

**The Upsurge in Infant and Child Mortality in Kenya During
the 1988-2003 Period: Levels and Determinants //**

By

Sam Wangila Wafula

**A thesis submitted in fulfilment for the degree of Doctor of Philosophy
in Population Studies of the University of Nairobi**

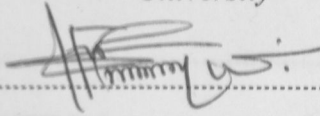


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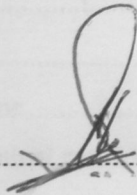
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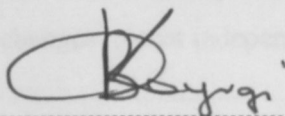
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LIST OF ABBREVIATIONS

AIDS	Acquired Immune Deficiency Syndrome
AFT	Accelerated failure time models
CBS	Central Bureau of Statistics
CMR	Child Mortality rate
DoMC	Division of Malaria Control
HIV	Human Immunodeficiency Virus
IMR	Infant mortality rate
ITNs	Insecticide Treated mosquito nets
KDHS	Kenya Demographic and Health Surveys
KEMRI	Kenya Medical Research Institute
KMICS	Kenya Multiple Indicator Cluster Survey
NCAPD	National Coordinating Agency for Population and Development
NMS	National Malaria Strategy
OLS	Ordinary Least Squares
PCA	Principal Component Analysis
SAPs	Structural Adjustment Programs
SPSS	Statistical Package for Social Sciences
UNICEF	United Nations Children's Education Fund

ABSTRACT

Kenya experienced an upsurge in infant and child mortality during the 1988 to 2003 period. The factors underlying this upturn are poorly understood. A better understanding of these factors is critical in designing appropriate child survival programs and strategies for the reduction of infant and child mortality as envisioned in major national and international public health programmes. This study established whether the observed upsurge in infant and child mortality during the 1988-2003 was real and if so, the extent to which it increased. Secondly, it identified some of the major factors that may have been associated with that upturn. Finally, the study investigated whether some of the factors behind the upturn in high HIV prevalence regions were different from those in low HIV prevalence regions and if so, to establish structural relationships that explained the mortality differences in the two HIV regions.

Data drawn from the 1993, 1998 and 2003 Kenya Demographic and Health Surveys (KDHS) were used. A set of demographic data assessment and evaluation techniques were used to address objective one. Parametric models (specifically the Weibull AFT models) and regression decomposition techniques were used to address objectives two and three respectively.

Preliminary findings showed that infant mortality increased by 30 percent while child mortality increased by 9 percent during the 1988-2003 period. Data assessment revealed that data were generally of high quality. In cases where there was heaping in age reporting of children such as at months 12, the effect would only have lead to an understatement of infant mortality and not its upsurge. Further, while there was heaping at months 12, 18, 24 and 36, the effect of this bias on child mortality estimation is negligible since all these ages are conventionally part of child mortality estimation.

Weibull AFT models revealed that compared to infants who were born during 1988-92 period, those born during the 1993-98 and 1999-2003 period were 43 percent more likely to die faster respectively and this association was statistically significant (p value < 0.05). During childhood, the risk of dying faster among children born during 1993-98 and 1999-2003 was 45 percent and 28 percent as compared to those who were born during the 1988-92 period (p value < 0.05). Overall, the Weibull AFT models explained an upsurge of 55 percent in infant mortality and 62 percent in child mortality (p value < 0.05).

The results obtained using regression decomposition techniques showed that there were differences in the covariate values of mortality between the two periods and HIV regions. Partially, these differences accounted for the differences in the observed infant and child mortality by period and regional HIV prevalence. The differences in infant and child mortality were largely due to the differences in the nature or structure of the relationship between mortality components and the explanatory variables. Duration of breastfeeding, maternal education, regional HIV prevalence and malaria endemicity appeared to explain much of the observed change in infant mortality between the time periods. If all live births that occurred in the 1996/03 period had the same mean values of all explanatory

variables as those of 1988/95 period, then infant mortality would have increased by a massive 14 deaths per 1000 live births. However, had live births that occurred in the 1988/95 period experienced the same mean values of all explanatory variables as those that occurred in the 1996/03 period, the upsurge in infant mortality would have been negligible.

The length of the preceding birth interval, belonging to a mother who received a tetanus injection during pregnancy, maternal education, and malaria prevalence were important explanatory variables behind the change in child mortality. The highest contribution to child mortality during the 1988-2003 period was due to the rise in Malaria endemicity (18 percent increase) followed by the differences in regional HIV prevalence (5 percent increase).

The difference in infant mortality between the high and low HIV zones was 9.2 deaths per 1000 live births while that of child mortality was 2.3 deaths per 1000 live births. An approximate 59 percent of the difference in infant mortality between the two HIV regions was accounted for by the socio-economic factors alone. This exceeded the total contribution of 56 percent increase in infant mortality that was observed between the two HIV regions. Among the socio-economic factors, it appears that the source of drinking water, type of toilet facility and household wealth index played a pivotal role in the observed differences in infant mortality. On the other hand, the study variables explained 39 percent of the difference in child mortality between the two HIV regions. However, it is maternal factors and socio-economic factors that contributed much to the child mortality differences between the two HIV regions (20.4 and 13.8 percent respectively).

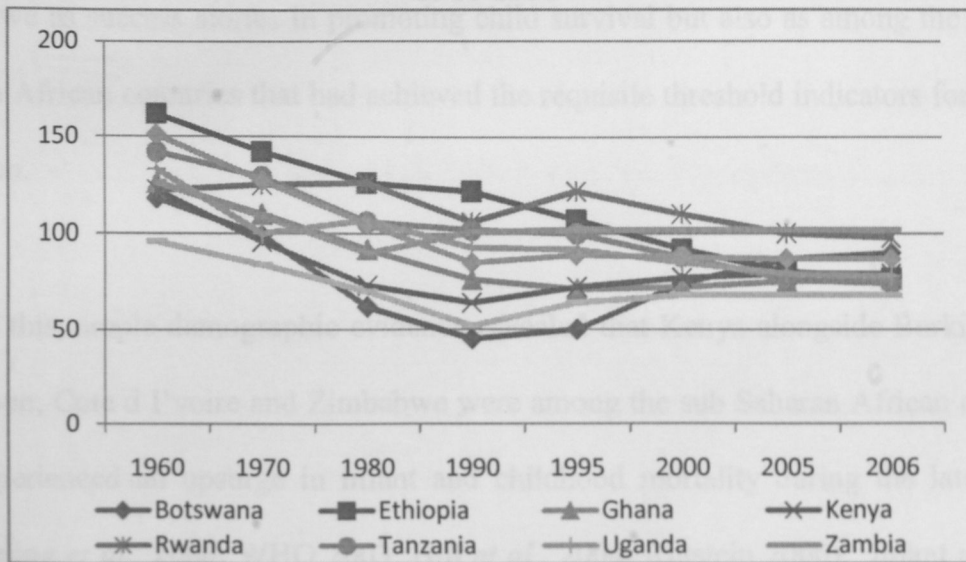
In summary, mortality increase was real during the 1988-2003 period. Efforts aimed at controlling and preventing malaria and HIV should be stepped up to avert childhood mortality. This could entail universal counselling and testing of HIV among pregnant mothers and strengthening prevention of mother to child transmission initiatives; intensified use of anti malaria prophylaxis among pregnant women and children as well as early diagnosis and treatment of Malaria. Additionally, programs aimed at increasing the distribution of Insecticide treated mosquito nets, in house spraying and other vector control strategies in high Malaria prone regions as well as biomedical research for malaria vaccine development need renewed emphasis. There is need to promote maternal education given its known role in reducing infant and child mortality. There is need to scale up family planning programmes to reduce short birth intervals and prevent high risk births. An affordable social health insurance system should be introduced to cater for low income households who cannot otherwise access any form of healthcare. Finally, provision of clean and portable water needs to continue especially through water purification techniques to households that do not have access to piped water.

CHAPTER 1: INTRODUCTION

1.1 Introduction and statement of the problem

Prior to the late 1980s, many studies indicated that sub Saharan Africa enjoyed a dramatic decline in infant and child mortality (Hill *et al.* 2001, Obungu *et al.*, 1994; UN 2005). According to the UN (2005), infant mortality in sub Saharan Africa in 1960 was 149 deaths per 1000 live births but dropped to 101 deaths per 1000 live births by 2000-2004 - a decline of 32.2 percent. However, since the late 1980s, trends in infant and child mortality in sub Saharan Africa started slowing, stalled or even reversed (Rutstein 2000; UNICEF 2001; Ahmad 2000). Figure 1.1 below illustrates the trend in infant mortality for select countries in sub Saharan Africa during the 1960 to the mid 2000s.

Figure 1.1: Trends in infant Mortality for selected sub Saharan African countries, 1960-2006



Source: UNICEF, *et al.*, 2007

Kenya recorded an average of 4 percent decline per annum in infant mortality between 1950s through the early 1980s- a rate that was almost twice the annual average decline of 2.5 percent for the rest of sub Saharan Africa during the same period (CBS, *et al.*, 2004; Hill, *et al.*, 2004, Obungu, *et al.*, 1994). The decline in early childhood mortality in

Kenya and the factors driving the decline prior to the mid 1980s were so clear that Brass and Jolly (1993: 169) noted that studies focusing on the same “had little to add”. The sentiments by Brass and Jolly (1993) were influenced by many local studies which extensively examined the levels, differentials and determinants of the declining infant and child mortality prior to the 1990s (Kibet, 1981; Muganzi 1984; Ewbank *et al.*, 1986; Obungu *et al.*, 1994; Ikamari, 1996; K'Oyugi 1992).

Further, the dawn of the 1990 decade in Kenya was marked by decisive views about Kenya's demography with some leading demographers concluding that Kenya had entered the long awaited demographic transition (Robinson and Cleland 1993; Caldwell *et al.*, 1991). Caldwell (*et al.*, 1992)¹ in particular rated Kenya alongside Botswana and Zimbabwe as success stories in promoting child survival but also as among the few sub Saharan African countries that had achieved the requisite threshold indicators for fertility transition.

Despite this, ample demographic evidence revealed that Kenya alongside Burkina Faso, Cameroon, Cote d'Ivoire and Zimbabwe were among the sub Saharan African countries that experienced an upsurge in infant and childhood mortality during the late 1980's (Houweling *et al.*, 2006; WHO 2003; Hill *et al.*, 2004; Rutstein 2000). Infant and child mortality rates in Kenya increased from 59 and 89 per 1000 live births respectively in 1989 to 78 and 115 deaths per 1000 live births respectively by 2003 (CBS, *et al.*, 2004).

¹ The authors argued that that for a country to enter the demographic transition, it must have threshold levels of infant mortality rate of below 70 per 1000 live births, under-five mortality rate of under 110/1000 live births, contraceptive use rates for married women of at least 27 per cent for all methods or 18 per cent for modern methods, a universal elementary schooling for all girls and that at least 30 per cent of girls should be attending secondary school. Kenya had attained all these thresholds by the onset of the 1990 decade.

This was a percentage increase of about 32 percent in infant mortality and 29 percent in child mortality during the 1988-2003 period.

Reversals in early childhood mortality in Kenya and some African countries during the late 1980s is currently the subject of much demographic and public health concern (Ahmad *et. al.*, 2000; Rutstein 2000; Hill *et.al.*, 2004; Rutstein 2006). The upsurge in Kenya's infant and child mortality is particularly a phenomenon in need for an urgent explanation for the following reasons: First, little is currently known regarding the factors behind the upsurge in infant and child mortality during the 1988-2003 period.

Secondly, there are no recent studies that have been done in Kenya to assess what changes in the commonly known covariates may have contributed to the upturn in infant and child mortality and what factors, if any, have played a key role in maintaining the high levels of mortality during the 1988-2003 period. Finally, systematic analysis of the relative roles of some of the potential correlates behind the rise in infant mortality during the study period remain scarce in Kenya. This study attempts to fill these gaps and establishes some of the factors associated with the upsurge in infant and child mortality during the 1988-2003 period in the country.

While this study focus on the causes behind the upturn in infant and child mortality during the 1988-2003 period, it is important to mention that the recent findings from the KDHS of 2008/09 which were released after this study was nearing completion shows that early childhood mortality has reversed since 2003 and is on the decline. In

particular, infant and child mortality have declined from 78 and 115 deaths per 1000 live births in 2003 to 52 and 74 deaths per 1000 live births respectively in 2008/09 (KNBS, *et al.*, 2010). Owing to the fact that we do not know whether this recent decline in early childhood mortality will be sustained, this study remains critical.

1.2 Literature review

Literature review was done with three main objectives: firstly, to find out the common factors that have been found to influence the change in infant and child mortality rates; secondly, to understand some of the strengths and weaknesses of past methodological approaches on this topic; and finally, to provide a firm basis upon which the study variables would be selected for further analysis.

1.2.1 A global review

Globally, there exists a rich literature on this topic. For instance, Rosero Bixby (1986) explained the factors behind the decline in infant mortality in Costa Rica from 60 deaths per 1000 live births in 1970 to 20 deaths per 1000 live births by 1980. In the study, Rosero-Bixby (1986) computed coefficients of correlation and multiple regression techniques to establish the statistical significance between a number of public health factors, socio-economic factors as well as a decline in fertility on the observed infant mortality rate.

The study findings revealed that the government initiated health programs in Costa Rica in the early 1970s accounted for up to 75 percent of the decline in infant mortality with

primary health care alone accounting for over 40 percent of the observed mortality decline. While Costa Rica enjoyed a period of economic progress during the 1970-1980s, the study attributed only 22 percent of the decline in mortality to the improvement in economic fundamentals. The author further found that a decline in fertility which may have averted high risk pregnancies independently accounted for only 5 percent of the observed infant mortality decline.

Rosero-Bixby's (1986) study however failed to recognize the fact that mortality changes are not only a function of changing levels in the covariates of infant and child mortality but rather, the structure of the relationship between the potential covariates and mortality could also have changed at the same time.

Da Vanzo and Habicht (1986) used a set of regression decomposition techniques to investigate the factors that were responsible for the decline in infant mortality rate in Malaysia from 100 deaths per 1000 live births in the 1940s to 30 deaths per 1000 live births in the 1970s. Using a temporal approach, the authors first performed a fixed structure analysis (i.e. they assumed that it is only the covariates that change over time rather than infant mortality) and estimated that on the overall, 16.5 percent of the decline in infant mortality during the 1946-60 and 1961-1975 was due to changes in the selected covariates. Their study found that maternal education played a critical role in infant mortality decline.

However, when Da Vanzo and Habicht (1986) ran models that allowed for structural changes, they found that much of the mortality decline (about 80 percent) could not be accounted for by factors considered in their study (Da Vanzo and Habicht 1986:152). Rather, most of the factors were unexplained –that is they were accounted by the intercept term. This finding is similar to a study conducted by Hale *et al.*, (2009:18) which found that the explanatory factors that were considered in their study explained less than 20 per cent of the decline in mortality between 1987-1994 and 1995-2002 in Matlab, Bangladesh during sub periods of infancy and about 40 per cent of the decline in child mortality.

In a similar study, Da Vanzo (1988) using regression decomposition techniques to study the concomitants of development found that an increase in maternal education in Malaysia's richer and poor states by four and five times was responsible for a reduction in infant mortality of about 10.3 and 14.8 deaths per 1000 live births respectively (Da Vanzo 1988:587). These results echo other findings which show a negative association between infant and child mortality and maternal education (Caldwell 1979; Hobcraft, *et al.*, 1984; K'Oyugi 1992; Ikamari, 1996).

Kintner (1988) studied the temporal and areal determinants of infant mortality decline during the 1871-1933 period in the administrative areas of Germany . He used regression decomposition techniques. Kintner found that on the overall, infant mortality rate fell by 69 percent during the study period. Furthermore, marital fertility, percentage rural, percentage catholic, access to medical care, percentage illegitimate, urbanization and

infant welfare centres accounted for 75 percent of the variance in the observed infant mortality rate (Kintner 1988:600).

Kintner (1988:606) found that a reduction in total fertility rate of one child per woman reduced infant mortality rate by 22 points in 1871 and 17 points in 1900. Like other studies (Da Vanzo and Habicht 1986; Da Vanzo 1988), Kintner (1988) found little evidence between mortality changes and the level of social and economic conditions during the study period. Similarly, some studies have also revealed that some states such as Sri-Lanka were “superior health achievers” and thus recorded a reduction in infant mortality rate independent of their low level of economic development (Caldwell, 1986; Da Vanzo 1988; Da Vanzo and Habicht 1986).

Bicego *et al.*, (1991) computed trends, age patterns and differentials in under-five mortality in Haiti during the 1960-1981 using both direct and indirect estimation methods. Their study computed the probabilities of dying before 1 month, 12 months, 24 months and 60 months by selected factors such as place of residence. Generally, the study found that as compared to other Caribbean countries, mortality was much higher in Haiti. In particular, the study found that although it was not possible to state precisely when the timing in childhood mortality decline started due to lack of maternity history data for 1977-1982, the decline seemed to have been more among girls than boys. The study concluded that owing to the fact that there was limited access to health services, a weak economy, decaying infrastructure, and social and political underdevelopment, the

prospects for a rapid acceleration of child mortality decline in Haiti would remain bleak. This supposition was however more speculative and not directly a result of their analysis.

The study by Hale *et al.*, (2009) similarly applied regression decomposition techniques to explain the factors associated with the decline in infant and child mortality in Matlab, Bangladesh. Using a sample size of nearly 94,000 singleton births from the Matlab Demographic surveillance system, they found that changes in reproductive patterns and socio-economic characteristics explained between 10 and 40 percent of the decline in mortality rates. Like the study of Da Vanzo and Habicht (1986), improvement in maternal education explained the largest portion of the reduction in infant and child mortality of all the explanatory variables considered. This was expected since mother's education has widely been found to have an inverse association with infant and child mortality (Caldwell 1979; Hobcraft, *et al.*, 1984; K'Oyugi, 1992).

However, one shortcoming with the study by Hale *et al.*, (2009) is that although insightful, the authors did not measure the effect of key factors such as changes in environmental conditions, breastfeeding and improved quality of and access to health care on the observed decline in infant and child mortality (Hale *et al.*, 2009:18). The omission of such important factors is bound to produce stochastic errors and bias the results.

In Kenya, the factors that are likely to have accounted for the upsurge in mortality during the 1988-2003 are likely to be very different from some of the factors in earlier studies

discussed above. For instance, my preliminary analysis did not show any evidence of variation in religion or fertility changes in Kenya during the 1988-2003 period. In fact, the upsurge in mortality was witnessed in an era when the country experienced a stall in fertility (Bongaarts, 2005) and hence marital fertility is unlikely to have been an important factor in the mortality upsurge.

1.2.2 The sub Saharan African region

A number of studies on mortality changes and its determinants has been done in sub Saharan African countries. Gakusi and Garenne (2007) in their study of mortality changes in Rwanda used a modelling approach that was premised on the fact that the effect of factors determining any given mortality change operate simultaneous or with a time lag. Their thesis was that mortality response to a deterioration in say income or other health determinant is not an immediate upsurge since health status is a 'stock' that normally provides a margin of health reserves above a critical threshold (Gakusi and Garenne 2007:413). Based on this premise, the authors reconstructed mortality trends by first constructing the life table using birth history data and then on the basis of the computed period estimates, determined slopes of mortality trends by fitting log linear trends.

The study found that while Rwanda experienced an annual mortality decline of about 2.8 percent during the 1950-65 period, the country experienced an upsurge in mortality of about 1.1 percent in mortality during the 1965-1977 period ($p < 0.001$) and a mean annual decline of 5.3 per cent over the 1977-90 period ($p > 0.001$). The study further found that in

the 1990s, there was no obvious trend in mortality that could be identified for the case of Rwanda. Arguing that the under-five mortality decline and reversals could be explained historically, the study cited the sudden departure of expatriates after independence, the relative international isolation, the lack of international aid, the flight of educated persons of Tutsi origin and the departure of herders as a result of ethnic conflicts between the Hutu and Tutsi as possible explanations for the high mortality during the 1965-77 period.

In another study based on 36 developing countries, Rutstein (2000) analysed 89 sets of The favourable social indicators such as increased international aid and internal political stability during the 1977-1990 were cited to have contributed to the rapid decline in under-five mortality during this period (Gakusi and Garenne 2007:426). The genocide and its aftermath was cited as the main reason behind the peak in mortality between the 1994-1998 period. For instance, the study cited that during genocide, not only were civilians (including doctors and nurses) killed and displaced but also massacre of the general population and destruction of health infrastructure led to low utilization of health facilities and increased malnutrition especially among children leading to an upsurge in under-five mortality.

However, the study by Gakusi and Garenne (2007) failed to demonstrate the extent to which each of the possible explanatory factors contributed to the under-five mortality changes during the different sets of time of study. In addition, like the study by Rosero-Bixby (1986), this study also failed to recognize the fact that mortality changes are not only a function of changing values in the covariates but rather, the structure of the relationship between the potential covariates and mortality components also changes.

Preston (1976), demonstrated that when demographic events such as infant mortality change, it is not only the factors that determine such events that could be changing over time and space but also the events could be changing at the same time. This phenomenon makes traditional statistical techniques (which assume that the dependant variable is constant) such as the modelling approach by Gakusi and Garenne (2007) inappropriate.

In another study based on 56 developing countries, Rutstein (2000) analysed 89 sets of Demographic and Health survey (DHS) data to explain factors associated with the changing trends in infant and child mortality in the 1990s. Discounting countries whose change in infant mortality was below 10 deaths per 1000 live births as having experienced sampling errors rather than a real change in infant mortality, Rutstein (2000) found that Brazil recorded the highest decrease in infant mortality (34 deaths per 1000 live births) while the highest upsurge in infant mortality was witnessed in Mali (17 deaths per 1000 live births). The study examined the relation between levels of selected covariates and infant and child mortality, followed by assessing the changes over time in the explanatory variables by relating them to the trends in infant and child mortality in each country. The author found that in general, explanatory variables explained between 45 and 70 percent of the variance in the trend in mortality.

Rustein (2000) concluded that since fertility behaviour, breastfeeding and infant feeding changed less during the period, their overall effect on mortality changes must be minimal. However, he cited factors such as maternal care, vaccinations (particularly measles), medical treatment of child illnesses, nutritional status, environmental health, education and other socio-economic factors to have explained much of the observed variation in

infant and child mortality. However, Rutstein's study which applied ordinary least squares (OLS) as the main multivariate analysis is inappropriate as discussed later under the study rationale.

Perhaps as recognition of this methodological shortcoming, Rutstein *et al.* (2009) adopted a different approach. The authors applied a simple regression decomposition technique to examine the changing health conditions and the decline in infant and child mortality rates in Benin. Their approach, entailed using survival analysis by first producing hazard ratios for explanatory variables for the 2006 survey using a backward stepwise regression procedure and then multiplying the coefficients from the multivariate analysis with the changes in the distributions of health conditions between the sample surveys. These products were then summed and the sum was exponentiated to explain the change in mortality due to the changing health conditions (Rutstein, *et al.*, 2009). Using this approach, Rutstein *et al.* (2009) were able to determine that the changing health conditions in Benin explained 56 and 60 percent of the decline in infant and under-five mortality respectively during the 2001 to 2006 period.

While the conclusions drawn by Rutstein (2000) were speculative, his suggestion of the resurgence of Malaria resistant strains and increasing HIV prevalence as factors responsible for the unexplained variance in early childhood mortality finds merit on the local scene. For instance, Kenya's malaria cases doubled in the 1990s due to higher levels of chloroquine treatment failure and also due to more frequent exposure of non-immune populations (Snow *et al.*, 2001; WHO and UNICEF 2003, Houwelling *et al.*, 2006).

In addition, the breakdown of malaria control programmes and the changes in climatic and environmental conditions (Omariba, 2005; K'Oyugi, 2000; WHO & UNICEF, 2003, WHO, 2006) contributed to the adverse effects of malaria on early childhood survival. For instance, a study conducted by K'Oyugi (1992) revealed that mortality estimates for children in the malaria endemic communities (such as Nyanza, Western, Coast and North Eastern provinces) was about six times higher as compared to the mortality rate in non-endemic communities when other factors were controlled for.

While the country experienced a breakdown in Malaria control programmes in the late 1980s, the renewed interests in the late 1990s in the control and prevention of Malaria is expected to have had a positive effect in reducing malaria morbidity and mortality in childhood. For instance, the change of the first line treatment of malaria from chloroquine to artemisinin combination therapy (ACT) for treatment of uncomplicated malaria (WHO *et al.*, 2003; IAEA, 2007, Houwelling *et al.*, 2006) ; the set up of the Division of Malaria Control (DoMC) within the Ministry of Health in 2000; the establishment of the national malaria strategic plan for 2001-2010; the increased external funding for Malaria from the Global Fund to Fight AIDS, TB and Malaria (GFTAM) and the implementation of an ITN social marketing program (MoH, 2001) could have reduced the adverse impact of Malaria on childhood mortality in the early 2000s.

On the other hand, spectrum models revealed that in Kenya, among children who had not attained age five, HIV infections increased from less than 1 percent in 1989 to about 8.1 percent in 1998 and declined to about 6.3 percent by 2003 (Stover, 2009). In absolute

numbers, Kenya registered an increase in HIV infections from about 3000 under-fives in 1989 to about 80,000 in 2003 (Stover, 2009). Generally, about 60 per cent of babies infected with HIV die before reaching their fifth birthday (Zaba *et al.*, 2003). The indirect effects of HIV/AIDS on child survival are manifested through death or frequent illness of the care-giver or breadwinner, unexplained trauma, and depletion of essential material (economic) and non material resources and even abuse from relatives which all tend to compromise child health (Adetunji, 2000; Nyambetha, *et al.*, 2003).

Equally, a study by Hill *et al.*, (2004) based on a merged dataset of 1993 and 1989 KDHS found that Kenya's upturn in infant and child mortality could not be largely explained by socioeconomic and bio-demographic or health status factors (apart from immunization coverage). Rather, much of the upsurge could be attributed to the increase in HIV prevalence (Hill *et al.*, 2004).

Other factors such as the decline in access to clean water and sanitation and changes in maternal factors may have worked to worsen infant and child mortality in Kenya over the period under study. For instance, only 30.6 per cent of the households in Kenya had access to piped water by 1999 as compared to 31.9 per cent recorded in 1989 (CBS, 2002). While this appears a marginal reduction in access to portable water, the population differences between 1989 and 1999 imply that a sizeable proportion of the population lacked access to portable water by 1999 as compared to the year 1989. Further, access to proper sanitation deteriorated from 84.1 per cent in 1992 to 81.1 per cent in 2000 in Kenya (UNICEF, 2002).

Safe water and sanitation can influence mortality through well known pathways. Firstly, safe water and sanitation positively influences infant and child mortality through a reduction in the exposure to water borne diseases (Merrick, 1985:5) particularly diarrhoea disorders. Secondly, increased water use leads to better hygiene and the elimination of bacteriological contamination (Merrick 1985:5; K'Oyugi, 1992; Ikamari, 2000). This in turn leads to reduced risks of infection through intake (Merrick, 1985:5; K'Oyugi, 1992; Ikamari, 2000).

Changes in some maternal factors may have contributed to the observed upsurge in mortality. For instance, 23 percent of Kenyan children were born less than 24 months after the preceding birth during the five years before the 1998 and 2003 KDHS (CBS, *et al.*, 2004). This rise in short birth intervals may have contributed to an upturn in infant and child mortality since short birth intervals predisposes both births to a heightened risk of death (Chidambaram *et al.*, 1985:21; Boerma and Bicego, 1992).

In addition, short birth intervals may increase the mortality risks for all the children in the family because of larger sibling sizes leading to competition for parental attention and resources. Some studies have shown that short intervals between successive pregnancies give women insufficient time to restore their nutritional reserves hence suffer from maternal depletion syndrome with adverse effect on foetal growth (Kabir and Amin 1993; Chidambaram *et al.*, 1985).

Literature also shows that educational attainment in Kenya increased in the early years of independence but started declining in the 1980s. For instance, in 1970, the primary gross enrolment rate (GER) was 62 percent and the gap between males and females was about 20 percent (Bedi, *et al.*, 2002). However, by 1980, primary GER had increased to 115% and the gap in gender enrolment had reduced to 10 per cent. Trends however took a sharp decline between 1984 and 1985 when GER fell from 107 to 99 per cent. The decline seems to have persisted through the 1990s (Bedi *et al.* 2002:33).

The reasons for the observed decline in the primary GER include the introduction of the cost sharing system as a result of the structural adjustment program by the Kenya Government in 1988 which increased the financial burden of education to parents and guardians (Bedi *et al.* 2002). The decline in GER after 1988 is likely to have adversely affected infant mortality owing to the widely known association between education (especially maternal education) and child survival (Caldwell 1979; 1986; Cleland and Ginneken 1988).

1.3 The research questions

The main research question in this study is: - What factors explain the observed upturn in infant and child mortality in Kenya during the 1988 - 2003? This question is preceded by the following specific questions:

- a) Is the observed upsurge in infant and child mortality real or is it a product of poor data quality?

- b) What factors contributed to the upsurge in infant and child mortality in Kenya during the study period (1988-2003)?
- c) What factors explain the upsurge in infant and child mortality in the low and high HIV/AIDS prevalence regions in Kenya?

This study sought to provide answers to the above questions using appropriate data as well as appropriate methodological approaches.

