between infant/child survival and maternal health. In ***chapter eight*** we have summary, general conclusions and recommendations.

CHAPTER 2

KENYA - COUNTRY PROFILE

In this chapter, the country profile and the policies and programmes which have shaped Kenya's demographic situation are discussed.

2.1. Geographical Setting

Kenya covers an area of 582,000 square kilometers. It borders Ethiopia in the north, Sudan in the northwest, Uganda on the west, Tanzania in the south and Somalia in the east. It has 400 kilometers of the Indian ocean coastline. It lies between 3 degrees north and 5 degrees south latitude and between 34 - 41 degrees east longitude. It is entirely within the equatorial zone. The country is almost bisected by the equator (see map).

The country falls within two distinct regions: lowland and highland (upland). This distinction affects the climate, patterns of human settlement and agricultural activities. Kenya has an unusually diversified physical environment; Savannah, Tropical Equatorial, Volcanic and Tectonic Plateau. Approximately 80 percent of Kenya's land is arid and semi-arid and only 20 percent is arable. A large part of the arid and semi-arid zones have been set aside for wildlife conservation.

Although the overall population density of Kenya is 38 persons per square kilometer, but the rich high potential district of west and central regions show densities of 200 - 300 persons per square kilometer. About 18 percent of the population lives in the urban centres and more than a one-third of the urban population live in Nairobi Metropolitan city.

The climatic feature is the long rainy season from March to May. This is followed by a long dry and cold spell from May to October. Short rains come between October and December. In the area around Lake Victoria in the west, rains are well distributed throughout the year.

Administratively, Kenya is divided into eight provinces which are further sub-divided into districts, divisions, locations and sub-locations. In all there are 57 districts in the country. The lowest administrative unit is the sub-location which is headed by an assistant chief.

2.2 History

Kenya became an independent nation from British rule on December 12, 1963. It was a multi-party state until 1982 when the constitution was amended to make it a one-party state. However, in November 1991, in line with political changes taking place in the world, parliament repealed the section of the constitution which made Kenya a one-party state and reverted back to a multi-party democracy. In 1992 the first multi-party elections were held.

There are 43 ethno-linguistic groups in Kenya. The major groups are Kikuyu, Luo, Luhyia,

Kamba, Kalenjin, Mijikenda, Meru, Embu and Kisii. Kikuyus primarily inhabit the Central Province, Luos in Nyanza Province, Luhyias in Western Province, Kambas in southern part of Eastern Province, Kalenjins in Rift Valley Province, Mijikenda in Coast Province, Merus in the central part of Eastern Province and Kisiis in Nyanza Province as well. Christianity and Islam are the major religions in the country. However, freedom of worship and association is enshrined in the constitution.

2.3 Economy

Agriculture is the main stay of the country's economy, accounting for about 25 percent of the gross domestic product (GDP); manufacturing accounts for about 13 percent of the GDP. Coffee, Tea and Tourism are the main foreign exchange earners. The vast majority of Kenyans are small-scale farmers but larger-scale dominate in the export-oriented sector of agriculture mainly coffee, tea, cereals and livestock production.

Economic Indicators

Table 2.3.1 indicates that during the first years of independence, Kenya achieved a commendable growth compared to other developing countries. The Gross Domestic Product (GDP) increased on average by 6.6 percent per annum during 1964 to 1973 period. The good performance following this period was however not maintained; from early 1980s, the performance of the economy has indeed been poor (GOK, 1996). The growth rate of GDP declined to an average

of 5.2 percent per annum in the period 1974-79 and to 4.1 and 2.5 in the periods 1980-89 and 1990-95 respectively.

Table 2.3.1: Average Annual Growth Rate of Real Gross Domestic Product (GDP) 1964 - 1995 (percent)

	1964-1973	1974-1979	1980-1989	1990-1995
Agricultture	4.6	3.9	3.3	0.4
Manufacturing	9.1	10.0	4.8	3.0
Private Households	3.5	14.5	10.0	10.5
Government Services	16.9	6.5	4.9	2.6
Finances, Real estate etc	9.8	12.4	6.7	6.6
Other Services	-	3.3	4.2	1.9
GDP	6.6	5.2	4.1	2.5

Source: National Development Plan 1997 - 2001 (1996)

It can be seen from the table that during the period 1964-1973, the manufacturing sector registered an average growth rate of 9.1 percent per annum and the agricultural sector 4.1 percent per annum. Whereas during the period 1980-89 and 1990-95 the annual growth rates in the manufacturing sector were 4.8 and 3.0 percent per annum respectively and in the agricultural sector 3.3 and 0.4 percent per annum respectively. This implies that the two major sectors have recorded declining performance over the last three decades.

The economic changes may be said to have had a substantial social and perhaps psychological impact in Kenya. All sectors and regions of the country have become linked to the cash/market economy. The mobility of the population has increased and modern media reach most groups. Values, perceptions and attitudes have changed rapidly and continue to change. These ideational

changes appear irreversible and are an important channel through which material changes affect demographic behaviour (Brass and Jolly, 1993).

2.4 Demographic Situation

On the basis of census statistics, Kenya's population increased from 5.4 million in 1948 to 21.4 million in 1989 (see Table 2.4.1). However, the trends show a general decline in demographic parameters over the years as depicted by the intercensal growth rates, crude birth rate (CBR), crude death rate (CDR), infant mortality rate (IMR), total fertility rate (TFR), and increase in life expectancy at birth.

Table 2.4.1: Demographic Indicators 1948-1989

INDICATORS	1948	1962	1969	1979	1989	*1996
POPULATION (in Million)	5.4	8.6	10.9	15.3	21.4	28.2
GROWTH RATE (% p.a)	2.3	3.0	3.3	3.8	3.3	2.7
DOUBLING TIME (years)	30	23	21	17.5	20.7	25
DENSITY (per sq. Km)	10	15	19	27	37	48
CBR (1000)	50	50	50	52	45	40
CDR (1000)	25	19	17	14	11	13
TFR	6-7	6.8	7.6	7.9	6.9	5.4
IMR (1000	184	174	119	84	74	62
LIFE EXP. (yrs)	35	45	49	52	59	51

Source: Census Vols. (various) CBS, MPND

* The 1996 figures are from the World Population Fact Sheet and can be deemed as estimates.

It can be seen from this table 2.4.2 that, differentials in childhood mortality by province are quite marked. Child mortality is highest in Nyanza province where 19% of children do not live to see their fifth birthday. It is lowest in Central, Eastern, and Rift Valley provinces respectively. Nyanza again leads with the highest IMR (it is important to note however that there are only two districts in Nyanza province which contribute to high mortality rates) followed by Coast and Western provinces respectively.

Table 2.4.2: Infant and Child Mortality Differentials by Region (Province)

Region	Neonatal Mortality	Postneonatal Mortality	Infant Mortality	Child Mortality	Under-Five Mortality
Nairobi	(33.8)	(10.6)	(44.4)	(39.5)	(82.1)
Central	16.8	14.1	30.9	10.7	41.3
Coast	28.5	39.8	68.3	43.4	108.7
Eastern	24.1	23.3	47.4	19.4	65.9
Nyanza	38.5	89.4	127.9	67.5	186.9
R.Valley	24.5	20.2	44.8	16.7	60.7
Western	26.9	36.6	63.5	49.3	109.6

Source: NCPD-KDHS, 1993

Maternal Mortality.

The above pattern of childhood mortality also prevails with regard to maternal mortality, although the hierarchy has changed. Kwale District in Coast province has the highest maternal mortality ratio followed by South Nyanza district in Nyanza province and Busia district in Western province (Table 2.4.3). The national ratio is 365 per 100,000 (KMMBS, 1994).

Table 2.4.3: District Maternal Mortality Estimates based on information on Survivorship of Sisters for the period 0-4 years Before the Survey

DISTRICT	EST. MMR. per 100,000 Births
Kwale (Coast)	2,221.7
South Nyanza (Nyanza)	1,072.9
Busia (Western)	1,002.3
Kitui (Eastern)	457.7
Baringo (R. Valley)	430.9
Kisumu (Nyanza)	294.3
Kisii (Nyanza)	233.6
Taita/Taveta (Coast)	211.0
Embu (Eastern)	136.5
Nyeri (Central)	18.8

Source: UNICEF/PSRI KMMBS (1994) (Unpublished).

Fertility Patterns

Table 2.4.4 shows that there have been marked declines in total fertility rates in all provinces between the period 1989 and 1993 with the exception of coast province which had an increase of 6 percent in the same period. It is important to note that coast province still experiences high levels of infant and child mortality and that one of the districts in coast province had the highest maternal mortality rate in the country.

Table 2.4.4: TFR By Province 1989 and 1993

Province	1989 KDHS	1993 KDHS	Percent Change
Nairobi	4.6	3.4	-26
Central	6.0	3.9	-35
Coast	5.0	5.3	6
Eastern	7.0	5.9	-16
Nyanza	7.1	5.8	-18
Rift Valley	7.0	5.7	-19
Western	8.0	6.4	-20
Total	6.7	5.4	-19

Source: NCPD-KDHS, 1993 pg.25.

The following table shows further evidence of fertility decline in Kenya for the period 1962-1993.

Table 2.4.5: Mean Births per Mother by Age Group, 1962 - 1993

Age Group	Census 1962	Census 1969	Census 1979	Census 1989	KDHS 1989	KDHS 1993
15-19	1.71	1.45	1.48	1.62	1.31	1.19
20-24	2.61	2.50	2.52	2.37	2.01	1.93
25-29	3.87	4.11	4.10	3.79	3.66	3.32
30-34	5.11	5.46	5.85	5.41	5.16	4.74
35-39	5.99	6.50	6.96	6.59	6.63	6.29
40-44	6.55	6.99	7.59	7.48	7.53	7.14
45-49	6.84	7.26	7.85	7.85	7.85	7.96

Source: CBS, Kenya Population Census Analytical 1989 Report Vol.4. pg.5.

In summary one may say that although changes in material conditions of life and also in attitudes, aspirations and motivations of the people seem to have been the driving forces behind the demographic changes in Kenya, government policies and programmes have also played a major role as well. Some of the policies and programmes which have played a role n Kenya's demographic situation are outlined in the following section.

2.5 Policies and Programmes

In 1965, the government of Kenya expressed concern with regard to the impact of rapid population growth on development in the Sessional Paper No.10 of 1965 entitled 'African Socialism and its implications to Planning in Kenya'. This led the government to request Population Council in 1966 to advise on programmes lower population growth. Subsequently in

1967, the National Family planning programme was established. However, the national census of 1969 (the first post-independence census) provided evidence of a high level of fertility in the country and as a result, the government decided to launch a five year (1974-1978) family planning programme. Similarly, at the world population Conference held in Bucharest in 1974, Kenya Country Statement indicated further the dire consequences of rapid population growth in the country and proposed her intent to expand voluntary family planning programs on a more systematic basis under the Ministry of Health. This led to the establishment of the family welfare centre (currently known as the Division of Family Health) in 1976 and thereafter the Population Studies and Research Institute was established in 1977.

The specific goals of 1974-1978 Family Planning Programme were to reduce annual rate of increase from 3.3 percent per annum in 1975 to 3.0 percent per annum in 1979 and to improve the health of mothers and their children aged under five. However, the family planning of the MCH programme had limited success. This was confirmed by the results of the 1979 National census which showed that the population growth rate was 3.8 percent per annum which was much higher than the projected figure of 3.0 percent per annum. The failure to meet the stated target was attributed to shortfalls in the assumptions used to arrive at the target. The plan to reduce the growth rate concentrated on the supply side of family planning services instead of putting emphasis on programmes aimed at changing family size norms.

With the realisation to improve on the earlier weaknesses of the family planning programmes, the Government of Kenya approved the establishment of the National Council for Population and

Development (NCPD) in 1982. Its mandate was to formulate and articulate population policies as well as to coordinate the implementation of all population activities in the country.

Other policy measures that were instituted in 1986, included the policy for District focus for rural development and guidelines for its implementation. As a result the NCPD decentralised population programmes at district level by posting population officers to district offices; to expand/modify service delivery to wider audience particularly in the rural areas and to enable subsidised sale of family planning commodities in urban areas. In 1989, the need to integrate a wide range of population variables in development plans as articulated in Sessional paper No.1 of 1986, on Economic Management and Renewed Growth (GOK, 1986).

CHAPTER 3

DATA SOURCE AND STUDY METHODOLOGY

3.1 Data Sources

In this chapter, we discuss the source of data for the study, the quality of the data to be used in the analysis and the general methods of analysis.

The data for the study is drawn from the Kenya Demographic and Health Survey (KDHS, 1993). Although the sample used for the survey was national in scope, it however excluded the whole of North-Eastern Province and four districts, two in the Rift Valley and two Eastern Provinces. The excluded areas account for less than four percent of Kenya's population. As such, the study utilizes information from only 15 rural districts of the total in which the survey was carried out. The districts used, are generally the larger districts in the provinces and were oversampled in the survey to allow for production of estimates for certain variables at district level. There are wide disparities in infant and child mortality as well as maternal mortality in these districts. Further, by using rural districts, it is hoped that one of the problem which has been associated with retrospective survey data, that is, information on place of residence and on all variables relating to socio-economic and environmental factors refer to the conditions prevailing at the time of the survey, while mortality data is pooled over twenty-five year span, will be minimised (Meegama, 1980). It has also been argued that mortality estimates of cities are necessarily very

crude because of the estimation procedures. The data on child survival for the cities is not limited to children born in the city or to persons-years of risk lived in the city, but includes some children who may have died in other areas before their mothers migrated to the city (Ewbank et al., 1986).

The information used in this study are obtained from the woman's questionnaire. The information used was based on retrospective birth histories collected from woman aged 15-49 at the time of the survey. The eligible women were identified through the household schedules. In all, 7952 women were identified as being eligible for the individual interviews and out of these, 7540 (95 percent) were successfully interviewed. The rural sample consisted of 6379 eligible women and out of these, 5370 women were drawn from the 15 focus districts whose data has been used in this study.

3.2 Data Quality

Although detailed consistency checks were in-built in the KDHS questionnaire to ensure accuracy of information obtained from birth histories, it is possible that some mothers may have omitted some information on the date of birth and the age at death. Date of birth data are important for any analysis of mortality by time period. It has been argued that ignoring cases with missing information would cause downward biases in childhood mortality estimates because typically information on year and month of birth is more likely to be missing for children who have died than those children who are still alive (Chitambaram and Sathar, 1984; Sullivan et al., 1990).

Further, trends and differentials in mortality would be distorted because in general the date of birth is more likely to be missing for events placed further back in time and for children in certain sub-groups of the population.

In order to overcome some of these problems, in Demographic and Health Surveys (DHS), if the year or month of birth is missing, a value is imputed using a standard imputation procedure developed by the Institute for Resource Development (IRD, 1987). This procedure uses other information reported by the respondent to establish a logical time-period in which the birth probably occurred and then randomly assigns a date within that period. All children are therefore included in the estimation of mortality rates (Curtis, 1995). With a well-designed imputation program, the impact of missing date of birth information on mortality rates and trends is expected to be minimal. This is particularly true if only the month of birth has to be imputed because imputation then occurs within a one year range.

Date of birth Data

This section examines the extent of imputation of date of birth data in the KDHS, 1993 in selected districts. The extent of missing information by survival status of the child is examined first. The second analysis looks at the extent of displacement of births out of the period covered by the health section of the questionnaire with particular emphasis on the relationship between any such displacement and the survival status. Table 3.1.1 presents the results of the extent of of births (year and month were reported, year was reported and month was imputed, year was

imputed from the reported and month was imputed, and nothing was imputed as both year and month were missing) by completeness of reporting, survival status and selected districts. The denominator of the percentage is births with a date of birth (reported or imputed) during the 15 year period prior to the survey.

Table 3.1.1: Percentage of births 0-15 years before the survey with incomplete information on date of birth by survival status in selected districts, KDHS,1993.

	Living Children					Dead Children				
District	1	2	3	4	5	6	7	8	9	10
Murang'a	97.1	1.4	2.0	0.1	731	85.2	11.1	0.0	3.7	27
Nyeri	97.1	0.1	2.5	0.2	690	94.7	5.3	0.0	0.0	19
Kilifi	98.4	0.3	1.2	0.2	746	90.8	7.5	1.7	0.0	120
T/Taveta	95.5	1.1	2.8	0.6	529	84.4	15.6	0.0	8.1	45
Machakos	94.8	0.8	4.4	0.0	1013	84.6	14.1	1.3	0.0	78
Meru	93.0	0.1	5.9	0.9	843	83.9	6.5	1.6	0.4	62
Kisii/Nyami	89.2	4.6	6.0	0.3	1076	76.8	23.2	0.0	0.0	142
Siaya	98.2	0.0	1.8	0.0	796	93.5	6.1	0.0	0.0	230
S.Nyanza	97.9	0.7	1.4	0.0	582	86.5	13.5	0.0	0.0	163
Kericho	76.4	2.0	21.2	0.4	848	53.6	44.9	0.0	0.0	69
Nakuru	98.4	0.8	0.8	0.0	608	85.7	14.3	0.0	0.0	28
Nandi	93.2	0.3	6.5	0.0	954	67.1	22.9	0.0	0.0	70
Uasin Gishu	94.6	0.4	4.2	0.7	684	82.6	17.4	0.0	0.0	46
Bungoma	98.9	0.8	0.2	0.1	932	95.6	4.3	0.0	0.0	117
Kakamega	98.0	0.5	1.6	0.0	832	72.4	27.6	0.0	0.0	105

NB:Due to rounding up, the row percentages may not add up to 100 percent

Col 1 = Nothing Missing Col 2 = Year and age-yr Ignored Col 3 = Month Imputed
Col 4 = All Missing Col 5 = Total Sample (for each district)
Col 6 = Nothing Missing Col 7 = Year Imputed Col 8 = Month Imputed
Col 9 = All Missing Col 10 = Total Sample

The above table indicates that the reporting of the date of birth for living children in the selected districts is fairly good with the exception of Kericho district where there was 21.2 percent imputation on the month for living children. However, the extent of imputation is high for most districts with regard to the year of birth. The highest levels of imputation are for Kericho, Kakamega, Kisii/Nyamira and Nandi districts. The table also shows that the percentage of births with missing date of birth information are higher for dead than for living children.

Displacement of Births.

Misplacement of births can affect estimation of childhood mortality in a number of ways. It has Generally if births are moved forward in time in a situation of declining mortality, the rates would tend to be overestimated for the periods into which they were moved. Conversely, if births are misplaced backwards in time in a similar situation of declining mortality, then the rates would tend to be underestimated in the periods in which they were shifted. The opposite would happen in a situation of increasing childhood mortality (which is however less common). Further, if the misplacement is related to the survival status of the birth, mortality levels and trends would be affected but the direction and magnitude of bias would depend on the nature and extent of the differential misplacement (Curtis, 1995). One way of detecting such errors is to look at birth ratios for adjacent calendar years. In the present study since detailed analysis will be based on information relating to births in the five year period prior to the survey, the ratios for this period are examined for both living and dead children. Birth ratios are defined as: $100*\{2B_5/(B_4+B_6)\}$, where B_4, B_5 and B_6 are the births in the fourth, fifth and sixth years preceding the survey. These are presented in table 3.1.2.

Table 3.1.2: Birth Ratios for the fifth calendar year preceding the survey by survival status in selected districts, KDHS, 1993.

District	Living Children	Number	Dead Children	Difference in Birth Ratios	Number
Murang'a	100.9	154	N.A	N.A	2*
Nyeri	67.9	150	133.3	-65.4	5
Kilifi	100.9	155	53.3	47.6	19
Taita/Taveta	81.7	100	57.1	24.6	9
Machakos	101.3	235	125.0	-23.7	13
Meru	111.9	170	166.7	-54.8	11
Kisii/Nyamira	87.5	207	35.3	52.2	20
Siaya	99.2	181	78.9	20.3	53
South Nyanza	135.1	124	46.7	88.4	37
Kericho	91.7	75	228.7	-136.9	15
Nakuru	90.5	122	N.A	N.A	5*
Nandi	129.0	204	120.0	9.0	8
Uasin Gishu	113.6	138	100.0	13.6	9
Bungoma	103.4	223	57.1	46.3	27
Kakamega	91.5	188	83.4	9.1	24

Source: Derived from KDHS, 1993.
(* There were no deaths in one or two calendar years used in the calculation of ratios).

It is observed from the table that there was some displacement of births from the fifth calendar year to the two adjacent years in some districts implied by a value of birth ratio of less than 100 for surviving and dead children. However, with the exception of Nyeri, Machakos, Meru and Kericho, the birth ratios are lower for dead than for surviving children. This indicates that there was some degree of displacement of births out of the health section of the questionnaire in some districts and that this was more pronounced for the dead children than for the surviving children.

Age at Death

Missing information on age at death causes problems because it is not possible to determine the allocation of death and exposure in the calculation of mortality rates (Curtis, 1995). If cases with missing information are ignored, it would result in a downward bias in the mortality rates. It would be severe when large numbers of dead children were missing because of information on age at death. In DHS, imputation is again used to assign a value to all such cases to be included in the analysis of mortality. A procedure is used which assigns a value to the missing age at death on the basis of last dead child with the same birth order. It has been argued that if omission of the age at death is systematically related to the age at death of the child, imputation could induce some distortion in the age pattern of mortality, although the overall under five mortality rate may not be affected. Trends and differentials in age pattern of mortality would also be affected by this process if omission of the age at death was more common for deaths that occurred further back in time and in some subgroups of the population (Curtis, 1995).

In order to assess the completeness of the age at death data in KDHS, 1993 and particularly in selected districts, deaths under five with incomplete information on age at death by period in which they occurred were tabulated from the date of birth and the imputed age at death in months. The results are presented in Table 3.1.3.

Table 3.1.3: Percentage of deaths under five with incomplete information on age at death by period in which the death occurred in selected districts, KDHS, 1993.

Districts	Years Preceding The Survey						
	0-4	5-9	10-14	15-19	20-24	0-24	No. of Deaths
Murang'a	0.5	0.4	0.3	0.7	0.4	2.3	68
Nyeri	0.1	0.3	0.4	0.3	0.5	1.7	48
Kilifi	1.2	1.6	2.4	1.6	1.1	7.8	197
Taita/Taveta	0.3	0.7	1.0	0.5	0.8	3.3	84
Machakos	0.7	1.3	0.9	1.0	1.0	4.9	140
Meru	1.0	0.8	0.7	0.9	0.8	3.4	96
Kisii/Nyamira	1.4	3.0	0.9	1.9	1.5	8.7	205
Siaya	2.9	5.4	2.5	2.2	1.5	14.6	332
South Nyanza	3.0	2.4	1.9	1.0	1.6	10.1	222
Kericho	0.6	1.1	0.5	0.6	0.7	3.5	78
Nakuru	0.4	0.6	0.2	0.3	0.1	1.7	44
Nandi	0.6	1.8	1.3	0.9	0.5	5.2	113
Uasin Gishu	0.4	0.7	1.0	0.5	0.6	3.3	82
Bungoma	1.7	1.9	1.7	1.1	0.9	7.0	163
Kakamega	1.2	2.0	1.6	1.5	1.4	7.7	173
							2045

Source: Derived from KDHS, 1993

The table indicates that Siaya district which also has the highest number of deaths, stands out as having the highest percentage of deaths with missing information on age at death followed by South Nyanza. For the period 0-24 years preceding the survey, about 15 percent of deaths with incomplete information on age at death occurred in Siaya and 10 percent in South Nyanza. In general, all districts have a high level of incomplete information on age at death for the period 0-24 years prior to the survey. This is particularly pronounced in the traditionally high infant and child mortality districts in the country, i.e, Siaya, South Nyanza and Kisii/Nyamira (Nyanza Province); Kilifi (Coast Province) and Bungoma and Kakamega (Western Province). Nyeri (Central Province) and Nakuru (Rift Valley Province) have the lowest level of incomplete information on age at death for the period 0-24 years prior the survey. Nyeri district, traditionally, has the lowest infant and child mortality level in the country.

Gross age misstatements may result if an interviewer systematically over or under estimates ages of mothers by five year age group. To detect such errors, the percentage of women in each five year age group is examined. When there are no serious defects with regard to age misreporting, the proportion of women in successive age groups would be expected to decrease monotonically with increasing age under conditions of constant fertility, declining mortality and closed migration.

Table 3.1.4: Percent distribution of women aged 15 - 49 years by five year age groups in selected districts, Kenya, KDHS,1993

Age Group	% distribution	No. of Women
15-19	24.5	1309
20-24	20.2	1080
25-29	18.8	797
30-34	18.9	808
35-39	11.9	508
40-44	11.8	505
45-49	8.0	340
	100.0	5347

Source: Derived from KDHS, 1993

The table shows that in general there were no serious age misstatement. However when data for the selected districts is examined individually, then there is some slight indication of age misstatement in some districts.