1.4 Study Objectives

This study aimed at investigating the factors underlying the upturn in infant and child mortality in Kenya between 1988 and 2003. The specific objectives were: -

- 1) To establish whether the observed upsurge in early childhood mortality was indeed real and if so, the extent to which early childhood mortality increased over the study period.
- 2) To establish some of the factors associated with the upturn in infant & child mortality in Kenya between 1988-1995 and 1996/2003.
- 3) To investigate the factors associated with the upturn in infant and child mortality in Kenya's high and low HIV prevalence regions during the study period.

1.5 Study Rationale

Worldwide, over 10 million children die globally and majority of these child deaths occur in sub Saharan Africa (UNICEF 2010). In Kenya, 111,000 children die annually (KNBS, 2010). A better understanding of the factors responsible for the upsurge in infant and child mortality is important in designing policies, programs and strategies to accelerate

the achievement of public health targets such as the Millennium Development Goal number IV of reducing infant mortality by two thirds by 2015. Early childhood mortality, especially infant mortality is regarded as a key indicator of social progress and economic development and rates of infant mortality reveal a society's overall ability and willingness to care for its most at risk members (UNICEF 2004).

The rise in infant and child mortality during 1988-2003 was too sharp and had never been witnessed in the post independence Kenya. Initial studies on this topic (e.g. Hill *et al*, 2001; Hill *et al.*, 2004) discounted deterioration in data quality as a probable factor behind the upsurge in mortality. Yet a clear understanding of the factors behind the upsurge is critical from a policy point of view for effective implementation of strategic public health priorities that can reverse the trends and prevent future upturns in early childhood mortality.

The rapid changes in Kenya's demography including an upturn in early childhood mortality during the 1988-2003 make this study urgent and important. After experiencing a rapid decline in mortality after independence to early 1980s, Kenya experienced a reversal in mortality in the mid 1980s through the 2003 period (Barbieri, 1994; Hill *et al.*, 2004; Ikamari, 2004; Omariba 2005). Kenya also experienced a fertility transition in the 1990s (Robinson and Cleland 1992, Brass and Jolly 1993, CBS, *et al.*, 1999) followed by what Shapiro and other scholars termed a mid transitional stall in fertility during the 1993-2003 period (Bongaarts, 2005; Shapiro and Gebreselassie 2007). With these demographic changes, it is urgent and important to understand the context under which

the country experienced an upturn in early childhood mortality and the factors explaining the upsurge.

This study was also motivated by the urgent need to fill some of the methodological gaps in the past studies on this topic. For instance, Rutstein (2000) used the ordinary least squares (OLS) as the main analytical model in studying changes in infant and child mortality in developing countries in the 1990s. However, the assumptions of the OLS are inappropriate for a dichotomous dependent variable as is the case in this study since the error terms are hardly normally distributed (K'Oyugi 1992:73). In addition, OLS are deficient in handling the problem of heteroscedasticity.² In this study, the risk of infant and child mortality vary across the data and hence the assumption of equal variance (technically referred to as homoscedasticity) among the correlates of infant and child mortality can no longer apply. This study used parametric analysis as well as regression decomposition techniques to address the above statistical problem.

In countries (such as Kenya) with a generalised HIV/AIDS epidemic, the assumption about the independence between the mother and the child is flouted (Mahy 2003; Zaba and Wald 2001). Consequently, in such countries, child mortality analysis should be limited to a period not exceeding five years prior to the survey (Mahy 2003; Zaba and Wald 2001). However, this is hardly used. For instance, Hill *et al.*, (2001) acknowledged this issue but went ahead to model trends and determinants in childhood mortality in

² Heteroscedasticity is a situation where the variance of the dependent variable varies across the data. Though not shown, preliminary exploration of KDHS data using the kernel density estimates revealed that the data were not normally distributed. Further, when other post estimation commands such as the *estat imtest* which is the Cameron & Trivedi's decomposition of LM-test for testing heteroskedasticity were applied, results revealed that the data were heteroskedastic ($\chi^2 \text{ test} = 141.38; p < 0.05$) and highly skewed ($\chi^2 \text{ test} = 130.07; p < 0.05$).

Kenya, using the data drawn from 1993 and 1998 KDHS for children born ten years preceding the surveys.

When analyses extend beyond 5 years before the survey, children belonging to mothers who have been infected recently with HIV are bound to be included in the sample more than those of mothers who were infected earlier leading to biased estimates (Omariba and Boyle 2007). To fill this gap, this study limits early childhood mortality analysis to the five year period before the 1993 and 2003 KDHS and to three years before the 1998 KDHS.

While the study by Hill *et al.*, (2004) is important in understanding the role of HIV/AIDS on the upsurge in infant and child mortality, the sentinel surveillance data upon which the study is based are not nationally representative. Studying the effect of HIV on child mortality remains a challenge in Kenya due to paucity of population based data. Even where population based data exist as is the case with the 2003 KDHS, no paediatric testing was done hence it is hard to determine the actual role of AIDS on the upturn in early childhood mortality.

This study seeks to contribute to a better understanding of the role of HIV/AIDS in the observed upturn in early childhood mortality. The study does this by adopting a regional approach in explaining probable reasons behind the upsurge in early childhood mortality in the high and low HIV prevalence regions. The argument here is that if indeed HIV/AIDS played a critical role in the upsurge in early childhood mortality, then its

effect (whether direct or indirect) on the covariates of early childhood mortality components in the high HIV prevalence zones should be different as compared to its effect in the lower HIV prevalence zones.

Consequently, if one decomposed the covariates of early childhood mortality by regional HIV prevalence status, one would expect such factors to account for the observed difference in mortality between the low and high HIV prevalence regions. Using regression decomposition procedures, Peterson *et al.*, (1986) did a similar regional analysis to explain the high infant mortality rates in the poorer states of Peninsular Malaysia.

1.6 Study scope

Generally, to fully disassociate the upsurge in infant and child mortality from the possibility of deterioration in data quality, one would require to use several data sets and methodological approaches of data collection. The data sets other than the KDHS would range from the Kenya Population and Housing Census; the Multiple Indicator Cluster Surveys (MICS), Kenya service provision data and Demographic Surveillance Data in DSS sites conducted in Kenya during the study period. It would also entail doing a qualitative study with the organizers of these surveys as well as look at the framing of questions for bias and the profile of all enumerators in each source of data. This study is, however, restricted to the use of data drawn from the 1993, 1998 and 2003 KDHS due to the limited resources available to the researcher.

Further, the study is restricted to births that occurred within five years preceding the 1993 and 2003 KDHS and 3 years preceding the 1998 survey. This is done in order to minimize recall bias but also due to the fact that some of the factors considered in this study such as place of delivery were only asked for births in the preceding five years prior to the 1993 and 2003 KDHS and three years prior to the 1998 KDHS.

1.7 Study limitations

Although this study examined changes in some of the selected factors that are likely to be associated with the recent upturn in early childhood mortality in Kenya, not all known factors that may have influenced the upturn in infant and child mortality were examined due to paucity of data. For instance, the effect of injury related factors on infant and child mortality components were not part of this study since KDHS do not collect information on these variables.

Likewise, the proportion of households using insecticide treated mosquito nets especially in Malaria endemic regions is a critical factor in influencing infant and child mortality levels and trends owing to the fact that Malaria alone accounts for about 26,000 deaths among the under-fives each year in Kenya (MoH, 2008). However, information on trends on the utilization of ITNs is scanty and was not collected in early rounds of KDHS such as during the 1992 and 1998 survey. Further, neither do we have national statistics on the household proportions that utilized ITNs before 2000.

Even where information on malaria control measures was collected such as during the KDHS 2003, information on ITN usage among deceased children was not available. Anecdotal evidence however shows that before the launch of the social marketing programme to promote the uptake of ITNs in Kenya in 2001, the utilization was low (KNBS, *et al.*, 2010). In fact, it was only in 2002 that the proportion of pregnant women sleeping under ITNs increased from 4.9 percent to 25.5 percent in 2005 (MoPHS 2009).

Even where data was available, not every known determinant of mortality was included in the analysis especially if it did not increase significantly over the years since such factors are unlikely to have contributed to the mortality upsurge (Da Vanzo 1988; Rustein 2000). For instance, while malnutrition would be expected to account for part of the upsurge in mortality, statistics show that by 1987, 32.1 percent of children were stunted and this percentage increased modestly to 32.7 in 1993 peaking at 36.8 percent in 1997 and dropping to 30.7 percent by 2003 (CBS, *et al.*, 2004). Hence on overall, the percentage of children stunted during the 1988-2003 period did not increase and hence could not have played a critical role behind the upsurge in Kenya's infant and child mortality.

Likewise, results from KDHS 1998 and 2003 revealed that at the national level, the percentage of wasted children were 6 percent and 5 percent in 1998 and 2003 respectively (CBS, *et al.*, 2004). On the other hand, percentage of underweight children in 1998 and 2003 were 22 and 20 percent respectively (CBS, *et al.*, 2004). Given these marginal differences, it is unlikely that malnutrition played a major role in the upturn in

infant and child mortality during the study period. Rutstein (2000) also discounted determinants of early childhood mortality that changed by less than 10 percent as potential candidates behind the mortality changes in countries and regions under his study and attributed such minor changes to sampling errors in survey data.

Further, literature shows that in countries with generalised HIV/AIDS epidemics such as Kenya, the assumption of independence between the survival status of the mother and her biological children is flouted (Mahy 2003, Zaba and Wald 2001). Consequently, it has been suggested that in such countries, child mortality analysis should be limited to a period not exceeding five years prior to the survey (Mahy 2003, Zaba and Wald 2001). To overcome some of the issues associated with generalized HIV/AIDS epidemic; this study limits its analysis of infant and child mortality to at most, five years preceding KDHS. The use of such truncated samples however may result in reduced sample sizes calling for the need to limit inclusion of too many covariates that may compromise the statistical power of the models.

This study does not also attempt to explain the factors responsible for the upsurge in all the standard definitions of early and late neonatal mortality, post neonatal mortality and child mortality (Hale *et al.*, 2009; Da Vanzo *et al.*, 2008). This is because such an attempt would have required a very large sample size (which we did not have) to maintain the statistical power of our analysis.

1.8 Theoretical Considerations

Several theoretical explanations have been put forward to explain the rapid decline in early childhood mortality after the World War II in most developing countries. The earliest of these is the demographic transition theory which attempted to explain the historical changes in mortality, fertility and subsequent population dynamics that occur as societies move from pre-transitional societies characterised by high mortality and fertility to post-transitional societies of low mortality and fertility (Kirk 1996).

According to the demographic transition theory, mortality decline precedes fertility decline though this has not always been the case. Among the many criticisms that bedevilled this theory, is the assumption that demographic changes are unidirectional, linear and progressive (Kirk 1996). That demographic changes are never unidirectional is best demonstrated by Kenya's demographic changes since the late 1980s. First, the country was perceived to have entered a demographic transition in the 1990s (Robinson and Cleland 1992, Brass and Jolly, 1993, CBS, *et al.*, 1999) followed by what Shapiro called a mid transitional stall in fertility during the 1993-2003 period (Bongaarts, 2005; Shapiro and Gebreselassie, 2007) and lastly, a marked upturn in Mortality during the 1988-2003 period (Barbieri 1994; Hill *et al.*, 2004; Ikamari, 2004; Omariba, 2005).

The failure of the demographic transition theory in foreseeing the possibility of stalling or even reversals in mortality as witnessed in the contemporary sub-Saharan Africa remain one of the weaknesses of these generalised propositions that have been put forward to

explain demographic dynamics. The theory also falls short in explaining the factors behind the mortality transitions.

The other dominant thinking before the 1960s was that mortality decline could be primarily explained by the success in the application of public health technologies (Vallin, 1992:407). For instance, it is believed that inoculation was instrumental in eradicating infectious diseases such as small pox (Vallin, 1992). Preston *et al.*, (1976) believes that 60 percent of the decline in mortality after the Second World War could be explained by the decline in parasitic and infectious diseases alone. Omran (2005) in his formulation of the epidemiologic transition theory also recognised the importation of public health measures and internationally sponsored medical package as key to mortality decline.

The argument, thus, was that irrespective of the society's level of socio-economic development, mortality decline was possible as long as there was a possibility to transfer health technology to the people (Vallin, 1992:407). Applying this theoretical argument, Omran (2005:754) explains that developing countries such as those in Africa are undergoing through what he terms as contemporary or delayed epidemiologic transition model. The delayed epidemiological transition model has been characterised by a rapid and truly substantial decline in mortality since the end of World War II.

However, counter-arguments on the efficacy of mere promotion of public health programmes abound. The argument has been that that public health efforts need to go

beyond medical tools (such as immunization and therapy) if they are to influence mortality (Vallin, 1992; Hill and Pebley 1989; Omran, 2005). Proponents of this school of thought argue that it is the socio-economic factors that provide the initial impetus to mortality decline. Further, they argue that exogenously developed and technically sophisticated public health programmes cannot result in sustained mortality decline in the face of only limited improvements in the living standards of third world countries (Hill and Pebley, 1989).

Consequently, once the initial impetus to improved standard of living is reached, improvements in medicine, increases in highly trained and skilled physicians, and easier access to medical care with urbanization would serve to enhance the survival chances of children (Hill and Pebley, 1989).

Using the same argument Omran (2005), while explaining mortality transition in Europe and most Western countries in the 19th century, agrees that medical advancement played a peripheral role in mortality decline. For instance, he asserts that if we discount smallpox, the recession of plague and many other pandemics in Europe, mortality decline was not in any way related to the progress made in medical science. Rather, he argues that ecobiologic factors (such as the recession of plague due to the mysterious disappearance of the black rat) and socio-economic factors (such as better standards of living, better health habits and hygiene and better nutrition) played a central role in mortality reduction (Omran, 2005:741).

In the case of developing countries, Omran (2005) argues that the delayed or contemporary model which is still ongoing explains the observed mortality changes. According to this model, importation of medical care preceded improvements in the standard of living (Omran 2005:754). Thus while the benefits of medical care followed improvements in the standard of living in the Developed World, Kenya like the rest of the developing countries first benefited from the importation of medical care prior to improvements in the standard of living (Preston, 1977; Omran, 2005).

The role played by the epidemiologic transition theory in explaining the causes in the shift of diseases as well as its contribution to the understanding of the cultural, social and behavioural determinants of health is indisputable (Fetter, 1997). However, just like the demographic transition theory, this theory is also faulted as being unidirectional, linear and progressive (Kirk 1996). For instance, the theory fails to explain the fact that in the course of mortality transition, there are possibilities of emergence of new infections which could be important causes of death as has been the case with the AIDS epidemic in some sub Saharan African countries.

Likewise, the theory fails to account for the re-emergence of diseases such as Malaria resistant strains and drug resistant strains of Tuberculosis which have increasingly become important causes of death in some developing countries such as Kenya. In fact, in Kenya, the development of malaria resistant strains in the mid and late 1980s has been pointed out as a potential candidate behind the rising mortality (WHO 2003; Houweling, 2006; IAEA, 2007).

In addition, the theory also failed to account for the fact that rather than a single mortality transition, there can indeed be many transitions in place and time (Fetter, 1997). The epidemiologic transition also left a number of unanswered questions. For instance, Fetter (1997) among other things, wonders whether there is indeed a minimum package of immunological conditions necessary for the occurrence of an epidemiologic transition. Fetter questions if human diseases that could cause death are sufficiently limited in number and type to allow for a package of medical and preventive interventions to be put in place so as to lower human mortality. Further, he argues that even if the diseases were limited in number and type, different people have varied immunological resistance to the diseases (Fetter, 1997) – issues which appear to have been overlooked in the theory.

He also demonstrates that perceiving medical interventions as independent in addressing morbidity and mortality among the human populations is inadequate since there exists a number of social, cultural and economic factors that need to be overcome if human mortality is to be lowered. Consequently then, a better theory of mortality needs not only to provide for medical/ public health interventions but should also allow for social, cultural and economic factors as important ingredients in explaining mortality levels.

The above argument is supported by Caldwell (1990) who showed that mortality transition in developing countries has not been largely driven by health technology nor material wellbeing since some transitional economies such as Sri-Lanka, Costa Rica and China have achieved low mortality levels even with very low levels of per capita income

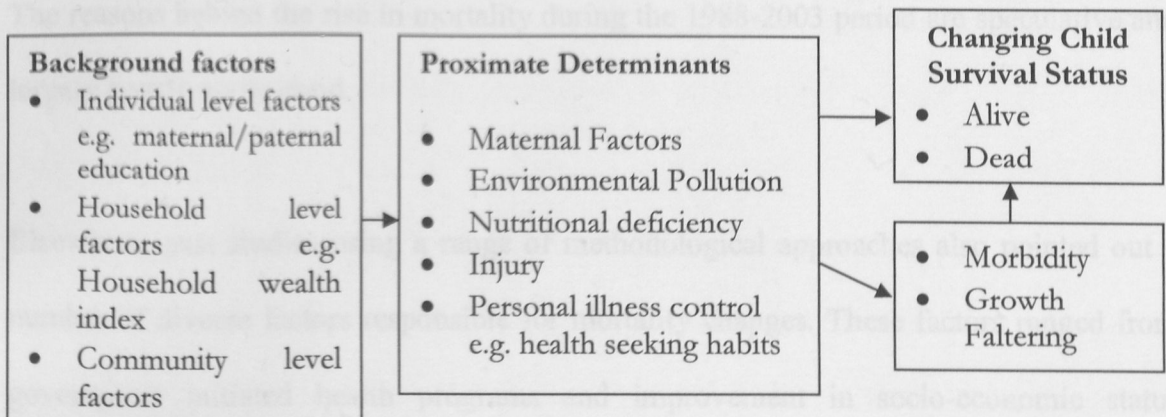
(Caldwell, 1990:46). Caldwell described such countries as “superior health achievers” as compared to their current level of economic development.

Intuitively, the Mosley and Chen (1984) framework and its variants prove useful as a theoretical guide in the study of human mortality. According to Mosley and Chen (1984) there are a finite set of proximate determinants through which background factors act through to affect mortality levels. The authors identify five groups of proximate determinants of child health: maternal factors (age, parity, birth interval); environmental pollution (such as exposure to disease); nutrient deficiency; injury and personal illness control. The authors argue that the list of proximate determinants is supposedly exhaustive, such that health will change if — and only if — one or more of the determinants change.

Further, the model holds that all the socio-economic or “background” factors (which include social, economic, cultural and health systems variables) influence a parsimonious but exhaustive set of proximate determinants which in turn directly influence child survival (Mosley and Chen 1984; Hill, 2003:2). The emerging upsurge in infant and child mortality in Kenya can therefore imply that the conditions which led to declines in the past (i.e. since the 1960s through the mid 1980s) must have changed. The conceptual framework as proposed by Mosley and Chen (1984) has widely been used in studies of child survival (Ikamari, 1996, 2000:125; K'Oyugi, 1992).

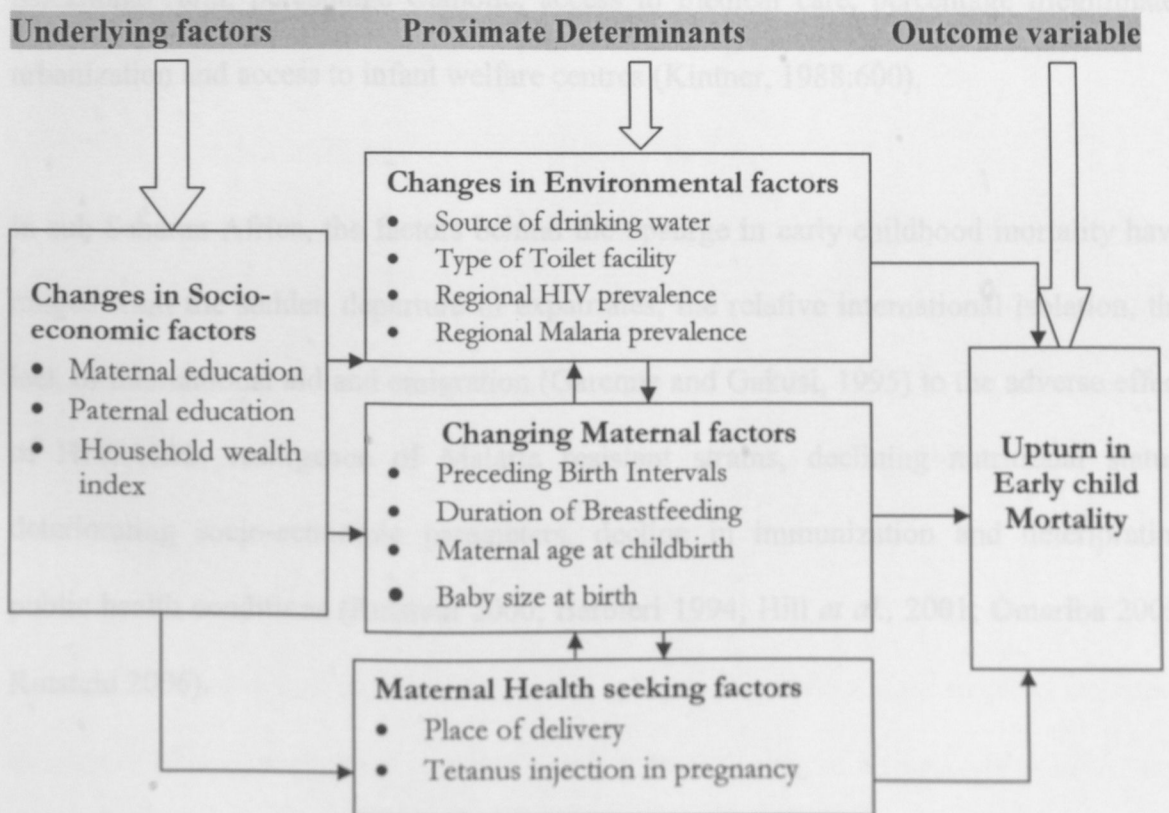
The framework shows that socio-economic and cultural factors affect childhood mortality through five proximate determinants as shown in Figure 1.2. Not all proximate factors are analysed in this study due to paucity of data and the need to maintain statistical power.

Figure 1.2: A summarized Mosley and Chen (1984) Conceptual Framework



Below is the operational framework used to guide the specification of variables for further analysis:-

Figure 1.3: The Operational Framework for studying childhood mortality in Kenya



1.9 A Synthesis of the theoretical considerations

The review shows that Kenya is among the few sub Saharan African countries that experienced reversals in early childhood mortality in the late 1980s throughout the 2003 period. Prior, the country had enjoyed a remarkable decline in mortality since the 1950s. The reasons behind the rise in mortality during the 1988-2003 period are speculative and largely poorly understood.

Elsewhere, past studies using a range of methodological approaches also pointed out a number of diverse factors responsible for mortality changes. These factors ranged from government initiated health programs and improvement in socio-economic status (Rosero-Bixby 1986; Bicego *et al.*, 1991); changing levels in maternal education (Da Vanzo and Habicht 1986; Da Vanzo 1998; Hale *et al.*, 2009), changes in marital fertility, percentage rural, percentage Catholic, access to medical care, percentage illegitimate, urbanization and access to infant welfare centres (Kintner, 1988:600).

1.9.1 Organisation of Thesis

In sub Saharan Africa, the factors behind the upsurge in early childhood mortality have ranged from the sudden departure of expatriates, the relative international isolation, the lack of international aid and emigration (Garenne and Gakusi, 1995) to the adverse effect of HIV/AIDS, resurgence of Malaria resistant strains, declining nutritional status, deteriorating socio-economic parameters, decline in immunization and deteriorating public health conditions (Rutstein 2000; Barbieri 1994; Hill *et al.*, 2001; Omariba 2005; Rutstein 2006).

The review also noted that in general, there is no single theory that can explain mortality changes. Rather, there are a number of potentially common elements within different theories. The Mosley and Chen's (1984) theory is among the few that attempted to bridge the gap between the public health and socio-economic viewpoints on mortality decline. According to Mosley and Chen (1984), the list of proximate determinants is supposedly exhaustive, such that health will change if — and only if — one or more of the determinants change.

This study used the Mosley and Chen framework to guide the specification of factors likely to have influenced the upsurge in infant and child mortality. This choice was influenced by the fact that this framework is popular, is well suited for studying covariates for changing mortality and has been widely used in the study of mortality in the past.

1.10 Organisation of Thesis

Chapter One presents an introduction to the study problem, rationale, research questions and objectives. It also presents the literature review and discusses the theoretical frameworks critical in understanding mortality transitions. Finally, it discusses the rationale on the choice of the Mosley and Chen Framework (1984) in guiding the specification of the covariates for this study.

Chapter Two presents Kenya's background information with a view to provide further insights on the circumstances under which Kenya experienced an upsurge in infant and

child mortality. Chapter Three describes the nature and sources of data as well as the data quality. The chapter applies a range of demographic data assessment and evaluation methods and also presents a set of techniques that are used to study the upsurge in infant and child mortality. In addition, the chapter discusses the variables that will be used in the study and how they were measured in this study.

Chapter Four presents bivariate results. These results show the extent to which early childhood mortality changed during the study period among the selected set of factors. The levels and trends in infant and child mortality that were computed using the synthetic cohort life table technique are presented and discussed.

Chapter Five examines the covariates underlying the upsurge in infant and child mortality in Kenya in the 1988-2003 period. The Weibull model in the accelerated failure time framework is used to assess the covariates that were associated with prolonged or reduced risk of dying as well as the overall model contribution to the upturn in infant and child mortality.

Chapter Six examines the factors associated with the upturn in infant and child mortality during the study period using regression decomposition techniques. Chapter Seven extends regression decomposition methods to study the upturn in infant and child mortality between the high and low HIV prevalence regions. The regression decomposition technique which was used by Hale *et al.*, (2009) is used.

Finally, Chapter Eight gives a summary of the research study by highlighting the objectives and methodological approaches as well a discussion of the key research findings and their implications. The chapter also speculates some of the other variables that are likely to have contributed to the upsurge in infant and child mortality that were not considered in this study. The chapter also presents the implications of the key findings for policy purposes as well as future research on this topic.

1.2 Kenya's Physical and Demographic Profile

Kenya, a country that became a republic in 1963, is one of the East African countries located between latitudes 4.21° N and 4.28° S and between longitudes 34° E and 42° E. It is located between the equator and vertically by the 38° E. The country has a total land area of 582,000 km² and is bordered by Ethiopia to the north, Sudan to the northwest, Uganda on the west, Tanzania in the south and Somalia to the east with a 400 kilometres of the Indian Ocean shoreline.

Kenya can be divided into two distinct regions based on the climatic conditions, patterns of human settlement and agricultural activities- the lowland and highland Kenya. Approximately 20 percent of the land is arid and semi-arid and only 20 percent is arable. Kenya's climate varies from a tropical climate on the coast characterized by hot and humid conditions to a temperate climate inland and to a very dry climate in the north. The coastal region receives two long rains starting in November and December. An exception to this is along the Lake Victoria

CHAPTER 2: STUDY CONTEXT

2.1 Introduction

This chapter presents the study context. It is divided into two main sections. The first section presents the background information on Kenya's demographic and socio-economic profile to shed some light on the contextual scenario under which the country experienced an upsurge in infant and child mortality. The second section provides a historical account of Kenya's socio-economic conditions that may have shaped trends in early childhood mortality since independence to the year 2003.

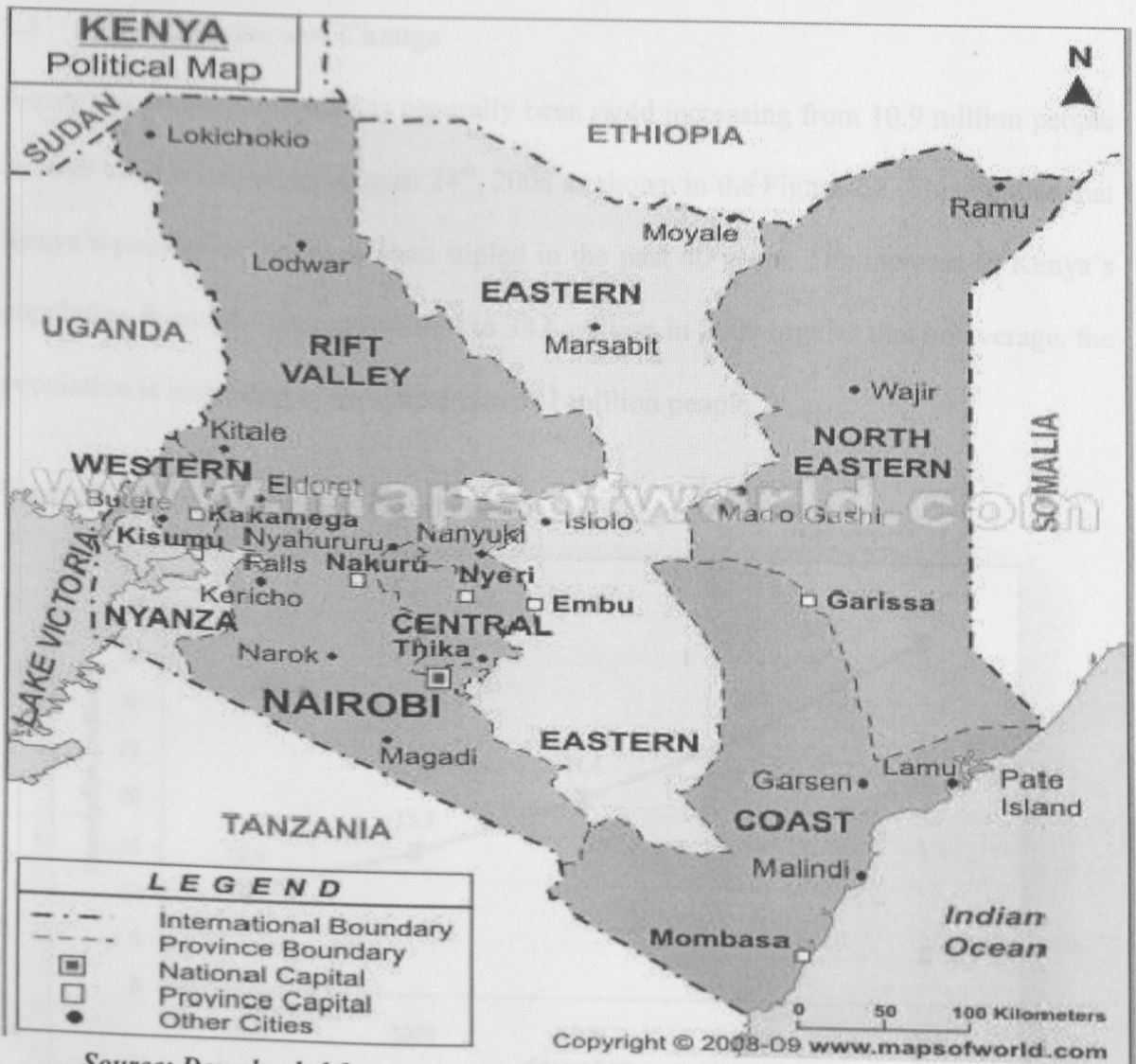
2.2 Kenya's Physical, and Demographic Profile

Kenya, a country that became a republic in 1963, is one of the East African countries located between latitudes 4.21° N and 4.28° S and between longitudes 34° E and 42° E. It is bisected into almost half horizontally by the equator and vertically by the 38° E meridian lines. The country has a total land area of $582,000 \text{ km}^2$ and is bordered by Ethiopia in the north, Sudan in the northwest, Uganda on the west, Tanzania in the south and Somalia in the east with a 400 kilometres of the Indian Ocean shoreline.