3.3. Study Methodology

As stated earlier, different statistical techniques have been used to model familial association in the risks of infant and child survival with varying results. Guo (1993) used a multivariate proportional hazard model with Gamma frailty to the familial association in the risks of child survival over the first five years of life in Guatemala. Zenger (1993) used the random effects

logistic and first order transitional models to examine familial association in the risks of neonatal mortality in Bangladesh. Similarly, Curtis et al., (1991, 1993) used the random effects logistic binomial model to examine familial associations in the risks of neonatal and postneonatal mortality in Brazil. Curtis and Steele (1996) also used the random effects model to examine the strength and structure of neonatal mortality in four populations. Basically all these methods yield similar results. In the present study, we plan to use the random effects model to examine factors affecting infant and child survival while accounting for any excess variation.

The random effects model assumes that the baseline risk of death varies across families due to heterogeneity in mortality risks. This model is an extension of the standardized logistic regression model. The response variable in the analysis will be death or survival within the first month of life i.e, 0-1 month, 1-11 months, 0-12 months etc. The random effects model is written as:

$$\text{logit}(p_{ij}) = x'_{ij}\,\beta + u_i, \qquad \ldots(3.1)$$

where, p_{ij} is the probability that the j^{th} child in the i^{th} family dies in the age intervals stated above, x_{ij} is the vector of covariates associated with the j^{th} child in the i^{th} family and ß is a vector of unknown parameters, u_i be the random effects associated with the i^{th} family. The term u_i is usually assumed to be normally distributed with mean zero and variance σ^2 forming the logistic-normal model (Piece and Sands, 1975 and Anderson and Aitkin, 1985), u_i is standardised to obtain a model of the form:

$$\text{logit}(p_{ij}) = x_{ij}\,\beta + \sigma\, v_i \qquad \ldots(3.2)$$

Where v_i has the standard normal distribution N(0,1) and σ is the scale parameter indicating the amount of variation across families on the logit scale. The model will be fitted using the statistical package EGRET (Statistics and Epidemiology Research Corporation, 1989) via maximum likelihood estimation. EGRET automatically modifies the likelihood ratio test for σ and gives a modified p-value.

However, in order to assess the presence of clustering in families (i.e. objective 1), an

examination will be made to determine whether the number of women with different numbers of child deaths exceeds that which would be expected if the risk were constant for all women and their children. Under the null-hypothesis of no clustering, the distribution of women by the number of child deaths will be assumed to be binomial and the probability of observing *k* deaths for women with family size *n* will be given by:

$$P(x=k)=\{n!/(k!(n-k)!\}* p^{k} (1-p)^{n-k} \quad \ldots(3.3)$$

The average number of child deaths among women with *n* children is *np* and the variance as *np(1-p)*. The parameter will be estimated from the observed proportion of children who have died among all live births of women of a given age. An excess of observed number of women with different number of deaths over the expected is taken to indicate clustering (Ronsmans, 1995; David and Zaba, 1996). It may be noted that rejecting H_0 implies the presence of clustering, but it does not distinguish between different causes.

CHAPTER 4

EXTENT OF DEATH CLUSTERING IN CHILDHOOD IN FAMILIES IN KENYA.

4.1 Introduction

In the present chapter we assess the extent of clustering of infant and child deaths in families in Kenya. The research question we expect to answer is whether some families experience higher proportions of infant and child deaths (death clustering) than others keeping family size fixed.

4.2 Methodology

A number of methodologies have been designed for assessing the extent of death clustering in families. One of the methods involves counting the number of women who have experienced more than one child loss (Das Gupta, 1990; Curtis et al.,1993; Guo, 1993; Curtis and Steele, 1993) while the other method entail the examination of whether the number of women with different numbers of child deaths exceeds that which would be expected if the risk were constant for all women and their children (Ronsmans, 1995; Zaba and David, 1996). The latter involves taking maternal demographic characteristics, preferably parity or age group and comparing these with a theoretical statistical distribution, viz; binomial, Poisson or negative binomial distribution depending upon the realisation of the event. The comparison between the application of these theoretical distributions allow the assessment of the extent of differences in the variability among women contributes to the observed child deaths after allowing for chance factors (Zaba and

David, 1996). If the risk is assumed to vary with parity then women of higher parity may experience higher child loss, and if within each parity group there are no differences in the risks of dying for births of different orders and between children of different mothers, then the distribution of numbers of deceased children will follow some theoretical statistical distribution. We discuss a few of the commonly used distributions namely binomial, Poisson and negative binomial distributions respectively.

The binomial distribution estimates the distribution of failures to be expected in a given number of trials with a constant probability of failure. In the case of mortality, it estimates the expected distribution of deaths in a group of live born children who are subject to a given mortality risk. In a large sample, this distribution should approximate the observed distribution of deaths. The assumption inherent in the use of the binomial model is that fertility decisions are made, by and large, without a clear notion of their outcome generally referred to as the "hoarding approach" (Wolpin, 1997).

The Poisson model is basically a generalized model of the binomial model. Hence, the assumptions inherent in the binomial model may also be true for the Poisson model. The negative binomial model, on the other hand, assumes that child bearing may be targeted towards a certain number of surviving children (i.e, the replacement approach). This implies that parents try to replace children who have died until the desired number is attained and then cease childbearing. The use of negative binomial model in such cases helps in generating a random distribution of deaths because it estimates the number of trials needed to obtain a particular

number of successes with a given probability of failure. Thus, the model tests the difference between the distribution of deaths and that which would prevail under conditions of pure targeting (Das Gupta, 1997). This model is therefore best suited for situations where families may have completed childbearing. Of course one may however, argue that these three models are variants of the same family of distributions.

In the present study, although the binomial distribution is preferred for reasons stated above but, the values for poisson and negative binomial are also given for comparative purposes only.

The probability of observing r deaths out of n births under the binomial distribution is given by:

$$p(x=r) = \frac{n!}{r!\,(n-r)!} q^r (1-q)^{n-r} \qquad \ldots (4.1)$$

where, q is the probability of dying of an individual child. The expected number of deaths out of n births is nq which is estimated from the proportion of dead children among women of parity n. The variance of this distribution will be $nq(1-q)$.

The Poisson distribution is derived as follows: let a random variable Y=0,1,2... denote the number of occurrences of an event of interest in a given time interval and y(t, t+dt) denote the number of events actually observed in the short time of interval (t, t+dt). The number of events in an interval of given length is Poisson distributed with the probability density function:

$$Pr(Y=y) = f(y;\mu) = \exp(-\mu)\,[\mu^y/y!]\; y=0,1,2\ldots,\mu>0 \quad \ldots(4.2)$$

The negative binomial distribution may be described as the probability distribution of the random variable Y defined as the number of failures encountered before the M^{th} success. Its Probability density is given by:

$$Pr[Y=y] = f(y;M,P) = [(M+y-1)!/y!\,(M-1)!]\,(P/Q)^y(1+P/Q)^M$$

$y=0,1,2,3 \ldots$ ...(4.3)

with parameters M and P, where $P=(1-p)/p$ and $Q=P+1$.

4.3 Results of Analysis

4.3.1 Extent of Death Clustering in Childhood in Families

In this section we assess the presence of death clustering in families by looking at the distribution of deaths per woman. The term 'family' is used here to refer to deaths of children belonging to a biological mother and thus excludes foster or any other children. The study sample relates to the total number of births in the last ten years. Note however that we have only selected deaths from only singleton births. This is because it has been shown that multiple births by their nature account for a disproportionately large percentage of infant mortality in both developed and developing countries (Guo and Grummer-Strawn, 1993). Thus, in excluding them from analyses of child mortality one is trying to minimise the bias they may introduce in the analysis.

Further, the distributions are divided into neonatal, postneonatal, infant and child deaths to reflect the age pattern of mortality in childhood. The sample used has also been restricted to fifteen rural districts in order to remove the exposure bias, i.e., the data on child survival for the cities may not be limited to children born in the city or to persons-years of risk lived in the city, but may also include some children who may have died in other areas before their mothers migrated to the city (Ewbank et al., 1986). Another reason for limiting the sample to the rural districts is from a policy perspective of the district focus for rural development which was adopted and implemented by the Kenya Government from mid-eighties. This policy was expected

to reduce urban bias in the provision of amenities. It is expected that results from this study would be useful in evaluating the suitability of this strategy. Moreover, the bulk of Kenya's population lives in the rural areas, i.e., about 80 percent of the total population. The total sample used in this analysis consists of 8745 births which occurred to 3377 women. Table 4.2.1. shows the distribution of the number of births contributed to the total sample by each woman. The mean number of births per woman is 2.6.

Table 4.2.1: Total number of births contributed to the sample by each woman in the ten years preceding the survey

Number of Births	No. of Women	Percent*	No. of Children
1	871	25.8	871
2	822	24.3	1644
3	835	24.7	2505
4	564	16.7	2256
5	246	7.3	1230
6	35	1.0	210
7+	4	0.1	29
Total	3377	100.0	8745

Source: Derived from KDHS, 1993

* The percentages may not add up to 100 due to rounding

Before presenting the death clustering patterns at various ages at infancy and childhood, it is important to highlight the rates of these outcomes for the sample being used and for the total national rural sample for the period under study. Table 4.2.2 shows the mortality rate patterns by various ages in children. It can be observed from the Table 4.2.2 that the rates for the fifteen districts (defined in the table as study sample) are more or less double for all ages of infant and

child mortality in relation to the national rural sample. The observed differences could be explained by the cluster selection effect for the other rural districts not included in this study sample. Secondly, the study sample is heavily weighted by the areas of high infant and child mortality in the country.

Table 4.2.2: Neonatal, Postneonatal, Infant and Child Mortality Rates for the Study Sample and National Rural Sample for the period under Study (1983 -1993).

	Neonatal Mortality Rate	Postneonatal Mortality Rate	Infant Mortality Rate	Child Mortality Rate
Study Sample (Rural)	47.1	61.7	118.9	61.2
National Sample (Rural)	27.5	37.3	64.9	49.7

Source:Derived From KDHS, 1993

Table 4.2.3 presents the distribution of the number of neonatal, postneonatal, infant and child deaths contributed to the study sample by each woman in order to gain some insight into the extent of familial clustering of deaths in Kenya. The proportion of women who have experienced neonatal, postneonatal, infant and child deaths in Kenya for the period under study are 9.5 percent, 12.6 percent, 22.0 percent and 10.1 percent respectively. The results show that 1.6 percent of women in the study sample experienced more than one neonatal death but the deaths to these women accounted for 35.2 percent of all neonatal deaths. Similarly in the postneonatal period, 2.3 percent of the women in the study sample experienced more than one postneonatal death and the deaths to these women accounted for 35.4 percent of all postneonatal deaths. In the infant period, 6.1 percent of the women in the study sample experienced more than one

infant death and the deaths to these women accounted for 48.5 percent of all infant deaths. Similarly, 2.2 percent of the women in the study sample experienced more than one child death which accounted for 38.6 percent of all child deaths.

Using the same method of counting the number of deaths to women who had experienced two or more child deaths for rural Punjab, Das Gupta (1990) observed that 12.6 percent of families in the study sample accounted for 62.2 percent of all child deaths. In Guatemala, Guo (1993) noted that 11 percent of the families with two or more child losses accounted for 53 percent of the total deaths in the sample. In Brazil, Curtis et al., (1993) also found evidence of death clustering in postneonatal deaths. Their findings showed that 2.5 percent of women in the study sample experienced more than one postneonatal death but the deaths to these women accounted for 43 percent of all postneonatal deaths.

The results obtained from the current study and those obtained from earlier studies are consistent and hence, these results may imply that clustering of deaths exists in some families in Kenya at every level of childhood mortality. It is more pronounced in the infant and childhood periods where perhaps the influence of biological, behavioural and household environmental factors play an important role. Also after the neonatal period, there could be other mortality risks such as cross-infection between siblings, particularly, where there are several children of close age. This is more so in cases where infectious diseases become important causes of mortality after the neonatal period (Curtis and Steele, 1996).

Table 4.2.3: Percentage distribution of the number of neonatal, postneonatal, infant and child deaths contributed by women to the study sample, and percent contributed by women experiencing multiple deaths, Kenya, 1993.

	Neonatal Period		Postneonatal Period		Infant Period		Childhood Period	
Number of Deaths	Number of Women	Percent	Number of Women	Percent	Number of Women	Percent	Number of Women	Percent
0	3055	90.5	2950	87.4	2634	78.0	3036	89.9
1	267	7.9	349	10.3	536	15.9	267	7.9
2	37	1.1	53	1.6	141	4.2	60	1.8
3	11	0.3	18	0.5	45	1.3	9	0.3
4+	7	0.2	7	0.2	21	0.6	5	0.1
Total	3377	100.0	3377	100.0	3377	100.0	3377	100.0
% of deaths Contributed by women with Multiple deaths	35.2		35.4		48.5		38.6	

Source: Derived from KDHS, 1993

Further, we can compare our results with that of Curtis and Steele (1996) who looked at variations in familial neonatal mortality risks in four countries where Kenya was included. They based their analysis on the Kenya Demographic and Health Survey data (KDHS) of 1989. The sample was confined to births in a 10 year period before the survey and to singleton births. The findings of this study and that of Curtis and Steele for the neonatal period differ to some extent as shown in table 4.2.4.

Table 4.2.4: Percentage distribution of neonatal deaths contributed to the sample by women and percent of all neonatal deaths contributed by women experiencing multiple deaths, Kenya 1989 and 1993.

Number of Deaths	1989 (KDHS)	1993 (KDHS)*
0	93.3	90.5
1	6.0	7.9
2	0.6	1.1
3	0.1	0.3
4+	0.0	0.2
Total	100.0	100.0
Number of Women	5006	3377
Percent of deaths contributed by women with multiple deaths	20.9	35.2
Neonatal Mortality Rate	28.2	47.1

Note: * implies data for Rural oversampled Districts only.

The proportion of women who experienced a neonatal death in Curtis and Steele (1989) study is 6.7 percent while it is 9.5 percent in the present study. The percentage contributing more than one neonatal death is 0.7 percent (using 1989 KDHS) and 1.6 percent (using 1993 KDHS). The percentage of all neonatal deaths contributed to the sample by women who experienced multiple deaths was 20.9 percent (1989 KDHS) and 35.2 percent (1993 KDHS).

The above differences between the two could be partly due to the different samples used in the two studies. The early study used the sample for the whole country including the urban sample, while the present study is limited to 15 rural districts (focus districts) and/or to the different time periods of the data collection. As earlier noted, the fifteen rural district comprise some of the districts with the highest infant and child mortality in the country. It can therefore be seen from the findings of the two studies that national figures tend to mask mortality differentials in the country by underestimating the real magnitude of the problem. The method does not however allow for parity effects on the probability of having a multiple death. High parity women will always be overrepresented among women with multiple deaths and the probability of observing a death increases with the number at risk. Thus, an alternative approach would be to allow for the systematic parity effects. This method is considered in the next section.

4.3.2 Observed and Expected distribution of childhood deaths among women under Theoretical Distributions

Another method of examining the extent of death clustering, as noted earlier, involves comparing the observed and expected distributions on the basis of some theoretical statistical distributions. In this study, the theoretical distributions are compared with the observed distribution of deaths for women of parities four, five, six and seven for the period 1983-1993 (KDHS, 1993). The methods used to fit the distributions to the data are described below.

The binomial distribution is fitted to the data by first calculating the values of p, i.e., proportion of dead children and $q=1-p$, from the observed distribution. The denominator

used is the total number of children born to women and the numerator is the total number of deaths to these women. The Poisson distribution is fitted to the data by first calculating the mean from the observed distribution i.e, $\mu=np$ and variance$=\mu$, where n is the parity and p is the proportion of dead children and this is then used in equation (4.2). The negative binomial is fitted to the data by first calculating p (i.e, the proportion of surviving children). To obtain the mean and variance, the transformation $P=(1-p)/p$, is made so that mean$=MP$, and the variance$=MP(1+p)$ where M is the parity. We then apply equation (4.3) to obtain the expected distribution of deaths.

If we compare the observed and the expected distributions (see tables 4.3.1 to 4.3.4 and Figs. 1 to 4), it can be seen that at parity four and probably at lower parities, the observed and expected distributions follow a similar pattern. This could be explained by the fact that at lower parities the distribution of women consists of those with fewer births and hence may not have experienced as many deaths as women of higher parities. Also, the unmeasured and/or unobserved factors may not be so pronounced at lower parities. Thus following Ronsmans (1995) and Zaba and David (1996), the observed pattern for higher parities does not follow that of the theoretical distributions. The wider dispersion for higher parities could be explained by the fact that as parity increases, women become increasingly more polarized in the apparent risks of their children's experience. Zaba and David (1996) observed that these differences persisted in spite of making allowances for parity-specific risk.

Table 4.3.1: Observed and Expected distribution of childhood deaths for women of parity 4, KDHS, 1993

Number of Deaths	Proportion of Women			
	Observed	Binomial	Poisson	Negative Binomial
0	0.732	0.713	0.723	0.713
1	0.216	0.251	0.234	0.231
2	0.048	0.033	0.038	0.047
3	0.004	0.002	0.004	0.008
4	0.000	0.000	0.000	0.001

Fig 1: Observed and Expected Distribution of Child Deaths for Women of Parity Four

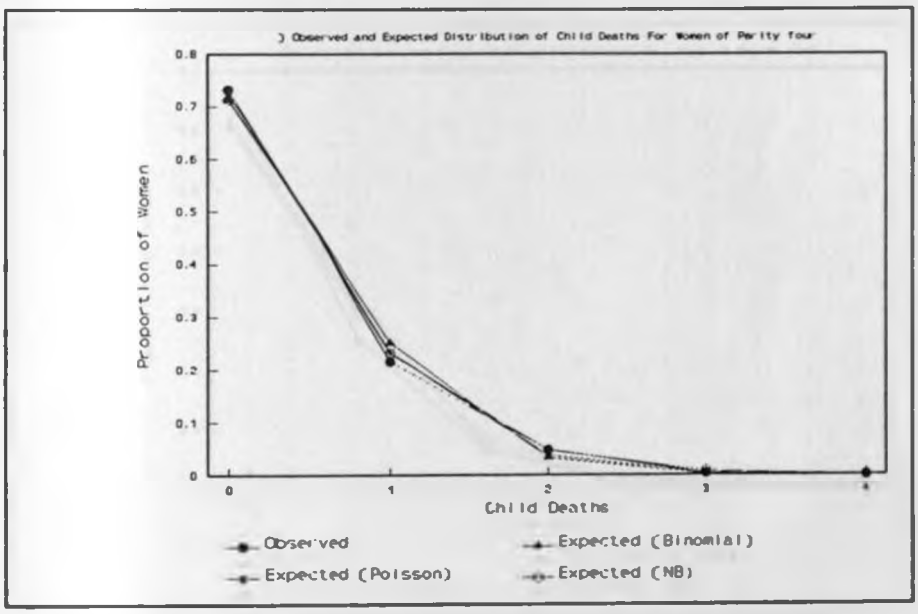


Table 4.3.2: Observed and Expected distribution of childhood deaths for women of parity Five, KDHS, 1993

Number of Deaths	Proportion of Women			
	Observed	Binomial	Poisson	Negative Binomial
0	0.655	0.604	0.618	0.604
1	0.249	0.321	0.298	0.290
2	0.064	0.068	0.072	0.083
3	0.023	0.007	0.012	0.019
4	0.009	0.000	0.001	0.004
5	0.000	0.000	0.000	0.001

Fig. 2: Observed and Expected Distribution of Child Deaths for Women of Parity Five

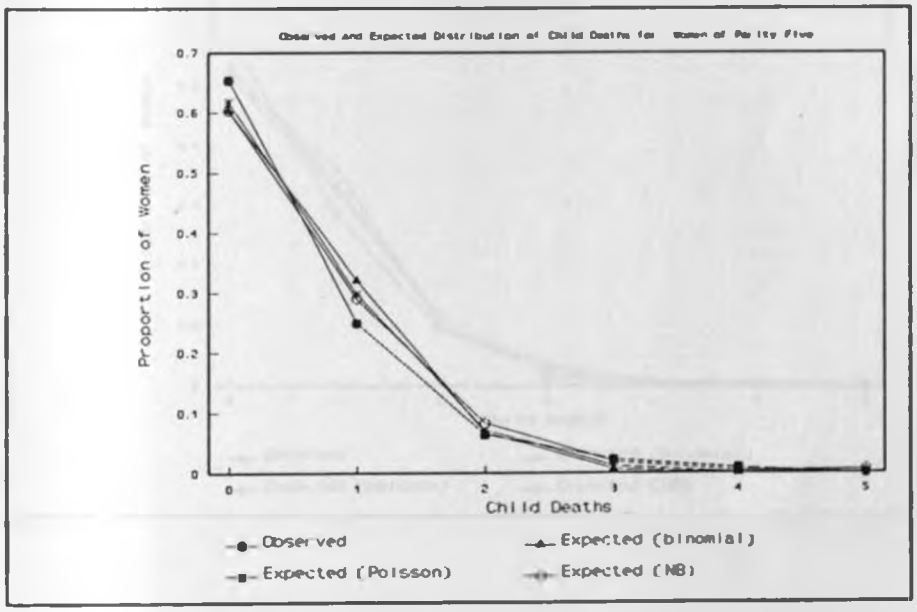


Table 4.3.3: Observed and Expected Distribution of Childhood Deaths for Women of Parity six, KDHS, 1993

Number of Deaths	Proportion of Women			
	Observed	Binomial	Poisson	Negative Binomial
0	0.573	0.514	0.531	0.514
1	0.285	0.362	0.336	0.323
2	0.096	0.106	0.106	0.119
3	0.034	0.017	0.022	0.033
4	0.006	0.002	0.004	0.008
5	0.006	0.000	0.000	0.002
6	0.000	0.000	0.000	0.000

Fig. 3: Observed and Expected Distribution of Child Deaths for Women of Parity Six

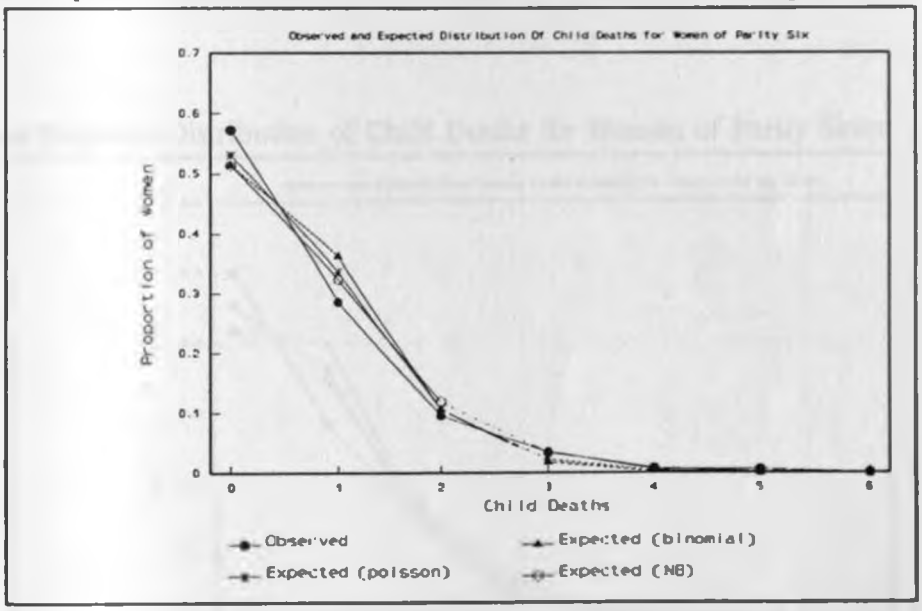
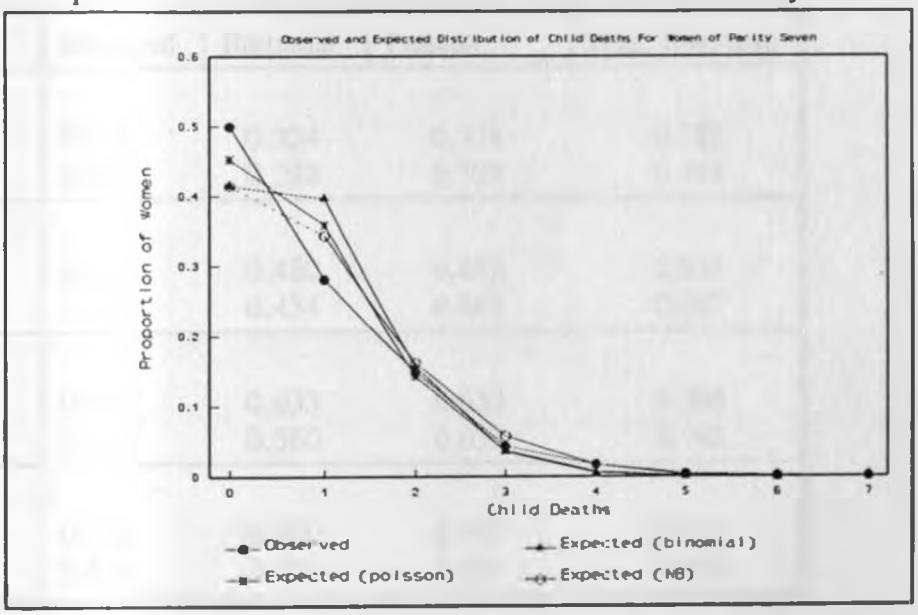


Table 4.3.4: Observed and Expected Distribution of Childhood Deaths for Women of Parity Seven, KDHS, 1993

Number of Deaths	Proportion of Women			
	Observed	Binomial	Poisson	Negative Binomial
0	0.500	0.415	0.453	0.415
1	0.281	0.397	0.359	0.343
2	0.149	0.156	0.142	0.162
3	0.043	0.035	0.037	0.057
4	0.017	0.005	0.007	0.017
5	0.010	0.0004	0.001	0.004
6	0.000	0.000	0.000	0.001
	0.000	0.000	0.000	0.000

Fig. 4: Observed and Expected Distribution of Child Deaths for Women of Parity Seven



The variances of the distributions in Table 4.3.5 attest further to the presence of overdispersion of the risk of child deaths by parity. It can then be concluded that the observed data appears to follow the theoretical distributions (see Figures 1-4), but it can be noticed that, at parity 7 there appears to be a slight disparity in the pattern (fit). It could be that despite controlling for parity, the theoretical distributions assume homogeneity while the observed data may still be having variations/dispersion as a result of some other factors (i.e., heterogeneity being present in the data). Also, while fitting the theoretical distributions we note that other factors have not been taken into consideration except parity.