Kenya can be divided into two distinct regions based on the climatic conditions, patterns of human settlement and agricultural activities- the lowland and highland Kenya. Approximately 80 percent of the land is arid and semi-arid and only 20 percent is arable. Kenya's climate varies from a tropical climate on the coast characterized by hot and humid conditions to a temperate climate inland and to a dry climate in the north. The major rainy seasons are two- long rains starting in early-mid March to May and short rains from October to December. An exception to this is around the Lake Victoria basin

in the west where, rains are well distributed throughout the year. Below is the map of Kenya.

Figure 2.1: The Map of Kenya



Source: Downloaded from www.mapsofworld.com accessed on January 5th 2012

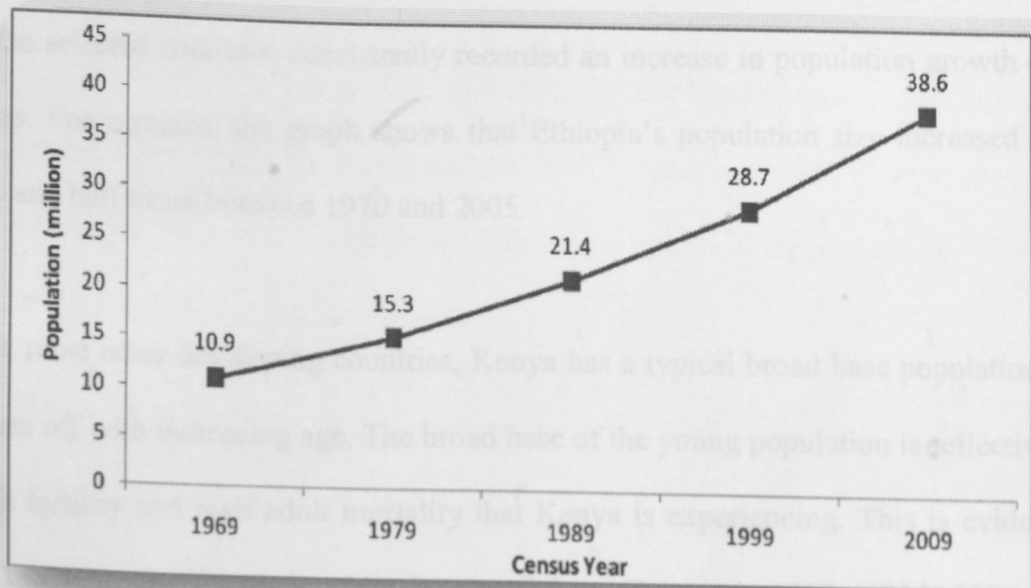
Kenya's different ecological zones determine human settlement, agricultural activities and also the distribution of certain diseases including Malaria- a leading cause of infant and child morbidity and mortality (MOH, 2006; Kimalu *et al.*, 2004). For instance, Western, Nyanza, Coast and highland parts of the Rift valley are Malaria endemic zones

while Nairobi, Central, Eastern and North Eastern provinces have lower prevalence of Malaria (K'Oyugi 1992).

2.3 Population size and Change

Kenya's population growth has generally been rapid increasing from 10.9 million people in 1969 to 38.6 million by August 24th, 2008 as shown in the Figure 2.2. This implies that Kenya's population has more than tripled in the past 40 years. The increase in Kenya's population from 28.7 million in 1999 to 38.6 million in 2009 implies that on average, the population is increasing at an annual rate of 1 million people.

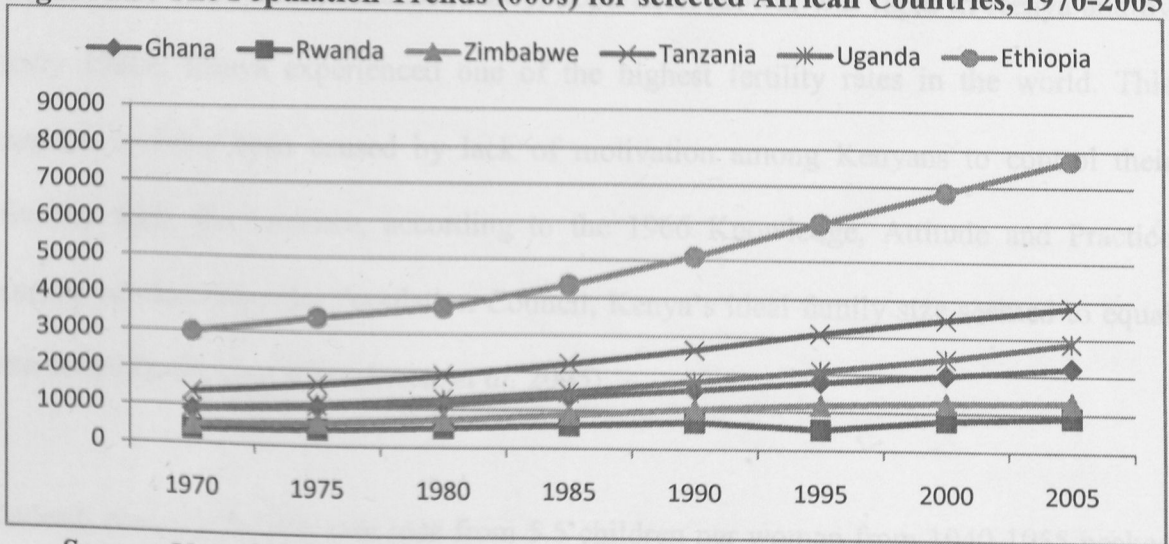
Figure 2.2: The Population Trends (000s) for Kenya, 1969-2009



Source: Kenya Housing and Population Census 2010

With a total land area of 581,677 km², the average population density by 2009 had reached 66 people per km². The change in population size is however not unique for Kenya but rather, is common among most sub Saharan African countries as well. Below is a graph showing the population growth in other selected African countries.

Figure 2.3: The Population Trends (000s) for selected African Countries, 1970-2005



Source: United Nations Department of Economic and Social Affairs/ Population Division, 2002

Figure 2.3 shows that apart from Rwanda which witnessed a slight decline in population growth in the mid 1990s due to civil war and its attendant countrywide genocide, the rest of the selected countries consistently recorded an increase in population growth over the years. For instance, the graph shows that Ethiopia's population size increased by over two and half times between 1970 and 2005.

Like most other developing countries, Kenya has a typical broad base population, which tapers off with increasing age. The broad base of the young population is reflective of the high fertility and high adult mortality that Kenya is experiencing. This is evidenced by the life expectancy at birth, which increased from 49 years in 1969 to 54 in 1979 and then to 59 years by 1989 but dropped to 57 and 47 years in 1999 and 2003 respectively (CBS, *et al.* 2004).

Further, although Kenya was the first sub Saharan African country to set up a population policy in 1967, it was only in the 1980s that the policy began to be actively implemented

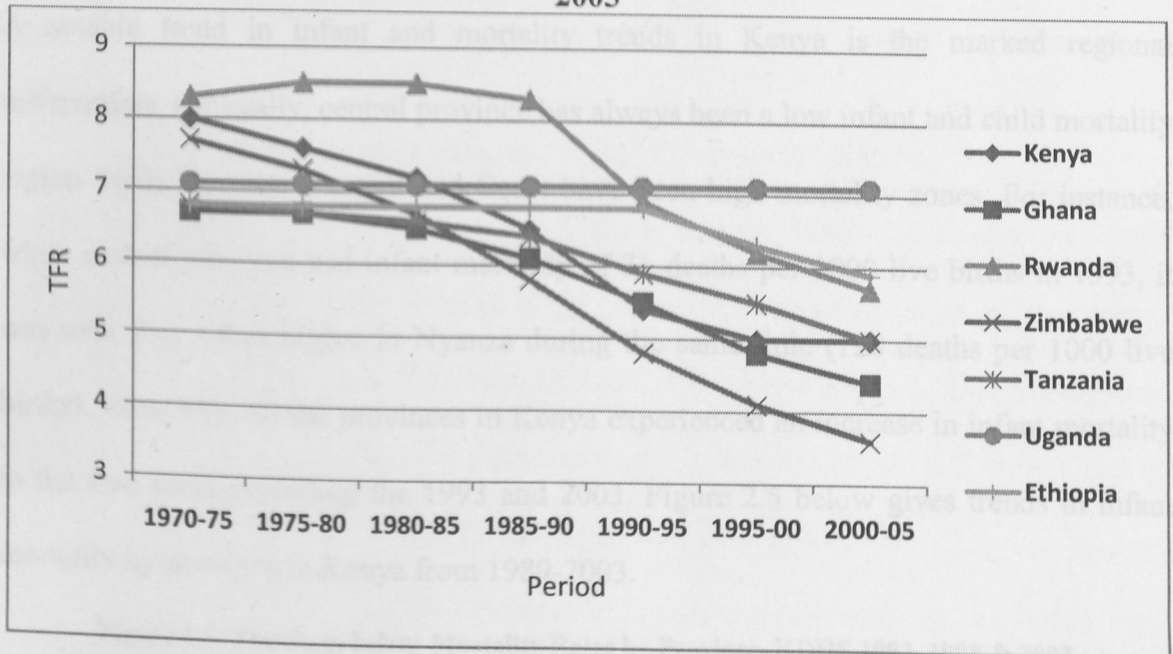
(Chimwete *et al.* 2005). Consequently, population growth was rapid in Kenya and by early 1980s; Kenya experienced one of the highest fertility rates in the world. This appears to have been caused by lack of motivation among Kenyans to control their fertility then. For instance, according to the 1966 Knowledge, Attitude and Practice survey conducted by the Population Council, Kenya's ideal family size seemed to equal the actual family size (Chimwete *et al.* 2005).

Indeed, Kenya's fertility rate rose from 5.5 children per woman from 1940-1955 peaked at 7.9 children per woman in the 1975-79 and then dropped to 4.7 in the 1993-1998 (CBS, *et al.* 2002). Below is a graph showing the trends in total fertility rate in Kenya and that of selected African countries. From Figure 2.4, the observed downward trends in fertility rates in most countries since mid 1980s epitomises the general fertility transition that is currently under way in most sub Saharan African countries. The exception to this is seen in the case of Uganda which exhibits a unique stall in fertility of about 7.1 children per woman throughout the period.

Kenya since the 1970s as shown in the figure above is in line with the general trend. Some scholars who concluded that Kenya had entered the demographic transition in the 1970s (Robinson and Cleland 1992, CBS and JICA 1994, CBS *et al.* 1999). Indeed, a reduction in total fertility rate from 7.9 in 1975 to 4.7 in 1998 and a reduction in inter-censal population growth rate from 2.8 to 2.7 percent in the same period (CBS, *et al.* 2002) was quite remarkable to warrant such conclusions. Despite the much talked demographic transition in Kenya, recent trends show a stall in fertility in the country since 1998 through 2003 (CBS, *et al.* 2004). The total fertility rate stood at 4.7 in 1998, 4.8 in 2003 and 4.6 in 2008 (KNBS, *et al.* 2009).

¹ For comparability purposes, the TFR of 4.8 excludes the Northern Province and other parts of Kenya which were not covered in earlier DHS rounds before 2003. If the omitted areas are included, the national TFR is 5.00 (CBS).

Figure 2.4: Trends in TFR in Kenya and other selected African Countries, 1970-2005



Source: United Nations Department of Economic and Social Affairs/ Population Division, 2002

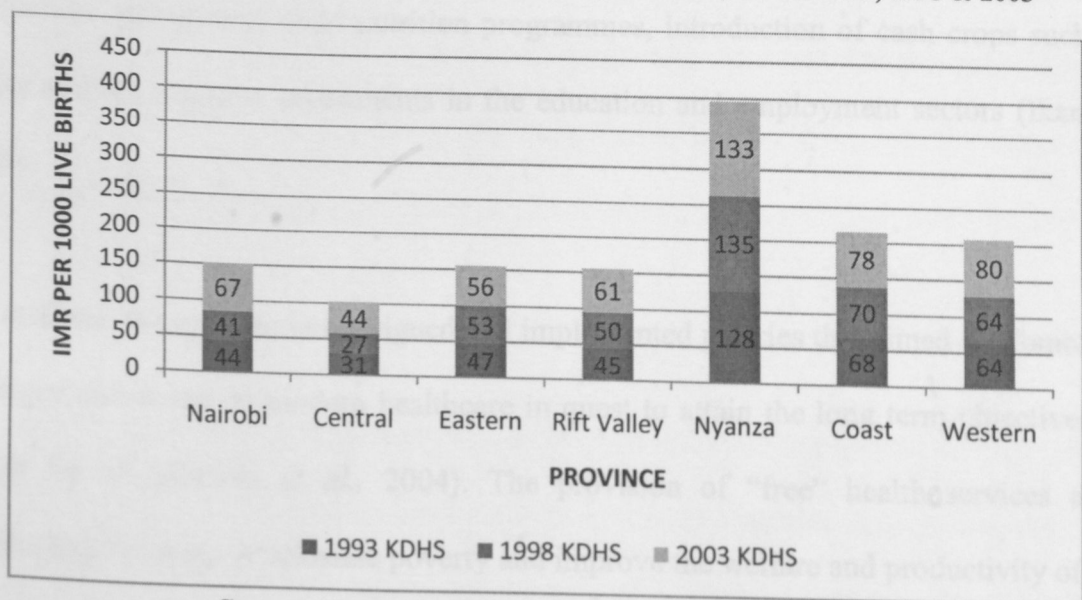
The consistent fertility decline in Kenya since the 1970s as shown in the figure above is consistent with the views of some scholars who concluded that Kenya had entered the once long awaited demographic transition in the 1990s (Robinson and Cleland 1992, Brass and Jolly 1993, CBS, *et al.* 1999). Indeed, a reduction in total fertility rate from 7.9 in 1979 to 4.7 in 1998 and a reduction in inter-censal population growth rate from 3.8 to 2.7 percent in the same period (CBS, *et al.* 2002) was quite remarkable to warrant such conclusions. Despite the much talked demographic transition in Kenya, recent trends show a stall in fertility in the country since 1998 through 2003 (CBS, *et al.*, 2004). The total fertility rate stood at 4.7 in 1998, 4.8 in 2003³ and 4.6 in 2008 (KNBS, *et al.*, 2010).

³ For comparability purposes, the TFR of 4.8 excludes the Northern Province and other parts of Kenya, which were not sampled in earlier DHS rounds before 2003. If the omitted areas are included, the national TFR in 2003 is 4.9.

2.4 Regional trends in infant and child mortality rates

A notable trend in infant and mortality trends in Kenya is the marked regional differentials. Generally, central province has always been a low infant and child mortality region while Nyanza, Western and Coast have been high mortality zones. For instance, while central province had infant mortality of 31 deaths per 1000 live births in 1993, it was over four times higher in Nyanza during the same time (128 deaths per 1000 live births). Generally, all the provinces in Kenya experienced an increase in infant mortality in the five years preceding the 1993 and 2003. Figure 2.5 below gives trends in infant mortality by province in Kenya from 1989-2003.

Figure 2.5: Trends in Infant Mortality Rates by Province, KDHS 1993, 1998 & 2003



Source: Author's Primary Analysis of KDHS data

Though Nyanza province has the highest level of infant mortality, it experienced only a 4 percent increase in infant mortality between 1989-2003 while Nairobi and Central experienced the highest increase (52 per cent and 42 per cent respectively). Rift valley and Western provinces also recorded a substantial increase in infant mortality rates (36 percent and 25 percent respectively). Likewise, Eastern and Coast provinces recorded an

upsurge of 19 and 15 percent in infant mortality during the 1988-2003 period respectively.

2.5 Structural and mortality changes in post independent Kenya

Ikamari (2004) provides a concise treatise of the economic phases that Kenya as country underwent through since independence to the early 1980s. The first phase, often referred as the “golden age” (Ikamari, 2004) was characterised by low inflation, high employment creation, a relative stable balance of payments position, and an annual GDP growth rate of about 6.5 per cent (Ikamari 2004). In addition, the first phase (1963-1973) was marked with an improvement in curative and preventive medical care services, increased food production, initiation of child nutrition programmes, introduction of cash crops such as coffee and tea, massive investments in the education and employment sectors (Ikamari 2004).

For instance, the government designed and implemented policies that aimed at promoting coverage and access to modern healthcare in quest to attain the long term objectives of health for all (Kimalu *et al.*, 2004). The provision of “free” health services as a development strategy to alleviate poverty and improve the welfare and productivity of the country soon after independence is likely to have played a critical role in the rapid under-five mortality decline until the early 1980s (Kimalu *et al.*, 2004; Ikamari, 2004). The above initiative was coupled with disease control measures as well as rapid development and expansion of health services and facilities including training of personnel, and tertiary healthcare delivery services (Kimalu *et al.*, 2004; UN 1973; Rutstein 2000).

The second phase from 1973-1979, was marred with external factors especially the Arab-Israeli war which resulted in a sharp increase in oil prices leading to among other things, internal imbalances in Kenya (Ikamari, 2004). During the 1976-77, Kenya experienced another shock when Brazil's coffee crop was destroyed by frost leading to a rise in Kenya's coffee prices with resultant improvements in the balance of payments position, but subsequently creating substantial internal imbalances since the country did not respond creatively to the windfall gains (Ikamari, 2004).

Thirdly, in 1977 there was the collapse of the East African Community due to ideological differences among the East African countries and fourthly, oil prices rose in 1979 leading to the slowing down of the GDP to 5.2 percent per annum. The fact that this era appears to have been accompanied by continued rapid decline in infant and child mortality underscores the fact that mortality responds to changes in socio-economic and health interventions after some lag (Garenne and Gakusi, 2005).

In the third phase (1980-1985), there was a slow growth in GDP of about 2.5 per cent per annum due to a wide range of factors including continued high cost of oil prices, the global recession of the 1980-1982, and poor internal economic management (Ikamari, 2004). Another important historical reason to consider is the political instability during the 1982 attempted coup d'état which might have served to scare investments in the country including cessation of some donor funded health programmes such as the UNICEF's expanded programme on immunization (Barbieri, 1994:30). These factors are likely to have been responsible for the decelerating mortality decline during this period.

The last phase that saw a consistent mortality decline at national level since Kenya's independence covered the 1986-1989 periods. This era was characterised by sluggish and generally fluctuating growth in the GDP reminiscent of the early 1980s. This phase was characterised with the infamous implementation of the structural adjustment programmes (SAPs) that were fronted by the International Monetary Fund and the World Bank.

The SAPs adversely affected health and education programs.

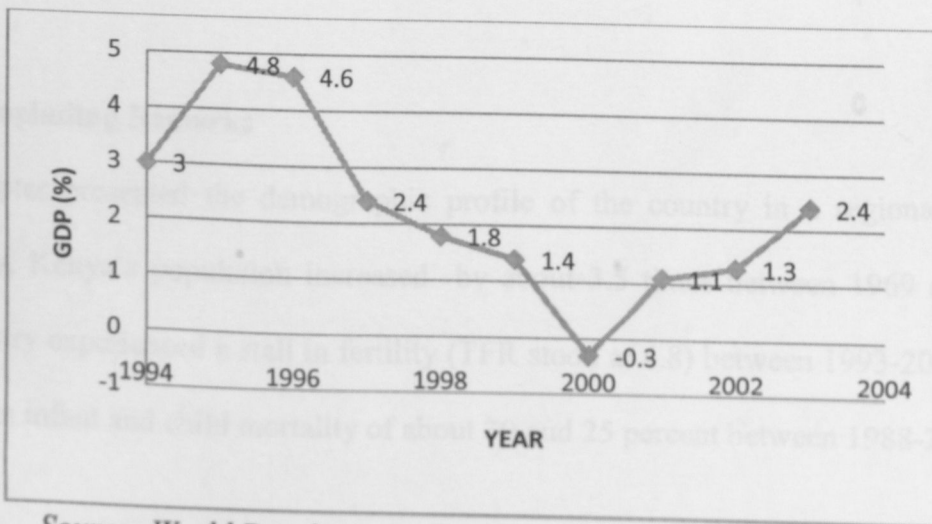
For instance, the decline in primary gross enrolment rate (GER) in 1984/5 from 107 to 99 per cent in the 1990s (Bedi *et al.* 2002:33) is largely attributed to the high cost sharing system by the Kenya Government in 1988 which increased the financial burden of education to parents and guardians (Bedi *et al.*, 2002). In particular, SAPs fronted for reductions in government spending on health and education, led to increases in food prices due to elimination of price supports, increased unemployment through retrenchment of civil servants and changes in exchange rates all of which resulted in sharp reductions in family income and less family expenditures on health (UNICEF, 1989).

One of the consequences was the increase in the cost of education leading to a decline in the quality of education and high school drop outs especially among girls and the poor (Mwega and Kukubo, 1993; Bwire, 1994). The decline in education worsened infant and child mortality rates since education especially among women encourages lifestyles supportive of good health and prenatal care (Hobcraft *et al.*, 1984, K'Oyugi, 1992; Ikamari, 1996).

Likewise, the introduction of cost sharing in healthcare as a result of SAPs coupled with mismanagement (such as lack of drugs and medical supplies and equipment in government health facilities) may have reduced accessibility to health services among the poor including facility based deliveries. Consequently, this may have slowed down or reversed the once declining mortality trend especially maternal and infant mortality (Bwire, 1994:41, Ikamari, 2004).

The decade of the 1980s and 1990s saw the country in experiencing weak and even negative economic growth. The country experienced a gross domestic product (GDP) of below 5 per cent since 1988 with some years such as 2000 recording negative growth (World Bank 2007). Part of this deterioration was due to the high population growth which outpaced GDP growth leading to a decline in per capita GDP and a decline in the standard of living. Figure 2.5 below shows the trends in Kenya's GDP in the mid 1990s to early 2000s.

Figure 2.6: Kenya's GDP (per cent real GDP) 1994-2003



Source: World Development Indicators, World Bank Group 2007

The sharp drop in GDP during the 1999-2000 has been explained by a severe drought which compounded Kenya's problems, causing the rationing of electricity and a reduction in agricultural output (CBS, 2003). During the same period, growth in manufacturing slumped as a result of competition from cheap imports, dilapidated infrastructure and lowered aggregate demand (CBS, 2003). Although 2001 saw the return of strong rains, weak commodity prices limited Kenya's economic growth. In general, the adverse economic downturn in the 1980s had an adverse impact on the health of the Kenyan population particularly on infant and child survival (CBS, MoF, 2002).

Though agriculture has been the backbone of Kenya's economy, the fall in agricultural production, particularly food crops since 1990, has forced the government to import major staple food crops virtually every year (CBS, 2003). Although the direct effects of food shortages and nutritional deficiencies are difficult to quantify, malnutrition is undoubtedly cited as a source of substantial demand for curative services and a major contributor of mortality and morbidity especially among children (CBS 2003).

2.6 Concluding Remarks

This chapter presented the demographic profile of the country in a regional context. Generally, Kenya's population increased by about 3.5 times between 1969 and 2009. The country experienced a stall in fertility (TFR stood at 4.8) between 1993-2003 and an upsurge in infant and child mortality of about 30 and 25 percent between 1988-2003.

Kenya's upturn in infant and child mortality coincided with an era of sluggish and fluctuating growth in GDP and the onset of structural adjustment programmes (SAPs) in the late 1980s. The conditions of SAPs such as cost sharing in education and health care, as well as retrenchment which led to a fall in household income may have increased infant and child mortality rates in the late 1980s throughout the early decade of 2000.

The erratic weather conditions may have also affected food supply in early decade of 2000. The increase in HIV prevalence, the persistence of Malaria resistant strains and the changing maternal factors are additional factors that may have influenced the upsurge in infant and child mortality. Some of these factors are investigated later in this study.

The data used in this study are drawn from the 1993, 1998 and 2003 Kenya Demographic and Health Surveys (KDHS). These sets of data were the most ideal because the upsurge in infant and child mortality was first observed in Kenya in the late 1980s and the data sets contain necessary history data that are necessary for estimating infant and child mortality. Secondly, where there is no complete and reliable vital registration system such as in Kenya, DHS data remain the most extensive source of relatively good quality data for population and health indicators at the national level (UN, 2004).

The KDHS are conducted by the Kenya Bureau of Statistics (KNBS), National Council for Science and Technology (NCST), and Macro-International Incorporation of Kenya among others. The samples for all the three rounds of KDHS were drawn using a two-stage national sample survey and evaluation programme (NASSEP) using a stratified multi-stage cluster sample design. Details of the sampling approaches are available in respective KDHS country reports.

CHAPTER 3: DATA AND METHODOLOGY

3.1 Introduction

This chapter has four sections. Section One describes the sources of data. Section Two explores the nature and quality of KDHS data in relation to infant and child mortality analysis. Section Three provides some important *caveats* on the basis of data quality assessment prior to data analysis. Section Four highlights the methods of data analysis used in this study.

3.2 The nature of sources of Data

The data used in this study are drawn from the 1993, 1998 and 2003 Kenya Demographic and Health Surveys (KDHS). These sets of data were the most ideal because the upsurge in infant and child mortality was first observed in Kenya in the late 1980s and the data sets contain maternity history data that are necessary for estimating infant and child mortality. Secondly, where there is no complete and reliable vital registration system such as in Kenya, DHS data remain the most extensive source of relatively good quality data for computing key population and health indicators at the national level (UN, 2004).

The KDHS was carried out by the Kenya Bureau of Statistics (KNBS), National Council for Population and Development (NCPD), and Macro-International Incorporation of Calverton, Maryland, U.S.A among others. The samples for all the three rounds of KDHS were drawn from the third national sample survey and evaluation programme (NASSEP III) using a stratified multi-stage cluster sample design. Details of the sampling approaches are available in respective KDHS country reports.

During the 1993 KDHS, a sample of 7540 women aged 15-49 years yielding a response rate of 94.3 per cent were interviewed while in the 1998 KDHS, 7,881 women of reproductive age (15-49 years) and 2,336 men aged 20-54 years were interviewed. The KDHS 2003 sampled 8195 eligible women (15-49 years) and 3578 eligible males yielding a response rate of 94 and 86 per cent respectively.

The merged KDHS datasets used in this study had a total of 14,137 live births which was about 97 percent of all live births born 5 years before the 1993 and 2003 KDHS and 3 years before the 1998 KDHS. The data used in this study excluded births that occurred during the month of interview as well as multiple births. The exclusion of births that occurred during the month of interview was informed by the fact that the survival status of such births was unknown. The exclusion of multiple births was necessitated by the fact that such births tend to experience a much higher rate of early childhood mortality as compared to that of singletons (Khasakhala 2000; Uthman, *et al.*, 2008; Kembo and Ginneken, 2009) and hence their inclusion could have biased our mortality estimates upwards.

In all the surveys, the information collected that is central to this study include the household data (which includes information on type of housing, household socioeconomic status, and environmental conditions of the household) as well as the full retrospective birth history of women aged 15-49 years. To obtain data from birth history, all women in the reproductive age groups (15-49 years) were asked to ascertain the sex

and date of birth of every child that was born; whether the child was still alive and if not, the age and/or date of death.

The 2003 KDHS was unique since in addition to collection of standard information on fertility, mortality, contraceptive use and fertility preferences, it collected dry blood samples (DBS) for HIV testing of adults. Out of the targeted 8486 respondents for HIV testing, 73 percent were successfully tested with 76 percent of adult women (15-49 years) tested against 70 percent of eligible males in the same age cohort. In all the three rounds, research ethical guidelines were followed.

3.3 Probable errors in childhood mortality data and their causes in Kenya

The common types of reporting errors in birth history data that may affect the quality of infant and child mortality measurement are omission of dead children, incomplete information on date of birth or age at death, displacement of events in time and errors in reported age at death (Becker and Suliman, 2005). In addition, sampling errors can also affect mortality estimation especially if the sampled households are not representative. However, an assessment of the comparison of the distribution of the population at risk by selected characteristics in the past has shown that the effect of sampling errors on mortality estimation are minimal (Becker and Suliman, 2005; Sullivan, 2008).

In Kenya, the number of dead children is under-reported in some societies due to socio-cultural beliefs and taboos (Muganzi, 1984; 2000). There is also evidence that mothers whose children died in the distant past may not recall when their children died leading to omission of such deaths in birth history data (Muganzi, 1984; 2000).

Some of the errors in mortality data such as shifting and displacement of births may understate or overstate mortality levels and it has been found to be common among more recent reporting especially among women with low literacy or where reporting is done by a foster mother (Muganzi, 1984; 2000; Kiersten *et al.*, 2005; 2009). In Kenya, children in their second year of life who die before they have been weaned may be reported as infant deaths (Muganzi, 1984; 2000) which may overstate the true rate of infant mortality. In DHS, there tends to be an aversion at age 4 by some research assistants so as to avoid asking additional questions on the child's anthropometry.

Generally, underreporting of early childhood deaths result in underestimation of mortality while misreporting ages at death tends to distort the age pattern of infant and child mortality (Obungu, *et al.*, 1994; Muganzi 2000). It is sometimes possible to detect these errors if they are systematic (Kiersten *et al.*, 2005; 2009).

In the next section, the study is devoted to assessing the quality of KDHS data by examining logistical issues that were put in place to ensure high data quality and by applying some of the demographic techniques for the assessment of data quality. In carrying out the assessment, the study aims to demonstrate whether the upsurge in infant and child mortality could have been a consequence of deteriorating data quality arising from any of the above four potential errors or was a genuine rise from changes in one or more of the proximate/distal determinants of mortality.

3.4 Logistical arrangements that enhanced quality of data collection in KDHS

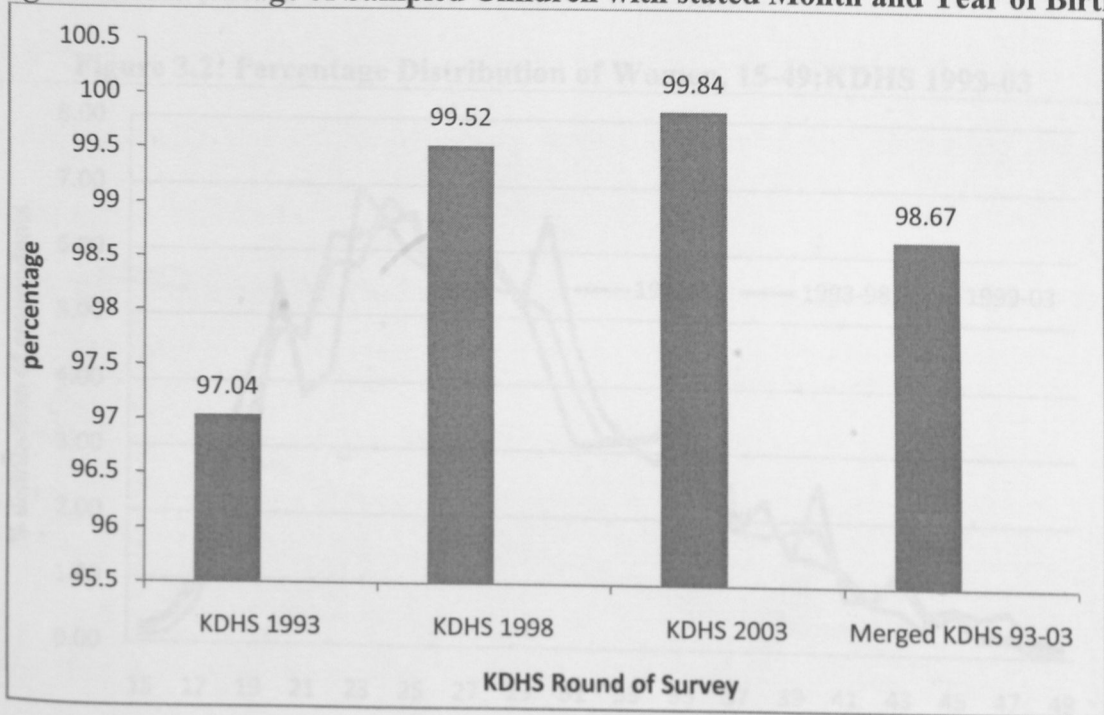
In all the three rounds of KDHS, a careful selection of supervisors, field editors and interviewers was done to ensure that data collected was of high quality. For instance, most of the supervisors were highly trained officers working with the National Coordinating Agency for Population and Development and Kenya National Bureau of Statistics (KNBS) as field officers, statistical officers or population officers. In addition, technical assistance in conducting of the surveys was provided by the MEASURE/DHS team. Secondly, the interviewers were mostly University graduates who were conversant with qualitative and quantitative methods of research with similar fieldwork experience in DHS or behavioral surveillance surveys (CBS *et al*, 2004).

The selected interviewing teams, supervisors and editors were also trained prior to the actual research in all the three rounds of KDHS considered in this study. Pretesting of questionnaires was also done to ensure flow and consistency (CBS *et al.*, 2004). On the other hand, completed questionnaires were returned periodically from the field to CBS offices in Nairobi, where they were edited and entered by data processing personnel specially trained for this task. In terms of verification, data were entered twice. The advantage with concurrent processing of data was that KNBS was able to advise field teams of errors detected and immediate measures would be put in place to ensure data quality.

3.5 Completeness of age reporting

A general analysis of KDHS data show that in general, the completeness of age reporting among the sampled children improved over the period. Figure 3.1 below shows that the percentage of children whose month and year of birth were reported increased from 97 percent during the five years preceding the 1993 KDHS to 99.8 percent during the five years prior to the 2003 KDHS. Suffice it to say, the percentage of children whose age was imputed declined over the years under study- a pointer to the fact that age reporting over the year improved.

Figure 3.1: Percentage of Sampled Children with stated Month and Year of Birth



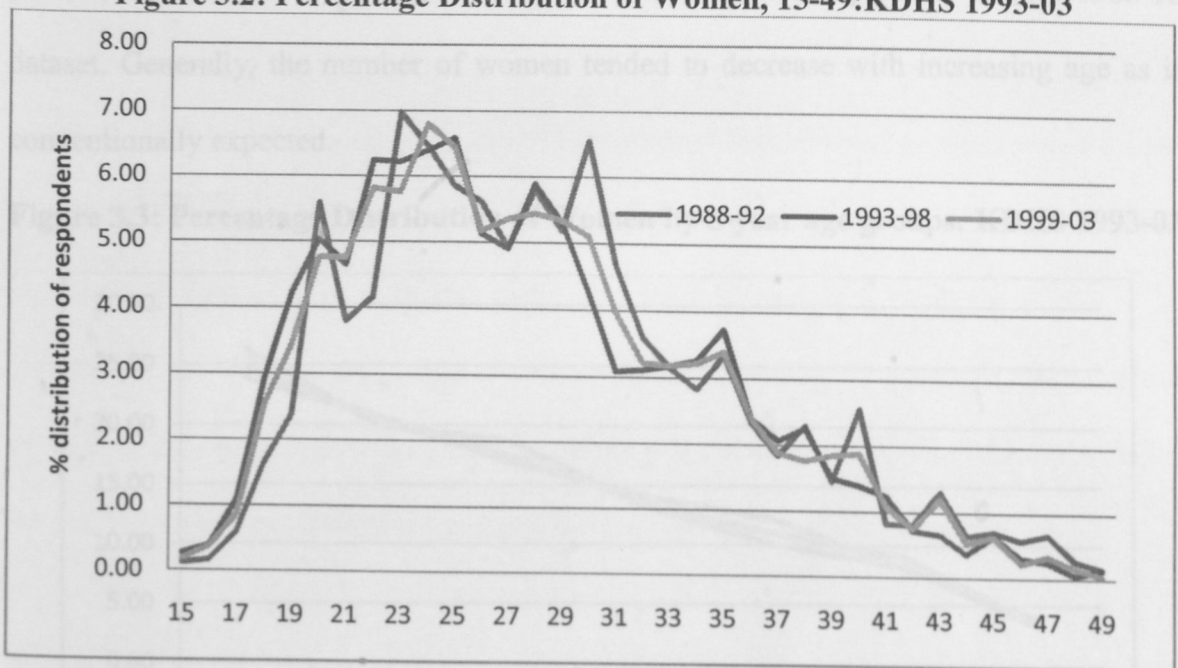
Source: Computed from KDHS, 1993, 1998 & 2003

The above finding echo earlier findings by Pullum (2006:36) who in his assessment of the 141 DHS for 66 countries did not find Kenya among the countries whose child incompleteness index exceeded 0.2 during the 1985-2003 period.

3.6 Quality of Age reporting

While age reporting improved as discussed in the preceding section, the question is to what extent was the accuracy of the reported ages? This section addresses this question. Normally, single year age distributions in censuses and surveys in developing countries tend to suffer from age heaping especially on ages ending with 0 and 5 but sometimes extent to other numbers as well. It is generally argued that if age reporting of the mother is reliable, then age reporting for children will equally be reliable (Ikamari, 1996). The graph below shows the age distribution of women 15-49 years that were sampled in all the three KDHS.

Figure 3.2: Percentage Distribution of Women, 15-49:KDHS 1993-03



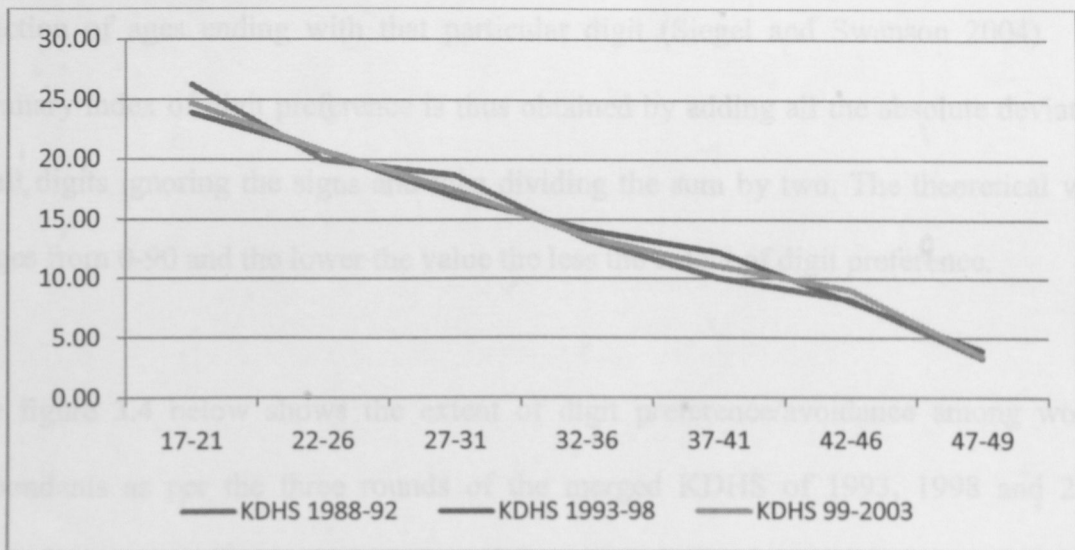
Source: primary analysis of KDHS 1993, 1998 & 2003

There appeared to be a marked age heaping on ages 20, 23, 25, 28, 30, 35, 40 and 43. On the other hand, the graphs show marked deficiencies in age reporting between ages 15 to 22, as well as ages 31, 32, 33, 34 and 44.

Some authors have proposed that to resolve the problem of age heaping, one can group women's ages in five years using unconventional groups whose terminal digits do not end with 0 or 5 (Palloni *et al.*, 2007). In doing so, digit preference that consists of rounding to terminal ages of 0 and 5 become tractable such that beyond the degenerate cases, such groupings will most certainly contain most of both the reported and the true age of individuals who rounded up their ages (Palloni *et al.*, 2007).

The adoption of this approach resulted in a marked reduction in the distortions in age reporting as shown in figure 3.3. As shown, women's reported ages in later surveys (1993-1998 and 1999-2003) appear to be the less noisy as compared to that for 1988-92 dataset. Generally, the number of women tended to decrease with increasing age as is conventionally expected.

Figure 3.3: Percentage Distribution of Women by 5-year age groups: KDHS 1993-03



Source: Primary Analysis of KDHS 1993-2003 Data

While the above preliminary analysis forms the crux of data quality assessment in demography, it does not allow us to explain the extent and nature of age heaping/digit preference or avoidance. As a solution, this study computed Myer's blended indices for

the three rounds of KDHS. The Myer's index aims at ascertaining the degree and pattern of digit preference (or avoidance) in age reporting (Siegel and Swanson 2004).

3.7 Computation of Myer's index

The computation of Myer's index is one of the most used approaches for checking data quality on age reporting. It entails constructing a blended population as a weighted sum of the numbers of people that report their ages ending in each of the ten terminal digits over a given age range. Generally, in the absence of any systematic distortions in age reporting, the sum at each terminal digit approximates 10 percent of the total blended population.

A sum in excess of 10 percent for any of the terminal digits indicates preference for ages ending in that digit and a negative deviation for a given digit means avoidance or under-selection of ages ending with that particular digit (Siegel and Swanson 2004). The summary index of digit preference is thus obtained by adding all the absolute deviations of all digits ignoring the signs and then dividing the sum by two. The theoretical value ranges from 0-90 and the lower the value the less the extent of digit preference.

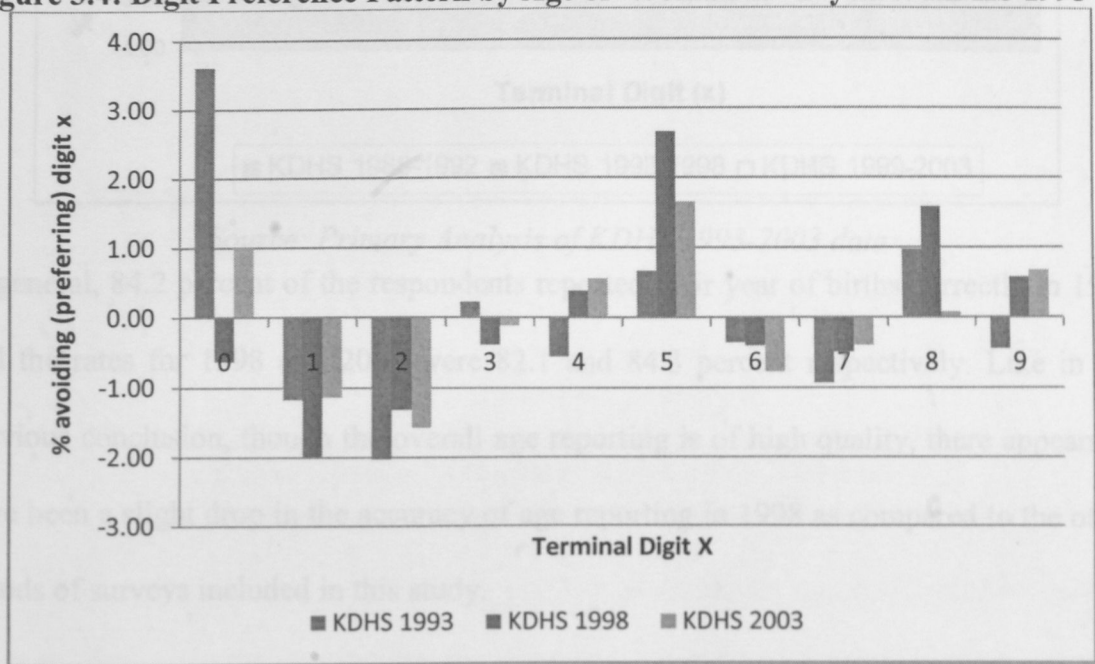
The figure 3.4 below shows the extent of digit preference/avoidance among women respondents as per the three rounds of the merged KDHS of 1993, 1998 and 2003. Generally, ages ending in 0, 4, 5, 8, and 9 seem to be preferred. On the contrary, ages with terminal digits of 1, 2, 3, 6, and 7 appear to be highly avoided in all the three rounds of KDHS. As shown in appendix IV, the overall index shows that 94.5 percent of the

women aged 20-49 years reported their ages within the correct terminal digits. The indices for the 1993-1998 and 1999-2003 merged KDHS were 94.8 and 96 percent respectively.

Figure 3.4: Digit Preference/Avoidance Pattern: KDHS 1993-2003

Figure 3.4 portrays that the extent of age heaping is low. There appears to have been a slight improvement in the quality of age reporting in the 1988-2003 period. The improvement in quality of age reporting could be as a result of close supervision and proper editing of filled questionnaires in the latter surveys and improvement in the intensity of training and quality of interviewers in latter rounds of surveys.

Figure 3.4: Digit Preference Pattern by Age of Woman 15-49 years: KDHS 1993-03

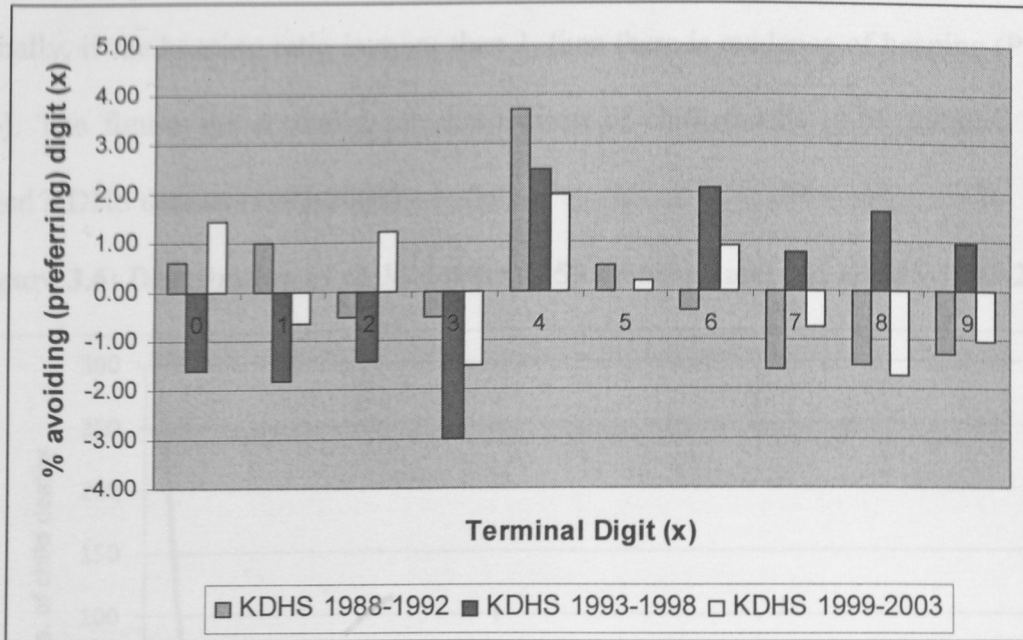


Source: Primary Analysis of KDHS 1993-2003 data

It is conventionally expected that respondents would accurately remember their year of birth as compared to their current age in years (Ngalinda, 1998). A close relationship exists between correct reporting of maternal age and that of the child (Ikamari, 1996). Consequently, a Myer's Blended index based on the respondent's year of birth was computed. Generally, births ending in terminal digit 4 were the most preferred in all the

three surveys. Other preferred terminal digits were 0, 5 and 6. On the contrary, ages ending in 1, 2, 3, 7, 8 and 9 tended to be avoided most as compared to the rest as shown in figure 3.5 below.

Figure 3.5: Digit Preference/Avoidance Pattern: KDHS 1993-2003



Source: Primary Analysis of KDHS 1993-2003 data

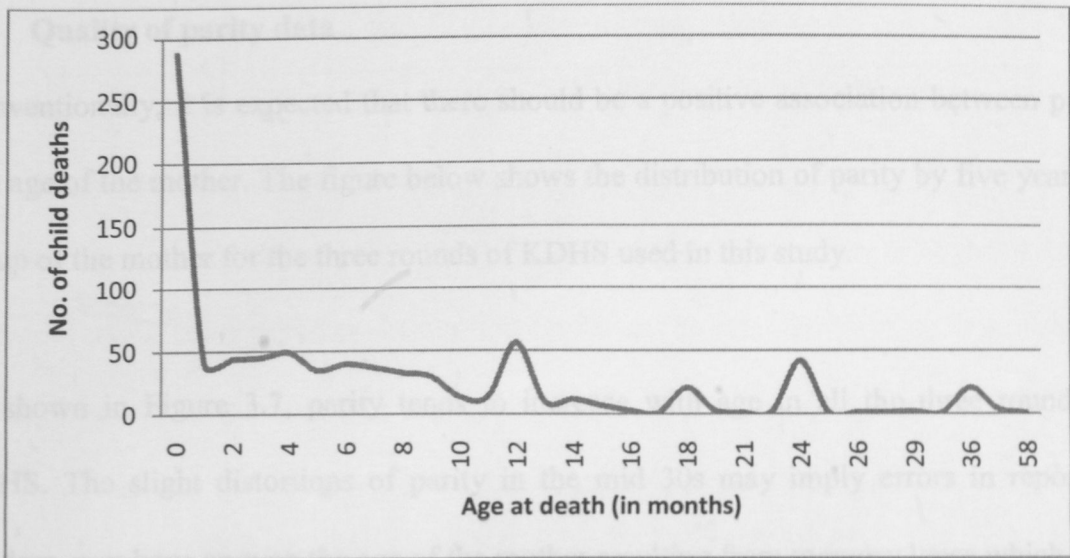
In general, 84.2 percent of the respondents reported their year of births correctly in 1993 and the rates for 1998 and 2003 were 82.1 and 84.3 percent respectively. Like in the previous conclusion, though the overall age reporting is of high quality, there appears to have been a slight drop in the accuracy of age reporting in 1998 as compared to the other rounds of surveys included in this study.

3.8 The extent of age heaping in reported age of child death

In DHS, children who die before the second birthday have their age at death recorded in months. Consequently, data often show some heaping at month 12 (and, to a lesser degree, at months 6 and 18). If the cause of the excess in mortality at month 12 months is partly due to misreporting of deaths that actually occurred at 10 or 11 months, this can

lead to an underestimate of the probability of dying in the first year of life (months 0-11) and lead to an overestimate of the probability of dying at ages 1-4 years (months 12-59). One way of assessing the extent of heaping at 12 months is to divide the reported number of deaths in month 12 by the average of the numbers in months 10, 11, 13, and 14. Normally, if the heaping ratio is more than 1, then there is evidence of heaping (Pullum, 2006). The figure below shows the distribution of child deaths (0-59 months) in the merged KDHS dataset (1993-2003).

Figure 3.6: Distribution of child deaths (0-59 months), merged KDHS 1993-2003



Source: Primary Analysis of KDHS 1993-2003 data

A computation of the heaping ratio from the graph above revealed a heaping ratio⁴ of 4.48. This is the ratio of the observed number of deaths in month 12 to the expected number, under a model of linear change in the true monthly frequencies from month 10 to month 14 (Pullum 2006:3). Since the ratio is greater than 1, it implies that there was heaping at the age of 12 months hence infant mortality estimates are bound to be understated.

⁴ Assuming that 10, 11, 12, 13 and 14 are (a), (b), (c), (d) and (e), heaping ratio at 12 months is computed as $c/((a + b + d + e)/4) = 4c/(a + b + d + e)$.

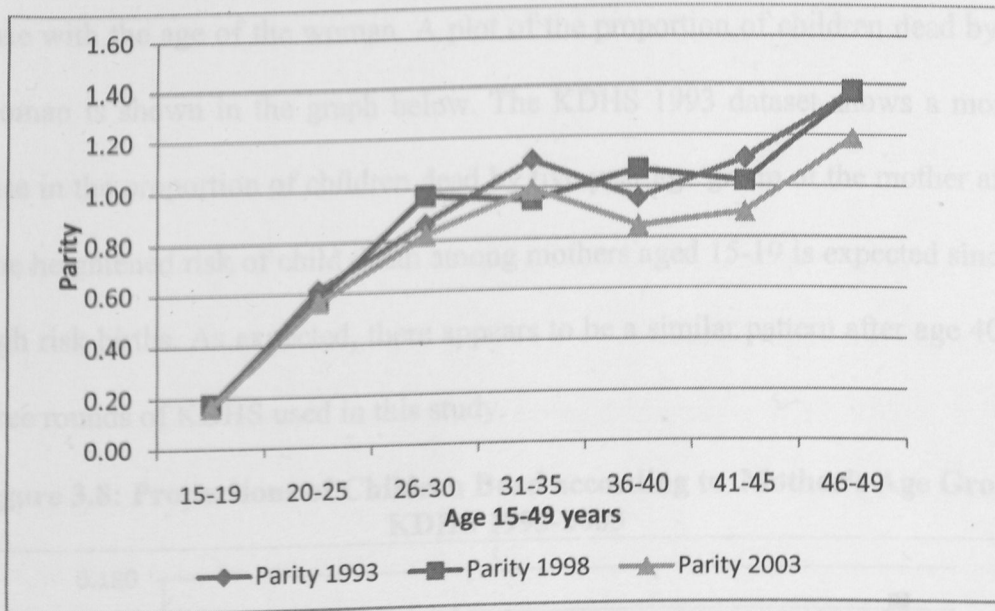
Figure 3.6 above also shows some heaping at months 18, 24 and 36. While there are no established methodological developments to handle this form of heaping in demography (Pullum 2006), it is evident that such heaping would lead to understating infant mortality and hence cannot be a consequence of its upsurge. Further, owing to the fact that birth transference of children dying at 18, 24 and 36 months fall within the estimation period of child mortality (12-59 months), its effect if any, on child mortality estimation would be minimal.

3.9 Quality of parity data

Conventionally, it is expected that there should be a positive association between parity and age of the mother. The figure below shows the distribution of parity by five year age group of the mother for the three rounds of KDHS used in this study.

As shown in Figure 3.7, parity tends to increase with age in all the three rounds of KDHS. The slight distortions of parity in the mid 30s may imply errors in reporting children ever born or even the age of the mother resulting from memory lapse which tend to increase with age.

¹ Though not shown, the weighted early neonatal deaths and all neonatal deaths were as follows for KDHS 1993, 1998 and 2005: 25.6 and 31.7 per 1000 live births respectively. Also for KDHS 1998 were 30.2 and 39.4 deaths per 1000 live births and for KDHS 2005, the rates were 34.3 and 45.7 deaths per 1000 live births respectively.

Figure 3.7: Parity of Women by age group

Source: Primary Analysis of 1993, 1998 and 2003 KDHS data

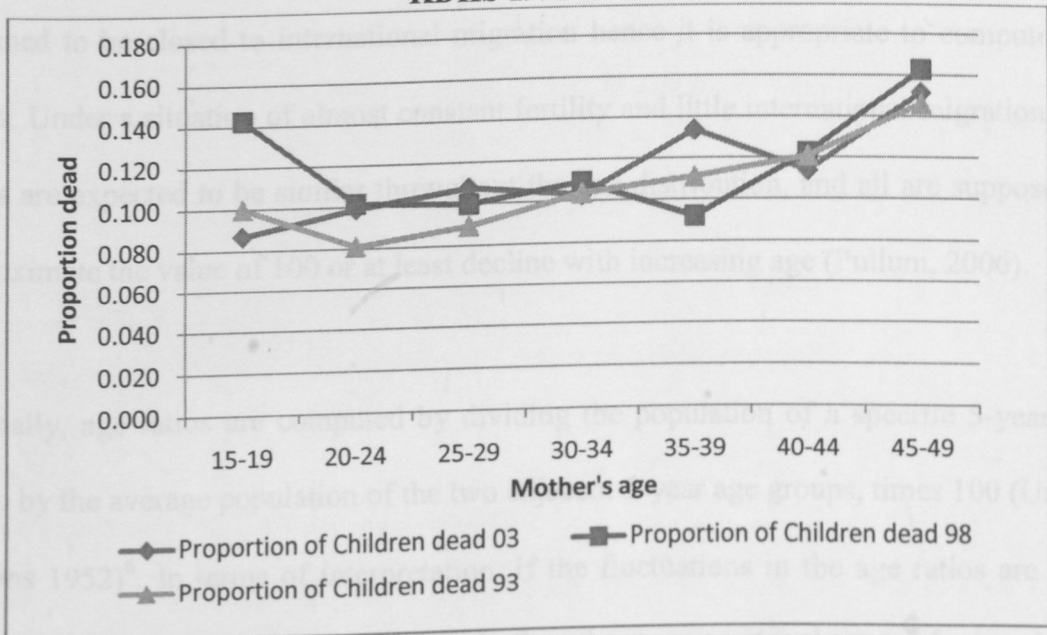
3.10 Reporting of children dead

Another key issue in data evaluation in this study related to the quality of reported deaths. Indirectly, differential reporting of omitted deaths can be detected by examining the ratio of early neonatal deaths (those in the first week of life) to all neonatal deaths (those in the first four weeks of life). It is expected that about 70 percent of neonatal deaths occur in the first six days of life (Sullivan, *et al.*, 1990; Obungu *et al.*, 1994; Becker and Suliman, 2005). In this study, the ratio of early neonatal deaths to all neonatal deaths⁵ in all the three surveys used was 72 per cent for KDHS 1993, 77 percent for KDHS 1998 and 76 percent for KDHS 2003— an indication of high data quality or lack of differential reporting of deaths.

⁵ Though not shown, the computed early neonatal deaths and all neonatal deaths were as follows: for KDHS 1993, they were 25.6 and 35.7 per 1000 live births respectively; those for KDHS 1998 were 30.2 and 39.4 deaths per 1000 live births and for KDHS 2003, the rates were 34.5 and 45.9 deaths per 1000 live births respectively.