Table 4.3.5: Distribution of observed and expected number of child deaths for women of parities four, five, six and seven

	Observed	Binomial	Poisson	Neg. Binomial
Parity 4				
Mean	0.324	0.324	0.324	0.353
Variance	0.603	0.298	0.324	0.384
Parity 5				
Mean	0.482	0.480	0.482	0.531
Variance	1.726	0.434	0.482	0.587
Parity 6				
Mean	0.633	0.633	0.633	0.704
Variance	2.704	0.560	0.633	0.786
Parity 7				
Mean	0.789	0.830	0.789	0.937
Variance	3.471	0.730	0.789	1.062

4.4 Conclusion

In this chapter the extent of childhood death clustering in families in Kenya has been assessed using two methods. The results indicate that there is evidence of death clustering at every level of childhood mortality. The results of the theoretical distribution indicate that at any parity, there are women who contribute higher than average deaths to the mortality risk. This may increase with parity because such women may try to replace their dead children or if 'hoarding' strategy is adopted in family building then they may have more children in anticipation that some may die. The differences in variances between the observed and expected distribution of deaths may be attributed to the above stated explanation. However, one limitation of these methods is that they are heavily weighted by women with no deaths. Secondly, some of the causes of clustering may arise out of the different characteristics of women which may be biological or behavioural thus, the method could be a necessary indicator of clustering but not sufficient to prove existence of death clustering in families. In the absence of well established methodologies to detect concentration of deaths to women in certain families, however, they suffice to indicate that certain families experience higher than average risks in infant and child deaths. This implies that the assumption of independence in siblings' observation may not hold good in the analysis of the determinants of infant and child mortality.

In the next two chapters we go on to examine factors that affect infant and child survival without taking into consideration the observed clustering of deaths in families in Kenya (chapter 5) and subsequently taking into account any excess variation (chapter 6) to fulfill the second objective of our study.

CHAPTER 5

FACTORS AFFECTING INFANT AND CHILD SURVIVAL IN FAMILIES IN KENYA

5.1 Introduction

In this chapter we examine factors that theory suggests to affect the risk of infant and child survival without taking into consideration the observed death clustering in families in Kenya, using data relating to births which have occurred five years prior to the survey. The reason for limiting analysis to this period is that most of the explanatory variables used in the analysis relate to this period. The analysis is further limited to singleton births of order two or more. Censored cases are also excluded from the analysis. Analysis is again limited to fifteen rural districts. The sample consists of 3681 births out of which 268 died in infancy and early childhood. Table 5.1.1 presents the distribution of deaths by age at death which indicate that the majority of infant deaths in the sample population occurred in the neonatal and postneonatal periods.

Table 5.1.1: Distribution of infant and child deaths by age at death, Kenya, 1993

Age at Death (months)	Number of Deaths	Percent Distribution
0	69	25.7
1-11	117	43.7
12-48	82	30.6
Tatal	268	100.0

Source: Derived from Kenya Demographic and Health Survey (KDHS, 1993)

While mean age at death is 9.5 months, the median is 6 months and 75 percent of all deaths occurred within 10 months from birth implying that the deaths in the sample districts were concentrated in the first year of life during the period of observation. As a result, the risk of death is higher at neonatal and postneonatal stages of life than at any other stage. One may therefore surmise that this has implications on the identification of factors affecting the mortality pattern i.e., whether predominantly biological, behavioural etc.

Table 5.1.2 further illustrates the death rates in infancy and childhood for the study period in the sample in the form of a life table whereby it can be seen that the greatest risk of mortality is at the neonatal and early postneonatal stages and that the risk decreases with age. Hence, we decide to carry out analysis of mortality in the neonatal, postneonatal, infant and childhood periods separately.

Table 5.1.2: Death rate in Infancy and Childhood, for the period 1988 - 1993, KDHS, 1993

Interval at Start of time	Number entering	Wdrawn During interval	Number Exposed to Risk	Number of Terminal Events	Proportion Terminating	Hazard Rate
.0	3618	25.0	3605.5	69	.0191	.0193
1.0	3524	59.0	3494.5	10	.0029	.0029
2.0	3455	54.0	3428.0	15	.0044	.0044
3.0	3386	57.0	3357.5	14	.0042	.0042
4.0	3315	58.0	3286.0	11	.0033	.0034
5.0	3246	53.0	3219.5	15	.0014	.0047
6.0	3178	64.0	3146.0	18	.0057	.0057
7.0	3096	70.0	3061.0	8	.0026	.0026
8.0	3018	61.0	2987.5	14	.0047	.0047
9.0	2943	63.0	2911.5	7	.0024	.0024
10.0	2873	63.0	2841.5	3	.0011	.0011
11.0	2807	59.0	2777.5	2	.0007	.0007
12.0	2746	276.0	2608.0	35	.0134	.0027
17.0	2435	246.0	2312.0	8	.0035	.0007
22.0	2181	289.0	2036.5	24	.0118	.0024
27.0	1868	266.0	1735.0	1	.0006	.0001
32.0	1601	276.0	1464.0	11	.0075	.0015
37.0	1314	245.0	1166.5	0	.0000	.0000
42.0	1019	300.0	869.0	0	.0000	.0000
47.0	719	283.0	577.5	3	.0052	.0010
52.0	433	285.0	291.0	0	.0000	.0000
57+	149	148.0	75.0	1	.0133	.0027

Source Derived from KDHS, 1993

Comparative analysis of the factors affecting infant and early childhood mortality will be thereby presented in this chapter.

5.2 Methodology

Standard logistic regression is applied to assess the effect of factors that are theoretically said to be associated with the risk of infant and child survival at different ages. The risk of survival as a function of the explanatory variables can be specified as follows:

$$p_{xij} = 1 / \{1 + \text{Exp-}(\beta_0 + \beta_1 X_{1j} + \ldots \beta_p X_{pj})\} \quad \ldots(5.1)$$

p_{xij}, is the probability of the dependent variable taking a value 1 given the explanatory variables $X_{1j}, X_{2j} \ldots X_{pj}$. Therefore, the probability of an infant with characteristics X_{1j}, $X_{2j}, \ldots X_{pj}$ surviving is given by equation 5.1.

However equation 5.1 is better modelled in its linear form where the left hand side is given by the natural logarithm of the ratio p_{xij}/q_{xij} (odds ratio) called the logit of p_{xij}. Where $q_{xij} = 1-p_{xij}$ the probability of not observing the event. Thus

$$\text{logit } p_{xij} = \ln(p_{xij}/q_{xij}) = \beta_0 + \beta_1 X_{1j} + \ldots \beta_p X_{pj} \quad \ldots (5.2)$$

The parameters β are interpreted in terms of logits, a unit change in the independent variable X_{ij} changes the logit of the dependent variable (In p_{xij}/q_{xij}) by the amount of β_i. This is

the additive form of interpretation of the effects. The alternative form of interpreting the logistic model is in terms of the odds of an event occurring. The odds of an event occurring is computed as follows:

Odds (event) ={ Probability (event)/Probability(No event)}

$$= \exp(\beta_0 + \beta_1 X_{1j} + \ldots + \beta_p X_{pj}) \qquad \ldots (5.3)$$

where $\exp(\beta_i)$ (where i=1,...,p) is the factor by which the odds change when the i^{th} independent variable increases by one unit. If β_i is positive, this factor will be greater than 1, which means that the odds are increased. If β_i is negative, the factor will be less than 1. When the β_i is 0, the factor equals to 1 which leaves the odds unchanged. The interpretation in terms of the odds ratio is the multiplicative form. Further, this formula assumes that all the variations in probability of survival with the pre-assigned age intervals can be explained by the set of covariates. Each child is assumed to be an independent observation.

The assumption of independence of siblings observations in the analysis of the factors affecting infant and child survival has been shown not to hold in recent studies. This is because siblings from the same parent, may share many attributes in common and also the assumption of independence implies larger sample (more information) than what is truly available. As a result, the estimated standard errors have been shown to be too small as the data contains more variability than is allowed for by the model. This would imply that some variables may appear to be significant when they are actually not (Curtis et al., 1993; Curtis and Steele, 1996). Additionally when important variables measured or unmeasured are not included in the models, we may obtain biased results (effect of unobserved variables).

5.3. Description of variables and their hypothesized relationship with the dependent variable.

The outcome variable is survival of the child and hence binary, i.e, dead or alive denoted by 0=dead and 1=alive. This is derived from information on survival status of the child at the time of survey and age at death (months imputed) which was drawn from the complete birth histories of women in the sample. The explanatory variables include socio-economic (maternal education), biological, behavioural, household exposure factors and region of residence as a proxy for the ecological zone. A brief description of these variables is outlned below:

Socioeconomic Factors: This comprises of maternal education which is used as a control for socio-economic status and for mother's skills and knowledge towards health related behaviour and child care practices. Education of mother has been shown in a number of studies to be correlated with levels of infant and child mortality although the causal mechanisms through which it operates are still a subject for debate (Cleland, 1980). It is hypothesized that the more educated (secondary and above) the woman is, the more the likelihood of utilizing child and maternal health care services maintaining hygienic practices in child care and avoiding the risks associated with short birth intervals etc.

Biological/Family Formation Factors: These comprise maternal age, birth order, birth interval, prematurity and whether the preceding child was dead or alive. Each of these variables has been shown to be important covariates of mortality in childhood as well as for maternal health in developing countries (Pandey et al., 1998). It is postulated that there is

an increased mortality risk for children with short birth intervals (less than 24 months). It has also been hypothesized that there is increased childhood mortality risk among first births and higher birth orders (5+). In the case of maternal health, it has been hypothesized that the risks of child death are highest among very young (under 20 years of age) and older women (over 35 years of age). Survival status of the preceding child has been shown to be a potential confounding factor since the death of the preceding child before the conception of the index child may lead to a shorter birth interval as a result of premature cessation of breast-feeding or through volitional "replacement" behaviour. Causes of prematurity are said to be outside the scope of an individuals control, although this is being questioned in recent studies (Geronimus, 1986; Sowards, 1997).

Behavioural Factors: Under this category, the variables which are included in the analysis are breastfeeding, access and utilization of maternal and child health care services. Breastfeeding has been shown to be associated with child survival, especially at durations of less than six months. In the case of access and utilization of maternal and child health care services, it has been hypothesized that pregnant mothers with short birth intervals are also likely to have young children requiring care and attention, and thus they are unlikely to attend prenatal care or will attend later when their pregnancy is at advanced stage. This is in contrast to women with longer birth intervals. Education of mothers as mentioned in the literature review section is a catalyst in this process.

Household Exposure Factors: Factors in this category include source of water and type of toilet facility. It has been hypothesized that these factors are expected to increase the risk of potentially fatal diarrhoeal diseases and acute respiratory infection (ARI) in cases of

overcrowding and where the ventilation is inadequate. On the other hand, these variables might be related to socio-economic status of the household/family.

Unmeasured and/or Unobserved Heterogeneity: The assumption inherent in this category of factors is that siblings share the same social and physical environment and consequently any risks associated with that environment. They also share risks associated with family behaviour and child feeding practices relating to infant feeding, use of health care facilities and general standards of hygiene. Also, because they come from the same parents they may inherit any genetic problems that may result from this association. Apart from these, there are other important variables such as causes of death and health seeking behaviour of the woman which are not included in the survey questions.

Region of Residence: It has been hypothesized that different regions may have different ecological and climatic conditions which may in turn represent different patterns of disease load. Table 5.3.1 presents the summary of variable descriptions and their measurements.

Table 5.3.1: Summary of Variables and their measurements

Variable Name	Measurement	Remarks
Infant and Child Survival	0=Dead 1=Live	The Dependent Variable (Outcome)
Maternal Age	< 20 20-34 (reference Category) 35+	
Length of Preceding birth Interval	< 14 14-19 20+ (Reference Category)	
Receipt of Tetanaus Toxoid Injection	No=Not Received Yes=Received (Reference Category)	Used only in neonatal survival analysis
Premature Birth	Premature=Not on Time On Time (Reference Category)	Used only in neonatal survival analysis
Receipt of BCG Vaccination	No = not received Yes = Recived (Reference Category)	Used only in neonatal survival analysis
Birth Order	First 2-4 (Refernce Category) 5+	First Birth Order is used only in the analysis of Child Survival where the survival status of the preceding child is not included.
Survival status of Preceding Child	0 = dead 1 = a live (Reference Category)	
Place of Delivery	Home Health Institution (Reference Category)	Used only in neonatal survival analysis
Region of Residence	Region 1 = high Disease Prevalence Region 2 = Low disease Prevalence (Reference Category)	

Table 5.3.1 (continued)

Variable Name	Measurement	Remarks
Maternal Education	1 = No Education 2 = primary Incomplete 3 = Primary complete 4 = Secondary and Above (Reference Category)	
Source of Drinking water	Unsafe Safe (Reference Category)	Used in postneonatal, infant and child survival analysis only
Type of Toilet Facility	Poor Good (Refence Category)	Used in postneonatal, infant and child survival analysis only
Prenatal Care	No = Not Received Yes = Received (Reference Category)	Used in postneonatal, infant and child survival analysis only
Intrapartum Care	No = Not Received Yes = Recived (Reference Category)	Used in postneonatal, infant and child survival analysis only
Immunization	No = Not Immunized yes = Immunized (reference category)	Used in postneonatal, infant and child survival analysis only
Breast Feeding	Not Breastfed Breastfed (Reference category)	Used in postneonatal, infant and child survival analysis only

5.4 Results of Analysis

5.4.1 Univariate Logistic Regression Models

The first stage of analysis is a univariate analysis using logistic regression to show the relationship of each independent variable to the dependent variable (gross effects). The models are estimated separately for neonatal, postneonatal and infant and early childhood periods. Tables 5.4.1 to 5.4.3 display the results. Univariate logistic regression is preferred over cross tabulations because it not only shows the association between two variables but also shows the direction of the association.

It can be seen from the tables that the variables that are associated with the child survival in the neonatal period are maternal age, receipt of BCG vaccination, prematurity (preterm) status, birth order and survival status of the preceding child. The variables with high predictive power in the univariate models are receipt of BCG vaccination and prematurity status respectively. It is important to note here that these two variables may be interrelated in that receipt of BCG might be as a result of premature birth and subsequently death. Thus, these variables suggest the influence of both biological and behavioural factors on survival status of the infant in the neonatal period.

In the postneonatal period, the variables that appeared to be associated with the survival of the infant are breastfeeding status, receipt of immunization, region of residence where there is high malaria prevalence, prenatal care, type of toilet facility, length of preceding birth interval, survival status of the preceding child, level of education and prematurity status. There is also a weak association of intra-partum care with survival status of the infant in the

postneonatal period. The variables that are highly significant in this period are breastfeeding, immunization and region of residence. These initial results suggest that behavioural and environmental factors may be crucial in determining the chances of survival of the child in this period.

In the infant period, birth order, prenatal care, immunization, survival status of the preceding child (dead), breastfeeding, prematurity status, type of toilet facility, intrapartum care, level of education, region of residence and lenghth of preceding birth interval are associated with survival status of the infant. The variables which appear to be important in this period are the same as those in the postneonatal period. In the infant period which combines both the neonatal and postneonatal stages, it is observed that biological, behavioural, social environment and ecological zone may play crucial roles in determining the survival status of the child.

On the basis of foregoing univariate results and the theoretical understanding of relationship between various variables and infant and child survival, multivariate logistic models are estimated. It may be noted here that although some variables which were not associated with the risk of survival in infancy and early childhood in the univariate analysis, they are still included in the multivariate analysis with the presumption that their presence may improve the fit of the model or when controlled for in a multivariate model, they may become important predictors of infant and child survival.

Table 5.4.1: Univariate Logistic Regression Models for Survival in the Neonatal Period, Kenya, 1993

Variable	ß	S.E	$e^{ß}$	Loglikel-ihood	Chi-Square	p-value	Pseudo-r^2
Maternal Age < 20	.11	.52	1.11	789.49	8.49	.834	
35+	-.75	.25	.47	789.49	8.49	.003	1.1
Preceding Birth Interval							
<14	-.17	.25	.84	807.25	3.68	.517	
14-19	.44	.31	1.56	807.25	3.68	.149	0.4
Receipt of Tetanus Toxoid (No)	-.48	.34	.62	732.68	1.71	.167	0.2
Prematurity (Premature)	-2.73	.26	.07	765.33	79.01	.000	10.3
Receipt of BCG (No)	-5.12	.40	.01	789.40	355.61	.000	45.0
Birth Order							
5+	-.63	.22	.53	830.23	8.18	.000	1.0
Survival Status of Preceding Child (Dead)	-1.31	.24	.27	830.23	24.60	.000	3.0
Place of Delivery (Home)	-.16	.24	.85	773.05	.44	.505	0.1
Region of Residence (Region 1)	-.31	.22	.73	830.23	2.07	.151	0.2
Maternal Education							
No Education	-.41	.39	.66	680.11	5.39	.291	
Primary Incomp	-.19	.37	.85	680.11	5.39	.651	
Primary Comple	.42	.44	1.52	680.11	5.39	.344	0.8

NB: Omitted categories are the reference or Baseline categories see Table 5.3.1

Table 5.4.2: Univariate Logistic Regression Models for Survival in the Postneonatal Period, Kenya, 1993

Variable	ß	S.E	$e^{ß}$	Loglikel-ihood	Chi-Square	p-value	Pseudo-r^2
Maternal Age: <20	.58	.52	1.79	968.30	1.52	.528	
35+	.06	.27	1.06	968.30	1.52	.261	0.2
Preceding Birth Interval							
<14	-.77	.21	.46	1037.25	15.65	.000	
14 -19	.06	.26	1.07	1037.25	15.65	.804	1.5
Source of Drinking water (unsafe)	-.21	.21	.81	1036.41	1.02	.319	0.1
Prematurity (Premature)	-.98	.38	.37	903.79	5.21	.010	0.6
Type of toilet Facility (Poor)	-.96	.20	.38	1036.67	20.58	.000	2.0
Birth Order							
4-6	-.33	.24	.72	1037.25	1.83	.176	0.2
7+	-.16	.22	.86	1037.25	1.83	.487	
Survival Status of Preceding Child (Dead)	-.82	.24	.32	1037.25	.44	.000	0.04
Intrapartum Care (No)	-.36	.21	.69	1037.25	3.10	.086	0.3
Region of Residence (Region 0)	-1.36	.21	.26	1037.25	48.67	.000	4.7
Maternal Education							
No Education							
Primary Incomp	-.36	.36	.69	1037.25	14.02	.305	
Primary Comple	-.80	.32	.45	1037.25	14.02	.012	1.4
	.00	.36	1.00	1037.25	14.02	.990	
Prenatal Care (No)	-.54	.25	.58	1037.25	4.18	.031	0.4
Immunization (No)	-2.01	.20	.13	960.23	91.04	.000	9.5
Breastfeeding (Not Breastfed)	-3.78	.25	.02	1657.40	271.46	.000	16.4

NB: Omitted categories are the reference or Baseline categories see Table 5.3.1

Table 5.4.3: Univariate Logistic Regression Models for Survival in Infancy, Kenya, 1993

Variable	ß	S.E	$e^{ß}$	Loglikel-ihood	Chi-Square	p-value	Pseudo-r^2
Maternal Age:							
<20	.14	.33	1.15	1489.15	3.52	.668	
35+	-.24	.18	.71	1489.15	3.52	.070	0.2
Preceding Birth Interval							
<14							
14-19	-.68	.16	1.98	1588.07	22.61	.000	
	.82	.20	2.28	1588.07	22.61	.000	1.4
Source of Drinking water (Unsafe)	-.07	.15	.93	1586.57	.22	.640	0.01
Prematurity (Premature)	-1.89	.23	.15	1393.49	47.72	.000	3.4
Type of toilet Facility (poor)	-.79	.16	.45	1587.03	22.35	.000	1.4
Birth Order							
4-6	-.45	.18	.64	1588.07	6.06	.014	
7+	-.23	.17	.79	1588.07	6.06	.178	0.4
Survival Status of Preceding Child (Dead)	-.90	.18	.41	1588.07	21.79	.000	1.4
Intrapartum Care	-.31	.16	.73	1588.07	3.94	.052	0.3
Region of Residence (Region 0)	-.93	.15	.39	1588.07	41.35	.000	2.6
Maternal Education							
No Education							
Primary Incomp	-.57	.26	.56	1588.07	17.44	.025	
Primary Comple	-.58	.24	.56	1588.07	17.44	.014	1.1
	.09	.27	1.09	1588.07	17.44	.743	
Prenatal Care (No)	-.66	.18	1.94	1588.07	10.99	.000	0.7
Immunization (No)	-2.96	.24	.05	1483.15	260.91	.000	18.1
Breastfeeding (Not Breastfed)	-4.36	.26	.01	1385.55	309.42	.000	22.3

NB: Omitted categories are the reference or Baseline categories see Table 5.3.1

5.4.2 Multivariate Logistic Regression Analysis

Neonatal Mortality

Although previous research have shown that variation in the risk of neonatal mortality can be explained largely by biological factors as deaths in this period are mainly due to endogenous rather than exogenous factors (Curtis and Steele, 1996), it has been noted that biological explanations of the risk of neonatal mortality may not provide the sole explanation. Also as indicated in the univariate analysis, the factors showing the highest predictive power for survival of the child in this period are both biological and behavioural. Hence, in this section we hypothesize that the variations in the risk of neonatal mortality in Kenya are not only associated with biological factors but also behavioural factors linked with utilization of health care services (more specifically preventive services such as receipt of tetanus toxoid injection during pregnancy and receipt of BCG vaccination). Utilization of services may also be linked with the social status of the mother, a product of her educational level and region of residence.

Table 5.4.4 presents the distribution of births and neonatal deaths by each of the explanatory variables used in the analysis. In this context, we have first fitted the biological and the behavioural models separately and then fitted a combined model consisting of both biological and behavioural variables. The analysis consisted of 3681 births, of which 69 died in the neonatal period. The missing cases have been omitted from the analysis. It is important to note here that the models are fitted without controlling for familial effects and unobserved/unmeasured factors.

Table 5.4.4: Distribution of Births and Neonatal Deaths by each explanatory variable

Explanatory Variables	Births		Deaths	
	Number	Percent	Number	Percent
Maternal Age				
<20	213	5.9	3	4.3
20-34	2649	73.3	45	65.2
35+	533	14.8	17	24.6
Missing	217	6.0	4	5.8
Maternal Education				
No Education	752	20.8	20	29.0
Primary Incomplete	1344	37.2	28	40.0
Primary Complete	948	26.2	11	15.9
Secondary & Above	568	15.7	10	14.5
Preceding Birth interval				
<14	1912	51.9	36	52.2
14-19	855	23.2	21	30.4
20+	914	24.8	12	17.4
Receipt of Tetanus				
No	288	7.9	10	14.5
Yes	3033	84.0	48	69.6
Missing	291	8.1	11	15.9
Receipt of BCG				
No	201	5.6	59	85.5
Yes	3192	88.4	6	8.7
Missing	219	6.1	4	5.8
Birth Order				
2-4	1912	52.6	26	37.7
5+	1700	47.4	43	62.3
Survival Status of Preceding Child				
Dead	372	10.3	17	24.6
A live	3240	89.7	52	75.4
Premature Birth				
Premature	94	2.6	19	27.5
on Time	3269	90.5	43	63.3
Missing	249	6.9	7	10.1
Region of Residence*				
Region 1	1660	46.0	36	52.2
Region 2	1952	54.0	33	47.8
Place of Delivery				
Home	2180	60.4	43	62.3
Health Instit.	1186	32.8	20	29.0
Missing	246	6.8	6	8.7

* 1= Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
2= Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.

Table 5.4.5 displays the parameter estimates and standard errors for the standard logistic regression model for neonatal mortality. The table also presents the exponential of the regression coefficients of the logistic model so as to assess the likely effects of a set of biological and behavioural variables on the dependent variable (survival status in the neonatal period). All independent ariables in the model are categorical (i.e. there is no continuous variables). Hence, parameter estimates may be interpreted as effect of each of these variables on the logit (log odds) of the dependent variable when the explanatory variable takes a different value from the reference category.

The biological model indicates that survival status of the preceding child, prematurity status and birth order have a negative significant effect on the survival chances of neonates. Notably these variables retain their significance as in the univariate model, whereas survival status of the preceding child is highly significant (1 percent level of significance) in the univariate model, it remains significant but at 5 percent level with the introduction of other explanatory variables in the multivariate model.

The behavioural model shows that non-receipt of BCG vaccination had a negative significant effect on the survival in the neonatal period. Although the effect of BCG is in the expected direction, one has to be cautious about its interpretation here because the infants who may have died prematurely and those who died in the first day of life and born outside a health institution may not have received this vaccination. Women who have no education show a positive significant relationship on survival in the neonatal period. Similarly, not all variables which are not significant in the univariate become significant under the multivariate set up e.g. having no education.

Table 5.4.6 presents the results of a combined model which includes both biological and behavioural variables. Most of the variables which are significant in the biological and behavioural models persist to be significant in the combined model too. However, birth order which is significant in the individual biological model, is insignificant in the combined model. This implies that the effect of this variable on neonatal survival may be acting through maternal behavioural factors, particularly when utilization of maternal and child health care service variables are included. This may possibly be explained by the fact that a mother becomes more careful with the care of the index child to avoid the possibility of another death. Also, social learning takes place as mothers become more experienced with child rearing i.e. they learn about childrearing from various social groups. Although receipt of BCG is highly significant in the individual behavioural model as well as in the combined model, the relationship may be spurious, because when the infant dies in early neonatal period or prematurely, this may not have been administered. However in this analysis, it can be taken as an important indicator of utilization of health care services.

Table 5.4.5: Parameter estimates and standard errors for the standard logistic regression model for neonatal mortality, Kenya, 1993

	Biological Model			Behavioural Model		
Variable	B	S.E	Exp (B)	B	S.E	Exp (B)
Maternal Age						
< 20	0.07	.66	1.08			
20-34	0.00	-	1.00			
35+	-.48	.38	.62			
Preceding Birth Interval						
<14						
14-19	.22	.30	1.24			
20+	.32	.37	1.37			
	0.00	-	1.00			
Receipt of Tetanus Toxoid						
No				.47	.41	1.61
Yes				0.00	-	1.00
Prematurity						
premature	-2.70***	.30	.07			
On Time	0.00	-	1.00			
Receipt of BCG						
No				-5.55***	.47	.00
yes				0.00	-	1.00
Birth Order						
2-4	0.00	-	1.00			
5+	-.61*	.31	.54			
Survival status of Preceding Child						
Dead	-.81*	.34	.44			
A live	0.00	-	1.00			
Place of Delivery						
Home				.54	.36	1.73
Health Inst.				0.00	-	1.00
Region of Residence						
region 0				.27	.32	1.30
Region 1				0.00	-	1.00
Maternal Education						
No Education				1.17*	.54	3.21
Primary Incomplete				.72	.50	2.06
Primary Complete				.77	.57	2.17
				0.00	-	1.00
constant	4.63***	.32	-	5.21***	.53	-

+ 1= Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
2= Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.
* p<.05 ** p<.01 *** P<.001

Table 5.4.6: Parameter estimates and standard errors for the standard logistic regression model for neonatal mortality, Kenya, 1993

Combined Model

Variable Name	B	S.E	Exp(B)
Maternal Age			
<20	-1.00	.79	.37
20-34	0.00	-	1.00
35+	-.17	.42	.85
Maternal Education			
No education	1.20*	.62	3.32
Primary Incomplete	.75	.56	2.13
Primary Complete	.58	.61	1.78
Secondary & Higher	0.00	-	1.00
Preceding Birth Interval (months)			
<14	-.04	.41	.96
14 - 19	.72	.47	2.06
20+	0.00	-	1.00
Receipt of Tetanus Toxoid			
No	.42	.43	1.52
yes	0.00	-	1.00
Received BCG Vacc.			
No	-5.45***	.49	.004
yes	0.00	-	1.00
Birth Order			
2 - 4	0.00	-	0.00
5+	-.08	.41	.92
Survival Status of Preceding Child			
Dead	-.68	.44	.51
A live	0.00	-	1.00
Premature Birth			
premature	-1.88***	.45	.15
On Time	0.00	-	1.00
Region of Residence			
region 0	.31	.35	1.36
region 1	0.00	-	1.00
Place of Delivery			
Home	.47	.39	1.60
Health Institution	0.00	-	1.00
Constant	5.53***	.58	-

+ 1= Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
2= Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.
* p<.05 ** p<.01 *** P<.001

Postneonatal Mortality

There were 3681 births of which 117 died in the postneonatal period and also 3681 births of which 206 had died in the infant period. Table 5.4.7 provides the distribution of births and postneonatal and infant deaths by each potential explanatory variable used in the analysis.

Table 5.4.8 shows parameter estimates and standard errors for survival in the postneonatal period and infancy. The factors which appear to be associated with survival in the postneonatal period are region of residence, level of education, immunization status, length of preceeding birth interval, type of toilet facility, breastfeeding and prematurity status, which all show a negative significant effect on the risk of survival in this period. Region of residence here relates to the division of the fifteen rural districts used in the analysis into two distinct groups based on their ecological zones and disease patterns. Those coded as '1' are regions where malaria is endemic. The high negative significant effect of the region of residence could be an indication that regional variations in survival chances are more pronounced in the postneonatal survival period. Similarly, the negative significant effect of type of toilet facility may be an indication of the general poor social environment in which the infant is reared. Further, the negative significant effect of breastfeeding could be an indication that it may not have been initiated before the death of the infant.

While most of the variables which appeared to affect survival chances in the postneonatal period in the univariate model persisted to be significant in the multivariate models, survival status of the preceding child and prenatal care which were significant in the univariate model, are not significant in the multivariate model. These variables may be highly correlated with

other explanatory variables included in the multivariate model. For example, survival status of the preceding child may be highly correlated with preceding birth interval more specifically, short birth interval. If the preceding child dies, the index child will be at an increased risk of death which in turn would lead to short preceding birth interval . Three mechanisms through which the short birth and survival status of preceding child operate to influence infant survival have been hypothesized in the literature (Pebley et al., 1991). The one which is of interest here is that short birth intervals may not allow sufficient time for recovery from the demands of pregnancy and lactation, which would lead to physiological depletion of the mother. This, in turn, is likely to damage the health of the child born after a short birth interval. Intrapartum care, which was significant in the univariate analysis at 10 percent level also ceased to be significant under the multivariate set up. In this case, prenatal care and intrapartum care may be highly correlated with the region of residence. In conclusion, the factors which appear to be affecting the risk of survival in the postneonatal period are both biological and behavioural, and to some extent, those indicative of the social environment of families.

Infant Survival

In the infant survival ~~period~~ period the variables that appear to have some effect are region of residence, immunization status, lenghth of preceding birth interval, type of toilet facility prematurity status and breastfeeding status, all have a negative significant effect on the risk of survival in infancy. Similarly, the high negative significance of the region of residence could be an indication that regional variations in disease patterns and social endowments are also evident. Further, negative significance of type of toilet facility may be an indication of

the general poor social environment in which the infant is reared. Apart from breastfeeding which appears to be important for the survival of the infant and was not important in the neonatal period, the pattern displayed here is similar to that of the postneonatal period and to some extent the neonatal period. Behavioural and environmental factors appear to exert more influence on infant survival.

Table 5.4.7: Distribution of Births and Postneonatal and Infant Deaths, by each explanatory variable

Explanatory Variables	Births				Deaths			
	Postneonatal		Infancy		Postneonatal		Infancy	
	Number	Percent	Number	percent	Number	Percent	Number	Percent
Maternal Age								
<20	212	5.9	206	5.9	4	3.4	10	5.2
20-34	2606	73.1	2551	73.4	88	75.2	143	69.4
35+	533	15.0	510	14.7	17	14.5	40	19.4
Missing	213	6.0	208	6.0	8	6.8	13	6.3
Maternal Education								
No Education	749	21.0	719	20.7	23	19.7	53	25.7
Primary Incomplete	1310	36.8	1277	36.7	62	53.0	95	46.1
Primary Complete	939	26.3	924	26.6	20	17.1	35	17.0
Secondary & Above	566	15.9	555	16.0	12	10.3	23	11.2
Preceding Birth interv.								
<14	809	22.7	1821	52.4	46	39.3	91	44.2
14-19	892	25.0	778	22.4	22	18.8	77	37.4
20+	1863	52.3	876	25.2	49	41.9	38	18.4
Prenatal Care								
No	281	10.3	365	10.4	20	17.1	38	18.4
Yes	3183	89.7	3112	89.6	97	82.9	168	81.6
Intrapartum Care								
No	2346	65.8	2283	65.7	31	26.5	149	72.3
Yes	1218	34.2	1192	34.3	86	73.5	57	27.7
Missing								
Birth Order								
2-4	1881	52.8	1848	53.2	57	48.7	90	43.7
5+	1683	47.2	1627	46.8	60	51.3	116	56.3
Survival Status of Preceding Child								
Dead	365	10.2	345	9.9	24	20.5	44	21.4
A live	3199	89.8	3130	90.1	93	79.5	162	78.6
Premature Birth								
Premature	105	2.9	86	2.5	8	6.8	27	13.1
on Time	3220	90.3	3162	91.0	92	78.6	150	72.8
Missing	239	6.7	227	6.5	17	14.5	29	14.1
Region of Residence*								
Region 1	1374	38.6	2144	61.7	83	70.0	126	61.2
Region 2	2190	61.4	1331	38.3	34	30.0	80	38.8
Immunization								
No	406	11.4	1044	30.0	55	47.0	19	9.2
Yes	2927	82.1	2221	63.0	53	45.3	173	84.0
Missing	258	6.5	210	6.0	9	7.7	14	6.8
Breastfeeding								
Not Breastfed	86	2.4	24	0.7	3	2.6	65	31.6
Breastfed	3220	90.3	3205	92.2	96	82.1	111	53.9
Missing	258	7.2	246	7.1	18	15.4	30	14.6
Source of Drinking Water								
Unsafe	1126	31.6	2366	68.1	32	27.4	144	69.9
Safe	1242	68.0	1096	31.5	85	72.6	62	30.1
Missing	13	0.4	13	0.4	-	-	-	-
Type of Toilet Facility								
Poor	589	16.5	567	16.3	40	34.2	62	30.1
Good	2966	83.1	2899	83.4	77	65.8	144	69.9
Missing	9	0.3	9	0.3	-	-	-	-

* 1 Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
2 Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.

Table 5.4.8. Parameter estimates and standard errors for the standard logistic regression model for survival in the postneonatal and infancy, Kenya, 1993

Variable	Postneonatal Model B	S.E	Exp (B)	Infant Survival Model B	S.E	Exp (B)
Maternal Age						
<20	.74	.55	2.09	.07	.45	1.07
20-34	0.00	-	1.00	0.00	-	1.00
35+	.15	.33	1.17	-.04	.31	.96
Preceding Birth Interval						
<14	-.83**	.26	.43	-.82	.24	.44
14-19	.01	.28	1.01	-.04	.26	.96
20+	0.00	-	1.00	0.00	-	1.00
Prenatal care						
No	.68	.50	1.96	-.55	.43	.58
Yes	0.00	-	1.00	0.00	-	1.00
Prematurity						
premature	-.96*	.48	.38	-1.01*	.43	2.75
On Time	0.00	-	1.00	0.00	-	1.00
Intrapartum Care						
No	.26	.25	1.29	.21	.22	1.23
yes	0.00	-	1.00	0.00	-	1.00
Birth Order						
2-4	0.00	-	1.00	0.00	-	1.00
5+	.09	.25	1.09	.11	.32	1.11
Survival status of Preceding Child						
Dead	-.02	.30	.98	-.03	.27	.96
A live	0.00	-	1.00	0.00	-	1.00
Source of Drinking Water						
Safe						
Unsafe	.02	.25	1.02	-.22	.21	.79
	0.00	-	1.00	0.00	-	1.00
Region of Residence						
region 0	-1.43***	.25	.24	-1.29***	.22	.27
Region 1	0.00	-	1.00	0.00	-	1.00
Maternal Education						
No Education	-.18	.47	.83	-.21	.40	.81
Primary Incomplete	-.83*	.40	.43	-.53	.36	.59
Primary Complete	-.26	.43	.77	-.06	.38	.94
Secondary and higher	0.00	-	1.00	0.00	-	1.00
Immunization						
No	-1.92***	.24	.15	-2.73***	.28	.06
yes	0.00	-	1.00	0.00	-	1.00
Type of Toilet Facility						
poor	-.74**	.24	.48	-.69	.22	.50
Good	0.00	-	1.00	0.00	-	1.00
Breastfeeding						
Not Breastfed	- 1.56*	.68	.02	-4.36***	.34	.01
Breastfed	0.00	-	1.00	0.00	-	1.00
Constant	5.45***	.55	-	6.04***	.74	-

* p<.05 ** p<.01 *** P<.001

The next section briefly examines factors that are said to influence survival in childhood. However, given that the majority of deaths used in this study occurred in infancy, further analysis will be restricted to these period.

Survival in Childhood

There were 2965 births in this period of whom 96 had died in childhood. A further censoring had to be done in this period. All those births which occurred in the year of the interview and had not had enough exposure time were removed from the analysis (see Table 5.5.6)

Table 5.4.9: Distribution of Births and Child Deaths by each explanatory variable

Explanatory Variables	Births		Deaths	
	Number	Percent	Number	Percent
Maternal Age				
<20	358	12.1	22	22.9
20-34	2192	73.8	51	53.1
35+	418	14.1	13	13.5
Missing	1	0.0	10	10.4
Maternal Education				
No Education	580	17.2	25	26.0
Primary Incomplete	1054	19.5	38	39.6
Primary Complete	824	35.5	25	26.0
Secondary & Above	511	27.8	8	8.3
Prenatal Care				
No	123	4.1	21	21.9
Yes	2846	95.9	75	78.1
Intrapartum Care				
No	2795	60.5	71	74.0
Yes	1174	39.5	25	26.0
Birth Order				
First	305	10.3	14	14.6
2-4	1436	48.4	37	38.5
5+	1228	41.1	45	46.9
Premature Birth				
Premature	79	2.7	4	4.2
on Time	2874	96.8	73	76.0
Missing	16	0.5	19	19.8
Region of Residence+				
Region 1	1864	62.8	68	70.8
Region 2	1105	37.2	28	29.2
Immunization				
No	659	22.2	25	26.0
Yes	2307	77.7	66	62.5
Missing	3	0.1	11	11.5
Breastfeeding				
<10 months	261	8.8	5	5.2
10-56	2650	89.3	72	75.0
Missing	58	2.0	19	19.8
Source of Drinkng water				
unSafe	2001	67.4	67	69.8
Safe	954	32.1	28	29.2
Missing	14	0.5	1	1.0
Type of Toilet Facility				
Poor	470	27.1	26	27.1
Good	2491	83.4	69	71.9
Missing	8	0.3	1	0.3

+ 1= Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
2= Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.

Table 5.4.10 shows the parameter estimates and standard errors for the logistic standard model for survival in the childhood period. The factors that appear to be associated with survival in this period are maternal age, region of residence and type of toilet facility. These variables are all negatively significant, indicating lower survival chances for children of women in these categories. These variables could be said to be highly associated with child rearing practices as well as social environment in which the child may be brought up (raised).

Table 5.4.10: Parameter Estimates and Standard Errors for the Standard Logistic regression model for Survival in Childhood Kenya, 1993

Childhood Survival

Variable	B	S.E	Exp (B)
Maternal Age			
<20	-1.14***	.36	.32
20-34	0.00	-	1.00
35+	.04	.37	1.04
Prenatal care			
No	-.37	.74	.68
Yes	0.00	-	1.00
Intrapartum Care			
No	-.14	.26	.87
yes	0.00	-	1.00
Birth Order			
First	.08	.40	1.09
2-4	0.00	-	1.00
5+	-.25	.49	.77
Source of Drinking Water			
Safe			
Unsafe	-.18	.25	.84
	0.00	-	1.00
Region of Residence			
region 0	-1.50***	.26	.22
Region 1	0.00	-	1.00
Maternal Education			
No Education	-.66	.48	.51
Primary Incomplete	-.56	.42	.57
Primary Complete	-.26	.44	.76
Secondary and higher	0.00	-	1.00
Immunization			
No	-.31	.29	.73
yes	0.00	-	1.00
Type of Toilet Facility			
poor	-.54*	.27	.58
Good	0.00	-	1.00
Breastfeeding			
Not Breastfed	.38	.48	1.47
Breastfed	0.00	-	1.00
constant	6.04***	.95	-

+ 0= Kilifi, Kisii/Nyamira, Siaya, South Nyanza, Bungoma, Kakamega
1= Nyeri, Murang'a, Taita/Taveta, Meru, Machakos, Nakuru, Nandi, Kericho, Uasin Gishu.
* p<.05 ** p<.01 *** P<.001

5.5 Effect of maternal education on Survival in Infancy

In this section, we examine the influence of maternal education on infant and child survival as a proxy for information and skills associated with the uptake of maternal and child health care services and general health care utilization in rural Kenya. The analyses to date have shown that maternal education is associated with behaviour that influence survival at various stages of infancy and that it acts mainly through behavioural factors. We have therefore assessed the effect of education on infant survival in Kenya at different stages of infancy when other variables are controlled for, assuming that the risks in individuals are uniformly distributed (homogenous) in factors that have been omitted so that the addition of these factors and the resultant variability in parameter estimates, is an indication of heterogeneity which has not been fully controlled for, in the present model.

We consider four categories of maternal education namely, no education, primary incomplete, primary complete and secondary and above in order to assess the effect maternal eduction on survival in the neonatal period.

Maternal education and neonatal survival

Table 5.5.1. presents the relative risk of the effect of maternal education on the risk of survival in the neonatal period in Kenya, 1993 according to the categories stated above. The results indicate that the odds of survival of infants of mothers with primary complete, in the neonatal period is 1.52 times greater than the baseline category (i.e., secondary and above) while the odds of survival of infants of mothers with primary incomplete, does not

differ from the baseline category (model 1, Gross effects). However the odds of survival for infants of mothers with no education, is marginally lower than the baseline category.

Addition of biological/family formation variables in model 2 shows that there is marginal change in the relative risk of survival of infants in the neonatal period for mothers with primary incomplete level of education. For the no education category, the odds of survival is almost the same as the baseline category while for primary complete which showed higher odds of survival in model 1, the odds decreases by about 20 percent. The changes observed with addition of these control variables, may be an indication that some of the added variables partially account for the effect of education on survival of the infant in the neonatal period although the differences are not statistically significant as in model 1.

However, addition of behavioural factors relating to utilization of health care services in model 3, substantially shifts the relative risk estimates for all categories of education upwards. Further, the odds of survival for neonates of women with no education is significant (at 5 percent level). The pattern displayed here could be an indication that if health campaigns (immunisation and information on maternal and child health care) had been successful, then the knowledge and skills relating to uptake of maternal and child health care services and general utilization of health services may be the same for all the women regardless of educational attainment, hence the comparative advantage of those with higher education may now be negligible.

Similarly, we have never assessed the impact of health/educational campaigns on women with no education. It may be case that those with no education take the messages more seriously

than educated women. It has also been argued that the continued advantage for women with no education with regard to infant and child survival, could be associated with continued observance or adherence to traditional taboos such as prolonged breastfeeding, longer birth intervals (Oppong and Abu, 1987; Pebley and Mbugua, 1989) and traditional methods of prenatal care (Amankwaa, 1996) which may be beneficial to the neonates. However, we have not yet accounted for income levels and community effects such as the contextual effects of education or the spill-over effect of knowledge from those with education to their relatives or peers without education. Income may be one of the key factor explaining differential utilization of health care more than education in Kenya given the introduction of fee based "cost-sharing" in health care by the government.

Addition of region of residence in model 4 marginally shifts the relative risk estimates for no education and primary complete levels of education and no change for primary incomplete. The odds of survival for neonates of women with no education still remain significant. Mott (1979) who used Kenya Fertility Survey Data (KFS) also noted that for older women, the infant deaths of better educated women are likely to occur in the neonatal period.

Addition of prematurity in model 5 reduces the relative risk estimates for all educational categories. The most noticeable change is that none of the estimates are significant. This may be an indication that prematurity picks up the influence of some of the variables that were introduced in models 3 and 4.

Table 5.5.1: Relative risk of neonatal survival associated with maternal education

Maternal Education	Model 1	Model 2	Model 3	Model 4	Model 5
No Education	.66	.98	3.22*	3.13*	2.50
Primary Incomplete	.85	.84	2.05	2.05	1.70
Primary Complete	1.52	1.29	1.91	1.96	1.59

Baseline Category=Secondary and above
Source: Derived From KDHS, 1993 (* p<.05) (** p<.001) (*** p<.0001)

Model 1 = Gross effects of maternal education
Model 2 = Addition of biological/family formation factors(age, preceding birth interval, birth order and survival status of the preceding child)
Model 3 = Addition of behavioural factors relating to utilization of maternal and child health care services (receipt of tetanus toxoid, BCG vaccination and place of delivery)
Model 4 = addition of region of residence (region 0 = high mortality districts and region 1 = low mortality districts)
Model 5 = Addition of Prematurity

Maternal education and Postneonanal Survival

The same procedure used to assess the effects of maternal education on neonatal survival, is also adopted in the case of postneonatal survival. Table 5.5.2 shows the relative risk of survival in the postneonatal period associated with maternal education. The effect of maternal education differs substantially from that observed in the neonatal period. The gross effects of maternal education in model 1 show that the relative risk estimates for infants of women with no education and those who did not complete primary level of education are less than one implying that the survival chances of infants of women in these educational categories are lower than those in the baseline category. However, only the estimate for primary incomplete level of education is statistically significant. For infants of women who completed

primary level of education, the survival chances are the same as the baseline category.

Table 5.5.2: Relative Risk of Survival in the postneonatal period associated with maternal education

Maternal Education	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
No Education	.69	.62	.96	.75	.91	.80	.82
Primary Incomplete	.45*	.38**	.46*	.40*	.46*	.41*	.43*
Primary Complete	1.00	.79	.85	.69	.73	.76	.77

Baseline Category=Secondary and above
Source: Derived From KDHS, 1993 (* $p<.05$) (** $p<.001$) (*** $p<.0001$)

Model 1 = Gross effects of Education on Postneonatal survival
Model 2 = Addition of biological/family formation factors
Model 3 = Addition of Health Care utilization Factors (Prenatal, intrapartum care and Immunisation)
Model 4 = Addition of Region of Residence
Model 5 = Addition of Household exposure factors (source of water and type of toilet facility)
Model 6 = Addition of Breastfeeding
Model 7 = Addition of Prematurity

Addition of biological/family formation variables in model 2 shifts downward the relative risk estimates for all educational categories. Bicego and Boerma (1990) also found that addition of family formation variables tended to shift the estimated education effect further away from the null value of one suggesting that in some countries (Kenya in particular), no or low education is correlated with safer categories of family formation variables. These findings are consistent to some extent with that of Bicego and Boerma (1990). However, only infants of women with primary incomplete level of education have significantly lower odds of survival in the postneonatal period.

As in the case of neonatal period, addition of behavioural factors associated with the utilization of health care services shifts the estimates upwards substantially for all categories of education. The effect is significant only for estimates of primary incomplete level of education. The odds of survival for children of women with no education and primary complete categories do not differ much from the baseline category.

Although the addition of region of residence in model 4 shifts the relative risk estimates for all educational categories downwards the pattern remains the same as n model 3. Addition of household exposure variables in model 5 shifts relative risk estimates for all educational categories upwards. The odds for no education category is again closer to one while those for primary incomplete and primary complete are pushed away from the null one. Only the estimate for primary incomplete level of education remains statistically significant.

In model 6 breastfeeding is added and we find that there are differential survival chances for infants of women with primary incomplete level of education when compared to other educational categories. The infants of women with primary complete and no education appear to have more or less the same survival chances as those of women with secondary and above level of education.

Addition of prematurity in model 7 changes the relative risk estimates for all educational categories marginally and follow more or the earlier pattern observed in model 6. In general the changes occurring in the relative riskr estimates, appear to be more pronounced for the no education category and the primary complete category. This could be an indication of the amount of variability which is not accounted for by the added variables. For the primary

incomplete category, the estimates remain significant throughout and without much variability. One noticeable aspect in the estimates for the primary incomplete category in this period, is the consistency. This is an indication that infants of women with primary incomplete level of education, have lower odds of survival than for the other educational categories. However, there are some marked attenuations in the no education and primary education categories.