On the other hand, it is conventionally expected that the proportion of dead children increase with the age of the woman. A plot of the proportion of children dead by age of the woman is shown in the graph below. The KDHS 1993 dataset shows a monotonic increase in the proportion of children dead by five year age group of the mother after age 20. The heightened risk of child death among mothers aged 15-19 is expected since these are high risk births. As expected, there appears to be a similar pattern after age 40 for all the three rounds of KDHS used in this study.

Figure 3.8: Proportions of Children Dead according to Mother's Age Group: KDHS 1993-2003



Source: Primary Analysis of 1993, 1998 and 2003 KDHS data

Apparently, minor distortions in the reported proportions of dead children exist in the 1998 and 2003 KDHS. These distortions could be due to recall errors or due to taboos associated with reporting dead children. If the children died shortly after birth before they were given names, some mothers are likely to have erroneously classified such births as stillbirths (Muganzi, 1984). The slight dip in the quality of data on children dead in 1998 and 2003 could be due to misreporting of child deaths during these periods as compared to the earlier survey period. The trend shown in the figure above echo the fact that

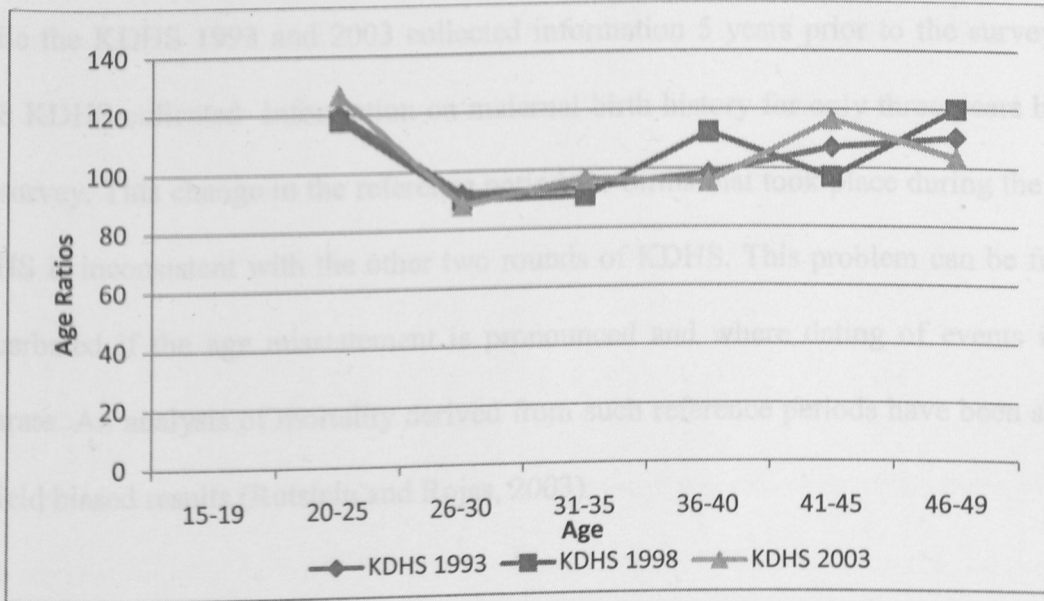
women aged 15-19 years tend to overstate 0 children ever born while those who are elderly tend to selectively forget children who died in the distant past (Ngalinda, 1998).

3.11 Age Ratios as a means of detecting age misreporting

The computation of age ratios for 5 year age groups can be used as indices for detecting possible age misreporting so long as fertility has not fluctuated much and the population has not been significantly exposed to international migration (UN, 1952). In Kenya, fertility appears to have stalled since 1993-2003 (CBS, 2004) and the population is assumed to be closed to international migration hence it is appropriate to compute age ratios. Under a situation of almost constant fertility and little international migration, age ratios are expected to be similar throughout the age distribution, and all are supposed to approximate the value of 100 or at least decline with increasing age (Pullum, 2006).

Normally, age ratios are computed by dividing the population of a specific 5-year age group by the average population of the two adjacent 5-year age groups, times 100 (United Nations 1952)⁶. In terms of interpretation, if the fluctuations in the age ratios are very large and their deviation from 100 is much, then there are greater chances of errors in the data. The figure below shows the calculated age ratios for all the three rounds of KDHS.

⁶ This can mathematically be represented as $nAR_x = \frac{2 \cdot N_{nx} \cdot (N_{nx-5} + N_{nx+5})}{N_{nx-5} + N_{nx+5}} \cdot 100$; where nAR_x is the age ratio of a given age group; N_{nx} is the actual age group and N_{nx-5} and N_{nx+5} are the preceding and succeeding age groups to N_{nx} .

Figure 3.9: Age ratios for women, 20-49 years, KDHS 1993-2003

Source: Primary analysis of KDHS 1993, 1998 & 2003

From the above figure, the calculated age ratios appear to be close and similar and further, they do not deviate much from the expected value of 100. However, beyond 35, the ratios become quite unstable- a cautionary note on inclusion of older cohorts (i.e. those above ages 35) in the computation of infant and child mortality.

3.12 Preparation of the data for analysis

A number of considerations ought to be made prior to a merged analysis of KDHS datasets. Firstly is the issue of reference period for infant and child mortality estimation. Secondly, for consistent estimation, there is need for computation of the household wealth index for KDHS 1993 which was not computed. The first issue is discussed in the next section below. The second issue which deals with the computation of the household wealth index which was done using the principal component analysis (PCA) approach is discussed under the section on operationalization of variables below.

3.13 Reference period for early childhood estimation

While the KDHS 1993 and 2003 collected information 5 years prior to the survey, the 1998 KDHS collected information on maternal birth history for only three years before the survey. This change in the reference period for births that took place during the 1998 KDHS is inconsistent with the other two rounds of KDHS. This problem can be further exacerbated if the age misstatement is pronounced and where dating of events is not accurate. An analysis of mortality derived from such reference periods have been shown to yield biased results (Rutstein and Rojas, 2003).

Three time five-year periods were created on the basis of the child year of birth in order to resolve this problem. These time periods are 1988-92, 1993-98 and 1999-2003.

3.14 Methods of Data Analysis

In order to estimate early childhood mortality, the synthetic cohort life table approach is used. Stata version 11 (Stata Corp, 2010) was used in the analysis of factors associated with the changing infant and child mortality. In particular, the Weibull models that take the accelerated failure time framework (AFT) were used to examine factors associated with the rise in early childhood mortality. These approaches are discussed in detail in the next sections.

3.14.1 Estimation of early childhood mortality

In this study we estimated the early childhood mortality by applying life table techniques to directly estimate the probability of dying (q_x) and cumulative probabilities of surviving

(ℓ_x). Direct estimation of early childhood mortality, especially in generalized HIV prevalence countries has been shown to be the most appropriate as none of the mortality estimates from indirect methods emulate any of the model life tables (Mahy, 2003; Wald and Zaba 2001). Additionally, the assumption about the independence of the mother from her biological child is greatly violated when indirect as opposed to when direct methods are used (Mahy, 2003).

This method entails the following steps: -

1. Cross tabulate all live births born within five years prior to each of the three surveys by age in months. In DHS, the century month code (CMC) can be used to convert the child's age in years to the number of months lived by the time of interview for living children.
2. Construct a life table. To do this, it is prudent to get the total live births disregarding all those cases that have no information. These total live births will form the number at start (i.e. the radix). The next columns of the table will be the number alive at interview tabulated by months since births (i.e. from 0-59 months).
3. Insert a column showing the number of children who died during any age interval (measured in months from 0-59 months). The DHS datasets have data on children's age at death in months and where data are missing on either age at death or birth, imputed cases are used.
4. Compute the number of children exposed to the risk of dying. Conventionally, it is assumed that those who die within any age interval do so halfway i.e. die halfway between age x to age $x+1$.

5. With these data, one is able to compute the probability of dying (${}_nq_0$) by simply dividing children who died within each age interval by the population of live births who were directly exposed to the risk of dying.

While the synthetic life table approach was used to compute the rates of the various components of early childhood mortality using the birth history data, one of the major gaps in this method is that it does not allow for the simultaneous analysis of potential determinants of early childhood mortality. Consequently, this study used two main additional sets of multivariate techniques of analysis: First, the Weibull parametric survival regression models that take the accelerated failure time framework and second, the use of the regression decomposition techniques.

Though the Weibull AFT models are important, they fail to account for changing values in the selected covariates over time and space. When variables change over time such as one would expect for variables such as source of drinking water, type of toilet facility or place of delivery among others, an important assumption of survival models is violated - that the event (in this case infant or child mortality) occurred at the time the selected factors were operating. In addressing this, the second approach - the regression decomposition techniques were used to examine the factors behind the upturn in early childhood mortality during the study period.

In the next section, a rationale for the extension of the Cox regression model is provided. A further discussion of the Weibull regression model as well as the regression decomposition technique appears in Chapters Five and Six, respectively.

3.14.2 The Cox Regression Hazard Model

The Cox Proportional Hazard Model is a commonly used multivariate approach for examining time to event data. Survival methods are superior as compared to standard regression techniques since they can account for censored cases in mortality analysis.

Mathematically, let us assume that $x=(x_1, x_2, x_3+...x_n)$ comprises the vector of predictor variables that we are interested in. The set of values of these predictor variables can be represented by x such that $x=(x_1, x_2, x_3+...x_n)$. Further if we can define $h_0(t)$ as the hazard function in which all the values of the predictor variables that make up the vector x are zero, then $h_0(t)$ is the baseline hazard function. Thus the hazard function for an individual child i aged between 0-59 months can mathematically be shown as: -

$$h_i(t) = h_0(t)\Psi(x_i) \dots\dots\dots\text{équation [1]}$$

Where $\Psi(x_i)$, is a function of the values of the vector of predictor variables for the i^{th} infant or child.

Since the baseline hazard is arbitrary, unspecified, and nonnegative apart from being a function of time (Bradburn *et al.*, 2003), it is convenient to write it as $\exp(\eta_i)$ where η_i is the linear component of the model and is represented as: -

$$\eta_i = \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \dots + \beta_n x_n \dots\dots\dots\text{équation [2]}$$

Where, the coefficients $\beta_1 \dots \beta_n$ represent unknown parameters that have to be estimated by Cox regression based on data obtained on the independent variables (x_1, x_n) . Each β_1 measures the change in the instantaneous risk resulting from a change of one standard

deviation in the variable x_1 . Consequently then, the hazard function for an infant or child i aged between 0-59 months at time t can be written as: -

$$h_i(t) = h_0(t) \exp (\beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \dots + \beta_n x_n) \dots \text{Equation [3]}$$

Or: -

$$\text{Log } h_i(t) / h_0(t) = \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \dots + \beta_n x_n) \dots \text{Equation [4].}$$

In the above models, $h_0(t)$ is the baseline hazard function and X_i is a vector of covariates. On the other hand, $h_i(t)$ is the hazard function which is the probability that an infant or child will die at time (t) .

Equation 4 above shows that in these models, the central statistical output is the hazard ratio i.e. $\{h_i(t)/h_0(t)\}$. A hazard ratio of 1.0 means the covariate has no effect on the occurrence or non-occurrence of infant and/or child mortality. However, if it is less than 1.0, it implies that the covariate acts to reduce the likelihood of infant and/or child mortality controlling for the effect of other covariates and finally, if it is more than 1.0, it will imply that the covariate increases infant and child mortality controlling for other covariates.

Although Cox regression is the most widely used multivariate approach for analyzing time to event data, it is of paramount importance that prior to its application, the proportionality assumption⁷ holds for each and every selected covariate (Bradburn *et al.*, 2003:432). When the proportionality assumption is not met, Gray (1996) reports that as much as 90 percent reduction in the power of significance tests (i.e. the chances of false

⁷ A covariate fails to meet the proportionality assumption if it interacts with time

negatives, rejecting the existence of true covariate effects) may be witnessed when rates cross rather than when they are proportionate.

The proportionality assumption prescribes that the duration of exposure to death of an infant or child yields the same effect on each infant or child. Therefore, the probability of each infant or child experiencing death since birth varies over time in the same way. The model holds that it is only the baseline hazard rate that varies from one live birth to another, depending on the attributes of each live birth.

The next section tests the proportionality assumption. To do this, one can run the log-log plots for the potential covariates. Normally, the log-log plots for categorical variables should be proportional if the proportionality assumption holds. This is an approach that was used by K'Oyugi (1992) in his study of the environmental factors that determined infant and child mortality in rural Kenya. Unfortunately, this approach is cumbersome and in this study, it would have meant that I construct almost 30 log-log plots.

Another highly recommended approach is to run a link test (Cleves, *et al.*, 2010). This test verifies that the coefficient on the squared linear predictor is insignificant. One can also interact the analysis time with the covariates and ascertain that the effects of the interacted variables are not different from zero since the proportionality assumption postulates that the effects do not change with time except in ways that the researcher has already parameterized (Cleves *et al.*, 2010). The disadvantage with this approach is that it is tedious especially if the number of the covariates is many unless one applies an

adjustment (the Bonferroni adjustment). In any case, this approach is complicated by the fact that not all covariates may need to be interacted with the same function of time yet there still remains no clear guidelines on choosing time functions (Cleves *et al.*, 2010).

Owing to the above shortcomings, this study used a test based on the Schoenfeld residuals. The logic behind this approach is to retrieve the residuals, fit a smooth function of time to them and then test whether there is a relationship with dependent variable(s). The scaled Schoenfeld residuals are recommended for assessing proportionality assumption since they provide usable information about the time dependence of the covariate effects.

The results of the Schoenfeld test (Table 3.1) showed that the duration of breastfeeding flouted the proportionality assumption ($p\text{-value} < 0.05$) hence the hazard ratios that increase over time for breastfeeding and its relative risk are bound to be overestimated on the one hand and its hazard ratios that decrease over time, the relative risk for such covariates are underestimated. In either case, the standard errors are incorrect and significance tests are decreased in power (Box –Steffensmeier and Zorn, 2001:972).

The table shows that on the overall, the models for infant and child mortality seem not to flout the proportionality assumption as shown by the global test values ($p > 0.05$). However, individual global test values (see appendix III) post-estimated in Stata 11 (Stata 2010 Corp) using the command *estat phtest* show that the duration of breastfeeding flout the proportionality assumption.

Table 3.1: The Schoenfeld residuals for selected covariates of infant and child mortality: merged KDHS 1993-2003

	Infant mortality				Child Mortality			
	rho	chi2	df	Prob>chi 2	rho	chi2	df	Prob>chi2
Maternal age at birth	-0.096	0.52	1	0.4689	-0.216	0.86	1	0.3531
Preceding Birth Interval	0.115	0.71	1	0.4010	-0.304	2.33	1	0.1271
Baby size at Birth	0.060	0.24	1	0.6216	0.149	0.53	1	0.4683
Breastfeeding period	0.201	5.04	1	0.0247*	0.074	0.11	1	0.7346
Maternal Education	-0.057	0.19	1	0.6592	-0.258	1.77	1	0.1832
Paternal Education	-0.004	0.00	1	0.9797	-0.237	1.37	1	0.2420
Marital Status	0.048	0.15	1	0.7027	0.172	0.73	1	0.3942
Water Source	-0.080	0.42	1	0.5193	0.118	0.39	1	0.5316
Toilet Facility type	-0.216	2.41	1	0.1206	0.192	0.95	1	0.3309
Household wealth Index	0.049	0.13	1	0.7175	0.163	0.68	1	0.4079
Place of delivery	0.041	0.11	1	0.7449	-0.096	0.19	1	0.6656
Tetanus injection	-0.129	1.06	1	0.3034	0.593	0.00	1	1.0000
Time period	0.038	0.07	1	0.7936	0.073	0.12	1	0.7251
Global test	$\chi^2 = 12.26$; df = 15 ; P-value = 0.6593				$\chi^2 = 19.37$; df = 15 ; P-value = 0.1514			

Source: Primary analysis of merged KDHS data

Apart from the proportionality assumption, the Cox PH model is also deficient in a study of this nature since it does not specify the baseline hazard yet mortality is known to follow a known and specific distribution. Consequently, parametric models (in particular, the Weibull models) are used to analyze the factors associated with the rise in early childhood mortality in Kenya during the study period.

In Chapter Five, the study presents a detailed discussion on the rationale for the choice of Weibull models that take up the accelerated failure time framework. In the next section, the study defines how the selected variables were measured and their expected relationship with the early childhood mortality components.

3.15 Variable definition and measurement

Using the empirical operational model that was modified from the Mosley and Chen (1984) as shown in Chapter One, the next section discusses how the selected variables were measured in this study. The choice of variables was guided by the past literature, availability of data and the statistical power anticipated.

Infant and child mortality

The dependent variables in this study were infant and child mortality. Since early childhood mortality varies with age, this variable was broken down into 0-11 months (infant mortality) and 12-59 months (child mortality). Infant mortality was measured as the number of deaths occurring before the first 12 months (i.e. 0-11 months) divided by all the live births 0-11 months multiplied by 1000 live births.

On the other hand, child mortality was measured as the number of deaths occurring between 12 -59 months divided by all children surviving between 12-59 months multiplied by 1000 children. It is expected that infant and child mortality has been increasing over the period under study.

Maternal age

This is the exact age of the mother at the birth of the index child. This variable was categorized as follows: 1= <20 years; 2= 20-34 years; 3= 35-49 years. It is hypothesized that the risk of death of a child varies with the age of the mother with young (<20 years) and old mothers (35+ years) associated with a heightened risk of experiencing early childhood mortality. In this study, it is expected that the proportion of children born by

mothers in the high risk ages (i.e. below 20 years or above 35 years) increased during the study period.

Birth Interval

This variable refers to the length in months of the preceding birth before the index child. The categories used were 1= less than 24 months; 2= 25-36 months; 3= 37 months or more. Normally, it is expected that shorter birth intervals are associated with a heightened risk of early childhood mortality.

Duration of breastfeeding

This was calculated in months based on whether the respondent was still breastfeeding the child or the child was breastfed until it died. The maximum period allowed during was the interval between the date of birth of the child and the date of interview. Categories used in this study were: 1= less than 10 months; 2= 11-19 months and 3 = 20 months or more. Conventionally, shorter periods of breast feeding are associated with increased chances of child death and vice versa.

Baby size

The variable refers to the physical size of the baby at birth. The categorization used was 0= small and 1= average/large. It is hypothesised that childhood mortality is a function of the size of the baby at birth with smaller than average baby size associated with heightened risk of experiencing death. While birth weight could have been a better measure than the size of the baby at birth, our preliminary analysis revealed that about 55 percent of babies in the KDHS datasets used in this study had missing values of the actual baby weight at birth. This is due to the fact that majority of babies in Kenya are born outside the formal health facilities and hence no measurement of their weight at

birth was taken. Size of baby at birth is used as a proxy measure since recent studies based on DHS data have shown an overall agreement between recorded and recalled weight and subjective assessment of birth size (Mbuagbaw, 2010).

Educational level

This refers to the educational level attained by the mother or father. This study used two variables: maternal education and paternal education. Categories used in both of these variables are 0 = none, 1= primary and 2= secondary and above. It is expected that children whose father or mother had no education were likely to experience higher risks of experiencing child mortality as compared to children whose father or mother had at least secondary schooling.

Household wealth Index

The household wealth index was computed using the principal component analysis (PCA) approach. The range of asset variables included in the construction of the index was broad enough to circumvent the problem of clumping⁸ and truncation⁹. Consequently, the study grouped households' socio-economic status into tertiles rather than the commonly used quartiles or quintiles which would otherwise have introduced the problem of truncation. This index was intended to capture the household resources available for childcare. The categories used were 1= Lower; 2= Middle and 3= Upper. It is expected that households in the lower tertiles will have higher risks of child mortality losing their children than the rest.

⁸ The problem of clumping also called clustering occurs when households are categorised together in a small number of distinct groups (Vyas and Kumaranayake 2006).

⁹ Truncation occurs when households are grouped into even distribution of socio-economic status bands but spread over a narrow range making it almost impossible to differentiate between households that belong to various socio-economic status bands (Vyas and Kumaranayake 2006).

Type of Toilet facility

This refers to how the household disposes human excreta. The options used were flush toilet (own/shared), traditional pit latrine, VIP latrine and other (no facility, bush, etc). It is expected that households without any form of facility for human waste disposal were more likely to experience child mortality due to poor sanitation as compared to the rest.

Source of water

This is defined as the source of water for drinking in the household. The categories used are 1=piped water; 2= well, and 3= lake, pond and stream.

Tetanus toxoid

This variable measures whether the mother received a tetanus toxoid injection during pregnancy or not. It is measured as 1=Yes and 2=No. Studies have shown that receiving a tetanus jab is associated with a reduced risk of experiencing childhood mortality from lockjaw disease especially during the first two weeks of life.

Place of delivery

This refers to the place where the child was born. The categories used here are 1= born at home; 2=being born in a government health facility and 3=being born in a private health facility. It is hypothesized that being born outside a health facility is associated with a heightened risk of experiencing child death.

3.16 Concluding remarks

This Chapter discussed the data sources, data quality and methods of data analysis. The thesis here was that if the data were to have deteriorated over the years in a way that overstates infant and child mortality, then they could partly be accountable for the upsurge in infant and child mortality independent of the changing covariates of early

childhood mortality. The results obtained show that the data quality remained the same or improved over the study period. Where there was evidence of heaping such as at month 12 (where the heaping ratio >1) the direction of the effect understated infant mortality and hence could not be a consequence of the observed upsurge in infant mortality.

A further discussion with staff from the Kenya National Bureau of statistics (KNBS) and National Coordinating agency for population and Development (NCAPD) in charge of organising these surveys did not show any compromise in the data quality procedures. Similar past studies in Kenya did not also find poor data quality as the main reason behind the upturn in early childhood mortality (Hill *et al.*, 2004).

The Chapter also discussed how the variables were measured. The synthetic life table approach and the Cox PH models were discussed. The duration of breastfeeding did not meet the proportional hazards assumption ($p < 0.05$) and hence the Weibull AFT models were recommended for studying covariates behind the rise in mortality during the 1988-2003 period. A further discussion of the application of the Weibull AFT models is provided in Chapters Five while regression decomposition techniques are discussed Chapters Six and Seven.

CHAPTER 4: A BIVARIATE ANALYSIS OF THE UPSURGE IN INFANT AND CHILD MORTALITY

4.1 Introduction

This chapter presents and discusses the main characteristics of the study population as well as the levels and trends in infant and child mortality over the study period. In order to achieve this, the study used cross tabulations with a total of 14,317 live births. Bivariate analyses based on the Weibull models in the accelerated failure time framework were performed in order to ascertain whether the association between the various levels of infant and child mortality are significantly associated with selected covariates.

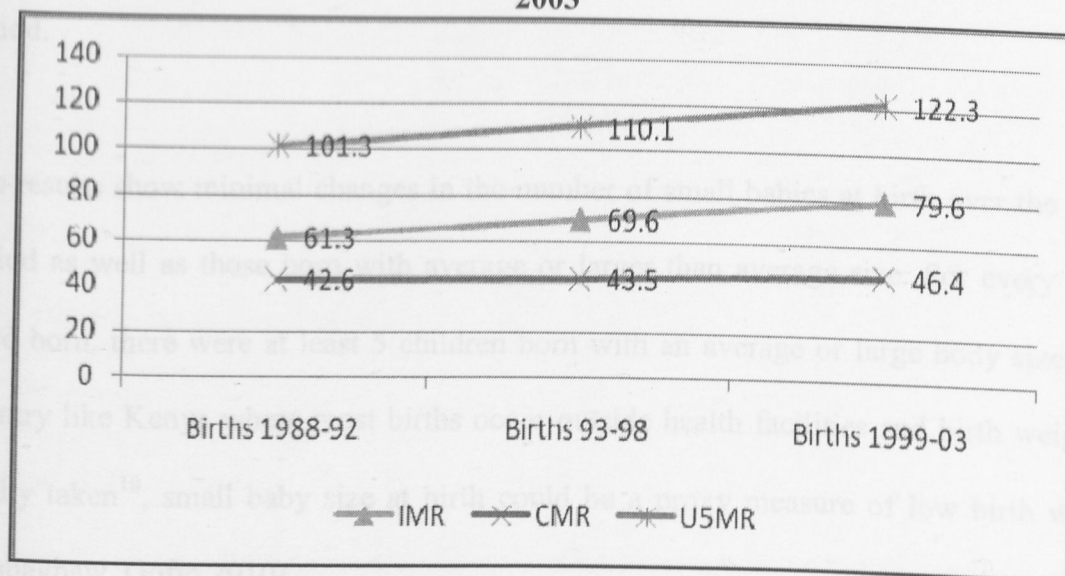
4.2 Levels and trends of infant and child mortality

In Figure 4.1 below, infant mortality rose from 61 deaths per 1000 live births during the 1988-92 time period to 70 deaths per 1000 live births during the 1993-98 time period and to almost 80 deaths per 1000 live births during the 1999-2003 time period. This represents an upsurge of 30 percent in infant mortality for the 1988-2003 time periods. The results compare well with the 32 percent increase in infant mortality reported in the main KDHS country report (CBS, *et al.*, 2004). In general, the upturn in infant mortality during the 1993-98 (14.3 percent) was slightly greater than that observed during the 1988-1992 (13.5 percent).

As shown in Figure 4.1, child mortality rose from 42.6 deaths per 1000 live births in the 1988-92 to 46 deaths per 1000 live births in the 1998-2003 time periods. This represents a rise of 9 percent over the period. The under-five mortality rose from 101 deaths per

1000 live births during the 1988-92 time period to 122 deaths per 1000 live births in 1998-2003 time period.

Figure 4.1: Trends in Early Childhood Mortality in Kenya, Merged KDHS 1993-2003



Source: Primary Analysis of Merged KDHS 1993- 2003

This represents a significant increase of 21 percent. These results confirm the rise in early childhood mortality during the 1988-2003 period reported in the KDHS Country report (CBS *et al*, 2004).

4.3 Distribution of selected characteristics

Table 4.1 below shows that in general, the percentage of mothers in the high risk reproductive age bands (<20 years and above 35 years) remained largely constant during the study period. For instance, in all the three time periods included in this study, more than a quarter of all births occurred among mothers in the high risk births. On the contrary, there was an apparent rise in the number of children who breastfed for very short durations (<10 months). The percentage increase in children breastfeeding for shorter durations rose by about 16 percent during the 1988-92 and 1993-1998 period and by about 29 per cent during the 1993-98 and 1999-2003 period. On the contrary, while

prolonged breastfeeding is usually associated with increased benefits of child survival (Lindstrom and Berhanu 1999), results from Table 4.1 show a 23 percent reduction in children who breastfed for more than 20 months during the 1988-92 and 1998-2003 period.

Table 4.1: Summary characteristics of the study population

Time period	Time period	Time period
1988-92	1993-97	1998-03

The results show minimal changes in the number of small babies at birth over the study period as well as those born with average or larger than average size. For every small child born, there were at least 5 children born with an average or large body size. In a country like Kenya where most births occur outside health facilities and birth weight is hardly taken¹⁰, small baby size at birth could be a proxy measure of low birth weight (Mbuagbaw, Gofin 2010).

The number of births with preceding birth intervals greater than 36 months decreased over the period. During the 1988-92 and 1993-98, this decrease was almost as much (19.1 percent) as that observed during the 1993-98 and 1999-2003 period (19.4 per cent).

The figures indicate that maternal education fluctuated less during the three periods. While there were as many mothers with no education as there were with secondary and above during 1993-98, results appear to indicate that during the 1999-2003 period, for every child belonging to a woman with no education that was included in the survey, there were two children of mothers with secondary schooling and above. Throughout the

¹⁰ In the preliminary analysis of the merged KDHS dataset, at least 53 percent of all the live births were not weighed at birth and hence were missing cases. Consequently, the use of birth weight as a variable could have biased our results. In any case, the effect of birth weight on mortality is bound to have been captured by size of the baby at birth and the preceding birth interval both of which are included in this study.

study period, for every child with no formal schooling whose household was randomly sampled, at least four children with primary schooling had their households randomly sampled.

Table 4.1: Summary characteristics of the study population

	<i>Time period</i> 1988-92	<i>Time period</i> 1993-97	<i>Time period</i> 1998-03
Maternal age at child birth			
< 20 years	14.3	15.0	14.1
20-34	72.4	70.5	72.9
35+	13.3	14.5	13.0
Duration of Breastfeeding			
< 10 months	23.0	26.6	34.2
11-19 months	42.3	39.9	39.3
>20 months	34.6	33.5	26.5
Size of Baby at Birth			
Small	16.0	14.5	15.3
Average/Large	84.0	85.5	84.7
Preceding Birth Interval			
<24 months	27.1	30.9	27.5
25-36	34.8	38.3	35.7
37+	38.1	30.8	36.8
Maternal Education			
None	14.4	16.6	13.8
Primary	62.3	66.6	60.6
Secondary+	23.3	16.8	25.5
Paternal Education			
None	10.2	11.0	8.0
Primary	52.7	58.1	49.8
Secondary+	37.1	30.9	42.2
Household wealth			
Lower	34.2	33.9	33.4
Middle	32.5	32.8	34.3
Upper	33.3	33.3	32.3
Source of drinking water			
Piped	38.7	20.3	32.7
Lake, pond, river, stream, rain	44.0	58.9	40.2
Well	17.2	20.8	27.2
Type of Toilet facility			
Flush toilet (own/shared)	9.1	3.0	10.1
Traditional pit latrine	67.5	73.2	67.5
VIP latrine	6.6	5.2	5.8

Other (no facility, bush, etc)	16.7	18.5	16.6
Place of delivery			
Home	53.9	57.9	57.0
Government Health facility	32.8	29.2	31.4
Private Health Facility	13.3	11.1	11.6
Tetanus Injection before birth			
No	12.4	8.8	8.6
Yes	87.6	91.2	91.4
Total number of cases	5551*	3915*	4851*

Source: Primary analysis of merged KDHS 1993, 1998 & 2003. * Some variables do not add up to these figures because of missing cases

During the 1988-92 period, for every three randomly sampled mothers with primary schooling, only one proceeded to secondary schooling. The ratio for 1993-98 and 1999-2003 periods was one in four and one in two respectively. However, children of fathers with no education were at least five times less likely to have their households sampled as compared to those with primary schooling. Children whose fathers had only primary education reduced from 10.2 percent to 8.0 percent. However, those whose fathers had only primary schooling reduced by 5.8 percent during the study period. The increase in children whose fathers had at least secondary schooling was 13.7 and 36.6 percent during the 1988-92/1998-2003 and 1993-97/1998-03 period respectively.