Maternal education and infant Survival

This section assesses the effect of maternal education on the risk of survival in infancy controlling for other factors. The results are presented in Table 5.5.3. The gross effects in model 1 indicate that the odds of survival in infancy are significantly lower than among women with no education and primary incomplete levels of education as compared to women with higher levels of education. Further, the relative risk estimates are statistically significant for the two categories of education whilst those for primary complete level are not statistically significant. The odds of survival for infants of women with primary complete category of education are not significantly different than the baseline category i.e., secondary and above level of education.

Addition of biological/family formation variables in model 2 does not alter the already observed pattern in model 1. However addition of health care utilization factors in model 3, alters the patterns as observed in models 1 and 2 but none of the maternal educational categories is statistically significant. The parameter estimates for all categories of education have shifted upwards.

Whilst the odds of survival in infancy for no education and primary complete levels of maternal education are closer to one, those for primary incomplete are away from one. This may indicate that the odds of survival in infancy are lower for children of women with primary incomplete level of education than for other educational categories when the health care utilization variables are controlled for. The reasons given for the neonatal period could also apply in this case. The persistence of lower odds of survival for children of women with

primary incomplete level of education in this period and the postneonatal period, calls for further research. It has often been said that little knowledge is no knowledge indicating that little knowledge acquired by women in this educational category may not be adequate to make a difference to survival chances of their children. Of course, the question one may ask here is why should this pattern persist?

Addition of breastfeeding in model 6 and prematurity in model 7 shifts the relative risk estimates for all categories of education downward with the most noticeable change being in the estimates for primary incomplete level of education. Similarly none of the categories remain statistically significant to show variation in the odds of survival. The relationships in the estimates for breastfeeding and prematurity could be ambiguous given that infants who are born prematurely may have died before breastfeeding was initiated. The estimates in the models are fairly consistent especially for primary complete level of education. One may argue that the observed differences in the estimates for primary complete may be due to chance errors.

Table 5.5.3: Relative risk of survival in infancy associated with maternal education

Maternal Education	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
No Education	.56*	.59*	.96	.85	.94	.81	.81
Primary Incomplete	.56*	.53*	.77	.73	.76	.58	.59
Primary Complete	1.09	.94	1.16	1.07	1.06	.95	.95

Baseline Category=Secondary and above
Source: Derived From KDHS, 1993 (* p<.05) (** p<.001) (*** p<.0001)

Model 1 = Gross effects of Education on infant survival
Model 2 = Addition of biological/family formation factors

Model 3 = Addition of Health Care utilization Factors (Prenatal, intrapartum care and Immunisation)
Model 4 = Addition of Region of Residence
Model 5 = Addition of Household exposure factors (source of water and type of toilet facility)
Model 6 = Addition of Breastfeeding
Model 7 = Addition of Prematurity

5.6 Conclusion

So far we have examined factors that may affect infant survival in families in Kenya at each survival stage in infancy without accounting for any excess variation. In the neonatal and postneonatal periods, the variables with high predictive power are receipt of BCG, immunization and prematurity. As noted earlier, the high significance of receipt of BCG in the neonatal period could be spurious, and that prematurity may be the main factor that is independently explaining the lower risk of survival in this period. This therefore indicates that the causes of survival in this period may have their roots in both biological and behavioural factors. It is argued that the distinction that separates causes such as congenital malformation and prematurity from infectious disease which are environmentally determined, should be re-evaluated. Geronimus (1986) and Sowards (1997) have noted that maternal infectious conditions which could be easily prevented through maternal health care interventions, may be responsible for the preterm labour that precipitates some premature births. Studies by Goldenberg (1996), Hauth et al., (1995) have supported this view. These findings have implications on strategies for intervention of preventing premature births, the majority of which appear to occur in the neonatal and postneonatal periods.

In infancy where the combined effects of neonatal and postneonatal plus deaths occurring in

the twelfth month are examined, biological, behavioural and environmental factors become more apparent. The effects of maternal education do not appear to be important here although the parameter estimates are in the expected direction. This could imply that maternal education may be acting through other variables which are not included here such as income and partner's educational level.

With regard to survival in childhood, the effect of maternal age (more specifically young age), which was not important in the earlier periods, become apparent. This may possibly be an indication of lack of experience in child rearing practices by these young women, who are having their first births. The effect of the social environment as proxied by type of toilet facility and region of residence are prominent in this period.

From the analysis of the effect of maternal education on survival at different stages of infancy, the relative risk estimates for women of all educational categories are not statistically significant for the gross effects and when the biological/family formation variables are included in the model. However, when utilization of health care factors are included in the model, there is a substantial change in the relative risk estimates for no education category and the estimate is significant indicating that children of women with no education have better survival chances than even those of women with secondary and above levels. This pattern does not however, conform to the findings of the majority of studies from other areas which have shown an inverse relationship between maternal education and level of infant and child survival, but is consistent with other studies carried out in Kenya (Boerma and Bicego, 1991). Hobcraft (1993) observed that this pattern may be due to deficiencies in the data. However no explanation exists to suggest why this phenomenon should be persistent in all

data sets so far used. It is important to note that in the neonatal period where health technology is crucial to the survival of neonates, the advantages of maternal education may not be important, rather, the household's social status may be the crucial factor (i.e, whether they can afford the available technology or not). It seems as earlier noted, that possibly the education advantage may not be important in this period.

In the postneonatal period, the odds of survival is significantly and consistently lower for infants whose mothers have primary incomplete level of education in all the models considered. One noticeable aspect of the relative risk estimates for all the categories of maternal education, are their low values compared with those for the neonatal period. The question one may ask is whether this could be an indication that the model may be inadequate in estimating the parameters (i.e., the standard errors are too large and hence the confidence intervals too narrow)? The relative risk ratios also indicate that survival chances are different with educational levels, however the non-significance of some of the estimates (for no education and primary complete) does not conform to the pattern suggested in literature. Is this due to chance errors only and/or a result of omitted variables (unobserved and/or unmeasured)?

The relative risk estimates for survival in infancy show a pattern that lies between that of neonatal and postneonatal periods. The results indicate when a wider grouping is considered may possibly minimise age misstatements but the issues raised above still persist. In the next chapter we examine factors affecting infant survival in families in Kenya when familial effects and unobserved/unmeasured factors are controlled for.

CHAPTER 6

FACTORS AFFECTING INFANT AND CHILD SURVIVAL IN FAMILIES IN KENYA: ANALYSIS INCLUDING RANDOM EFFECTS

6.1 Introduction

In the previous chapter we examined factors affecting infant and child survival in families in Kenya using the standard logistic regression model. The standard logistic regression assumes that each child is an independent observation. This assumption may not always hold (Curtis et al., 1993; Guo, 1993; Zenger, 1993; Das Gupta, 1990 and 1997). Ignoring completely the dependence among observations of siblings of the same family may lead to standard errors that are understated and parameter estimates that are both biased and inconsistent when estimating non-linear models (Sastry, 1997; Wolpin, 1997). In other words, estimates obtained under the standard logistic regression although may be reasonable, have too small variances leading to spurious results. Also, the standard logistic regression does not take into consideration the unobserved and/or unmeasured heterogeneity.

Having examined the presence of death clustering among families in chapter four, in this chapter, we examine factors affecting infant and child survival in families in Kenya taking into account sibling dependence and unobserved and/or unmeasured heterogeneity using logistic regression with random effects which is a generalised form of standard logistic regression model.

6.2 Methodology

The logistic regression model with randoma effect is applied to analyse the data on the occurence of neonatal, postneonatal and infant deaths with the data specifications remaining the same as in chapter five. The random effects models are natural generalization of the standard logistic models for analyzing proportions or dichotomous variables (relating to individual attributes). The model assumes that the baseline risk of death varies in families due to heterogeneity in mortality risks or due to unobserved and/or unmeasured heterogeneity. The model takes the following mathematical form:

$$\text{logit}(p_{ij}) = x'_{ij}\,\beta + u_i, \qquad \ldots (6.1)$$

where p_{ij} is the probability that the j^{th} child in the i^{th} family dies in the age intervals stated above, x_{ij} is the vector of covariates associated with the j^{th} child in the i^{th} family and ß is a vector of unknown parameters, u_i is the random effects associated with the i^{th} family. When the term u_i is assumed to be normally distributed with zero mean and variance σ^2 then we have the logistic-normal model (Pierce and Sands, 1975 and Anderson and Aitkin, 1985 cf EGRET Reference Manual, 1997). However, equation 6.1 can be modified to obtain a model of the form:

$$\text{logit}(p_{ij}) = x_{ij}\,\beta + \sigma\, v_i \qquad \ldots (6.2)$$

where v_i has the standard normal distribution and σ is the scale parameter indicating the amount of variation across families on the logit scale. This form is referred to as the logistic binomial model (Mauritsen, 1984 cf EGRET Reference Manual, 1997) and is applied in the present study. The extra variation postulated in this model namely the random perturbations may be described by the single extra parameter σ. This extra parameter is used to account

for global excess variation in the data. Sometimes, σ is set to be equal to $u_i\sigma_i$ where u_i is a vector of covariates associated with the i^{th} family (assumed to be omitted) and σ_i are parameters (excess variation being thought to differ by levels of these covariates). It is important to note that this parameter is slightly different from other parameters of the logistic regression model in that it can only be nonnegative which implies that with these models, one can only model too much as opposed to too little variation (EGRET Reference Manual, 1997).

Strategy for Fitting Models

When fitting the random effects models with a moderate to large number of parameters, there are strategies that may produce faster fits with fewer problems. The most acceptable strategy is to first fit all the fixed effects terms with no random effects terms in the model and then extend the model to include the random effects term or scale parameter denoted as %SCL. An alternative method to the above could be to first fit the model consisting of only the constant or grand mean (denoted by % GM) in the fixed effects model and the scale parameter of the random effects term (%SCL). This can then be extended by adding few fixed effects terms at each extension (EGRET Manual, 1997). For the present study, the second method is adopted by adding only one variable at each stage of the extension for the biological/family formation variables. The behavioural variables relating to the utilization of health care services and the household exposure variables (postneonatal and infant survival period only) are added to the models in a group while maternal education and region of residence are also added as single terms as control varibles. Models are fitted separately for neonatal, postneonatal and infant survival periods.

6.3. Results of Analysis

Neonatal Survival Period

Various models are fitted to the data for the neonatal survival period. Table 6.3.1 presents the results of the parameter estimates and standard errors for the logistic binomial models of neonatal survival. Model 1 shows the results for the effect of prematurity (in this case whether the birth was premature or not) with only the grand mean (constant) and the scale parameter (%SCL) for the random effects term. In model 2, birth order of the child (5+) is added whereas in model 3, survival status of the preceding child (dead) is added; in model 4, length of birth interval is added; in model 5, maternal age is added; model 6, the variables associated with utilization of health care services (receipt of BCG vaccination, receipt of tetanus toxoid injection and place of delivery) are added; in models 7 and 8 maternal education and region of residence are added respectively as the control variables.

It may be observed that in model 1, the variances are very large and as a result, the parameter estimates are not statistically significant. This may be due to the omission of a number of covariates. The scale parameter is close to one. In the subsequent models where a number of covariates are included, the magnitude of the estimates of the scale parameter becomes very small (near zero) except in model 6 where the health care variables are included. The effect observed in model 6 could be as a result of attenuation or it is possible that there could have been excess variation associated with utilization of health care services. In all the models, the scale parameters are not statistically significant.

In the earlier standard logistic regression model, we estimated the effects of the variables (covariates) without accounting for excess variation. When the standard models are compared with the random effects models (accounting for possible excess variation), the direction as well as the magnitude of the parameter estimates remain the same. The standard errors of the estimates of parameters also remain the same in almost all models except model 6 (see Table B.1 in appendix 1). However, when we include control variables, i.e maternal education (model 7) and region of residence (model 8), the excess variation observed in model 6 disappears (becomes negligible). This could be due to the omitted covariates (in this case education and region of residence).

Among the factors which appear to influence survival status in the neonatal period, prematurity remains by and large the most important biological variable. It persists to be negatively significant throughout all the models. It may be mentioned here that although the effects of prematurity have been considered to be biological, Geronimus (1986) has argued that maternal behavior such as substance abuse during pregnancy could be a contributing factor to premature or preterm birth. Birth order and survival status of the preceding child, remain statistically (negative) significant but cease to be significant with the addition of health care utilization services covariates. Preceding birth interval displays some attenuation effects. The medium preceding birth interval (14-19 months) appears to be associated with higher survival chances, but the association is only significant at the 5 percent level in models 4 and 7. Age of mother at the birth of the infant is not found to be statistically significant at any stage.

The introduction of utilization of health care service variables (model 6) indicates that non

receipt of BCG vaccination is highly (negative) significant almost everywhere while the place of delivery is positively associated with higher survival in only model 6. Receipt of tetanus toxoid injection by the mother before the birth of the child is not found to be significant in any of the models. As observed in the fixed effects models, the results show that even when accounting for the presence of excess variation, children of women with no education still have higher survival chances in the neonatal period.

Table 6.3.1 Parameter Estimates and Standard Errors for the Logistic Binomial Models of Neonatal Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	4.77 (.50E+05)	4.78*** (0.242)	4.86*** (0.248)	4.77*** (0.273)	4.79*** (0.285)
Premature Birth	-2.73 (.18E+05)	-2.59*** (0.315)	-2.55*** (0.316)	-2.55*** (0.318)	-2.56*** (0.319)
Birth Order (5+)		-.77** (0.283)	-.75** (0.284)	-.77** (0.284)	-.72* (0.323)
Survival Status of Precedng Child					
Preceding Birth Interval < 14			-.69* (0.341)	-.64# (0.354)	-.63# (0.355)
14-19				-.15 (0.321)	-.16 (0.324)
Maternal Age < 20				.67* (0.403)	.66 (0.405)
35+					-.12 (0.645)
					-.21 (0.349)
%SCL	.95 (.57E+05)	.106E-12 (1.51)	.475E-12 (1.21)	.132E-12 (1.21)	.120E-12 (1.28)

Standard Errors are in Parenthesis
* p<.05 ** p<.01 *** P<.001 #<.10

Table 6.3.1 (cont')

Variable Name	Model 6
Constant (%GM)	6.10*** (1.21)
Premature Birth	-2.04*** (0.757)
Birth Order (5+)	.17 (0.380)
Survival Status of Preceding Child (dead)	-.56 (0.454)
Preceding Birth Interval <14	-.09 (0.414)
14-19	.70 (0.476)
Maternal Age <20	-.77 (0.882)
35+	-.14 (0.422)
Receipt of BCG (No)	-5.33*** (0.795)
Receipt of Tetanus Toxoid Injection (No)	.47 (0.467)
Place of Delivery (Home)	.68* (0.399)
Maternal Education	
No Education	
Primary Incomplete	
Primary Complete	
Region of Residence	
Region 1	
%SCL	.38 (2.56)

Standard Errors are in Parenthesis

* p<.05 ** p<.01 *** P<.001 #<.10

Model 7	Model 8
-5.59*** (0.582)	5.53*** (0.588)
-1.88*** (0.453)	-1.88*** (0.454)
-.03 (0.403)	-.08 (0.407)
-.01 (0.403)	-.04 (0.406)
.77* (0.465)	.72 (0.468)
-.95 (0.785)	-1.00 (0.789)
-.19 (0.414)	-.17 (0.417)
-5.44*** (0.491)	-5.45*** (0.489)
.38 (0.427)	.42 (0.429)
.55 (0.375)	.47 (0.386)
1.24* (0.618)	1.20* (0.619)
.75 (0.560)	.75 (0.561)
.56 (0.614)	.58 (0.615)
	.31 (0.349)
.232E-13 (1.53)	.102E-13 (1.12)

Postneonatal Survival Period

A similar model building procedure as employed in the neonatal survival period is also adopted in this section. Table 6.3.2 presents the parameter estimates and standard errors for the logistic binomial models for postneonatal survival. The results in model 1 show the effect of preceding birth interval when only the grand mean (constant) and scale parameter for the random effects term are considered; in model 2 the effect of prematurity is added; in model 3 the effect of the survival status of the preceding child is added; in model 4 the effect of maternal age at the birth of the child is added; in model 5 the effect of birth order is added; in model 6 the effect of health services utilization factors (prenatal care and intrapartum care) are added; in model 7 effect of household exposure factors (type of toilet facility and household water source) are added; in models 9 and 10 the control variables (maternal education and region of residence) are included.

From model 1, it may be observed that the parameter estimates are not significant due to high variances. This was also the case in the neonatal survival period and similar reasons may be given for this. When other covariates are gradually introduced in the models, the magnitude of the scale parameter estimate is very small (near zero) as is observed in case of neonatal period. The introduction of utilization of health care service variables do not alter the observed pattern of the scale parameter estimates which is not significant.

It is observed that the direction as well as the magnitude of parameter estimates in fixed effects models (see Table B.2 in Appendix 1) are the same as in the random effects models. This is found in the case of neonatal survival period as well and as such, the same explanations may be offered in this section.

Among the factors that appear to be associated with postneonatal survival, short preceding birth interval and premature births are found to be statistically significant (from model 2 to model 9). The level of significance also remains the same throughout all the models. Survival status of the preceding child is statistically significant at 5 percent level of significance in models 4 to 6 when only the biological/family formation variables are taken into consideration. In model 7 when the utilization of health care service variables are introduced, survival status of the preceding child remains significant but at 10 percent level. In model 8 when the household exposure variables are introduced, survival status of the preceding child ceases to be significant more specifically when the random effects term (or scale parameter) is included in the model.

Inclusion of maternal education and region of residence in models 9 and 10, shows that as in the fixed effects model, children of women with primary incomplete educational level have lower chances of survival in this period. Similarly, children of women residing in region 1 also have lower survival chances.

Table 6.3.2: Parameter Estimates and Standard Errors for the Logistic Binomial Models of Postneonatal Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	4.13 (.92E+05)	3.79*** (0.192)	3.83*** (0.164)	3.80*** (0.171)	3.88*** (.198)
Preceding Birth Interval <14	-.74 (.64E+04)	-.73** (0.235)	-.64** (0.241)	-.67** (0.242)	-.66** (.242)
14-19	-.07 (0.485)	-.09 (0.272)	-.07 (0.272)	-.08 (0.273)	-.07 (.273)
Premature Birth		-.92* (0.405)	-.87* (0.409)	-.88* (0.409)	-.87* (.410)
Survival Status of Preceding Child			-.53* (0.276)	-.55* (0.278)	-.54* (.278)
Maternal Age <20				-.60 (0.521)	-.51 (.882)
35+				-.07 (0.289)	-.16 (.311)
Birth Order (5+)					-.19 (.283)
%SCL	.93 (.11E+06)	.118E-11 (5.08)	.238E-10 (1.21)	.241E-12 (3.28)	.188E-12 (2.63)

Standard Errors are in Parenthesis
* p<.05 ** p<.01 *** P<.001 #<.10

Table 6.3.2 (cont')

Varible Names	Model 6
Constant (%GM)	4.32*** (0.254)
Preceding Birth Interval <14	-.75** (0.248)
14-19	-.07 (0.277)
Premature Birth	-.90* (0.436)
Survival Status of Preceding Child	
Maternal Age <20	-.50# (0.279)
35+	-.53 (0.532)
Birth Order (5+)	-.18 (0.312)
Prenatal Care (No)	-.16 (0.231)
Intrapartum Care (No)	.05 (0.374)
Source Of Drinking Water	-.31 (0.231)
Unsafe	
Type of Toilet Facility	
Poor	
Maternal Education	
No Education	
Primary Incomplete	
Primary Complete	
Region of Residence	
Region 1	
%SCL	.32E-12 (2.86)

Model 7	Model 8	Model 9
4.25*** (0.289)	4.56*** (0.406)	5.39*** (0.442)
-.68** (0.244)	-.71** (0.246)	-.75** (0.249)
-.03 (0.274)	-.02 (0.276)	-.08 (0.279)
-.91* (0.414)	-.97* (0.418)	-.96* (0.429)
-.39* (0.283)	-.33 (0.285)	-.001 (0.290)
-.60 (0.533)	-.68 (0.535)	-.84 (0.540)
-.12 (0.314)	-.08 (0.316)	-.08 (0.320)
-.12 (0.232)	-.09 (0.239)	-.002 (0.244)
.20 (0.478)	.15 (0.480)	-.15 (0.483)
-.16 (0.236)	-.09 (0.240)	-.03 (0.243)
-.11 (0.235)	-.07 (0.236)	-.004 (0.238)
-.98*** (0.227)	-.95*** (0.232)	-.87*** (0.232)
	-.05 (0.453)	-.33 (0.458)
	-.82* (0.382)	-.93* (0.387)
	-.12 (0.420)	-.29 (0.425)
		-1.57*** (0.247)
.226E-14 (1.84)	.153E-13 (2.61)	238E-13 (1.47)

Infant Survival Period

In this section we have examined factors that may infuence survival in the infant period by combining neonatal, postneonatal and survival in the twelfth month. The same model building procedure as used in the neonatal and postneonatal periods is used in this case as well. Table 6.3.3 and B.3 (appendix 1) show the parameter estimates and standard errors of infant survival with and without the random effects. The results are presented according to the sequence in which the various variables (covariates) are added to the models (Table B.3 is in appendix 1).

As noted in the neonatal and postneonatal survival periods, in the case of infant survival period also, the variances of the estimates of parameters are very high in model 1 when the random effects term is included. However, the parameter estimates are not significant. Hence similar reasons as given in the case of neonatal and postneonatal survival periods may also suffice. When other covariates are introduced gradually in the model, the magnitude of the random effects term (scale parameter) is reduced substantially.

When statistical significance of factors which may influence survival in infancy period are examined, it is observed from Table 6.3.3 that with the inclusion of the random effects, short birth interval persists to be highly (negative) significant throughout all the models. But in models 3, 4, 5 and 6, although still significant, the level of significance is reduced marginally, perhaps this could be as a result of interaction with some of the added variables in subsequent models. Prematurity also remains highly significant (negative) in models 3 to 5. However, when breastfeeding is introduced (model 6) its level of significance is reduced ($p < 0.01$) and introduction of health care services utilization variables (model 7) further reduces its significance ($p < 0.05$). This may be taken as an indication that management and prevention of premature births is highly dependent on availability and utilization of health care services. The trend persists in models 8, 9 and 10. Survival status of the preceding child remains negatively significant in models 4 to 6, but ceases to be significant with the introduction of health care utilization services in model 7. This could perhaps be explained by the fact that parents may realize the individual frailty of the child as such take appropriate action to ensure its survival through utilization of health care services.

The behavioural factors that remain statistically significant (negative) are breastfeeding (not breast-fed); and whether the child was fully immunized. Among the household exposure variables, type of toilet facility has persisted to be highly significant while among the control variables, region of residence has persisted to be significant. This may be due to the spatial distribution of disease patterns and health care utilization services. The effect of maternal education is nullified in this period perhaps as a result of "technology" (i..e, utilization of health care services as well as access to information regarding child care).

Table 6.3.3 Parameter Estimates and Standard Errors for the Logistic Binomial Models of Infant Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	3.41 (.29E+06)	3.22*** (0.122)	3.27*** (0.124)	3.32*** (0.132)	3.51*** (0.159)
Preceding Birth Interval <14	-.66 (.30E+05)	-.68*** (0.179)	-.57** (0.184)	-.60** (0.186)	-.59** (0.186)
14-19	-.14 (.49E+04)	-.10 (0.214)	-.13 (0.214)	-.10 (0.215)	.13 (0.216)
Premature Birth		-1.87*** (0.405)	-1.87*** (0.247)	-1.83*** (0.248)	-1.82*** (0.249)
Survival Status of Preceding Child					
Maternal Age <20			-.66* (0.209)	-.64** (0.210)	-.62** (0.210)
35+				.31 (0.360)	.09 (0.372)
Birth Order (5+)				-.29 (0.202)	-.07 (0.219)
					.44 (0.182)
%SCL	.89 (.39E+06)	.698E-13 (1.57)	.275E-09 (1.21)	.695E-10 (.968)	.158E-10 (.787)

Standard Errors are in Parenthesis
* p<.05 ** p<.01 *** P<.001 #<.10

Table 6.3.3 (cont')

Varible Names	Model 6	Model 7
Constant (%GM)	3.92*** (0.186)	5.52*** (0.323)
Preceding Birth Interval <14	-.70** (0.217)	-.80*** (0.230)
14-19	-.10 (0.243)	-.11 (0.249)
Premature Birth	-.90** (0.362)	-.89** (0.249)
Survival Status of Preceding Child	-.65** (0.246)	-.41 (0.262)
Maternal Age <20	-.01 (0.411)	-.07 (0.422)
35+	-.14 (0.263)	.16 (0.272)
Birth Order (5+)	-.42 (0.207)	-.11 (0.220)
Breastfeeding (Not breast Fed)	-4.26*** (0.274)	-4.02*** (0.320)
Prenatal Care (No)		.49 (0.415)
Intrapartum Care (No)		-.003 (0.214)
Immunization (not immunized)		-2.11*** (0.278)
Source of Drinking Water (unsafe)		- .11 (0.235)
Type of Toilet Facility Poor		-.98*** (0.227)
Maternal Education		
No Education		
Primary Incompl		
Primary Complete		
Region of Residence		
Region 1		
%SCL	.558E-13 (1.55)	.289E-13 (.589)

Standard Errors are in Parenthesis; * p<.05 ** p<.01 *** P<.001 #<.10

Model 8	Model 9	Model 10
5.51*** (0.344)	5.61*** (0.420)	6.25*** (0.450)
-.79*** (0.232)	-.81*** (0.233)	-.80*** (0.238)
-.10 (0.249)	-.08 (0.250)	-.03 (0.256)
-.93* (0.414)	-.94* (0.415)	-1.00* (0.435)
-.32 (0.265)	-.27 (0.269)	-.03 (0.274)
-.04 (0.424)	-.01 (0.428)	-.05 (0.438)
-.08 (0.274)	-.07 (0.274)	.06 (0.278)
-.09 (0.221)	-.08 (0.229)	-.04 (0.233)
-4.08*** (0.321)	-4.09*** (0.321)	-4.37*** (0.339)
.58 (0.422)	.53 (0.426)	.55 (0.432)
-.09 (0.219)	.12 (0.222)	.21 (0.225)
-2.80*** (0.279)	-2.79*** (0.280)	-2.74*** (0.285)
.16 (0.211)	.20 (0.212)	.23 (0.214)
-.81*** (0.232)	-.79*** (0.222)	-.69 (0.225)
	.02 (0.398)	-.18 (0.406)
	-.40 (0.350)	-.51 (0.355)
	.05 (0.377)	-.06 (0.384)
		-1.29*** (0.219)
.131E-14 (.599)	.131E-14 (.599)	258E-13 (0.670)

6.4 Discussion

The main objective of this chapter has been to re-examine the effect of covariates that have been shown to influence infant survival in families in Kenya taking into account sibling dependence and unobserved and/or unmeasured heterogeneity, using the logistic regression model with the random effects term. The variant used in fitting the models is the logistic binomial regression (programmed in EGRET Package).

In all the models fitted, the random effects term (scale parameter) is not significant. This is not consistent with what has been observed in other similar studies which have used the same methodology (Curtis et al., 1993; Madise and Diamond, 1995; Curtis and Steele, 1993) but is consistent with the findings of Guo, (1993) who used proportional hazards model instead of variants of logistic model. One of the possible reasons for such inconsistency could be due to omitted variables such as prematurity, utilization of health care services and physical environment and social status factors such as type of toilet facility, which are included in the present study but are not in the afore mentioned studies. Another possible reason for the observed differences in the studies could be that the data used in the present study is more homogeneous in the sense that it relates only to a rural sample. The extent to which the differences could be due to sampling variability is unknown and as such this calls for further research on such influences on estimation of the models.

From the fitted models, it is observed that most of the covariates which are statistically significant in the fixed effects models, persists to be significant in the random effects models. The direction of the estimates also remains the same. Further, most studies which have

studied the phenomenon of interest as in the present study, have identified preceding birth interval and survival status of the preceding child as factors that may uniquely be associated with familial effects (Curtis et al, 1993; Zenger, 1993). The reason for this could be that previous birth interval probably reflect past fertility-related decisions and as such will be correlated with family-specific frailty endowments (Wolpin, 1997). Hence, preceding birth interval in the neonatal period (table 6.3.2.) shows some attenuation effects, where it is significant in some models and not significant in others. The reasons for this are not easy to discern in the present analysis. In the postneonatal and infant survival models, the effect of short birth interval remained statistically significant (negative) throughout the models. The available literature regarding the effect of short birth interval on survival in infancy, appears to be inconclusive and mixed. For example, Zenger (1993) argues that, from her Bangladesh study, the results provided moderately strong evidence that little or no excess mortality risk was associated with a short preceding birth interval if the preceding child died, but that some excess risk was present if the preceding child survived. However, Curtis et al., (1993) have argued that in their results of their Brazil study (postneonatal mortality), the fact that the birth interval effects are not attenuated when the "family effects" are controlled for, suggests that they are not caused by spurious associations with other aspects of family behaviour which may influence health outcomes.

In the present study, short birth interval persists to be statistically significant in some models, but the pathways through which it operates are still unknown and hence a subject of further research. For example, unless specific causes of death are known, it is not very easy speculate as to whether the short birth interval was detrimental to the survival status of the index child (the same applies to the survival status of the preceding child). The argument that

prior birth intervals reflect family frailty endowments as suggested by Wolpin (1997), may not hold because families' behaviour towards the survival of their children, may be altered through social learning and interaction. More specifically, parents may recognize an unfavourable genetic inheritance in their children and accordingly may make extra efforts to keep them healthy, thus counterbalancing the genetic disadvantages. If this is the case, the total familial effects will not be the sum of genetic and environmental factors and the interpretation of the upper bound of the shared additive genetic factors will not be guaranteed (Guo, 1993). It has also been argued that most estimates of consequences of prior birth intervals in low-income countries, are potentially biased, for they treat the prior birth interval and the health of a child and mother as an estimate of causal effect (c.f. Shultz, 1997). It is however not yet clear whether the increments to birth intervals in a relatively healthy and well-fed population are indeed important for the health of the mother and child except in extreme cases (Schultz, 1997).

Another source of familial effects which has featured prominently in the literature is parental competence as proxied by maternal education (Das Gupta, 1990; 1997). In all the models considered here, the effects of maternal education are introduced after the inclusion of household exposure variables (source of drinking water and type of toilet facility). The surprising result that emerged from the study is that in the neonatal survival period, children of women with no education seem to have higher odds of survival than those of women in the other categories of education. These results are consistent with those of Boerma and Bicego (1991), who used data from the Kenya Demographic and Health Survey of 1989 (KDHS,1989). They noted that the effect of inclusion of variables that capture variation in the pattern of family formation is either substantial or works to amplify the education

advantage. Kenya was seen as the most notable country where these effects were visible, suggesting that less education was associated with family formation patterns that reduce rather than increase neonatal mortality risk. This could also be due to the fact that in this period, most of the deaths may occur as a result of biological factors associated with congenital malformations which may not reflect maternal competence.

The other unique finding with regard to maternal education was that at the postneonatal stage, children of women who had primary incomplete level of education had a lower odds of survival. This type of finding is difficult to explain especially with regard to the maternal competence hypothesis. Guo (1993) also found that parental competence hypothesis may not hold in all communities and should therefore not be generalized due to different rearing practices. Further, education is related to income, which may in part be related to the utilization of health care services (where cost is a factor for utilization). Hence, an alternative way of looking at the effect of maternal education would be to control for family income and other socio-economic status factors. Perhaps this could be the reason why type of toilet facility was persistently statistically significant (negative) which could be a reflection of the socio-economic status of the household rather than the inadequacy of the toilet facilities since infants do not use toilet facilities at such age. Wolpin (1997), argues that it may be the case that toilet facilities are more expensive to instal in the rural areas such that rural households with flush toilet facilities will be of higher wealth or possibly with greater preference for health.

6.5 Conclusion

Although random effects models seem to provide the more unified approach to logistic regression analysis, interpretation of the estimates of parameters of the covariates in these models is however difficult. The random effects term(s) may be considered to represent the totality of cluster-constant covariate effects omitted from the model that are independent of those already present. Since the term measuring the random effects is negligible in the present study, the standard logistic model suffices for interpretation of the covariate effects. It may however be noted that, even the random effects models will only be without bias as long as the omitted variables are independent of those included in the models. In demographic analysis, it is also not sufficient to conclude that when scale parameter in the model is significant therefore there is presence of familial effects since factors not included may nest individual frailty, mother/household and community characterestics. A test for the presence of familial effects may only be possible in an experimental situation where the study sample is taken from the same or related universe, some of which can be held constant (e.g. two eyes) or where a cluster-specific variable which would identify children of the same woman is included in the analysis and possibly where the cluster size is fixed for all observations.

Hence, our results of the analysis showing that the random effects are not significant may indicate that a number of covariates accounting for infant survival may have been included in the models. This conclusion follows the interpretation of the random effects term as that which accounts for factors not included in the model rather than a measure of familial (cluster) specific risks as have been used in past literature (Curtis et al., 1993; Madise and Diamond, 1995; Curtis and Steele, 1996; Das Gupta, 1997).

Appendix 1

Table B.1 Parameter Estimates and Standard Errors for the Logistic Models of Neonatal Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	4.77 (.50E+05)	4.78*** (0.242)	4.86*** (0.248)	4.77*** (0.273)	4.79*** (0.285)
Premature Birth	-2.73 (.18E+05)	-2.59*** (0.315)	-2.55*** (0.316)	-2.55*** (0.318)	-2.56*** (0.319)
Birth Order (5+)		-.77** (0.283)	-.75** (0.284)	-.77** (0.284)	-.72* (0.323)
Survival Status of Precedng Child					
Preceding Birth Interval <14			-.69* (0.341)	-.64# (0.354)	-.63# (0.355)
14-19				-.15 (0.321)	-.16 (0.324)
Maternal Age <20				.67* (0.403)	.66 (0.405)
35+					-.12 (0.645)
					-.21 (0.349)

Standard Errors are in Parenthesis

* p<.05 ** p<.01 *** P<.001 #<.10

Table B.1 (cont')

Variable Name	Model 6	Model 7	Model 8
Constant (%GM)	6.10*** (1.21)	5.59*** (0.582)	5.53*** (0.588)
Premature Birth	-2.04*** (0.757)	-1.88*** (0.453)	-1.88*** (0.454)
Birth Order (5+)	.17 (0.380)	-.03 (0.403)	-.08 (0.407)
Survival Status of Preceding Child (dead)	-.56 (0.454)	-.01 (0.403)	-.04 (0.406)
Preceding Birth Interval <14	-.09 (0.414)	.77* (0.465)	.72 (0.468)
14-19	.70 (0.476)	-.95 (0.785)	-1.00 (0.789)
Maternal Age <20	-.77 (0.882)	-.19 (0.414)	-.17 (0.417)
35+	-.14 (0.422)	-5.44*** (0.491)	-5.45*** (0.489)
Receipt of BCG (No)	-5.33*** (0.795)		
Receipt of Tetanus Toxoid Injection (No)	.47 (0.467)	.38 (0.427)	.42 (0.429)
Place of Delivery (Home)	.68* (0.399)	.55 (0.375)	.47 (0.386)
Maternal Education			
No Education		1.24* (0.618)	1.20* (.619)
Primary Incomplete		.75 (0.560)	.75 (.561)
Primary Complete		.56 (0.614)	.58 (.615)
Region of Residence			
Region 1			.31 (.349)

Standard Errors are in Parenthesis * p<.05 ** p<.01 *** P<.001 #<.10

Table B.2 Parameter Estimates and Standard Errors for the Logistic Models of Postneonatal Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	4.13 (.92E+05)	3.79*** (0.192)	3.83*** (0.164)	3.80*** (0.171)	3.88*** (0.198)
Preceding Birth Interval <14	-.74 (.64E+04)	-.73** (0.235)	-.64** (0.241)	-.67** (0.242)	-.66** (0.242)
14-19	-.07 (0.485)	-.09 (0.272)	-.07 (0.272)	-.08 (0.273)	-.07 (0.273)
Premature Birth		-.92* (0.405)	-.87* (0.409)	-.88* (0.409)	-.87* (0.410)
Survival Status of Preceding Child			-.53* (0.276)	-.55* (0.278)	-.54* (0.278)
Maternal Age <20				-.60 (0.521)	-.51 (0.882)
35+				-.07 (0.289)	-.16 (0.311)
Birth Order (5+)					-.19 (0.283)

Standard Errors are in Parenthesis
* p<.05 ** p<.01 *** P<.001 #<.10

Table B.2 (cont')

Varible Names	Model 6	Model 7	Model 8	Model 9
Constant (%GM)	4.32*** (0.254)	4.25*** (0.289)	4.56*** (0.406)	5.39*** (0.442)
Preceding Birth Interval <14	-.75** (0.248)	-.68** (0.244)	-.71** (0.246)	-.75** (0.249)
14-19	-.07 (0.277)	-.03 (0.274)	-.02 (0.276)	-.08 (0.279)
Premature Birth	-.90* (0.436)	-.91* (0.414)	-.97* (0.418)	-.96* (0.429)
Survival Status of Preceding Child	-.50# (0.279)	-.39* (0.283)	-.33 (0.285)	-.001 (0.290)
Maternal Age <20	-.53 (0.532)	-.60 (0.533)	-.68 (0.535)	-.84 (0.540)
35+	-.18 (0.312)	-.12 (0.314)	-.08 (0.316)	-.08 (0.320)
Birth Order (5+)	-.16 (0.231)	-.12 (0.232)	-.09 (0.239)	-.002 (0.244)
Prenatal Care (No)	.05 (0.374)	.20 (0.478)	.15 (0.480)	-.15 (0.483)
Intrapartum Care (No)	-.31 (0.231)	-.16 (0.236)	-.09 (0.240)	-.03 (0.243)
Source Of Drinking Water				
Unsafe		-.11 (0.235)	-.07 (0.236)	-.004 (0.238)
Type of Toilet Facility				
Poor		-.98*** (0.227)	-.95*** (0.232)	-.87*** (0.232)
Maternal Education				
No Education			-.05 (0.453)	-.33 (0.458)
Primary Incomplete			-.82* (0.382)	-.93* (0.387)
Primary Complete			-.12 (0.420)	-.29 (0.425)
Region of Residence				
Region 1				-1.57*** (0.247)

Standard Errors are in Parenthesis

* p<.05 ** p<.01 *** P<.001 #<.10

Table B.3: Parameter Estimates and Standard Errors for the Logistic Models of Infant Survival.

Variable Name	Model 1	Model 2	Model 3	Model 4	Model 5
Constant (%GM)	3.41 (.29E+06)	3.22*** (0.122)	3.27*** (0.124)	3.32*** (0.132)	3.51*** (0.159)
Preceding Birth Interval					
<14	-.66 (.30E+05)	-.68*** (0.179)	-.57** (0.184)	-.60** (0.186)	-.59** (0.186)
14-19	-.14 (.49E+04)	-.10 (0.214)	-.13 (0.214)	-.10 (0.215)	.13 (0.216)
Premature Birth		-1.87*** (0.405)	-1.87*** (0.247)	-1.83*** (0.248)	-1.82*** (0.249)
Survival Status of Preceding Child					
Maternal Age			-.66* (0.209)	-.64** (0.210)	-.62** (0.210)
<20				.31 (0.360)	.09 (0.372)
35+				-.29 (0.202)	-.07 (0.219)
Birth Order (5+)					.44 (0.182)

Standard Errors are in Parenthesis

* p<.05 ** p<.01 *** P<.001 #<.10

Table B.3 (cont')

Varible Names	Model 6	Model 7	Model 8	Model 9	Model 10
Constant (%GM)	3.92*** (0.186)	5.52*** (0.323)	5.51*** (0.344)	5.61*** (0.420)	6.25*** (0.450)
Preceding Birth Interval <14	-.70** (0.217)	-.80*** (0.230)	-.79*** (0.232)	-.81*** (0.233)	-.80*** (0.238)
14-19	-.10 (0.243)	-.11 (0.249)	-.10 (0.249)	-.08 (0.250)	-.03 (0.256)
Premature Birth	-.90** (0.362)	-.89** (0.249)	-.93* (0.414)	-.94* (0.415)	-1.00* (0.435)
Survival Status of Preceding Child	-.65** (0.246)	-.41 (0.262)	-.32 (0.265)	-.27 (0.269)	-.03 (0.274)
Maternal Age <20	-.01 (0.411)	-.07 (0.422)	-.04 (0.424)	-.01 (0.428)	-.05 (0.438)
35+	-.14 (0.263)	.16 (0.272)	-.08 (0.274)	-.07 (0.274)	.06 (0.278)
Birth Order (5+)	-.42 (0.207)	-.11 (0.220)	-.09 (0.221)	-.08 (0.229)	-.04 (0.233)
Breastfeeding (Not breast Fed)	-4.26*** (0.274)	-4.02*** (0.320)	-4.08*** (0.321)	-4.09*** (0.321)	-4.37*** (0.339)
Prenatal Care (No)		.49 (0.415)	.58 (0.422)	.53 (0.426)	.55 (0.432)
Intrapartum Care (No)		-.003 (0.214)	-.09 (0.219)	.12 (0.222)	.21 (0.225)
Immunization (not immunized)		-2.11*** (0.278)	-2.80*** (0.279)	-2.79*** (0.280)	-2.74*** (0.285)
Source of Drinking Water (unsafe)		- .11 (0.235)	.16 (0.211)	.20 (0.212)	.23 (0.214)
Type of Toilet Facility Poor		-.98*** (0.227)	-.81*** (0.232)	-.79*** (0.222)	-.69 (0.225)
Maternal Education No Education				.02 (0.398)	-.18 (0.406)
Primary Incompl				-.40 (0.350)	-.51 (0.355)
Primary Complete				.05 (0.377)	-.06 (0.384)
Region of Residence Region 1					-1.29*** (0.219)

Standard Errors are in Parenthesis

* p<.05 ** p<.01 *** P<.001 #<.10

CHAPTER 7

INFANT/CHILD SURVIVAL AND MATERNAL HEALTH: AN EXPLORATORY ANALYSIS

7.1 Introduction

In the previous chapter, we examined factors that have been shown theoretically to influence infant and child survival in families in Kenya taking into consideration sibling dependence and unobserved and/or unmeasured heterogeneity. In the present chapter, we explore the relationship between infant/child survival and maternal health. It has been argued that most of the reproductive health variables that affect child survival (age, parity etc) also affect maternal health. Age, especially very young age is said to be associated with the disability that results from pregnancy and child birth e.g., vesico-vaginal fistulae (VVF) which is common among young mothers, who are more likely to experience prolonged labour resulting from immature pelvises (cf. McCarthy and Maine, 1992). Prolonged labour may also lead to foetal stress, which could in turn result in a perinatal death. Similarly, parity may influence one of the other major maternal disabilities that results from pregnancy and subsequently, childbirth i.e, uterine prolapse. This is much more common among high parity women (cf. McCarthy and Maine, 1992). Uterine prolapse may be a consequence of too many births at too close intervals which in turn may be a consequence of previous child loss resulting from prematurity and low birth weight of both the mother and child.

It has been argued that prematurity and low birth weight may be indicators of poor nutritional status of the mother (Gondotra, Das and Dey, 1982) and short birth intervals may result in repeated infant deaths. Thus, short birth spacing appear to influence both the outcome for

the child as well as for the mother (reverse causality). Although the detrimental effects of short birth interval on child survival have been extensively examined and documented in literature (Srivastava, 1990; Hobcraft, McDonald and Rutstein, 1984), the reverse effect of short birth interval on maternal health has not been examined. This is mainly due to a paucity of data. Winikoff, (1983) has noted that since both shorter birth intervals and higher risk of repeat infant mortality occur in families that have experienced one or more early infant deaths, the death of a second or subsequent child after a short birth interval may not be due to the interval but to some other factor that also caused the first death and shortened the interval. This could imply that the second child may have died as a result of the effect of the health of the mother i.e, depletion syndrome due to impaired nutritional status of the mother or other genetic or behavioural factors.

As stated in the literature review chapter, the importance of family formation or bio-demographic variables and their association with infant and child survival and subsequently maternal ill-health, has continued to be a subject of research interest among demographers and related fields. The relationship has been explained through the maternal depletion syndrome hypothesis i.e, when a woman gives birth to too many children at short birth intervals she does not get enough time to recover from the pregnancy and replenish her energy. The problem is exacerbated if the birth results in child loss.

The maternal depletion syndrome operates through the socio-economic, cultural, region of residence, behavioural and bio-demographic factors to influence child survival and maternal ill-health. For example, maternal education (through its association with late age at marriage) is likely to be associated with lower fertility and hence with fewer pregnancies. Education

can also be associated with development of fewer complications among pregnant women if better educated women in general are in better health than others before and during pregnancy; and that, education could be associated with a greater likelihood of receiving appropriate care for complications that may arise given that better educated women might also be better informed about the symptoms of complications and could therefore be more likely to make timely decision to seek care (McCarthy and Maine, 1992). Unfortunately, due to lack of data, these relationships have not been explored.

In the present study, some of these relationships are partially explored. Body Mass Index (BMI) is used as a measure of maternal nutritional status (proxy for maternal health) and reflects primarily the current nutritional status of the woman. It is also a net measure in the sense that it reflects the balance between/and the claim to food intakes. Although height is determined by the cumulative nutritional status during an entire developmental age span of the woman (including childhood and adolescence), BMI fluctuates with the current balance between nutrient intakes and energy demands especially during pregnancy, breast feeding and illness (Fogel, 1997). Thus, BMI is a useful measure of relative thinness in adults. Levels of below 18.5 indicate chronic undenutrition (James et al., 1988)

7.2. Methodology

In order to explore the relationship between child survival and maternal health, we use log-linear models. Log-linear models have been formulated for the analysis of categorical data. These models are useful for uncovering the potentially complex relationships among variables in a multiway crosstabulation. In log-linear models all variables that are used for

classification are independent variables and the dependent variable is the number of cases in a cell of the crosstabulation (SPSSPC Manual). The guide to choosing an appropriate model includes whether it fits the data and how easily it can be interpreted as well as its simplicity (parsimonious). e.g, if models with and without higher order interaction terms fit the data well, the simpler models are usually preferable since higher-order interaction terms are difficult to interpret.

When using a log-linear model, the number of cases in each cell can be expressed as a function of the main effects and the interaction between the main effects. To obtain a linear model, the natural logs of the cell frequencies, rather than the actual counts, are used. In general, the model for the log of the observed frequency in the i^{th} row and the j^{th} column is given by:

$$\ln(F_{ij}) = \mu + \lambda_i^{A} + \lambda_j^{B} + \lambda_{ij}^{AB} \qquad \ldots(7.1)$$

The term denoted by μ is comparable to the grand mean in the analysis of variance. It may be described as the average of the logs of frequencies in all the table cells. The lambda parameters represent the increments or decrements from the base value (μ) for particular combination of values of the row and column variables. Each individual category of the row and column variables has an associated lambda. Thus, the number of cases in a cell is a function of the values of the row and column variables and their interactions. In particular, F_{ij} is the observed frequency in the cell, λ_i^A is the effect of the i^{th} category of the first variable, λ_j^B is the effect of the j^{th} category of the second variable and λ^{ijAB} is the

interaction effect for the i^{th} value of the first variable and the j^{th} value of the second variable. The lambda parameter and μ are estimated from the data.

In general, the effect of the i^{th} category of a variable, called a main effect is estimated from

$$\lambda_i^{var} = \mu_i - \mu \qquad \ldots(7.2)$$

where μ_i is the mean of the logs in the i^{th} category and μ is the grand mean. Positive values of lambda occur when the average number of cases in a row or a column is larger than the overall average. The interaction parameters indicate the amount of difference between the sums of the effects of the variables taken individually and collectively. They represent the 'boost' or 'interference' associated with particular combinations of values. The estimate for the interaction parameter is the difference between the log of the observed frequency in a particular cell and the log of the predicted frequency using only three lambda parameters for the row and column variables. To obtain a unique estimate for the lambda, certain constraints have to be imposed on them e.g. the lambdas must sum to 0 across the categories of the variables. Similar constraints are imposed on interaction terms as well where the lambdas must also sum to 0 over all categories of the variable. The model described above is a fully saturated model.

The first step in determining a suitable model will be therefore to fit a fully saturated model (i.e, one which contains all main effects and interaction terms) and examine the standardized values for the parameter estimates. Effects with small estimated values can usually be deleted

from the model. Another strategy is to systematically test the contribution made by terms in a particular order to the model e.g, a model with interaction terms may first be fitted followed by a model with main effects only. The change in the chi-square value between the two models is then attributable to the interaction effects. In order to have a simpler model, terms whose contribution is negligible should be eliminated. The first step in this elimination process is to fit a hierarchical log-linear model. In hierarchical linear-model, if a term exists for the interaction of a set of variables, there must be a lower-order term for all possible combinations of these variables. This means that for a two-variable model, the interaction term can only be included if both main effects terms are present. For a three-variable model, if the term λ^{ABC} is included in a model, then the terms $\lambda^{A}, \lambda^{B}, \lambda^{C}, \lambda^{AB}, \lambda^{BC}$, and λ^{AC} must also be included. To describe a hierarchical log-linear model, it is sufficient to list the highest-order terms in which variables appear which is called the generating class of a model.

To obtain the best generating class model, backward elimination procedure is applied. This begins with the inclusion of all the variables (effects) in the model and systematically removes those that do not satisfy the criterion for remaining in the model. Although hierarchical log-linear models are fairly adequate in describing the data, they however do not produce parameters for unsaturated models to allow for specification of contrasts for parameters or display correlation matrix for parameters. For this, the procedure 'loglinear' is used as a general procedure that does model fitting, hypothesis testing and parameter estimation for any model that has categorical variables as its major components.