The proportion of children belonging to lower, middle and upper class households remained generally unchanged over the study period and across the categories. Almost a third of all the children belonged to either of the three categories of the household wealth index throughout the period.

A notable finding was that the number accessing piped water for drinking declined during the study period from 38.7 percent during the 1988-92 to 32.7 percent during the 1998-

03. This was a 15.5 percent reduction in households with piped water during the study period. On the other hand, about one sixth of the households did not have any form of toilet facility.

Further, Table 4.1 shows that throughout the period under study, mothers who delivered at home accounted for over a half of all deliveries. The number giving births at home peaked during the 1993-97 period and appears to have stalled throughout the 1999-2003 period. The percentage delivering in private health facilities remained the least throughout the period.

Past evidence shows that the tetanus causing organism- *Clostridium tetani* accounts for a large number of infant deaths by the end of the first two weeks of a baby's life (Boerma and Stroh 1993). Results revealed that mothers who had received tetanus toxoid prior to giving birth increased by 3.8 percentage points during the 1988-92 and 1999-2003 periods as shown in Table 4.1 above. Generally, for every 7 mothers who received a tetanus toxoid, one mother did not receive the injection. In the next section, the study presents the changes in infant and child mortality rates by selected characteristics.

4.4 Changes in Infant and Child Mortality Rate according to selected factors

Table 4.2 below shows that infant mortality increased by maternal age at child birth during the 1988-92 through the 1999-2003 period. In particular, children belonging to mothers in the 20-34 ages experienced the highest increase (43 percent) in infant mortality rate during the 1988-92 and 1999-2003 period. However, when child mortality

was considered, children belonging to mothers with at least 35 years and above exhibited the highest increase in mortality during the study period (71 percent).

Further, the increase in the percentage of infant mortality among babies born with small body sizes was three times less as compared to the upsurge in infant deaths experienced among babies with average or large body sizes. However, during childhood, babies born with small body sizes recorded a huge upsurge in mortality (272 percent) during the 1988/92 and 1999/2003 period. Among those with average or large body sizes, there was a slight decline (7 percent) in child mortality during the study period.

Generally, shorter birth intervals are associated with a heightened risk of early childhood mortality (Hobcraft 1985, Chidambaram 1985). Results confirmed that infants with a preceding birth interval of <24 months experienced an upsurge in mortality of 71 percent during the 1989-2003 period. Interestingly, longer birth intervals (37 months +) were associated with a higher upsurge in mortality as compared to preceding birth intervals of 25-36 months. For instance, for every 2 children who died before attaining the fifth birthday but whose preceding birth interval was at least 37 months during the 1993-98 and 1999-03, one child with a preceding birth interval of between 25-36 months died.

The duration of breastfeeding especially during the first six months of an infant has been found to improve infant survival due to the nutritional value of mother's milk, the immunities against communicable diseases that it provides, and because it is hygienic (Lindstrom and Berhanu 1999). Table 4.2 below shows that infant mortality rose by 143

percent among infants who breastfed for less than 10 months during the study period while the upsurge in mortality was 184 percent among children (12-59 months). Generally, children who breastfed for <10 months experienced the highest mortality during the study period. As expected, results revealed that throughout the study period, children who breastfed for the longest period (>20 months) exhibited the highest decline in early child mortality.

Results from Table 4.2 show that all dummies for children belonging to mothers with various levels of education recorded an upsurge in infant mortality with children belonging to mothers having no formal schooling experiencing the highest upsurge during the 1988-92 and 1999-2003 time periods (61 percent). However, when the risk of dying was assessed in childhood, children belonging to mothers with no education experienced a 22 percent reduced risk of dying during the study period while those who had some primary schooling experienced the highest increase in child mortality during the study period (16 percent).

The upsurge in infant mortality rate as assessed by paternal education showed interesting results. Unlike children belonging to mothers with no formal schooling who exhibited the highest upsurge in infant mortality during the study period, children belonging to fathers with some primary education exhibited the highest upsurge in infant mortality rate (39 percent) while those belonging to fathers with no education or secondary schooling experienced almost a similar percentage in mortality upsurge during the study period. When paternal education was assessed vis-à-vis child mortality, results revealed that children belonging to fathers with no education exhibited the highest upsurge in mortality

rates (54 percent) during the study period. On the contrary, children belonging to fathers with at least secondary schooling recorded a decline in child mortality (17 percent) during the study period. This could be due to educational homogamy where educated fathers are more likely to marry educated women both of which experience lower rates of infant and child mortality as compared to their counterparts.

Table 4.2: Levels and trends of infant and child mortality by selected characteristics

	Changes in IMR, 1988-03				Changes in CMR, 1988-03			
	88-92	93-98	99-03	% Δ 88-03	88-92	93-98	99-03	% Δ 88-03
Mother's Age								
<20years	74.9	93.5	87.2	16.4	48.6	83.6	57.6	18.5
20- 34	51.4	58.2	73.6	43.2	34.8	33.7	35.5	2.0
35+	77.6	82.8	90.6	16.8	66	55.4	112.7	70.8
Baby size at birth								
Small	86.2	107.5	97.2	12.8	18.6	56.5	69.2	272.0
Av./ Large	53.7	58	73.6	37.1	44.1	40.9	41	-7.0
Birth Interval								
<24 months	76.7	82.9	130.8	70.5	43.0	38.6	41.8	-2.8
25-36	51.5	78.2	72.5	40.8	45.2	22.5	35.7	-21.0
37+	47.1	50.5	67	42.3	47.6	45.9	72	51.3
Breastfeeding Period								
<10 months	108.8	227.4	264.3	142.9	15.8	96.2	44.9	184.2
10-19 months	1.6	0.7	7.9	393.8	47.7	60	48.7	2.1
>20 months	92.9	6.5	1.5	-98.4	41.8	11.2	39.1	-6.5
Maternal education								
None	59	84.9	95.2	61.4	43.7	30.5	34.3	-21.5
Primary	69.5	77.4	85.4	22.9	50.1	50.3	58.2	16.2
Sec+	40.1	41.6	54	34.7	20.9	32.9	21.4	2.4
Paternal Education								
None	81.5	91.5	104.9	28.7	44.3	1.6	68.2	54.0
Primary	63.4	83.5	87.8	38.5	52.7	63.2	63	19.5
Sec+	47.1	42	61.2	29.9	26.7	26.5	22.2	-16.9
Household wealth								
Lower	42.1	60	75.6	79.6	26.7	22.8	49.5	85.4
Middle	56.5	74.2	80.1	41.8	33.5	24.5	28.8	-14.0
Upper	79.4	76.4	62.3	-21.5	69.7	56.6	41.7	-40.2

Place of delivery								
Home	68.8	78.8	83.4	21.2	56.8	56.3	53.6	-5.6
Gov't Health facility	53.1	56.2	84	58.2	25.8	27.8	27.2	5.4
Private Health Facility	33.7	23.1	54.7	62.3	17.1	20.9	54.9	221.1
Tetanus Toxoid								
No	105.1	81.1	63.9	-39.2	35.7	29.5	50	40.1
Yes	54.4	63.8	53.6	-1.5	41.3	41.3	50.2	21.5

Source: Primary analysis of merged KDHS 1993, 1998 & 2003. * Some variables do not add up to these figures because of missing cases

We computed the household wealth index using the principal component analysis (PCA) approach in order to determine the changes in the household wellbeing and early childhood mortality components. The results obtained indicate a decline in infant mortality among mothers belonging to the upper socio-economic class (22 percent) while those in the middle and lower tertiles experienced an upsurge in infant mortality during the 1988/92 and 1993/98 period. The decline in mortality among the upper class households persisted throughout early childhood beyond infant mortality.

On the other hand, the upsurge in early childhood mortality persisted beyond infancy among the lower households with children from such households exhibiting 85 percent increased chances of dying between 1988 and 2003 study period. It seems that generally, rich parents have resources to provide better healthcare for their children hence their lower infant and child mortality as compared to the rest. Children belong to such wealthy households also experienced the greatest decline in infant and child mortality during the study period.

Finally, Table 4.2 shows that giving home delivery was associated with the highest risk of infant mortality during the study period. During the study period, infant mortality rate

among mothers who delivered at home rose by 21 percent. While being born in a government or a private health facility was associated with a lower risk of dying as compared to being born at home, generally deliveries at a health facility (both government or private) recorded the highest upsurge in infant mortality rate (58 percent and 62 percent respectively) during the study period.

Tetanus injection before birth is important in reducing tetanus related mortality during the neonatal period. Generally, infant mortality rate decreased among mothers irrespective of whether they had received tetanus injection or not. As shown in Table 4.2 above, among children born of mothers who had not received the tetanus injection, infant mortality decreased by 39 per cent from 105 deaths to 64 deaths/1000 live births. The results also show a minor decrease in mortality among children whose mothers had received tetanus injection prior to the time they were born (1.5 percent decrease).

Finally, an upsurge in child mortality among mothers who had not received a tetanus jab was 40 percent while the upsurge among mothers who has received a tetanus jab was 22 percent during the 1988-2003 period. While the above analysis is insightful in bringing out the changes between infant and child mortality components and the selected socio-economic characteristics, it fails to answer the question on whether the observed changes were statistically significant. To address this, the next section presents the bivariate analysis between each of the selected factors against the components of mortality controlling for other factors.

4.5 Bivariate Weibull AFT Analysis Results

This section presents the results of bivariate Weibull analysis in the accelerated failure time framework (AFT). The time ratios (TR) for the dummy variables are presented to explain whether infant and children were at a higher risk of dying faster or not as compared to the reference category.

As compared to children born to mothers aged 20-34 years, those born to mothers aged below 20 years experienced an increased risk of dying faster in infancy and this association was statistically significant ($p\text{-value} < 0.05$). Similarly, during childhood, children born to mothers aged below 20, experienced 45 percent elevated mortality risks compared to those born to mothers aged 20-34 years. These results are similar to those found in other past studies (Hobcraft *et al.*, 1985; Miller 1993, Da Vanzo *et al.*, 1983; Chidambaram *et al.*, 1985).

As expected, compared to children with a preceding birth interval of at least 37 months, children whose preceding birth interval was < 24 months or 25-36 months were significantly associated with a 45 percent and 28 percent increased chances of dying faster during infancy ($p\text{-value} < 0.05$). The effect of the preceding birth interval on infant and child mortality appears to diminish and in a large measure was reversed when assessed across children (12-59 months). When the risk of death was modelled among children (12-59 months), belonging to mothers with a preceding birth interval of less than 24 months was associated with a 19 percent increased chances of dying faster as compared to belonging to mothers whose preceding birth interval was 37 months or higher ($p\text{-value} < 0.05$). However, children whose preceding birth interval was 25-36

months were associated with a 22 percent prolonged chances of survival in childhood as compared to our reference category (37+ months) and this association was statistically significant ($p < 0.05$).

Table 4.3: Factors associated with the changing Infant and child mortality in Kenya: KDHS 1993-2003

Characteristic	Infant Mortality		Child mortality	
	TR [Exp(β)]	S.E(β)	TR [Exp(β)]	S.E(β)
Maternal age				
20-34 years (RC)				
<20 years	0.754*	.114	0.558*	.244
35-49 years	0.958	.134	0.773	.284
Preceding Birth Interval				
37 months+ (RC)				
<24 months	0.554*	.128	0.815*	.263
25-36 months	0.723*	.127	1.224*	.273
Baby Size at birth				
Av./large (RC)				
Small	0.965	.124	1.455	.313
Duration of Breast feeding				
10-19 months (RC)				
<10 months	0.049*	.259	1.195*	.296
>20+ months	0.128*	.249	1.537*	.209
Maternal Education				
Sec+ (RC)				
None	0.681*	0.165	0.580	0.364
Primary	0.559*	0.13	0.436*	0.293
Paternal Education				
Sec+ (RC)				
None	0.671*	0.164	0.757	0.358
Primary	0.639*	0.107	0.639	0.227
Source of Drinking water				
Piped (RC)				
Well	0.710*	0.149	0.499*	0.338
Lake, Pond, Stream	0.684*	0.128	0.427*	0.296
Type of toilet Facility				
Flush (own/shared) (RC)				
Trad. Pit Latrine	0.612*	0.219	0.482	0.500
VIP	0.674	0.287	0.497	0.638
Other	0.419*	0.23	0.261*	0.524

Household Wealth Index				
Upper (RC)				
Lower	0.563*	0.112	0.477*	0.255
Middle	0.851	0.119	0.611*	0.260
Place of Delivery				
Government facility (RC)				
Home	0.710*	0.105	0.608*	0.226
Private Health Facility	1.346	190	1.768	0.437
Tetanus Injection				
No (RC)				
Yes	1.042	0.176	1.740	0.489

Source: Primary analysis of merged KDHS 1993- 2003. * P -value <0.05 ; Coef. =coefficient; S.E. =Standard error of the coefficient; TR = time ratios. These are computed as 1 minus the exponents of the AFT Weibull coefficients (Box -Steffensmeier and Zorn, 2001).

Generally, there appeared to be no significant association between size of the baby at birth and the risk of dying in infancy or childhood (p -value >0.05). For instance, being born with a small body size was associated with a marginal 3.5 percent increased chances of dying during infancy, as compared to being born with average/large body sizes at birth. However, the association was not statistically significant ($p>0.05$).

The duration of breastfeeding appears to have one of the strongest effects on infant mortality (p -value <0.05). Table 4.3 above shows that during infancy, children who breastfed for a shorter duration (less than 10 months or >20 months), tended to experience a 95 and 87 percent increased chances of dying faster as compared to those who breastfeed for 10-19 months and this association was statistically significant (p -value <0.05).

However, beyond infancy, children who breastfed for a shorter or longer duration (less than 10 months or >20 months), tended to experience a 20 and 54 percent prolonged

chances of surviving as compared to those who breastfed for 10-19 months and this association was statistically significant ($p\text{-value}<0.05$).

The results also indicate that children whose mother had no formal schooling or those with primary schooling alone was associated with 32 percent and 45 percent increased chances of dying faster in infancy as compared to children whose mothers had at least secondary education and the association was statistically significant ($p\text{-value}<0.05$). On the contrary, children whose mothers had primary education were significantly associated with a 56 percent increased chances of dying faster in childhood as compared to children whose mothers had at least secondary education ($p\text{-value}<0.05$). However, there was no statistically significant difference in the child mortality risk of children whose mothers had no education and those whose mothers had at least secondary education.

The association between paternal education and infant mortality was expected. Children whose fathers had no education and those whose fathers had primary education were associated with a 33 and 36 percent, respectively, higher chances of dying during infancy as compared to children whose father had at least secondary schooling ($p\text{-value}<0.05$).

The results show that paternal education did not have a statistically significant effect on child mortality.

As expected, using water from any other source other than piped water supply was associated with increased infant and child mortality risks. For instance, as compared to children belonging to households that had piped water supply, children belonging to

households that had water supply from the well or from the lake, pond or stream, were significantly more likely to die faster during infancy as well as during childhood and this associated risk was statistically significant ($p < 0.05$).

Generally, the use of any other form of toilet facility other than the use of flush toilet was associated with an increased chance of dying faster during infancy and childhood. For instance, as compared to children from households with flush toilets or those who were sharing flush toilets, those who belonged to households that used the traditional pit latrines experienced 39 percent increased chances of dying in infancy and this association was statistically significant ($p\text{-value} < 0.05$). On the other hand, those that belonged to households that used other toilet facilities including the bush had 58 percent increased chances of dying faster in infancy and this association was statistically significant ($p\text{-value} < 0.05$).

Interestingly, as compared to using flush toilets, use of VIP toilets was not significantly associated with infant and child mortality. Further, the use of the traditional pit latrine was not significantly associated with the risk of dying in childhood ($p > 0.05$). However, as compared to children belonging to households who had flush toilets, those that belonged to other types of toilet facilities such as the bush had 74 percent increased chances of dying faster in childhood and this association was statistically significant ($p\text{-value} < 0.05$).

Belonging to households that were in the upper wealth tertile was associated with the lowest chances of dying during infancy and childhood (1-4 years). For instance, children belonging to households that were in the lower and middle wealth tertiles were 44 and 15 percent more likely to die earlier during infancy as compared to infants belonging to household in the upper wealth tertile. However, it was only the children that belonged to lower household wealth tertile that had a statistically significant elevated risk of experiencing infant mortality as compared to children belonging to wealthy /rich household (upper tertile) ($p\text{-value}<0.05$).

During childhood, children belonging to lower tertile households and those belonging to middle tertile household were significantly associated with 53 percent and 39 percent increased chances of dying faster in comparison to children belonging to wealthy /rich households (upper tertile) and this association was statistically significant ($p<0.05$).

This study also examined the role of some maternal health seeking factors in relation to the upsurge in infant mortality during the study period. Table 4.3 above shows that home delivery was associated with a 29 percent and 40 percent increased risk of dying faster at infancy and childhood as compared to being born in a government health facility and this association was statistically significant ($p < 0.05$). Children whose mothers had received a tetanus injection during pregnancy enjoyed a 4 percent prolonged chances of surviving at infancy although this association was not statistically significant ($p>0.05$). During childhood, children whose mothers had received a tetanus injection during pregnancy had

74 percent increased chances of living longer as compared to their counterparts although the association was not statistically significant ($p > 0.05$).

5.1 Introduction

4.6 Concluding remarks

This chapter revealed that there was an upturn in infant and child mortality during the 1988-2003 period in Kenya. The overall upturn in infant mortality was much higher than that of child mortality. Infant and child mortality increased by a number of selected background and proximate factors. In particular, many of the selected covariates were significantly associated with increased mortality risk apart from whether the mother received a tetanus injection or not and the size of the baby at birth. These results however do not show the net effect of the selected covariates on infant and child mortality. In the next chapter, we address this issue by running multivariate analysis.

CHAPTER 5: RESULTS OF THE MULTIVARIATE ANALYSIS

5.1 Introduction

This chapter used a set of survival analysis methods to study the determinants of infant and child mortality. This is important because prior to establishing the factors behind the upturn in mortality, it is logical to first establish what factors influenced the level of early childhood mortality. The choice of survival models was motivated by the fact that when data on infant and child mortality components are analysed using standard regression such as logistic regression, there is a likelihood of bias arising from the failure to account for censoring.

5.2 Analytic Approach

The Weibull model in the accelerated failure time (AFT) framework was used. This is because it is widely documented that the distribution of human mortality is theoretically known to follow a Weibull distribution (Cleves *et al.*, 2010; Omariba, 2007). Further, Weibull models have been shown to be appropriate for hazards that are either monotonically increasing or decreasing (Blossfeld and Rowher, 2002) as is the case in this study.

Preliminary analysis which entailed running hazard plots as a way of assessing the appropriateness of statistical models for parametric survival analysis (appendix III) paints this type of mortality distribution as the most appropriate. Generally, when the empirical data follow a Weibull distribution, then a plot of the log $[-\log s(t)]$ against $\log t$ (commonly known as a “log-log survivor plot” or “log cumulative-hazard plot”) should

yield a straight line (Guo, 2010:104). The log-log survivor plot in this study yielded a straight line as shown in appendix III hence the choice of the Weibull AFT model.

The study used the *streg* command in Stata 11 (StataCorp, 2009) to run Weibull models. In an AFT model, the dependent variable is the event time (which in this study is the time to experiencing death before the first 12 months after birth or between 12 months and 59 months). The AFT assumption is that all observations have the same shape hazard function, but the time axis varies such that some groups of children as is in our case pass through stages of the hazard curve faster than others.

The Weibull AFT model can mathematically be presented as

$$S(t)=S_0(\psi t) \dots\dots\dots \text{equation [4]}$$

Where $S_0(t)$ is the baseline survivor function and ψ is an 'acceleration factor' that depends on the covariates according to the formula

$$\psi = \exp\{(b_1x_1+b_2x_2+\dots+b_px_p)\} \dots\dots\dots \text{equation [5]}$$

The principle here is that the effect of a covariate is to stretch or shrink the survival curve along the time axis by a constant relative amount ψ . The most intuitive way of expressing AFT model coefficients is to convert them into time ratios (TR). Normally, time ratios are computed as the exponents of the regression coefficient ($\exp(\beta)$) but one can run the model with the '*tr*' option in Stata to get the output of time ratios for the explanatory variables directly. Two models of Weibull regression can be expressed in terms of time ratios- the Weibull proportional hazards (PH) models and the Weibull accelerated failure time (AFT) models. Whereas the Weibull PH models assumes that the covariate effect is

to multiply the hazard by some constant, the Weibull AFT models assume that the effect of a covariate is to multiply the predicted event time by some constant. Consequently, time ratios have different interpretations in the AFT and PH Weibull modelling.

This study applied the Weibull AFT models to circumvent the problem of non proportionality that was presented and discussed in Chapter Three. In these models, $TR > 1$ is associated with a prolonged survival time, or more accurately, an expansion of time to failure and vice versa. Suffice it to say, a time ratio above 1 for a given covariate implies that the covariate 'slows down', or prolongs the time to the event, while a time ratio below 1 indicates that an earlier event is more likely (Bradburn, *et al.*, 2003:432). If $TR=1$, it implies that the covariate has no effect on survival time.

5.3 Effects of selected covariates on infant mortality

Table 5.1 below shows an analysis of the covariates of infant mortality as guided by the Mosley and Chen framework. Model I shows the period variable. Model II shows the period variable and background characteristics considered in the analysis. The background characteristics include maternal and paternal education, source of drinking water, type of toilet facility and household wealth index. Finally, Model III is the full model showing results of the period variable, background factors and proximate determinants of mortality. The proximate determinants assessed in this study include maternal age at child birth, preceding birth interval, duration of breastfeeding, size of the baby at birth, place of delivery and whether or not the child's mother got a tetanus toxoid during pregnancy.

Results from Table 5.1 show that all the dummies of time variable were critically important in explaining the upsurge in infant mortality before and after they were adjusted for other factors. When time was analysed as the only variable as shown in Model I, infants born during the 1993-1998 and 1999-2003 were 41 percent and 30 percent more likely to die earlier as compared to those who were born during the 1988-92 period and this association was statistically significant ($p\text{-value} < 0.05$).

When background factors were added to the period variable as shown in Model II, the increased risk of dying earlier among infants born during 1993-1998 and 1999-2003 remained statistically significant at about 42 percent and 30 percent respectively as compared to being born during the 1988-1992 period. Further, maternal and paternal education, type of toilet facility used by the household and the household's socioeconomic status as measured by the household wealth index were not significantly associated with the risk of dying in infancy ($p\text{-value} > 0.05$) even though most of the dummies for these variables were in the expected direction.

The only exception to this was the association between the household's source of drinking water and infant mortality. As compared to infants belonging to households that used piped water (whether owned or shared), those belonging to households that drew water from the lake, pond or stream had a surprising 10 percent increased chances of surviving longer and this association was statistically significant ($p\text{-value} > 0.05$). While DHS do not collect information on whether the source of water is treated or not, the results in this study could be a reflection of the concerted efforts in the use of water

treatment and purification techniques in the early 2000s which consequently negated the health risks that are associated with unsafe water sources such as lake, pond and stream.

For instance, according to results from KDHS 1998 (CBS, *et al.*, 1999), 66 percent of the population in Nyanza province alone lacked access to safe drinking water and 47 percent of the under-five children had experienced diarrhoea in the preceding two weeks before the survey. However, a safe water system that was implemented by Care Kenya and Centers for Disease Control (CDC) in 2000 saw an adoption of the approach by 33.5 percent of the households (Makutsa, *et al.*, 2001). Field evaluations have shown that use of safe water system results in a 25-85 percent reduction in diarrhoea (Migele, *et al.*, 2007) and a significant reduction in deaths owing to the fact that diarrhoeal is the second leading cause of death among the under-five children worldwide (UNICEF and WHO, 2009).

Model III presents results of the full model. The effect of time still remains strongly associated with the risk of dying in infancy. Results show that as compared to infants who are born during 1988-92 period, those born during the 1993-98 and 1999-2003 period were 43 percent more likely to die faster and this association was statistically significant (p value < 0.05). The deterioration in Kenya's economy as a result of structural adjustment programmes in the late 1980s could have reduced household purchasing power due to retrenchment and reduced income due to inflation which adversely affected access to medical care as well as better nutrition. At the national level,

GDP reduced from less than 5 percent in 1988 to about 1.4 percent in 2002 (World Bank 2007).

While the dummies of maternal education were in the expected direction, none was significantly associated with the risk of dying in infancy when assessed against the reference category ($p\text{-value} > 0.05$). Interestingly, children belonging to fathers with primary level of education were 7 percent more likely to survive for a longer time as compared to those whose fathers had at least secondary education and this association was statistically significant ($p\text{ value} < 0.05$). This could be due to diffusion of ideas on better child care from fathers with at least secondary education to their primary level counterparts with the later having a lot of time with their families to translate the knowledge gained on better child care practices into actual practice unlike the former who are more likely to be engaged in gainful employment that leaves them with little time for child care.

Table 5.1: Time ratios for selected factors associated with the changing infant mortality: KDHS, 1993-2003

Characteristic	Model I		Model II		Model III	
	TR	S.E(β)	TR	S.E(β)	TR	S.E(β)
Time period						
88-92 (RC)	1.000		1.000		1.000	
93-98	0.591**	0.019	0.575**	0.019	0.570**	0.022
1999-03	0.704**	0.022	0.705**	0.028	0.576**	0.026
Maternal Education						
Sec+ (RC)			1.000		1.000	
None			0.960	0.055	0.985	0.063
Primary			0.929	0.036	0.947	0.043
Paternal Education						
Sec+ (RC)			1.000		1.000	
None			1.068	0.066	1.098	0.072
Primary			1.060	0.036	1.074*	0.041
Source-Drinking Water						
Piped (RC)			1.000		1.000	

Well	1.054	0.048	1.085	0.055
Lake, Pond, Stream	1.099*	0.045	1.096*	0.050
Type of Toilet Facility				
Flush-own/shared (RC)	1.000		1.000	
Trad. Pit Latrine	0.946	0.058	0.902	0.064
VIP	0.912	0.070	0.822*	0.073
Other	0.900	0.066	0.886	0.074
HWI				
Upper (RC)	1.000		1.000	
Lower	0.970	0.045	0.962	0.049
Middle	0.979	0.041	0.962	0.043
Maternal age at child birth				
20-34 years (RC)			1.000	
<20 years			1.093	0.049
35-49 years			1.014	0.043
Prec. Birth Interval				
37 months+ (RC)			1.000	
<24 months			1.021	0.043
20-36 months			1.088	0.043
Baby Size at birth				
Small (RC)			1.000	
Av./large			1.097*	0.047
Breastfeeding period				
10-19 months (RC)			1.000	
<10 months			0.962	0.035
>20+ months			0.944	0.038
Place of Delivery				
GoK facility (RC)			1.000	
Home			0.914*	0.035
Private Health Facility			1.041	0.038
Had Tetanus Injection				
No (RC)			1.000	
Yes			1.192**	0.062
Model Parameters				
Log Likelihood	-8933.9	-6172.1	-4779.9	
Ln_p	0.403	0.433	0.440	
P	1.500	1.542	1.553	
1/p	0.668	0.649	0.644	

Notes * refers to p -value < 0.05 , ** p -value < 0.01 ; TR = time ratio, HWI = household wealth index; RC = reference category.

Infants belonging to households with piped water were not in any way different from those who obtained their drinking water from the well (p value > 0.05). However, those

who drew water from the lake, pond or stream enjoyed a 10 percent increased chances of surviving much longer as compared to those who drew piped water and this association was statistically significant ($p\text{-value} < 0.05$).

The relationship between the dummies of type of toilet facility are in the expected direction. Generally, infants belonging to households that use flush toilets experience a lower risk of experiencing mortality as compared to those who use other forms of facilities. However it is only infants that belong to households that used ventilated improved pit latrines (VIP) that were significantly associated with 18 percent increased chances of dying faster as compared to those who belonged to households with flush toilets.

As expected, belonging to households in the upper class was associated with a lower risk of dying faster as compared to those who belonged to households from middle or lower socio-economic status. However, this association was not statistically significant ($p\text{-value} > 0.05$).

None of the dummies for maternal age at child birth, preceding birth interval and duration of breastfeeding was significantly associated with the risk of dying in infancy ($p\text{-value} > 0.05$). However, as compared to babies who were small during child birth, those who were born with average or large body sizes during birth, were 10 percent more likely to survive longer ($p\text{-value} < 0.05$).

Similarly, as compared to infants that were born in a government health facility, those who were born at home were 9 percent more likely to die faster and this association was statistically significant ($p\text{-value}<0.05$). On the other hand infants belonging to mothers who received at least a tetanus injection during pregnancy were 19 percent more likely to survive longer as compared to those whose mothers did not ($p\text{-value}<0.05$).

One clear issue from this model is that generally, the risk of dying in infancy appeared to increase throughout the period as seen from the values of the Weibull AFT ancillary parameter p . In particular, in model I when time was considered as the only variable, the risk of experiencing infant mortality increased by 50 percent. When background factors were considered, the increased risk dying was 54 percent and when the full model was assessed, the increase in infant mortality during the study period stood at 55 percent.

5.4 Effect of the selected covariates on child mortality

Results presented in Table 5.2 show that when time was considered as the only variable (Model I), the risk of dying faster in childhood stood during the 1992-98 and 1999-2003 was 33 and 12 percent respectively as compared to the risk of dying during the 1988-92 period ($p\text{-value}<0.05$).

In model II, the period variable remained an important explanatory variable with children born during 1993-98 and 1999-2003 more likely to experience 34 percent and 13 percent increased chances of dying much faster as compared to those born during 1988-1992 period ($p\text{ value}<0.05$).

However, maternal education, paternal education and source of drinking water were not significantly associated with the risk of dying in childhood ($p\text{-value} > 0.05$). On the contrary, as compared to children who belong to households that used flush toilets, those that used other means of faecal disposal including the bush were 9 percent more likely to die faster and this association was statistically significant ($p\text{-value} < 0.05$). Equally, children belonging to the lower socio-economic class experienced a 5 percent increased chances of dying faster as compared to those who belonged to upper socio-economic households and this association was statistically significant ($p\text{-value} < 0.05$).

Model III presents results of the full model. Time variable explained 45 and 28 percentage increase in the risk of dying faster during the 1993-98 and 1999-2003 as compared to the risk of dying during the 1988-1992 period ($p\text{-value} < 0.05$). The deterioration in economic growth after 1988 through 2003 may have decreased the household level spending on health leading to deterioration in child health.

On the other hand, paternal and maternal education, source of drinking water, type of toilet facility, household wealth index, size of the baby at birth and whether or not the mother received at least a tetanus toxoid during pregnancy were not statistically important in explaining the risk of dying in childhood ($p\text{-value} > 0.05$).

While there was no statistical association between the risk of dying among children belonging to mothers in the prime age (20-34 years) as compared to those belonging to mothers who were below 20 years, the association was in the expected direction.

However, as compared to children belonging to mothers in the prime age (20-34 years), those belonging to mothers who were 35 years and above were 9 percent more likely to live longer and this association was statistically significant ($p\text{-value} < 0.05$). Results from Table 5.2 also show that children whose preceding birth interval was less than 24 months were almost 5 percent more likely to survive longer as compared to those whose preceding birth interval was at least 37 months ($p\text{-value} < 0.05$).

Likewise, as compared to children belonging to mothers who breastfed for less than 10 months, children who breastfed for 10-19 months had 5 percent increased chances of surviving longer and this association was statistically significant ($p\text{-value} < 0.05$). However, breastfeeding for at least 20 months was not in any way significantly associated with the risk of dying in childhood as compared to breastfeeding for less than 10 months ($p\text{-value} > 0.05$).

Table 5.2: Time ratios for selected factors associated with changing Child mortality: KDHS, 1988-2003

Characteristic	Model I		Model II		Model III	
	TR	S.E(β)	TR	S.E(β)	TR	S.E(β)
Time period						
88-92 (RC)	1.000		1.000		1.000	
93-98	0.773**	0.013	0.655**	0.012	0.554**	0.011
1999-03	0.875**	0.013	0.867**	0.017	0.716**	0.020
Maternal Education						
Sec+ (RC)			1.000		1.000	
None			1.034	0.031	1.006	0.035
Primary			1.016	0.021	1.005	0.026
Paternal Education						
Sec+ (RC)			1.000		1.000	
None			1.045	0.034	1.024	0.037
Primary			1.007	0.018	1.005	0.022
Source-Drinking Water						
Piped (RC)			1.000		1.000	
Well			1.014	0.025	0.979	0.028

Lake, Pond, Stream	1.034	0.022	0.996	0.026
Type of Toilet Facility				
Flush-own/shared (RC)	1.000		1.000	
Trad. Pit Latrine	0.950	0.030	0.957	0.038
VIP	0.982	0.039	0.991	0.051
Other	0.910*	0.035	0.941	0.045
HWI				
Upper (RC)	1.000		1.000	
Lower	0.950*	0.022	0.971	0.028
Middle	0.994	0.021	1.012	0.026
Maternal age at child birth				
20-34 years (RC)			1.000	
<20 years			0.964	0.041
35-49 years			1.091**	0.027
Prec. Birth Interval				
37 months+ (RC)			1.000	
<24 months			1.045*	0.024
20-36 months			1.021	0.022
Baby Size at birth				
Av./large (RC)			1.000	
Small			1.013	0.026
Breastfeeding period				
<10 months			1.000	
10-19 months (RC)			1.051**	0.024
>20+ months			1.021	0.031
Place of Delivery				
GoK facility (RC)			1.000	
Home			0.978	0.020
Private Health Facility			1.082*	0.033
Had Tetanus Injection				
Yes (RC)			1.000	
No			1.037	0.033
Model Parameters				
Log Likelihood	-11224.8	-7877.1	-5630.1	
Ln_p	0.448	0.476	0.485	
P	1.566	1.610	1.624	
1/p	0.639	0.621	0.615	

Notes *refers to p -value < 0.05 at 95 percent Confidence Interval; ** p -value < 0.01;

RC=reference category

Source: primary analysis of merged KDHS: 1993-2003

As expected, being born at home carried a higher chance of diminished survival in childhood although this association was not statistically significant (p value > 0.05) when

compared with those born in a government health facility. However, as compared to children who were born in a government facility, those who were born in a private health facility had 8 percent increased chances of surviving longer and this association was statistically significant ($p\text{-value} < 0.05$). This could be due to the mass departure of medical doctors and other middle level health officers especially in rural areas from government health facilities to the private sector and elsewhere during the study period. Kimalu *et al.*, (2004:32) note the heavy staffing of low cadre staff in government health facilities but the rapidly diminishing number of middle and high professionals in government health facilities especially in rural areas where majority of Kenya live as others leave for private practice as evidenced by the mushrooming private health facilities.

Normally, mothers are given a tetanus toxoid jab during pregnancy at least once to prevent tetanus infection to the mother and the baby which can be deadly to the child especially during the first two weeks of life. Results however show that there appears to be no association between whether the mother received tetanus injection during pregnancy or not and the risk of dying in childhood ($p\text{-value} > 0.05$).

An assessment of model parameters as shown in Table 5.2 reveal that the Weibull AFT ancillary parameter p showed a positive time dependence. This implies that the risk of child mortality increased with time during the study period. In particular, there was an increase of 57 percent in child mortality rate when time was considered as the only variable and 61 percent increase when the time variable was adjusted background factors

as shown in model II. Finally, when the full model was considered, the model accounted for 62 percent increase in child mortality during the study period.

5.6 Concluding Remarks

The results show that the period played a critical role in the upsurge in infant and child mortality and remained significant all through. As compared to those who were born during the 1988-92 period, those who were born during the 1993-98 and 1999-2003 were more likely to die faster and this association was statistically significant. During infancy, paternal education, type of toilet facility, source of drinking water, type of place of delivery, size of the baby at birth and whether or not the mother received tetanus toxoid were important explanatory factors behind the upsurge in infant mortality. In terms of child mortality, maternal age, study period, preceding birth interval, duration of breastfeeding and place of delivery were important explanatory factors.

As expected, the effect of shorter birth intervals and duration of breastfeeding on infant and child mortality were in the expected direction though not statistically significant ($p\text{-value} > 0.05$). Unlike most past studies, paternal education appeared to be an important determinant of infant mortality in this study and not maternal education as is conventionally believed. It is likely that although the introduction of structural adjustment programs (SAPS) in the late 1980s which was accompanied with cost sharing in education may have increased the cost of educating girls, the full effect of SAPS on mortality is likely to have occurred beyond the study period when the girls who may have been adversely affected by the cost sharing in education become mothers.

CHAPTER 6: RESULTS OF REGRESSION DECOMPOSITION

The prolonged survival among infants from households that drew water from the lake, pond or stream as compared to households who were using piped water could be due to the positive effect of intentional use of water purification techniques among other sources of drinking water besides the piped water. The late 1990s in particular saw concerted efforts on water purification especially through the social marketing of water treatment chemicals for households in diarrhoea endemic regions that more often than not accessed water from other sources besides piped water and hardly boiled the water prior to drinking (Makutsa *et al.*, 2001).

With such widespread programs in place, the source of water supply increasingly becomes a less important determinant of mortality as seen from the results from child mortality. Finally, the Weibull AFT ancillary parameter (p) showed a positive time dependence which implies an upsurge in infant and child mortality during the 1988-2003 study period.

CHAPTER 6: RESULTS OF REGRESSION DECOMPOSITION TECHNIQUES

6.1 Introduction

In this chapter, regression decomposition techniques were used. Basically, when variables change over time such as one would expect for variables such as source of drinking water, type of toilet facility or place of delivery among others, an important statistical assumption of survival models is violated- that the event (in this case the chance of experiencing infant or child mortality) occurred at the time the selected factors were operating.

Preston (1976) demonstrated that when demographic events (such as infant mortality) change, it is not only the factors that determine such events that could be changing over time and space but rather, the events themselves could be changing at the same time. This phenomenon makes traditional statistical techniques inappropriate because most of them assume that the dependant variable is constant. Regression decomposition techniques appear appropriate in such situations and have been widely used to study changes in mortality (Kintner 1988; Da Vanzo and Habicht 1986; Peterson *et al.*, 1986; Hale *et al.*, 2009; Ikamari 1996; 2000).

This study extended these techniques to assess the correlates behind the upsurge in infant and child mortality between the two periods (i.e. 1988-1995 and 1996-2003) and between regions with high and low HIV prevalence. The key questions we attempt to address using these techniques are: Supposing we assumed that mortality did not change on its own, what factors could have accounted for the upturn in infant and child mortality in

Kenya during the 1988-2003 period? If we assumed that apart from the covariates, mortality changed as well, what was the structure of the relationship between mortality and each explanatory variable in each period?

6.2 Analytic Approach

In order to address the preceding questions, this chapter decomposed the differences in the observed rates of infant and child mortality in the two periods of 1988-1995 and 1996-2003 into components attributable to group differences in the characteristics and group differences in the effects of the covariates. To do this, all selected explanatory variables were converted into dichotomous variables as is conventionally done (Peterson, *et al.*, 1986; Da Vanzo and Habicht 1986; Ikamari 1996 and 2004:186).

The covariates were dichotomized based on theory and preliminary analysis to allow the variables' strongest effect on infant and child mortality. For instance, children belonging to mothers who were young (below 20 years) or old (above 35 years) are theoretically known to experience an elevated risk of infant and child mortality as compared to children born to mothers in the prime age (between 20-34 years) hence children of such high risk mothers were grouped together. Except for maternal and paternal education which are continuous, the rest of the variables were dichotomized as shown below:-

- a) Maternal age at birth (1=Below 20 years & above 35 years; 0= 20-34 years)
- b) Length of the preceding birth interval (0 < 24 months; 1 = > 24 months)

- c) Size of the baby at birth¹¹ (0=Small; 1=Normal/Big)
- d) Duration of breastfeeding (0= Less than 10 months; 1>10 Months)
- e) Source of water (1= Piped water; 0= Other)
- f) Presence of toilet facility (1= Yes; 0= No)
- g) Maternal education: Years of schooling
- h) Paternal education: Years of schooling
- i) Household wealth status¹² (1=Low; 0=High)
- j) Place of delivery (1 = Health facility; 0= Home)
- k) Had tetanus injection (1 =Yes; 0=No)
- l) Period (0 =1988-95; 1=1996/03)
- m) Malaria endemicity¹³ (0=Low; 1=High)
- n) Level of HIV prevalence¹⁴ (0=Low; 1=High)

The next step entailed computing means of these dichotomised variables for each period while assessing whether the means were statistically different using the t-test statistic.

The choice of whether to use a t-statistic with equal or unequal variance in assessing

¹¹Conventionally, infants born with a birth weight of less than 2500grams are categorized as low birth weight (LBW) and vice versa. This study did not use this variable due to the large number of missing cases (55% of the total sampled live births did not have recorded birth weight) as a result of low health facility deliveries in Kenya (only 42% of births were delivered in a health facility) and thus its use could have biased our results. Consequently, baby size at birth was used as a proxy measure for LBW. Recent studies based in Africa have confirmed a close correlation between size of the baby at birth and birth weight (Mbuagbaw, 2010).

¹² Household wealth index was computed using the principal component analysis (PCA) based on the household possessions of each sampled household. Those with low and medium income were categorized as low while those with high income were categorized as "high." In our preliminary analysis (presented in Chapter Four), we found that low and medium households experienced an upsurge in mortality while the high income households experienced a decline in mortality hence this rationale behind the proposed dichotomization.

¹³ The categorization of whether a region was low or high malaria endemicity was based on the categorization of Kenya Medical and Research Institute which was used in a study by K'Oyugi (1992). Accordingly, Nyanza, Western, Coast and North Eastern provinces were categorised as high malaria endemic regions while Central, Eastern, Nairobi and Rift valley were categorized as low malaria endemic regions. For consistency, we excluded North Eastern from our analysis since this region was not sampled prior to the 2003 KDHS.

¹⁴ In this study, we categorized regions with higher than national HIV prevalence (6.7%) as high HIV prevalence regions and vice versa (CBS *et al.*, 2004). Using this categorization, Nyanza (18.3%); Central (7.6%); Nairobi (11.9%); Rift Valley (6.9%) were grouped as high HIV prevalence while Western (5.8%); Coast (6.6%); Eastern (6.2%) and North Eastern (0.0%) were categorized as low HIV prevalence. For the purposes of consistency, we excluded North Eastern from our analysis since this region was not sampled prior to the 2003 KDHS. In any case, the region had a negligible HIV prevalence (almost 0%) and a very small sample size (CBS, *et al.*, 2004).

whether the two sets of groups were statistically different is controversial in statistical literature. However, the general consensus is that if the standard deviations of the two groups are similar and the sample sizes of the two groups are large¹⁵, then the options of using equal or unequal variance will yield close and marginally inconsequential differences in the estimated t-statistic values (Ruxton 2006; Dupont 2009:38).

In other words, the loss in statistical power of not assuming that the two samples have equal standard deviations or variance becomes trivial (Dupont 2009). This study equally found trivial differences when the t-test with equal or unequal variance was applied to the available data (not shown).

Once the means of the explanatory variables for the samples are computed, it is usually important to establish the structure of the relationship between each explanatory variable and mortality in each time period, or between high and low mortality regions as is the case in this study. In past studies, this has been accomplished by running logistic regression to obtain the logit coefficients or even ordinary least squares (OLS) and the associated standard errors of each explanatory variable in the two regions or time periods (Da Vanzo 1986; Peterson, *et al.*, 1986; Kintner 1994; Ikamari 1996, 2004).

In addition, a test of significance of the difference between the logit coefficients of each explanatory variable in the two mortality regions or periods has been conventionally computed to gauge whether they are significantly different (Peterson *et al.*, 1986; Ikamari

¹⁵ The t-distribution tends to be normal for large sample sizes (i.e. where $n > 30$) and hence in such cases, concerns about unequal variance and non normality of the samples tends to be trivial (Ruxton 2006).

1996, 2004). The logit coefficients are then used to account for the relative contribution of the differences in the structure of the relationship in explaining the difference in the level of infant and child mortality between the two periods or mortality regions.

The critical question in using logit coefficients to predict the role of each explanatory variables to the observed level of early childhood mortality in two mortality regions or periods is: *Ceteris paribus*, what level of infant and child mortality would we have, say in the later time period (1996-2003) if a given explanatory variable in the initial period (1988-1995) had the same mean value as in the later period (1996-2003)? This is a counterfactual analysis since the question is repeated for each and every explanatory variable in assessing its contribution to the change in the dependent variable for each period or mortality region. This is illustrated using the example below:-

Assuming we would like to know the contribution of any covariate, say, household wealth Index (HWI) on infant mortality (IMR) in 1988-1995 period had this variable values been applied to the 1996-2003 period, we would have to use the following formula:-

$$\Delta \text{ in IMR}_0 = \beta \text{ HWI}_0 * \text{IMR}_0 * (1 - \text{IMR}_0) * (\text{HWI}_0 - \text{HWI}_1) * 1000$$

Where

$\Delta \text{ in IMR}_0$ = change in infant mortality rate for the 1988-1995 period

$\beta \text{ HWI}_0$ = the regression coefficient of HWI for the 1988-1995 period

HWI_0 = Mean value of the Household wealth Index for the 1988-1995 period

$\beta \text{ HWI}_1$ = the regression coefficient of HWI for the 1996-2003 period

HWI_1 = Mean value of the Household wealth Index for the 1996-2003 period

In the above formula, the equation was multiplied by 1000 to assess the contribution of the household wealth index to infant mortality per 1000 live births. In this study, the Cox proportional hazard regression was used in computing regression coefficients instead of logistic regression. This is because statistically, both logistic regression and Cox proportional hazards models yield similar estimates of regression coefficients in studies with short follow-up period and low incidence of event occurrence (Armitage *et al.*, 2002) as is the case in this study.

In addition, Cox regression unlike logistic regression accounts for censoring by ensuring that children who were born more recently who may not be exposed to the full risk of dying are included in the analysis hence eliminating mortality measurement biases. Further, some recent studies have used hazard rate models in decomposing group differences in effects (Hale *et al.*, 2006; Hale *et al.*, 2009; Powers and Yun, 2009).

Mathematically, the Cox regression model was presented and discussed in Chapter three of this thesis. A detailed discussion of the use of Cox regression can be found in any standard biostatistics textbook (see Armitage, *et al.*, 2002; Collett 2003; Fisher and Belle 2004; Dupont 2009).

In the next section, the study presents results on the observed rates in infant mortality among the two periods. To achieve this, the mean values of the explanatory variables between the two study periods were computed as well as the t-statistic to assess whether the mean differences in the explanatory variables were statistically significant. Generally,

explanatory variables that were statistically significant (i.e. where $t\text{-statistic} > 1.96$) implies that those particular variables partly accounted for the observed upsurge in infant mortality between the two periods and vice versa.

6.3 Results of decomposition analysis

6.3.1 Explanatory variables of infant mortality between the time periods

In Table 6.1 below, columns (a) and (b) show the actual changes in the covariates that took place during the 1988-1995 and 1996-2003 period. The last column (c) presents the t -statistics indicating whether the differences were significantly different from zero. Results show that duration of breastfeeding, maternal education, HIV prevalence and malaria endemicity were important explanatory variables responsible for the differences in infant mortality between the two periods.

Over the time span, duration of breastfeeding and maternal education increased. These two variables could potentially result to a decline in infant mortality as is evidenced in past literature (Caldwell 1979; Hobcraft, *et al.*, 1984; K'Oyugi 1992; Ikamari 1996). However, there was a significant rise in the means of regional HIV status and malaria endemicity both of which could have potentially contributed to the upsurge in infant mortality during the study period. The actual rise in malaria endemicity was 18 percent higher $[100 - (0.345/0.421) * 100]$ while regional HIV prevalence was 5 percent higher during the 1996-2003 period as compared to the values observed during the 1988-1995 period.

Past studies have also pointed out the emergence of malaria resistant strains and the rise in HIV prevalence as key causes for the upturn in infant and child mortality in some sub Saharan African countries (Hill *et al*, 2001; 2004; Rutstein 2000; Omariba 2005). The rest of the variables were not statistically different between the two time periods (t-statistic<1.96). However, as expected, most variables showed an increase in the means during the 1988-1995 and 1996-2003 period which reflects that they may have contributed to the differences in infant mortality during the two periods under this study. The results from Table 6.1 imply that the observed changes in infant mortality in Kenya during the 1988-95 and 1996-2003 periods could be partly accounted for by the four variables whose means were statistically different between the two periods.

Table 6.1: Means of explanatory variables for infant mortality by time period:
Kenya, 1988-2003

Characteristic	1988-95 (n=3115) (a)	1996-03 (n=2150) (b)	differences in means (t-statistic)
Maternal factors			
Maternal age at child birth (D)	1.672	1.688	-0.951
Baby size at birth (D)	0.818	0.816	0.175
Duration of breastfeeding (D)	0.810	0.908	-7.525**
Preceding birth interval (D)	0.699	0.736	-1.93
Maternal health-seeking factors			
Place of delivery (D)	0.624	0.593	1.43
Tetanus Toxoid (D)	0.883	0.867	1.297
Socio-economic factors			
Paternal education (years)	5.471	5.789	-1.141
Source of water (D)	0.268	0.299	-1.746
Type of Toilet Facility (D)	0.795	0.812	-1.145
Maternal education (Years)	5.149	5.490	-4.068**
Household Wealth Index (D)	0.558	0.580	-1.92
Community level factors			
HIV regional status (D)	1.364	1.429	3.388**
Malaria endemic region (D)	0.345	0.421	-4.344**

Notes: D denotes dichotomous variable. **p<0.001.

Source: Primary analysis of the merged KDHS dataset 1988/89 and 1997/98

Section 6.4, assesses the role of the explanatory variables in relation to the rising levels of infant mortality between 1988-95 and 1996-2003 periods before and after other covariates were controlled for.

6.4 Covariate contribution to infant mortality variation during 1988-2003 Period

Table 6.2 shows that in Model I the risk of dying in infancy during 1996/2003 period was 19 percent higher as compared to dying in infancy during the 1988/92 time period. When maternal factors were added to the period variable (Model II), the risk of dying in infancy during the 1996/2003 was not in any way statistically different as that observed during the 1988-1995 period (t-test <1.96). However, when socio-economic factors were added to Model I (Model III), the risk of dying in infancy was 23 percent higher during the 1996/2003 as compared to the 1988-1995 period and this increase was statistically significant ($t > 1.96$).

Table 6.2: Hazard Ratios and likelihood chi-square values indicating the effects of controlling for explanatory variables on infant mortality differential between the two time periods, merged KDHS 1993-2003

Explanatory variable	Model I	Model II	Model III	Model IV	Model V	Model VI
LR χ^2	7.18	33.07	64.3	38.19	110.59	102.57
d.f.	1	1	1	1	1	1
P-value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
Time period						
1988-95	1.0	1.0	1.0	1.0	1.0	1.0
1996-2003	1.19 (2.69)*	1.03 (0.35)	1.23 (2.42)*	1.10 (5.17)*	1.28 (2.46)*	1.38 (2.41)*

Notes: Model I= Time period indicator only, Model II= Model I plus all maternal factors, Model III=Model I plus all socio-economic factors (SES); Model IV= Model I plus all maternal health seeking factors; Model V= Model I plus community level factors and Model VI= Model I plus all other models. T-statistics are shown in the parentheses; *significant at p-value<0.05. The LR χ^2 values refer to those associated with the time period indicator variable

Source: Primary analysis of pooled KDHS Datasets, 1993, 1998 and 2003

In Model IV, maternal health seeking factors were added to the time variable. Results showed that the risk of dying in infancy was 10 percent higher during the 1996-2003 period as compared to the 1988-1995 period and this increase was statistically significant at 95 percent confidence interval.

Similarly, in Model V where community level factors (regional HIV prevalence and regional malaria endemicity) were added to Model I, results showed that being born during the 1996/2003 era was associated with 28 per cent increased risk of dying during infancy as compared to being born during the 1988-1995 time period and this was statistically significant ($p < 0.05$). Finally, when all the variables were added to Model I as shown in Model VI above, the risk of dying during infancy in the 1996-2003 period was 38 percent higher as compared to the 1988-1995 period and this was statistically significant.

When all explanatory variables were added to Model I, the likelihood ratio test satisfied $p < 0.001$ for all the models which implies that the explanatory power of the additional variables more than compensated for the loss in degrees of freedom. Generally, the results presented in Table 6.2 above imply that the differences in the values of explanatory variables, taken together, during the two periods contributed to differences in the observed infant mortality. The above analysis however is a fixed structure analysis since it assumes that only the covariates are bound to change and not infant mortality. In the next section, we assess the contribution of explanatory variables taken together in accounting for the observed differences in infant mortality between the two time periods.

6.5 Structure of the relationship between infant mortality and explanatory variables

As a follow up to the discussion in the previous section, it is important to ascertain whether there were any structural differences in the effects of the explanatory variables vis a vis infant mortality in the two periods under analysis. As earlier presented and discussed in Table 6.2, the differences in a number of covariates between the two periods imply that the nature of the structural relationship could also differ as evidenced in past studies (Preston 1976; Da Vanzo and Habicht 1986; Ikamari 1996, 2004). Results from Table 6.3 shows that apart from maternal age at child birth, having received a tetanus toxoid during pregnancy, and household wealth index, the effect of the rest of the explanatory variables were significantly different during the 1988-1995 and 1996-2003 periods (t -statistic > 1.96).

A detailed analysis shows that the size of the baby at birth had a significant but negative effect on infant mortality in both periods. The effect of the baby size at birth was however much greater during the 1996-2003 period as compared to its effect during the 1988-1995 period. Other explanatory variables that exhibited a negative effect on infant mortality in the two periods were the preceding birth interval and household wealth index both of which showed a significant negative effect on infant mortality during the 1988-1995 period and not in the later period (1996-2003). On the contrary, malaria endemicity exhibited a negative significant effect on infant mortality in the two periods. The negative effect of malaria endemicity on infant mortality was particularly more pronounced during 1988-1995 as compared 1996-2003 period.

Duration of breastfeeding and regional HIV rates had a positive significant effect on infant mortality in both periods. The effect of duration of breastfeeding was however greater during the 1996-2003 than the 1988-1995 period. Likewise, the effect of HIV seemed to have been more pronounced during the 1988-1995 period as compared to the 1996-2003 period. Its reduced effect in the later period is likely due to the introduction of HIV interventions which saw the peak in HIV prevalence (of about 13.2 percent) dropping after 2000 to 6.7 percent by 2003 (CBS, *et al.*, 2004).

Table 6.3: Regressions explaining infant mortality between 1988-1995 and 1996/2003, allowing for different coefficients between the time periods

Characteristic	Coefficients (1988-95) (n=3115) (a)	Coefficients (1996-03) (n=2150) (b)	t-statistic if (a) and (b) differ (c)
Maternal factors			
Maternal age at child birth (D)	-0.430 (-1.02)	0.035 (0.08)	-0.114
Baby size at birth (D)	-0.456 (-2.01)***	-0.367 (-2.40)***	5.897***
Duration of breastfeeding (D)	1.032 (5.82)**	4.179 (8.12)***	6.969***
Preceding birth interval (D)	-0.402 (-2.29)**	-0.589 (-1.72)	6.094***
Maternal health-seeking factors			
Place of delivery (D)	0.228 (1.16)	0.103 (0.47)	2.624*
Tetanus Toxoid (D)	-0.423 (-1.56)	-0.129 (-0.40)	0.864
Socio-economic factors			
Paternal education (years)	-0.052 (-1.19)	0.015(0.77)	-64.717***
Source of water (D)	0.066 (0.260)	-0.392(-1.30)	-19.901***
Type of Toilet Facility (D)	-0.399 (-1.85)	0.243(0.82)	-2.316**
Maternal education (Years)	-0.040 (-0.94)	-0.017(-0.38)	56.965***
Household Wealth Index (D)	-0.497(-2.37)**	-0.167 (-0.72)	1.757
Community level factors			
HIV regional status (D)	1.082 (5.15)***	0.933 (3.86)***	4.289***
Malaria endemic region (D)	-1.265(-5.06)***	-1.332 (-4.78)***	3.831***

Notes: D denotes dichotomous variable. ***p<0.001, **p<0.01, *p<0.05. Column C was computed using the following formula: $(P_2 - P_1) / (1.96 \text{SQRT}(X_1 + X_2))$ where P_2 & P_1 are the coefficients of the two time periods and X_1 & X_2 are the standard errors for each explanatory variable for the two time periods

Source: Primary analysis of the merged KDHS dataset 1993-2003

The results also showed that there were statistically significant differences in the coefficients of size of the baby at birth, duration of breastfeeding, preceding birth interval, maternal education, regional HIV status, regional malaria endemicity, paternal education, source of drinking water and type of toilet facility and maternal education on infant mortality in the two time periods, (t-statistic > 1.96).

While the source of drinking water supply, place of delivery, maternal and paternal education and type of toilet facility had insignificant effect on infant mortality in both the two time periods, the difference in their effect in the two time periods was statistically significant at the 5 percent level.

On the overall, the effect of paternal education, source of water and type of toilet facility between the two time periods was significant but negative which implies they contributed to the decline in infant mortality. The rest of the variables that were significant between the two periods were positive (which implies that they directly contributed to the upsurge in infant mortality). The question that arises thus is to what extent did each of the explanatory variables contribute either to the rise or decline in infant mortality during the two periods? This is a counterfactual analysis and is examined in the next section below.

6.6 What would be the rate of infant mortality during the 1988-95 period had infants born during that time experienced the same values as those of the 1996-2003 period and vice versa?

In Table 6.4, the length of breastfeeding, maternal education, the regional level of HIV prevalence and regional Malaria prevalence appeared to account for the largest share of

the differences in mortality between the two periods. Had children belonging to mothers in 1988-1995 period had the same mean values of duration of breast feeding as those of children belonging to mothers in the 1996-2003 period, infant mortality would have reduced by 5 deaths per 1000 live births. However, had children belonging to the 1996-2003 period experienced the same mean values of duration of breastfeeding as those belonging to the 1988-1995 period, infant mortality could have increased by a massive 19 deaths per 1000 live births.

However, had children belonging to the 1996-2003 period experienced the same mean values of malaria endemicity as those of children belonging to the 1988-1995 period, then infant mortality would have reduced by 7 deaths per 1000 live births. On the contrary, had children belonging to the 1988-1995 period experienced the same mean values of malaria endemicity as those belonging to the 1996-2003 period, infant mortality would have increased by 5 deaths per 1000 live births.