7.3 Variable Description

In this section the variables that are used in the analysis are described. Given that the main objective of this chapter is to explore the relationship between infant/child survival and maternal health, the variables that are used in the analysis have been selected to reflect this aspect. The variables selected are those which have been shown to theoretically influence infant/child survival and maternal health directly and those indicative of the socio-economic status of the household, use of health technologies and physical environment of the household (see theoritical framework in chapter 1).

The dependent variable is the body mass index (BMI). Although there are few studies which have investigated the mortality and morbidity risk of low BMI in developing countries, increasing evidence suggests that individuals with low BMI (BMI=wt in kg/ht in m^2) suffer grreater illness, impaired work capacity, reduced social activity and lower income (Shetty and James, 1994). As earlier indicated, levels of BMI of less than 18.5 is an indication of chronic undernutrition. Thus in this study BMI is coded as V445A and takes the value of zero if less than 18.5 and one otherwise.

The independent variables are proportion of closed birth intervals which are short (PSBINT), children ever born (V201), current age of mother (V012), maternal education (S109B), region of residence (V023), childhood place of residence (V103), receipt of tetanus toxoid injection(TTI), place of delivery (PLACED) and contraceptive use (V302). These can be categorised into socio-economic (maternal education, childhood place of residence and region of residence); demographic/family formation (proportion of short birth intervals, current age

of mother and children ever born/parity) and utilization of health care services (receipt of tetanus toxoid injection, place of delivery and contraceptive use).

Proportion of short birth intervals has been computed by taking the ratio of the number of closed birth intervals less than 14 months to the total number of closed birth intervals (length of inteval less gestation period taken to be equal to 9 months). This variable takes the value of zero if there are no short birth intervals and one if some or all intervals are short. Children ever born or parity takes the value of zero if number is less than 5 and one otherwise while current age of mother takes the value of less than or equal to 30 or greater than 31. Maternal education, region of residence, place of delivery and receipt of tetanus toxoid injection remain the same as in the previous chapters (five and six).

Childhood place of residence is included because studies have pointed out that it determines the height (part of the nutritional status) of a woman during the entire developmental age span (including childhood and adolescence). This variable is coded as zero if rural and one if urban. Contraceptive use has been shown theoretically to influence birth spacing hence a distinction is made for ever use of modern, traditional or never. Table 7.1.1 presents the summary of variable descriptions and their measurement.

Table 7.1.1: Variable Description

Variable Name	Code	Measurement	Remarks
Body Mass Index (BMI)	V445A	0, if BMI less or equal 18.5 1, if BMI greater than 18.5	Used as dependent variable in loglinear model
Proportion of short birth intervals	PSBINT	0, if no short birth intervals 1, if birth intervals are short	
Total Children ever born (CEB)	V201	1, if V201 less than 5 2, if V201 greater than 5	
Maternal Age	V012	v012 less than or equal to 30 2, if v012 greater than 31	
Maternal Education	S109B	1=No Education 2=Primary Incomplete 3=Primary Complete 4=Secondary and Above	
Childhood Place of Residence of Residence	V103	0, if V103 is Rural 1, if V103 is urban	

Table 7.1.1 (continued)

Variable Name	Code	Measurement	Remarks
Receipt of Tetanus Toxoid	TT	0, if TT= none 1, if TT=1 dose 2, if TT=2 or more doses	
Place of Delivery	Placed1	0, if Placed1=Home 1, if placed1= Health Inst (Govt) 2, if Placed1= health inst (private)	
Region of Residence	V023	0, if V023=high disease prevalence 1, if V023= low disease prevalence	
Ever Use of Contraceptives	V302	0, if V302 is never use 1, if folkloric or traditional 2, if modern methods	

7.4 Results of Analysis

Bivariate Results

Bivariate analysis was performed on eleven variables using the pearson chi-square test for association between the Body Mass Index (BMI) and other explanatory variables. Table 7.1.2

presents the results which show that 10.4 percent of the women in the sample fall below the cut-off level of BMI of 18.5.

The results of the bivariate analysis indicate there is a strong association between BMI maternal age and parity. There is a higher proportion of women with low BMI among older women (age 31 and above) than young women. Similarly, higher proportion of women have a low BMI among high parity (5 and above) than low parity women. These two variables (maternal age and parity) may be related in the sense that women with higher parity could also be older. However, there is not much difference between the proportion of women with short birth intervals and those without and low body mass index as well as between the proportion of women who had experienced child loss and those who had not and low body mass index. This could be due to the fact that these two variables may be interacting with other variables for their association to be visible.

With regard to the association between maternal education and body mass index, the results indicate that women with no education have higher proportion with low body mass index. Thus proportion of low BMI decreases with the level of education. There are no significant differences in the body mass index (BMI) between women who had received the tetanus toxoid injection and those who had not. However, with regard to place of delivery, those women who deliver in government health institutions display a low body mass index as compared with those who deliver in private health institutions possibly due to the differentials in socio-economic status of the women in the two institutional deliveries. The results also show that there is a higher proportion of women with low body mass index among those women who have never used a contraceptive and those who used folkloric and/or traditional

methods. Childhood place of residence (proxy for nutritional status in childhood and adolescence) although not statistically significant, indicates that those women who previously resided in the rural areas tend to have low body mass index.

From these initial results, it appears that there may be some association between low BMI and age, parity, education, place of delivery, contraceptive use and region of residence. The other variables which do not show association at this stage, may be interacting with some other variables. The high significant association of the place of delivery and BMI could be an indication that those women who avail themselves of the services (especially government based) may be those who have experienced complications but cannot afford services offered in the private institutions.

Table 7.1.2: Distribution of women by BMI and Biodemographic, socio-economic, health care utilization, behavioural and Household Environment Variables

Variable Code	BMI < 18.5	BMI ≥ 18.5	P-Value	Chisquare
Maternal Age ≤30	9.2 (147)	0.8 (1429)		
31+	12.4 (114)	87.6 (808)	0.013	6.21 (df.1)
Children Ever Born				
<5	8.8 (124)	91.2 (1409)		
≥5	12.4 (135)	87.6 (952)	0.003	8.61 (df.1)
Proportion with short Birth Intervals				
0	9.9 (113)	90.1 (1034)		
1	10.8 (146)	89.2 (1203)	0.428	0.63 (df.1)
Proportion Dead				
0	10.0 (176)	90.0 (1587)		
1	11.3 (83)	88.7 (650)	0.317	1.00 (df.1)
Maternal Education				
1	15.3 (66)	84.7 (365)		
2	10.8 (90)	89.2 (747)	0.001	17.48 (df.3)
3	9.0 (67)	91.0 (675)		
4	7.4 (36)	92.6 (450)		
Receipt of Tetanus Toxoid				
0	10.6 (147)	89.4 (1239)		
1	9.3 (68)	90.7 (661)	0.472	1.50 (df.2)
2	11.5 (44)	88.5 (337)		
Place of Delivery				
0	8.2 (75)	91.8 (842)		
1	12.8 (159)	87.2 (1084)	0.000	17.47 (df.2)
2	6.4 (18)	93.6 (264)		
Contraceptive Use				
0	13.0 (146)	87.5 (981)		
1	12.6 (42)	87.4 (292)	0.000	23.55 (df.2)
2	6.9 (71)	93.1 (964)		

Table 7.1.2. (continued)

Variable Code	BMI < 18.5	BMI ≥ 18.5	P-Value	CHI-SQ
Region of Residence				
1	11.7 (164)	88.3 (1237)	0.014	6.09
2	8.7 (95)	91.3 (1000)		(df.1)
Childhood Place of Residence 1	10.7 (236)	89.3 (1971	0.189	1.73
2	8.2 (23)	91.8 (259)		(df.1)
Total	10.4 (259)	89.6 (2237)		

In order to explore these relationships further, loglinear models are fitted. The analysis is divided into five models for convenience. Model 1 relates the health of the mother to proportion of short birth intervals, her current age and to children ever born. It is expected that women who experience short birth intervals are also likely to experience higher parities. Short birth intervals and higher parities do not allow the woman to replenish her strength before the next pregnancy. It is thus expected that with such depleted health status, the resultant birth may be premature and as such end up in child loss. It is also expected that the women of higher parities are older.

Model 2 relates the health of the mother to her socio-economic status (proxied by education, childhood place of residence) and the ecological zone of residence. It is expected that the health of the mother (nutritional status) is influenced by her socio-economic status in that women with higher incomes are more likely to have better nutrition so that even when the body is depleted due to repeated pregnancies, survival status of the infant may be high.

Similarly women with higher education are more likely to be more knowledgeable in terms of preventive measures that can be taken to prevent the depletion syndrome and subsequently child loss. Childhood place of residence, as already noted, reflects childhood and adolescent nutritional status which is in turn reflected in the height of the woman (body stature). The ecological zone of residence may influence the woman's health status prior to and during pregnancy. This may lead to her developing and surviving complications. The leading ecological health conditions that may be exacerbated by pregnancy and delivery are malaria, anaemia and malnutrition.

Model 3 relates maternal health to the utilization of health care technologies such as receipt of tetanus toxoid injection and prenatal care. It is expected that women who utilize these technologies are aware of their benefit. Model 4 relates the health of the mother to utilization of intrapartum care. It is expected that who give birth in a health institution and are assisted by trained health personnel may have higher survival chances for themselves and their infants even if their health status may be depleted due to repeated pregnancies and child loss.

Model 5 relates the health of the mother to contraceptive use and breastfeeding practices. It is expected that women who are breastfeeding may have a lower BMI due to the nutritional demands of the infant. With regard to contraceptive use, women who perceive health risks as a result of using modern contraceptives may not use them and as such may be exposed to pregnancy within a short birth interval especially where child loss has occurred.

The first step is to fit hierarchical loglinear models to test whether the higher-order effects are important in each of the five models. Tests for collective importance of effects of various

orders but not individual terms in representing the data are displayed in table 7.1.2. Using model 1 as an example, the first line is a test of the hypothesis that the fourth order of interactions is zero. The goodness of fit is given by the likelihood ratio chi-square statistic that tests the hypothesis whether omitting the fourth order interactions is zero or not. The second line is the goodness of fit test without third and fourth order effects. The last line when $k=1$ corresponds to a model that has no effects except the grand mean. The column labelled 'probability' gives the observed significance for the test that k and higher order effects are zero. If the observed significance level for the test that k and higher order terms are zero is large, then the hypothesis that the k and higher order interactions are zero, should not be rejected which implies that a model with k and lower order effects is adequate to represent the data. From Table 7.1.3, it is observed that the probabilities for the third and higher order effects for each of the models is high therefore the hypothesis that third and higher order effects are zero should not be rejected. This implies that if we are to use the principle of parsimony, then each of the models can adequately be represented by third order effects.

Although some of the results from Table 7.1.3 show that third-order effects or 3-way effects may be sufficient to represent the data, they do not indicate which among the effects is important. Hence, we use backward elimination procedure in order to determine the best generating class effects to represent the data. The results for each of the five models are presented in Table 7.1.4. It is observed from this table that except in model 3 where the third order effects adequately represent the data, in the other models, a two order effect appears to be adequate.

Table 7.1.3: Test for K-way and Higher-order Effect

Model 1			
K	DF	LR-Chisq	Probability
4	2	5.73	0.057
3	9	12.34	0.194
2	18	2130.59	0.000
1	23	6181.46	0.000
Model 2			
K	DF	LR-Chisq	Probability
4	3	1.29	0.732
3	13	22.71	0.045
2	25	239.75	0.000
1	31	3943.46	0.000
Model 3			
K	DF	LR-Chisq	Probability
3	4	2.85	0.583
2	12	322.57	0.000
1	17	3319.64	0.000
Model 4			
K	DF	LR-Chisq	Probability
4	4	1.67	0.797
3	9	11.21	0.797
2	28	1201.13	0.000
1	35	4339.54	0.000
Model 1			
K	DF	LR-Chisq	Probability
3	2	1.06	0.589
2	7	156.79	0.000
1	11	2287.83	0.000

From the hierarchical linear models, the observation that data can adequately be presented by second or lower-order effects, shows that the variables in each of the models are not independent or that the sample of women according to the classification given by the categories are not homogenous. This implies that there exists association between BMI (maternal nutrition) and the variables indicated. However, some of the factors may operate

through the other variables e.g, birth spacing may operate through number of children ever born.

Table 7.1.4: Results of the Best Generating Class Models Using Backward Elimination Procedure

Model 1			
K	DF	LR-Chisq Change	Probability
PSBINT*v201	2	850.50	0.000
V445A*V201	2	8.57	0.014
V012*V201	2	1286.07	0.000
Goodness of Fit	12	18.46	0.217
Model 2			
K	DF	LR-Chisq Change	Probability
V445A*V103*S109B	3	14.83	0.002
V445A*V023	1	7.81	0.005
V103*V023	1	140.72	0.000
V023*S109B	3	47.73	0.000
Goodness of Fit	10	7.88	0.640
Model 3			
K	DF	LR-Chisq Change	Probability
PRENAT*TT	4	317.29	0.000
V445A	1	1769.82	0.000
Goodness of Fit	8	5.28	0.727
Model 4			
K	DF	LR-Chisq Change	Probability
PLACED*ASSTA	4	1140.59	0.000
PSBINT*ASSTA	2	17.91	0.000
V445A*PLACED	2	23.51	0.000
Goodness of Fit	21	19.12	0.577
Model 1			
K	DF	LR-Chisq Change	Probability
V445A*V302	2	24.61	0.000
V404*V302	2	131.08	0.000
Goodness of Fit	3	1.09	0.778

Further, loglinear procedure is used in the next section to investigate some of the relationships observed in table 7.1.4. The parameter estimates, can be transformed to obtain regression-like coefficients which are again translated into log-odds or odds as in table 7.1.5. The log-odds equals to twice the parameter estimates or coefficients.

The regression-like model implied by the coefficients is given by:

$$\ln(F_{ijk1}/F_{ijk2}) = B + B(A)_i + B(B)_j + B(C)_k + B(BC)_{jk} \quad \ldots(7.3)$$

where, F is the expected frequency of the log-odds. The meaning of other notations are explained using model 1 of table 7.1.5 as an example;

B = -2.1684 (mean or overall effect),

$B(A)_i$ = -0.2484 for i = 1 (Children ever Born <5),

= 0.2484 for i = 2 (Children ever Born ≥ 5),

$B(B)_j$ = 0.0788 for j = 1 (Proportion with short birth interval=0),

-0.0788 for j = 2 (Proportion with short birth interval=1),

$B(C)_k$ = 0.1800 for k = 1 (Region of Residence=1),

=-0.1800 for k = 2 (Region of Residence=2).

To evaluate the model in terms of odds, the multiplicative form of the models is obtained by finding the antilog of the 2*coefficients and are given in column 4 of Table 7.1.5, i.e,

$$F_{ijk1}/F_{ijk2} = T * T(A)_i * T(B)_j * T(C)_k * T(BC)_{jk} \quad \ldots(7.4)$$

where, T = the antilog for -2.1684 = 0.1144 and so on.

The model decomposes the expected frequency of having low BMI into the above

components; 0.1144 is the overall effect, 0.7799 is the effect of low parity relative to high parity on the likelihood of having low BMI. The last column of the Table 7.1.5 gives the Z - values through which we can compare whether the association is statistically significant or not. Keeping the above interpretations in mind, the results of the analysis in Table 7.1.5 indicate that women with low parity (fewer children ever born) are less likely to have low BMI as compared to women of high parities. Further, women with lower parity may have longer birth intervals and subsequently fewer child deaths. However, this does not take into consideration miscarrages, abortions or stillbirths. Also, the effect of short birth intervals on BMI on its own, is not statistically significant. This is consistent with the results of the bivariate analysis in table 7.1.2 but the effect may act through children ever born.

With regard to socioeconomic status, the results indicate that there are significant differences in the proportion of women having low BMI between regions. The odds of women in region 1 having low BMI is 1.2 times greater than those women in region 2, when other factors are held constant. This could be as a result of ecological differences in disease patterns, cultural eating habits etc. Similarly, the results for maternal education show the odds having low BMI among women with no education is 1.5 times greater than those with secondary and above levels. There are no significant differences in the proportion of women having low BMI in other educational categories.

The results further indicate that women who have never used any form of contraception and those who have used folkloric/traditional methods are more likely to have a low BMI. The reason for the non- or usage of folkloric/traditional methods of contraceptives by these women could be that prior knowledge of their health status for those with low BMI who may

then consider contraceptive use as an additional risk to their health. Further, those with low BMI may not have used contraceptives at all and as such may have been exposed to short birth intervals more so if there is premature child loss. These women are also likely to have high parities and short birth intervals. The results also show that women who used government health facilities for delivery are more likely to have low BMI (Odds=1.5 times) than those who did use private health facilities. These could be those women who may have experienced complications and were therefore referred to these health facilities.

Table 7.1.5: Presents Parameter estimates of the Relationship between Infant/Child Survival and Maternal Health

Model 1 Effects	Coeffecient	2*Coeffecient	Antilog	Z-Values
V445A	-1.0842	-2.1684	0.1144	-31.574
V445A by V201	-0.2484	-0.2484	0.7799	-3.093
V445A by PSBINT	0.0390	0.0780	1.0811	0.964
V445A by V023	0.0900	0.1800	1.1972	2.638
Model 2				
Effects	**Coeffecient**	**2*Coeffecient**	**Antilog**	**Z-Values**
V445A	-1.0645	-2.1290	0.1189	-28.206
V445A by V201	-0.1180	-0.2360	0.7897	-2.911
V445A by PSBINT	0.0207	0.0414	1.0422	0.504
V445A by V302 (1)	0.1209	0.2418	1.2735	2.661
V445A by V302 (2)	0.1166	0.2332	1.2626	1.916
Model 3				
Effects	**Coeffecient**	**2*Coeffecient**	**Antilog**	**Z-Values**
V445A	-1.0642	-2.1284	0.1190	-28.239
V445A by V201	-0.1203	-0.2406	0.7861	-3.003
V445A by PSBINT	0.0453	0.0906	1.0948	1.117
V445A by TT (1)	0.0120	0.0240	1.0242	0.266
V445A by TT (2)	-0.0638	0.1276	1.1361	-1.215
Model 4				
Effects	**Coeffecient**	**2*Coeffecient**	**Antilog**	**Z-Values**
V445A	-1.1533	-2.3066	0.0995	-24.145
V445A by V201	-0.1117	-0.2234	0.7997	-2.731
V445A by PSBINT	0.0373	0.0746	1.0774	0.908
V445A by PLACED (1)	-0.0345	-0.0690	0.9333	-0.586
V445A by Placed (2)	0.1929	0.3858	1.4707	3.578
Model 1				
Effects	**Coeffecients**	**Coeffecients**	**Antilog**	**Z-Values**
V445A	-1.0729	-2.1458	0.1169	-30.705
V445A by V201	-0.0781	-0.1562	0.8554	-1.841
V445A by PSBINT	0.0482	0.0964	1.1012	1.185
V445A by S109B (1)	0.1982	0.3964	1.4864	3.139
V445A by S109B (2)	0.0166	0.0332	1.0338	0.313
V445A by S109B (3)	-0.0570	-0.1140	0.8922	-0.076

7.5 Conclusion

Some of the variables that appear to be associated with low BMI (maternal health indicator) are the same as those which were associated with infant/child survival namely high parity, region of residence, maternal education and use of health technology for intervention. Although this exploratory analysis reveals that there may be association between child survival and maternal health, the mechanisms through which they may operate are still merely speculative. Hence, there is a need for study designs as well as information to be collected in such study designs that can enable analysts to discern reverse causality between infant/child survival and maternal health outcomes.

Further, in terms of health intervention, rather than focusing on child survival alone, there is growing need for a holistic approach which should also encompass the mother's health needs. Moreover the mother is usually the main respondent in most of these surveys and yet no information is solicited regarding their health status. In short we may recall the statement made by Winikoff (1988), that in the past the major health problems of a community were addressed in terms of their effects on children, hence, if the focus is now to be on women's health problems, it would be an important complement.

CHAPTER 8

SUMMARY AND GENERAL CONCLUSIONS

This study has attempted to examine the extent of familial association in the risks of infant and child survival and maternal health. A number of demographic studies in recent years have focused on the problem of death clustering or inter-family heterogeneity in child survival. Death clustering refers to the concentration of infant and child deaths among certain mothers and is a consequence of siblings sharing many of the same mortality risks. It has been noted that ignoring the effects of death clustering in the estimation of parameters of covariates of infant and child survival may bias the results. Obtaining unbiased estimates of parameters relating to various covariates is of great value to policy makers because of the need to distinguish real from spurious effects of various covariates in order to design appropriate intervention measures. Further, death clustering in families could have considerable implications for the reproductive health and child survival programmes.

In developing countries, health services are made available largely in response to demand. Hence, if child deaths are heavily concentrated in certain families, this would suggest that substantial improvements in child mortality could be achieved by adopting the more cost-effective techniques of focusing on the sub-group of families with a high risk of child deaths

In this light, the present study set out to achieve the following objectives:

(a) Assess the extent of clustering of infant and child deaths in families in Kenya at neonatal, postneonatal and child mortality levels.

(b) Identify factors affecting infant and child survival in Kenya without and with accounting for excess variation in child deaths.

(c) Examine the implications of child survival on maternal health.

In order to achieve the first objective, two methodologies which have been used by other researchers in the same area, have been applied. The first method involved counting the number of women who had experienced more than one child loss and the second method was an examination of whether the number of women with different numbers of child deaths exceeded that which would be expected if the risk were constant for all women and their children.

The results indicate that there is evidence of death clustering at every level of childhood mortality. The results of the theoretical distribution indicate that at any parity, there are women who contribute higher than average deaths to the mortality risk. This may increase with parity because such women may try to replace their dead children or if 'hoarding' strategy is adopted in family building then they may have more children in anticipation that some may die. However, one limitation of these methods is that they are heavily weighted by women with no deaths. Some of the causes of clustering may arise out of the different characteristics of women which may be biological or behavioural, thus the method may be a necessary indicator of clustering but not sufficient to prove existence of death clustering in families. But in the absence of well established methodologies to detect concentration of deaths to women in certain families, they suffice to indicate that certain families experience higher than average risks in infant and child deaths. This implies that the assumption of independence in siblings' observation may not hold good in the analysis of determinants of

infant and child mortality.

For the second objective, we have examined factors that may affect infant survival in families in Kenya at each survival stage in infancy without accounting for any excess variation. In the neonatal and postneonatal periods, the variables with high predictive power are receipt of BCG, immunization and prematurity. The high significance of non receipt of BCG in the neonatal period, could be spurious, implying that prematurity may be the main factor that is independently explaining the lower risk of survival in this period. This therefore indicates that the causes of survival in this period may have their roots in both biological and behavioural factors. It is argued that the distinction that separates causes such as congenital malformation and prematurity from infectious diseases which are environmentally determined, should be reevaluated. These findings have implications on strategies for intervention of preventing premature births, the majority of which appear to occur in the neonatal and postneonatal periods.

In infancy where the combined effects of these periods plus deaths occurring in the twelfth month are examined, biological, behavioural and environmental factors became more apparent. The effects of maternal education do not appear to be important here although the parameter estimates are in the expected direction. This implies that maternal education may be acting through other variables which are not included here such as income and partner's educational level. And in childhood, the effect of maternal age (more specifically young age), which is not important in the earlier periods, become apparent. This may possibly be an indication of lack of experience in child rearing practices by these young women, who may be experiencing their first births. The effect of the social environment as proxied by type of

toilet facility is also prominent in this period, similarly region of residence is also important in this period.

From the analysis of the effect of maternal education on survival at different stages of infancy, it is observed that the parameter estimates for women of all educational categories are not statistically significant in the gross effects and when the biological/family formation variables are added to the model. However, when utilization of health care factors are included in the model, there is a substantial upward shift in the magnitude of the relative risk estimates for no education category, and the estimates are significant indicating that children of women with no education have better survival chances than even those of women with secondary and above levels. This pattern does not, however, conform to the findings of the majority of studies which have shown an inverse relationship between maternal education and level of infant and child survival, but are consistent with other studies carried out in Kenya. It is important to note that in the neonatal period where health technology is crucial to the survival of neonates, the advantages of maternal education may not be important, rather, the household's social status may be the crucial factor (i.e., whether they can afford the available technology or not). The relative risk estimates for children of women with primary incomplete and primary complete levels of education in the neonatal period despite showing higher survival odds, are not statistically significant in all the models. As stated earlier, possibly the education advantage may not be important in this period.

In the postneonatal period, children of women with primary incomplete level of education have lower survival odds in all the models. The relative risk estimates for primary incomplete category remain significant and consistent throughout while those for no education

and primary complete remain insignificant throughout. One noticeable aspect of the relative risk estimates for all the categories, are their low values compared with those for the neonatal period. The relative risk estimates for survival in infancy, showed a pattern that lies between that of neonatal and postneonatal periods.

In order to fully achieve objective two, we re-examined factors affecting infant and child survival in families in Kenya using the generalised logistic regression model (logistic regression with random effects term). The standard logistic regression assumes that each child is an independent observation. This assumption may not always hold since children from each woman are pooled and treated as if they are independent observations although they share a number of characteristics of their mother. Similarly, children belonging to the same community may share a number of characteristics. Ignoring completely the dependence among observations of siblings of the same family may lead to standard errors that are understated and parameter estimates that are both biased and inconsistent when estimating non-linear models. In other words, estimates obtained under the standard logistic regression although may be reasonable, would tend to have too small variances leading to spurious results. Also, the standard logistic regression does not take into consideration the unobserved and/or unmeasured heterogeneity. There are factors which significantly affect survival chances of children (frailty of each child, biological or behavioral characteristics of mother, ecological setting) which are not observed or measured. The extension of the standard logistic regression to the generalised model can also be viewed as a model which accounts for the extra variation that arises due to omitted variables. The extra parameter then measures the amount of deviation from the standard logistic distribution.

Taking the above into consideration the results however, indicate that in all the models fitted, the random effects term (scale parameter) was not significant. This is not consistent with what has been observed in other similar studies which have used the same methodology (Curtis et al., 1993; Madise and Diamond, 1995; Curtis and Steele, 1993) but is consistent with findings of Guo (1993) who used proportional hazards model. The possible reasons for such inconsistencies could be due to omitted variables such as prematurity, utilization of health care services and household environment and social status factors such as type of toilet facility, which are included in the present study but not in the afore mentioned studies. Another possible reason for the observed differences in the studies could be that the data used in the present study may be more homogeneous in the sense that it relates only to a rural sample. **The extent to which the differences could be due to sampling variability is unknown and as such this calls for further research on such influences on estimation of the models.**

From the results obtained in the previous sections, it is observed that most of the covariates which are statistically significant in the fixed effects models, persist to be significant in the random effects models. The direction of the estimates also remains the same. Further, most studies which have studied the phenomenon of interest as in the present study, have identified preceding birth interval and survival status of the preceding child as factors that may uniquely be associated with familial effects (Curtis et al., 1993; Zenger, 1993). The reason for this could be that previous birth interval may reflect past fertility-related decisions and as such will be correlated with family-specific frailty endowments (Wolpin, 1997).

The preceding birth interval during the neonatal period shows some attenuation effects, where

it is significant in some models and not significant in others. The reasons for this are not easy to discern from the present analysis. In the postneonatal and infant survival models, the effect of short birth interval remained statistically significant (negative) throughout the models. The available literature regarding the effect of short birth interval on survival in infancy, appears to be inconclusive and mixed. **Although short birth interval persists to be statistically significant in some models, but the pathways through which it operates are still unknown and hence a subject of further research. For example, unless specific causes of death are known, it is not very easy to speculate as to whether the short birth interval was detrimental to the survival status of the index child (the same applies to the survival status of the preceding child).** The argument that prior birth intervals reflect family frailty endowments as suggested by Wolpin (1997) may not hold because families' behaviour towards the survival of their children may be altered through social learning and interaction since parents may recognize an unfavourable genetic inheritance in their children and accordingly may make extra efforts to keep them healthy, thus counterbalancing the genetic disadvantages. It is however not yet clear whether the increments to birth intervals in a relatively healthy and well-fed population are indeed important for the health of the mother and child except in extreme cases (Schultz, 1997).

Another source of familial effects which has featured prominently in the literature is parental competence as proxied by maternal education (Das Gupta, 1990; 1997). In all the models considered here, the effects of maternal education are introduced after the inclusion of the household exposure variables (source of drinking water and type of toilet facility). The surprising result that emerged from the study is that in the neonatal survival period, children of women with no education seem to have higher survival odds than those of women in the

other categories of education. These results are consistent with those of Boerma and Bicego (1990), who used data from the Kenya Demographic and Health Survey (KDHS) of 1989. This could be due to the fact that in this period, most of the deaths may occur as a result of biological factors associated with congenital malformations which may not reflect maternal competence.

The other unique finding with regard to maternal education is that at the postneonatal stage, children of women who had primary incomplete level of education had a lower odds of survival. This type of finding is difficult to explain especially with regard to the maternal competence hypothesis. Guo (1993) also found that parental competence hypothesis may not hold in all communities and should therefore not be generalized due to different rearing practices.

Further, education is related to income, which may in part be related to the utilization of health care services (where cost is a factor for utilization). Hence, an alternative way of looking at the effect of maternal education would be to control for family income and other socio-economic status factors. Perhaps this could be the reason why type of toilet facility was persistently statistically significant (negative). This could be a reflection of the socio-economic status of the household rather than the inadequacy of the toilet facilities since toilet facilities are more expensive to instal in the rural areas such that rural households with flush toilet facilities are of higher wealth.

Although random effects models seem to provide the more unified approach to logistic regression analysis, interpretation of the estimates of parameters of the covariates in these

models is however difficult. The random effects term(s) may be considered to represent the totality of cluster-constant covariate effects omitted from the model that are independent to those already present. Since the term measuring the random effects is negligible in the present study, the standard logistic model suffices for interpretation of the covariate effects. However, it should be noted that even the random effects models will only be without bias as long as the omitted variables are independent of those included in the models which in a way is not sufficient to conclude that when scale parameter in the model is significant therefore accounts for the presence of familial effects. A test for the presence of familial effects may only be possible in an experimental situation where the study sample is taken from the same or related universe some of which can be held constant or where a cluster-specific variable which would identify children of the same woman, is included in the analysis and possibly where the cluster size is fixed for all observations.

Hence our results of the analysis showing that the random effects are not significant may indicate that a number of covariates accounting for infant survival may have been included in the models. This conclusion follows the interpretation of the random effects term as that which accounts for factors not included in the model rather than a measure of familial (cluster) specific risks as have been used in past literature (Curtis et al., 1993; Madise and Diamond, 1995; Curtis and Steele, 1996; Das Gupta, 1997). One way in which familial effects could be studied in such models, would be to analyse the mortality risk of one child per mother. But, if a mother has more than one child, which one should be selected for study? Also, the characteristics of the child, mother and community may be interrelated as such, models which consider nested frailty may be appropriate in such studies but their demographic interpretation need to be reconsidered.

In the third objective, we have explored the association between child survival and maternal health. The findings of the analysis show that some of the variables that appear to be associated with low BMI (maternal health indicator) are the same as those which are associated with infant/child survival namely high parity, region of residence, maternal education and use of health technology for intervention. **Although this exploratory analysis reveals that there may be association between child survival and maternal health, the mechanisms through which these may operate are still merely speculative. Hence, there is a need for study designs as well as information collected in such designs that can enable the analysis of reverse causality between infant/child survival and maternal health outcomes.** This will enable the investigation of the much touted **"maternal depletion syndrome"** which is cited in almost every literature on child survival and birth spacing but with little or no empirical evidence to support the hypothesis. Perhaps this would allow for the evaluation of Schultz's (1997) statement that most estimates of consequences of prior birth intervals in low-income countries are potentially biased, for they treat the prior birth interval and the health of a child and mother as an estimate of causal effect but it is not yet clear whether the increments to birth intervals in a relatively healthy and well-fed population are indeed important for the health of the mother and child except in extreme cases.

Further, in terms of health intervention, rather than focusing on child survival alone, there is growing need for a holistic approach which should also encompass the mother's health needs. Moreover, the mother is usually the main respondent in most of these surveys yet no information is solicited regarding their health status. Winikoff (1988) noted that in the past the major health problems of a community are addressed in terms of their effects on children, hence, if the focus is now to be on women's health problems, it would be an important

complement.

Policy Implications

Child survival is closely linked with the pace (timing and spacing) and number of births and to the reproductive health of mothers. Early, late, numerous and closely spaced pregnancies are major contributors to high infant and child mortality and morbidity rates, especially where health care services are scarce. Where infant mortality remains high, couples often have more children than they otherwise would to ensure that a desired number survives (ICPD, 1994). Subsequently Kenya's draft population policy document (1996) and the national Development plan for the period 1996-2001 do not list infant and child survival as a critical population issues of concern. However under the safe motherhood issues, the areas of concern include provision of family planning services, antenatal care, clean and safe delivery, postnatal care, promotion of breast feeding and maternal nutrition, all of which are derived from the ICPD designated actions to increase child survival.

These policy documents, however, do not distinguish between age patterns of infant and child survival more specifically in terms of the different rates of mortality and determinants of survival at different ages. The present research and other researches have shown that different factors influence infant and child survival at different ages and that as infant and child mortality begins to decline, the bulk of infant deaths occur in the perinatal and generally in the neonatal period. This has obvious implications on the basis of action suggested in the a fore mentioned documents. From the findings of the present study, it has been observed that prematurity is a major contributor to neonatal and postneonatal deaths.

By implication, this will shorten birth interval lengths without being amenable to the effects of family planning. This calls to question the viability of family planning as a policy instrument to induce further declines in infant and child mortality. The other basis for action is the expansion of immunization and breastfeeding. But if prematurity is a major determinant of survival in the neonatal and postneonatal periods, then infants dying in these periods are not likely to benefit from such interventions.

Given the above scenario, suitable policy objectives and instruments must address the issue of prematurity and also immaturity (although not investigated in the present study). This calls for monitoring of pregnancies and management of delivery. While monitoring involves utilization of antenatal care services, management involves utilization (availability, accessibility and acceptability) of intrapartum care services. Whereas in Kenya, there is high utilization of antenatal care services, the use of intrapartum services is very low especially in the rural areas. Therefore, the basis for policy instruments must seek to understand the reasons behind low utilization of intrapartum care services in rural areas, which are necessary for management of preterm births. Because of the linkage between preterm births and frequent pregnancies, management of such births will be beneficial to the health of the mother and child.

Study Limitations

The study suffers from limitations of data derived large scale surveys in that variables collected for other uses when used as proxies, may not accurately measure the phenomenon of interest.

It is also possible that some of characteristics of the mother, child and community are interrelated and as such models that consider nested frailty may be more appropriate for such studies, however, their demographic interpretation need to be reconsidered. Other limitations of the study have been outlined and highlighted in the text and suggested as areas for further research.

REFERENCES

Amankwaa, AA (1996)
Prior and Proximate causes of infant survival in Ghana, with special attention to polygyny. J. Biosoc. Sci. vol.28 (3), pp.281-295

Ashton, LS (1995).
New Perspectives on Population: Lessons Learnt in Cairo. Population Bulletin. Vol.15 (1),pp.28.

Bankole, A and Olaleye, D (1991)
The Efffects of Breastfeeding on infant and Child Survival in Kenya. Demographic and Health Surveys, Vol.11, pp.1045-1072.

Bicego, GT and Boerma, JT (1990),
Maternal Education and Child Survival: A comparative Analysis of DHS Data. Demographic and Health Surveys, Vol.1,pp.177-204.

Blacker, et al., (1987).
Mortality Differentials in Kenya. A paper presented to IUSSP Seminar on Mortality and Society in Sub- saharan Africa, Younde.

Boerma, JT and Bicego, GT (1990).
Preceding Birth intervals and Child Survival: Searching for Pathways of Influence. Demographic and Health Survey, Vol.1,pp.1183-1197.

Bongaarts, J (1987)
Does Family Planning reduce Infant Mortality Rates? Pop. and Dev. Rev. Vol.13 (2),pp.323-334

Brass, W and Jolly, CL (1993)
Population Dynamics of Kenya, National Academy Press, Washington D.C.

Caldwell, JC (1979)
Maternal Education as a Factor in Mortality Decline an Examination of Nigeria Data. Population Studies Vol.33(3),pp.395-413

Caldwell, JC (1986)
Routes to Low Mortality in Poor Countries. Pop. and Dev. Rev Vol.12 (2) pp.171-220

Chidambaram, V.C and Zeba A. Sathar (1984)
Age and Date Reporting. WFS Comparative Studies No.5. Voorburg, Netherlands: International Statistical Institute

Cleland, JG and Sathar, ZA (1984)
The effect of birth spacing on childhood mortality in Pakistan. Pop. Studies 38 (3)

pp.401-418

Cleland, JG (1990)
Maternal Education and Child Survival: Further Evidence and Explanations. What Do We Know About Health Transition: The Cultural, Social and Behavioural Determinants of Health Vol.1 pp.400-419 (eds) J.Caldwell et al.,Canberra: Health transition Centre, ANU

Curtis, S.,(1995)
Assessment of The Quality of data used for Direct Estimation of Infant and Child Mortality in DHS-11 Surveys. Occasional Paper No.3 Demographic and Health Surveys. Macro International Inc. Calverton, Maryland

Curtis et al,. (1993).
Birth Interval and Family Effects on Postneonatal Mortality in Brazil. Demography Vol.30 (1),pp.3-4

Curtis and Steele, (1996).
Variations in Familial Neonatal Mortality Risks in Four Countries. J. Biosoc. Sci.VOl.28 (2),pp.141-159

Das Gupta, M, (1990).
Death Clustering, Mother's education and Determinants of Child Mortality in Rural Punjab, India. Population studies vol.44(3),pp.489-505

_____ (1997).
Socio-economic Status and Clustering of Deaths in Rural Punjab. Population Studies. 51:191-202

Da Vanzo, J and Starbird, EH (1991)
Correlates of Short Interbirth Intervals in Peninsular Malaysia: Their Pathways of Influence Through Breastfeeding and Contraceptive Use. Stud. In Fam. Planning Vol.27 94) pp.241-254

Ewbank et al., (1986)
An Integration of demographic and epidemiologic research on mortality in Kenya. In Determinants of Mortality Change and Differentials in Developing Countries: A Five Country Case Study Project. United Nations, New York.

Farah, A and Preston, SH (1982).
Child Mortality Differentials in Sudan. Pop. and Dev. Rev. Vol.8 (2) pp.365-383

Fogel, WR (1997)
New Findings on Secular Trends in Nutrition and Mortality: Some Implications for Population Theory. A handbook of Population and Family Economics vol.1a. pp.433-481. Mark R. Rozenzweig and Oded Stark (eds), Elsevier

Gandotra, Das and Dey (1982)

Infant Mortality and It's causes in Gujarat. Population Research Centre, Baroda

Geronimus (1986)
"Comment on 'Toward a reformulation of of Neonatal Mortality Rate' by Dudley L. Poston and Richard Rogers". Social Biology,33 (3-4)pp.326-327

Goldenberg, RL (1996)
"Editorial: Inttrauterine Infection and Why Preterm Prevention Programs Have Failed". American Journal of Public Health, 86 (6)pp.781-83

Gubhaju et al., (1991)
Socio-economic, Demographic and Environmental Determinants of Infant and Child Mortality in Nepal. J. of Biosoc. Sci:23, pp425-435.

Guo, G (1993).
Use of Sibling Data to Estimate Family Mortality Effects in Guatemala. Demography Vol.30 (1),pp.15-32

Guo, G and Grummer-Strawn (1993)
Child Mortality Among Twins in Less Developed Counties. Population Studies:47 pp.495-510

Hauth et al., (1995)
"Reduced Incidence of Preterm Delivery With Metronidazole and Erythromycin in Women With Bacterial Vaginosis". The New England Journal of Medicine,333 (26)pp.1732-36

Hill, A (1989).
Trends in Childhood Mortality in Sub-Saharan Mainland Africa. Mortality and society in Sub-Saharan Africa, Van de Walle, Pison and Sala-Diakanda (eds). Clarendon Press, Oxford.

Hobcraft et al (1984).
Socioeconomic Factors in Infant and Child Mortality: Cross-National Comparison. Population Studies Vol.38 (2),pp.193-223.

Hobcraft, J et al,. (1985).
Child Spacing effects on Infant and Early Child Mortality. Pop. Index Vol.49,pp.485-618.

Hobcraft, J et al,. (1985).
Demographic Determinants of Early Child Mortality: A Comparative Analysis: Pop. Studies Vol.39(3),pp.363-385.

Hobcraft, J (1993)
Women's Education, Child Welfare and Child Survival: A review of the Evidence. Health and Transition Review Vol.3. (2) pp.159-175

Ikamari, L (1996)
Factors affecting Child Survival In Kenya. Unpublished Ph.D Thesis, Australian National University (ANU), Canberra

Jain, AK. (1984)
Determinants of Regional Variations in Infant Mortality in Rural India. Infant Mortality in India: Differentials and Determinants (eds). A.K.Jain and P.Visaria, Sage Publication.

James et al., (1988)
Definition of Chronic Ernegy Deficiency in Adults. European Journal of Clinical Nutrition, 42 (12)pp. 969-981

Kibet, MK (1981)
Mortality Differentials in Kenya. Unpublished M.Sc. Thesis, University of Nairobi

Koenig et al. (1990)
Birth Intervals and Childhood Mortality In Bangladesh. Demography Vol.27 (2),pp.251-265

Koyugi, BO (1994)
Household and Environmental Factors affectng Child Survival in Kenya. Unpblished Ph.D. Thesis, University of Nairobi

LeVine et al., (1991)
Women's Schooling and Child Care in the Demographic Transition: A Mexican Case Study: Population and Development Review, 17 (3) pp. 459-496

Lindenbaum, S (1990)
Maternal education and Child Care in Bangladesh: The Health and Hygiene of the Middle Classes:What Do We Know About Health Transition: The Cultural, Social and Behavioural Determinants of Health Vol.1 pp.425- (eds) J.Caldwell et al.,Canberra: Health Transition Centre, ANU

Madise and Diamond (1995)
Determinants of infant mortality in Malawi: An analysis to control for death clustering within families. J. Bisoc. Sci:25 pp95-106

Majumder, AK (1990)
"Child Survival and its Effects on Mortality of Siblings in Bangladesh". J. of Biosoc. Sci Vol.22 (22),pp.333-347

McCarthy and Maine
A Framework for Analyzing the Determinants of Maternal Mortality. Stud. in Fam. Plan.Vol.23 (1),pp.23-33 (1992)

McKeown, T (1976)
Modern rise of Population. London: Edward Arnold (Pub)

Meegama, A. (1980)
Socioeconomic determinants of infant and child mortality in Sri Lanka: An analysis of Post-war experience. Scientific Report No.8 (London: WFS,1980).

Mensch et al., (1985)
Socioeconomic Differentials in Child Mortality in Developing countries, New York. Dept. of International and Social Affairs, UN, (ST/ESA/SER.A/97).

Miller,J (1989)
'Is the relationship between birth interval and perinatal mortality spurious? Evidence from Hungary and Sweden'. Pop Studies vol.43 (4) pp.479-95.

Miller, J (1994)
Comment on James Gribble's Birth Intervals, Gestational age and Low Birth Weight: are the relationships Confounded? Population Studies Vol.48 () pp.359-363

Miller et al., (1992)
Birth Spacing and Child Mortality in Bangladesh and The Philippines. Demography. Vol.29 (2),pp.305-318.

Mosley and Chen (1984)
An analytical Framework for the Study of Child Survival in Developing Countries. Child Survival Strategies for Research. (Ed). W.H. Mosley and Lincoln C. Chen,pp.25-45. Pop. and Dev. Rev.10. Supplement. New York: Population Council.

Mott, F. (1982)
Infant Mortality in Kenya: Evidence from the Kenya Fertility Survey. Scientific Reports No.32. World Fertility Survey

Muganzi, Z (1984)
Contextual Factors affecting Child Mortality In Kenya. Unpublished Ph.D Thesis, Florida University

NCPD/IRD (1989)
Kenya Demographic and Health Survey. Columbia, Maryland

_____ (1993)
Kenya Demographic and Health Survey. Columbia, Maryland

Oppong, C and Abu, (1987)
Seven Roles of Women: Impact of Education , Migration and employment on Ghanaian Mothers. Women. Work and Development (13), ILO, Geneva.

Palloni and Millman (1986)
Effects of inter-birth intervals and Breastfeeding on Infant and Early Child Mortality. Pop. Stud. Vol.40 (),pp.215-236

Pandey et al., (1998)

Infant and Child Mortality in India. National Family Health Report (Unpublished). International Institute for Population Sciences, Mumbai, India and East West Centre Program on Population, Honalulu, Hawaii, USA

Pant, DV (1991).
Effects of Education and Household Characteristics on Infant and Child Mortality In Urban Nepal. J. of Biosoc. Sci:23, pp.437-443

Pebley and Elo (1989)
The relationship of Birth spacing and Child Health. International Population Conference. New Delhi. Vol.1,pp.403-415.

Pebley, A and Mbugua, W (1989)
Polygyny and Fertility in Sub-saharan Africa. Reproduction and Social Organisation in Sub-Saharan Africa. Lesthaegue, RH (eds). University of Carlifonia Press, Berkeley

Pebley, A and Millman, S (1986)
Birth Spacing and Child Survival. Intern. Fam. Plan. Persp Vol.12 (20 pp.71-79

Pebley, A and Stupp, P.W (1987).
Reproductive Patterns and Child mortality In Guatemala. Demography Vol.24 (1),pp.43-60

Potter, J (1988)
Birth Spacing and Child Survival (A cautionary Note regarding Evidence from WFS). Pop. Stud. Vol.42 (),pp.443-450

Preston, SH and Nelson, VB (1974)
Structures and change in the causes of death: An internationalsummary. Population Studies. Vol.20, pp19-51

PSRI/UNICEF (1994)
Kenya Maternal Mortality Baseline Survey (KMMBS).Unpublished Report

Republic of Kenya (1996)
National Development Plan 1997 - 2001

Republic of Kenya (1996)
Kenya Population Census 1989 Analytical Report Vol.4. Fertility and Nuptiality, CBS, OVP&MNPD.

Retherford et al (1989)
To what extent does breastfeeding explain birth-interval effects on early childhood mortality? Demography Vol.26 (3),pp.439-450

Ronsmans, C., (1995).
Patterns of Clustering of Child deaths in a Rural Area of Senegal. Population Studies

Vol.49 (3),pp.443-461

Rutstein, SH (1984).
Socio-economic Differentials in Infant and Child: Preliminary Tables. WFS Comparative Studies. Cross-National Summaries, International Statistical Institute, The Hague, Netherlands.

Sastry, S (1997)
Nested Frailty Model for Survival Data: With an Application to The Study of Child Survival in North Brazil. Journal of The American Statistical Association: Applications and Case Studies Vol.92 (438) pp. 426-435.

_____ (1997)
Family-Level Clustering of Childhood Mortality Risk in Northeast Brazil. Population Studies, 51:245-261

Schultz, TP (1997)
Demand for Children in Low Income Countries. A handbook of Population and Family Economics vol.1a. Mark R. Rozenzweig and Oded Stark (eds), Elsevier

Sodermann et al., (1997)
High Mortality Despite Good care Seeking Behaviour: A Community Study of Childhood Deaths in Guinea-Bissau. Bulletin of The World Health Organisation Vol.75 (3) pp.205-212

Sowards, KA (1997)
Premature Births and the Changing composition of Newborn Infectious Disease Mortality:Reconsidering 'Exogenous' Mortality. Demography Vol.34 (3) pp.399-409

Streatfield et al (1990)
Maternal education and Child Immunization. Demography Vol.27 (3),pp.447-455.

The Cebu Study Team (1990).
Underlying and Proximate determinants of Child Health: The Cebu Longitudinal Health and Nutrition Study. Am. Journ. of Epid. Vol.133 (2),pp.185-201

Trussell, J and Pebley, A (1984).
The potential Impact of Changes in Fertility on Infant, child and Maternal Mortality. Stud. in Fam. Plan Vol.15 (6),pp.267-280

Trussell, J and Menken, J (1984)
Estimating Levels, Tends and Determinants of Child Mortality in Countries with Poor Statistics. Child Survival Strategies for Research, Ed. W.H. Mosley and Lincoln C.Chen, pp.325-346. Pop. and Dev. Rev. Supplement Vol.10. New York: Population Council.

United Nations (1991).
Child Mortality in Developing Countries: Socio-economic Differentials, Trends and

Implications, United Nations, New York

United Nations (1994).
ICPD, Cairo. Programme of Action. United Nations, New York.

United Nations (1994).
World Population Prospects. United Nations, New York.

UNICEF (1995).
State of the World Children Report. United Nations, New York.

Venkatacharya, K. (1985)
An Approach To the Study of Infant and Child Morbidity and Mortality. International Population Conference, Florence Vol.2. Liege IUSSP

Ware, H (1984)
Effects of Maternal Education, Women's Roles and Child Care on Child Mortality. Child Survival Strategies for Research: Pop. And Dev. Rev. Supplement to Vol.10,pp.191-214

Winkoff, B. (1983)
"The Effects of Birth spacing on Child and Maternal Health". In Studies In Fam Plan.Vol.14 (10),pp.231-245

Wolpin, K.I (1997)
Determinants And Consequences of the Mortality and Health of Infants and Children. A handbook of Population and Family Economics vol.1a. Mark R. Rozenzweig and Oded Stark (eds), Elsevier

World Health Organisation (1994)
SAFE MOTHERHOOD, Mother-Baby Package: Implementing safe Motherhood in Countries. WHO, Geneva.

Zaba and David (1996)
"Fertility and the Distribution of Child Mortality Risk Among Women: An Illustrative Analysis". Population Studies,50, pp.263-278

Zenger,E (1993)
Siblings' Neonatal Mortality Risks and Birth Spacing in Bangladesh. Demography Vol.30 (3) pp477-488