Results also show that had infants for 1996-2003 period experienced the same mean values of high HIV region as those of the 1988-1995 period, then infant mortality would have increased by 4 deaths per 1000 live births. However, had infants belonging to the 1988-1995 period experienced the same mean values of HIV region as those belonging to the 1996-2003 period, then infant mortality could have reduced by 3 deaths per 1000 live births. Likewise, had infants belonging to the 1988-1995 period experienced the same mean values of maternal education as those belonging to the 1996-2003 period, then

infant mortality could have increased by 1 death per 1000 live births. The inverse could have resulted in a negligible decrease in infant mortality.

The results generally seem to indicate that if the rest of the explanatory variables for the 1996-2003 period had values as those of 1988-1995 period, then infant mortality rate in the 1996-2003 period could have remained largely unaltered and vice versa. Generally, the conditional nature of each of the variables considered in this analysis makes it difficult to assess their actual roles to the changes in mortality. For instance, some of the effect of maternal education on infant mortality is subsumed by other correlates such as household wealth index, place of delivery and type of toilet facility among others. It is therefore important to look at the overall effect of these variables on infant mortality at their mean value.

Table 6.4 shows that had all infants in the 1996-2003 period experienced the same mean values of all explanatory variables as those of the 1988-1995 period, then infant mortality would have increased by 14 deaths/1000 live births. However, had all the infants in the 1988-1995 period experienced the same mean values of all explanatory variables as those of 1996-2003 period, then the rise in infant mortality could have been negligible (less than 1 death per 1000 live births).

Table 6.4: Changes in infant mortality rate had each time period experienced the same value of explanatory variables

Characteristic	Mean values 1988-95	Mean values 1996-03	β 's for 1988-95	β 's for 1996-03	Change in IMR if 1988-1995 had the same mean value of this variable as 1996-2003 (e)	Change in IMR if 1996- 2003 had the same mean value of this variable as 1988-1995
	(a)	(b)	(c)	(d)		(f)
Maternal factors						
Maternal age (D)	1.672	1.688	0.011	-0.077	-0.010	-0.080
Baby size (D)	0.818	0.816	-0.122	-0.0568	-0.014	0.007
Duration of breastfeeding (D)	0.81	0.908	0.858	3.053	-4.838	19.375
Preceding birth interval (D)	0.699	0.736	-0.294	-0.373	0.626	-0.894
Maternal health seeking behaviour						
Place of delivery (D)	0.624	0.593	0.284	0.207	0.507	-0.416
Tetanus Toxoid (D)	0.883	0.867	-0.251	-0.174	-0.231	0.180
Socio-economic factors						
Paternal education (years)	5.471	5.789	0.005	0.011	-0.091	0.227
Source of water (D)	0.268	0.299	0.075	-0.31	-0.134	-0.622
Type of Toilet Facility (D)	0.795	0.812	-0.399	0.082	0.390	0.090
Maternal education (Years)	5.149	5.49	-0.057	-0.019	1.118	-0.420
Household Wealth Index (D)	0.558	0.58	-0.373	-0.177	0.472	-0.252
Community level factors						
HIV regional status (D)	1.364	1.429	0.829	0.899	-3.101	3.784
Malaria endemic region (D)	0.345	0.421	-1.21	-1.405	5.292	-6.915
					0.112	14.159
IMR	0.0613	0.0696				
P*(1-P)	0.0575	0.0648				

Notes: D denotes dichotomous variable.

Source: Primary analysis of the merged KDHS dataset 1993-2003

6.7 Mean values of explanatory variables of child mortality

Unlike at infancy, maternal factors appear to take less precedence to socio-economic and health seeking factors in explaining the differences in child mortality between the two periods. For instance, apart from preceding birth interval, none of the rest of maternal factors considered in this study accounted for the actual changes in child mortality rates between the 1988-1995 and 1996-2003 periods. Table 6.5 below shows that place of delivery, whether or not the mother had received a tetanus toxoid, source of drinking water, type of toilet facility, maternal education and regional malaria endemicity were all significantly associated with the differences in child mortality between the two time periods ($t > 1.96$).

In particular, the decrease in the number of children belonging to mothers receiving at least one tetanus jab during pregnancy, the decline in the values of type of toilet facility and the increase in the value of malaria endemicity during the 1988-1995 and 1996-2003 could have potentially contributed to the upsurge in child mortality. However, the increase in the values of the preceding birth interval, place of delivery, source of drinking water and maternal education are bound to have suppressed the rapid rise in child mortality during the 1988-1995 and 1996-2003 period.

Table 6.5: Means of explanatory variables of child mortality between 1988-1995 and 1996-2003: KDHS 1993-2003

Characteristic	Time period 1988-95 (n=4650)	Time period 1996-03 (n=4872)	Significance of Differences in Means (t-statistic)
Maternal factors			
Maternal age at child birth (D)	1.688	1.689	-0.085
Baby size at birth (D)	0.860	0.854	0.835
Duration of breastfeeding (D)	0.144	0.113	0.224
Preceding birth interval (D)	0.690	0.730	-3.659**
Maternal health-seeking behaviour			
Place of delivery (D)	0.550	0.577	-2.487**
Tetanus Toxoid (D)	0.926	0.895	4.508**
Socio-economic factors			
Paternal education (years)	5.561	5.595	-0.234
Source of water (D)	0.281	0.318	-3.273**
Type of Toilet Facility (D)	0.835	0.806	3.598**
Maternal education (Years)	5.100	5.56	-9.221**
Household Wealth Index (D)	0.606	0.591	1.439
Community level factors			
HIV regional status (D)	1.340	1.329	1.015
Malaria endemic region (D)	0.410	0.446	-3.369**

Notes: D denotes dichotomous variable. **p<0.001

Source: Primary analysis of the merged KDHS dataset 1988/89 and 1998/99

In the next section, we discuss the role of selected covariates on the upsurge in child mortality.

6.8 Effects of explanatory variables on child mortality

Table 6.6 below shows the effect of controlling different sets of explanatory variables on child mortality between the two time periods. Generally, some of the models (e.g. Models I and II) were not statistically significant implying that the differences in the values of explanatory variables, either as individual sets or taken together for the two periods contributed to differences in the observed child mortality.

Table 6.6: Hazard Ratios and likelihood chi-square values indicating the effects of controlling for explanatory variables on child mortality differentials between the two periods: KDHS 1993-2003

Explanatory variable	Model I	Model II	Model III	Model IV	Model V	Model VI
LR χ^2	0.04	9.16	20.31	12.44	48.48	28.83
d.f.	1	1	1	1	1	1
P-value	> 0.05	> 0.05	< 0.05	< 0.05	< 0.001	< 0.05
Time period						
1988-95	1.0	1.0	1.0	1.0	1.0	1.0
1996-2003	0.97 (-0.21)	0.90 (-0.57)	0.88 (-0.56)	0.98 (-0.10)	1.02 (0.13)	1.00 (-0.01)

Notes: Model I= Time period indicator only, Model II= Model I plus all maternal factors, Model III=Model I plus all socio-economic factors (SES); Model IV= Model I plus all maternal health seeking factors; Model V= Model I plus community level factors and Model VI= Model I plus all other models. T-statistics are shown in the parentheses; The LR χ^2 values refer to those associated with the time period indicator variable

Source: Primary analysis of the pooled KDHS dataset

Below, we present results of the effect of explanatory variables on child mortality in the two periods. Maternal age at birth had a positive effect on child mortality in the 1988-1995 period but had no statistical significance on child mortality in the 1996-2003 period. On the overall, maternal age at birth, place of delivery, maternal and paternal education significantly accounted for the difference in the effects of the covariates between the two periods.

In general, maternal and paternal education both had a negative effect on child mortality in both periods and appeared to explain a large portion of the observed upsurge in child mortality rates. The strong influence of education could be due to the fact that education exerts more influence on child mortality as compared to infant mortality. During the study period, education was adversely affected by the structural adjustment programmes which resulted in massive increase in school fees, a reduction in the quality of education

offered and a massive school dropout especially among girls (Mwega and Kukubo 1993) hence its effect on child mortality is expected.

Table 6.7: Coefficients of explanatory variables of child mortality between 1988-1995 and 1996/2003: KDHS 1993-2003

Characteristic	Coefficients For 1988-1995 (n=2366) (a)	Coefficients for 1996-03 (n=1314) (b)	Significance of differences in coefficients for 1988-1995 & 1996-2003 (t-statistic) (c)
Maternal factors			
Maternal age at child birth (D)	0.887 (1.96)**	0.150 (0.14)	2.134***
Baby size at birth (D)	1.107 (1.53)	-0.257 (-0.33)	-1.938
Duration of breastfeeding (D)	0.335 (0.75)	0.538 (0.68)	0.388
Preceding birth interval (D)	0.095 (0.29)	-0.915 (-1.60)	-0.122
Maternal health seeking factors			
Place of delivery (D)	0.493 (1.45)	0.095 (0.15)	5.051***
Tetanus Toxoid (D)	1.076 (1.06)	-0.168 (-0.21)	-1.950
Socio-economic factors			
Paternal education (years)	-0.040 (-0.54)	-0.057 (-0.44)	16.067***
Source of water (D)	-0.300 (-0.71)	0.612 (0.82)	-0.343
Type of Toilet Facility (D)	-0.312 (-0.77)	-1.123 (-1.66)	0.227
Maternal education (Years)	-0.084 (-1.15)	-0.033 (-0.26)	59.415***
Household Wealth Index (D)	-0.170 (-0.488)	-0.316 (-0.47)	0.476
Community level factors			
HIV regional status (D)	-0.014 (-0.037)	0.171 (0.25)	-0.067
Malaria endemic region (D)	-1.185 (-2.19)**	-1.246 (-1.47)	0.481

Notes: D denotes dichotomous variable. *** $p < 0.001$, ** $p < 0.01$, Column C was computed using the following formula: $(P_2 - P_1) / (1.96 \sqrt{X_1 + X_2})$ where P_2 & P_1 are the coefficients of the two time periods and X_1 & X_2 are the standard errors for each explanatory variable for the two time periods

Source: Primary analysis of the merged KDHS dataset 1993-2003

While malaria endemicity appeared to account for the statistical difference in child mortality during the 1988-1995 period, it was not significantly associated with the observed child mortality rates in the 1996-2003 period. In Kenya, malaria resistance was first detected in the 1980s and it peaked by 1997 (when resistance to chloroquine ranged from 66-87 percent). It was then (1997) that the government of Kenya changed the first

line treatment of malaria from chloroquine to artemisinin combination therapy (ACT) for treatment of uncomplicated malaria (WHO *et al.*, 2003; IAEA, 2007, Houwelling *et al.*, 2006).

Improvements in public health interventions including the establishment of the Division of Malaria Control (DoMC) in 2000 to serve as the control arm of the national malaria control programme as well as the roll out of the National Malaria Strategic Plan for 2001-2010 went along way in accelerating efforts for Malaria control (MoH, 2001). Further, the country has received external funding especially from the global fund to fight malaria. Consequently, it is expected for Malaria to have played a greater role in child mortality during the earlier years (1988-1995) as compared to the later years. The increase in the use of insecticide treated mosquito nets in the early 2000 is an additional factor behind the reduced role of malaria in the later period. For instance, while earlier rounds of KDHS did not collect data on ITN usage, the 2003 KDHS shows that initial campaigns to promote ITN usage saw the uptake of the nets at 4.4 and 4.6 percent among pregnant women and children under five by 2003 (KNBS, *et al.*, 2004).

Although the duration of breastfeeding, the length of the preceding birth interval and receiving tetanus toxoid during pregnancy were not significantly associated with mortality differences during the study periods, the nature of the results found in this study were unique and could be a result of problems associated with use of household characteristics in differential childhood mortality analysis. For instance the positive association in the values of these variables in the 1988-1995 period could be due to the

fact that some of the children could have died much earlier resulting to the observed positive association. Further, in generalized HIV prevalence regions such as Kenya, prolonged breastfeeding could be associated with increased mother to child transmission leading to higher deaths hence the positive association.

While source of drinking water was negatively associated with child mortality during the 1988-1995 period, it was positively associated with child mortality differences during the 1996-2003 period though in both, the relationship was not statistically significant ($p\text{-value} > 0.05$). This could be due to the fact that the increase in the water purification techniques during the later cohort could have eliminated source of water supply as an important factor in accounting for mortality differentials during the study period.

6.9 Explanatory variables versus structural relationships: A counterfactual analysis

Table 6.8 below presents detailed results of the decomposition between individual effects on child mortality differentials during the study period. The results show that malaria endemicity, maternal education and belonging to mothers who had received tetanus injections during pregnancy accounted for the largest portion of child mortality change during the 1988-1995 period. On the contrary, malaria endemicity, type of toilet facility and preceding birth interval explained much of the difference in child mortality during the 1996-2003 period.

If the values of malaria endemicity during the 1996-2003 were to have the same mean value as those for the 1988-1995, then child mortality in the later could have increased by

1.9 deaths per 1000 live births. However, if the values of malaria endemicity for the 1988-1995 were to have fixed mean values as those of the 1996-2003, then child mortality could have reduced by 1.7 deaths per 1000 live births. However, had children belonging to the 1996-2003 period experienced the same mean values of type of toilet facility as those of the 1988-1995 period, then child mortality would have increased by 1.4 deaths per 1000 live births. The inverse could have led to a reduction in child mortality by 0.4 deaths per 1000 live births.

Had children belonging to mothers who had received a tetanus jab during pregnancy in 1988-1995 experienced the same mean values as those of 1996-2003 period, then child mortality could have increased by 1.4 deaths per 1000 live births. The inverse could have led to a reduction in child mortality by 0.2 deaths per 1000 live births. While tetanus injection during pregnancy is only an imperfect proxy measure of immunization coverage, the results here echo DHS findings relating increased childhood mortality to deterioration in the overall immunization coverage (CBS, *et al.*, 2004).

The rest of the explanatory variables in both periods appear to have contributed to a negligible change to child mortality. For instance, had children belonging to the 1988-1995 period experienced the same mean values of all explanatory variables as those belonging to the 1996-2003 period, then child mortality would have increased by 6 deaths per 1000 live births. However, the inverse could have reduced child mortality by almost 2 deaths per 1000 live births.

Table 6.8: Changes in child mortality rate had each time period experienced the same value of explanatory variables

Characteristic	Means 1988-95	Means 1996-03	Coefficients 1988-95	Coefficients 1996-03	Change in CMR if 1988-95 had same mean of this variable as 1996-03	Change in CMR if 1996-03 had the same mean value of this variable as the 1988-95
Child level factors						
Maternal age at child birth (D)	1.688	1.689	0.887	0.15	-0.036	0.006
Baby size at birth (D)	0.86	0.854	1.107	-0.257	0.272	0.065
Duration of breastfeeding (D)	0.144	0.113	0.335	0.538	0.425	-0.706
Preceding birth interval (D)	0.69	0.73	0.095	-0.915	-0.156	-1.550
Maternal health-seeking behaviour						
Place of delivery (D)	0.55	0.577	0.493	0.095	-0.545	0.109
Tetanus Toxoid (D)	0.926	0.895	1.076	-0.168	1.367	0.220
Socio-economic factors						
Paternal education (years)	5.561	5.595	-0.04	-0.057	0.056	-0.082
Source of water (D)	0.281	0.318	-0.3	0.612	0.455	0.959
Type of Toilet Facility (D)	0.835	0.806	-0.312	-1.123	-0.371	1.379
Maternal education (Years)	5.1	5.56	-0.084	-0.033	1.583	-0.643
Household Wealth Index (D)	0.606	0.591	-0.17	-0.316	-0.104	0.201
Community level factors						
HIV regional status (D)	1.34	1.329	-0.014	0.171	-0.006	-0.080
Malaria endemic region (D)	0.41	0.446	-1.185	-1.246	1.748	-1.899
Sum					5.752	-1.815
Child Mortality	0.043	0.044				
$p(1-p) = \text{CMR} * (1 - \text{CMR})$	0.041	0.042				

Notes: D denotes dichotomous variable

Source: Primary analysis of the merged KDHS dataset 1993-2003

6.10 Concluding remarks

This chapter examined the factors behind the early childhood mortality differentials during 1988-2003 period. The results showed that on the overall, there was an upsurge in infant mortality during the 1996-2003 as compared to the 1988-1995 period. The duration of breastfeeding, maternal education, regional malaria endemicity and regional HIV prevalence were found to be important explanatory variables that accounted for the mean differences in infant mortality between the 1988-95 and 1996-2003 period.

Apart from maternal age at child birth, tetanus toxoid and household wealth index, the effect of the rest of the explanatory variables were significantly important in accounting for time differences in mortality during infancy. While the baby size at birth, duration of breastfeeding, preceding birth interval, place of delivery, maternal education, regional HIV prevalence and regional malaria endemicity accounted for the upsurge in infant mortality, paternal education, the source of water supply and the type of toilet facility appeared to have contributed to infant mortality decline during the 1988-1995 and 1996-2003 period.

On the other hand, the duration of breastfeeding, place of delivery, whether or not the mother received a tetanus toxoid during pregnancy, source of drinking water, type of toilet facility, maternal education, and whether the household lived in a malaria endemic region or not were accountable for the mean differences in child mortality between the two periods. In accounting for the differences in group effects, maternal age at child birth, place of delivery, paternal education and maternal education were important.

Results appeared to point out that preceding birth interval, having received a tetanus toxoid injection, maternal education and malaria prevalence were important explanatory variables that contributed to the changing child mortality during the study period.

The main research question was to investigate the factors that accounted for the upturn in infant and child mortality in Kenya during the 1988-2003 period. The results presented in this chapter do not only confirm the existence of the upturn but also provide the much needed answers regarding the explanatory factors that played a critical role in the observed upsurge in early childhood mortality. The results also points out the factors that suppressed the observed levels of infant and child mortality from increasing beyond the levels that were observed during the study period.

CHAPTER 7: A REGIONAL ANALYSIS

7.1 Introduction

This chapter aimed at investigating the factors associated with the upturn in infant and child mortality in Kenya's high and low HIV prevalence regions during the study period. Most past studies have tended to attribute the upsurge in infant and child mortality to the increase in HIV prevalence in some sub-Saharan African countries (Gregson, *et al.*, 2001; Hill *et al.*, 2001). The following two key research question were investigated: Were the levels in the factors underlying the upsurge in infant and child mortality in the high HIV prevalence regions the same as those for the low HIV prevalence regions? What was the actual contribution of the selected factors in the observed infant and child mortality differences between the two HIV prevalence regions?

7.2 Analytic Approach

The chapter and analytic approach adopted were guided by a number of factors: firstly, in sub Saharan Africa, data on longitudinal studies where mothers HIV status is repeatedly measured and where the mortality of their children (0-59 months) can be linked to maternal HIV status are scarce and where available, they are not population based. Even in population based surveys such as the 2003 and 2008-2009 KDHS that included biomarkers and tested a sample of mothers to determine their HIV status, it is difficult to obtain population attributable fractions (PAF) to ascertain the contribution of HIV to infant and child mortality due to the retrospective nature of KDHS (Urassa *et al.*, 2001; Nakiyingi *et al.*, 2003; Zaba *et al.*, 2003).

Secondly, the assumption of independence among covariates and the dependent variables upon which most standard regression techniques are based is flouted in generalised HIV settings like Kenya. For instance, Zaba and Gregson (1998) argue that in high HIV settings, the assumption that the mother's survival status is independent of the survival status of her biological children is flouted. HIV positive women tend to experience a heightened risk of dying and reduced fertility as compared to HIV negative women (Zaba and Gregson 1998; Zaba and Wald 2001; Zaba, *et al*, 2003; Mahy 2003). This leads to biased estimates since the assumption of independence between the mortality experience of mothers and children no longer holds (Zaba and Wald 2001; Zaba, *et al*, 2003; Mahy 2003; Omariba 2007) and hence makes conventional methods of mortality analysis unrealistic.

7.2 Analytic Approach

The study findings by Montaña *et al.*, (2007) which mapped the HIV prevalence at provincial level using the geographical information systems (GIS) technology are used to map low and high HIV prevalence zones. Montana *et al.*, (2007), segregate the regional distribution of HIV prevalence by gender. On the basis of HIV prevalence results from KDHS 2003 among adult women (15-49 years), the following provinces which had a higher than national HIV prevalence rates were categorised in this study as high HIV prevalence regions: Nyanza, Central, Nairobi and Rift Valley¹⁶.

¹⁶ While the national HIV prevalence was 6.7 percent by 2003 (CBS *et al.*, 2004), the HIV prevalence among women (15-49 years) were as follows: Nyanza (18.3%); Central (7.6%); Nairobi (11.9%); Rift Valley (6.9%) Western (5.8%); Coast (6.6%); Eastern (6.2%) and lastly, North Eastern (0.0%).

On the other hand, Coast, Eastern, North Eastern and Western were categorised as low HIV prevalence zones since their actual prevalence was lower than the national HIV prevalence of 6.7 percent. This categorization yielded a total of 6770 children (0-59 months) in the high HIV regions and 3625 children in the low HIV regions. For purposes of consistency, this study excluded North Eastern from the analysis since this region was not sampled in earlier surveys prior to KDHS 2003.

Using this regional categorization, this study computed the rates of infant and child mortality in the two categorized HIV regions and examined the plausible factors responsible for the upsurge in mortality in the two HIV prevalence zones in Kenya during the study period. The thesis here was that if HIV adversely affected infant and child mortality (either directly or indirectly), then were the higher rates of mortality in the high HIV prevalence regions due to lower levels of factors known to reduce mortality? Could the differences between the high HIV prevalence and the rest of the low HIV prevalence regions be a result of the nature of the relationships between infant and child mortality rates and their correlates?

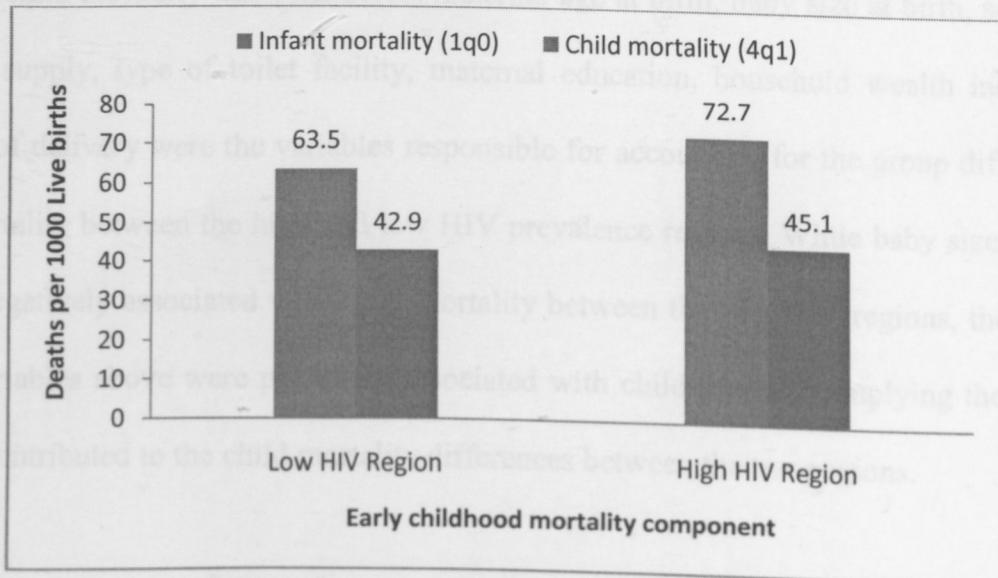
This chapter used regression decomposition techniques that were recently used by Hale *et al.*, (2009) in explaining early childhood mortality decline in Matlab, Bangladesh. However, unlike Hale *et al.*, (2009) who had access to a large dataset of over 93,000 singleton births and hence could calculate very many components of early childhood mortality, this study used infant and child mortality to estimate the role of select covariates in accounting for the observed mortality rates. This approach was the most

appropriate since a detailed analysis of all the early childhood mortality components would have compromised the statistical power in our analysis. In the next sections, we show and discuss the results based on this method.

7.3 Early childhood mortality changes in the low and high HIV regions

The synthetic cohort life table approach was used to compute direct measures of early childhood mortality in the high and low HIV regions. Infant and child mortality rates were higher in the high HIV region as compared to the low HIV region. Figure 7.1 below shows that the difference in infant mortality between the two HIV regions was 9.2 deaths per 1000 live births. On the other hand, child mortality variation between the two HIV regions was only 2.3 deaths per 1000 live births.

Figure 7.1: Mortality rates in early childhood in the high and low HIV Regions, Kenya



Source: Primary Analysis of pooled KDHS data

In an effort to explain the differences in mortality in the two HIV regions, this study sought to examine the changes in the values of the explanatory variables. This was done by computing the mean values of all the explanatory variables in both HIV regions and

assessing their significance using the t-statistic. All explanatory variables whose t-statistic value was at least 1.96 were considered as important correlates of infant and child mortality.

Results from Table 7.1 show that during infant mortality, the preceding birth interval, paternal education, source of drinking water, type of toilet facility, household wealth index and place of delivery seemed to be significantly different between the two HIV regions ($p < 0.05$). While preceding birth interval, paternal education, source of drinking water, type of toilet facility and household wealth index appeared to be positively associated with the increase in infant mortality and place of delivery were negatively associated with infant mortality in the two HIV prevalence regions.

When child mortality was considered, maternal age at birth, baby size at birth, source of water supply, type of toilet facility, maternal education, household wealth index and place of delivery were the variables responsible for accounting for the group differences in mortality between the high and low HIV prevalence regions. While baby size at birth was negatively associated with child mortality between the two HIV regions, the rest of the variables above were positively associated with child mortality implying they could have contributed to the child mortality differences between the two regions.

Table 7.1: Means of explanatory variables for infant and child mortality by Regional HIV status: Kenya, KDHS

	Infant Mortality Rate			Child Mortality Rate		
	Means LHIV	Means HHIV	Sig. of differences in means (t-statistic)	Means LHIV	Means HHIV	Sig. of differences in means (t-statistic)
Maternal factors						
Maternal age at child birth (D)	1.693	1.661	1.749	1.699	1.672	2.461**
Baby size at birth (D)	0.810	0.832	-1.480	0.849	0.874	-3.067**
Duration of breastfeeding (D)	0.887	0.846	1.922	0.117	0.116	0.126
Preceding birth interval (D)	0.756	0.693	3.136**	0.717	0.721	-0.365
Socio-economic factors						
Paternal education (years)	5.884	5.373	2.003**	5.688	5.629	0.347
Source of water (D)	0.336	0.262	3.741**	0.334	0.288	3.647**
Type of Toilet Facility (D)	0.821	0.716	6.202**	0.840	0.717	11.752**
Maternal education (Years)	5.447	5.303	1.551	5.478	5.182	5.011**
Household wealth index (D)	0.655	0.501	7.971**	0.666	0.507	13.318**
Maternal health - seeking factors						
Place of delivery (D)	0.541	0.655	- 5.970**	0.648	0.583	-10.50**
Tetanus Toxoid (D)	0.868	0.871	-0.188	0.887	0.863	1.395

Notes: D denotes dichotomous variable. ***p<0.001, **p<0.01, *p<0.05. Preceding birth intervals referred to second and higher order births.

Source: Primary analysis of the merged KDHS dataset 1988/89 and 1998/99

In Table 7.2 below, we attempt to answer the following question: what is the resultant early childhood mortality effect of a child born in a high HIV region as compared to the one born in a low HIV region? In order to answer this question, this study computed the hazard ratios with HIV as the only variable (model I) for both the two components of early childhood mortality followed by the full model which was the model with HIV adjusted for all the other explanatory variables. The results from the two models were then used to determine the percentage by which the change in hazard ratios accounted for infant and child mortality components.

Table 7.2: Effect of being born in a high HIV region relative to the low HIV region on mortality prior to attaining the first and fifth birthday ^a

Mortality component	Model I ^b	Model II ^c	% by which Hazard ratio changes by the additional explanatory variables in model II
Infant mortality (_{1q0})	3.08	1.92	56
Child mortality (_{4q1})	2.82	3.96	39

All hazard ratios are statistically significantly different from 1.0 at $p < 0.001$

^a Numbers shown are hazard ratio on the high HIV prevalence region from running a Cox PH model

^b In model I, a dummy variable for the high regional High prevalence is the only explanatory variable

^c Model II contains the dummy variable for the high regional HIV prevalence plus all the rest of the explanatory variables considered in this study: maternal age at child birth, size of the baby at birth, preceding birth interval, duration of breastfeeding, paternal education, maternal education, source of drinking water, type of toilet facility, household wealth index, place of delivery, whether or not the mother received a tetanus toxoid and malaria endemicity.

Source: Primary analysis of the pooled KDHS data

The results show that the hazard ratio for infant mortality in model I where HIV region was considered as the only variable was 3.08 implying that infant mortality was 208 percent higher in the high HIV region as compared to that of the low HIV region. When additional explanatory variables were added as shown in model II, the risk of experiencing infant mortality was 92 percent higher in the high HIV prevalence zone as

compared to the low HIV region. About 56 percent ($[1.92-3.08]/[1-3.08]$) of the upsurge in infant mortality between the two HIV regions could be attributed to changes in the explanatory variables we examined in this study.

During child mortality, the hazard ratio in model I was much lower (2.82) than it was during infant mortality (3.08). This implies that the differences in mortality between the two HIV regions was much greater during infancy than it was during child mortality. The increase in the hazard ratio from 2.82 in model I to 3.96 in model II implies that approximately 39 percent of the upsurge in child mortality ($[3.96-2.82]/[1-3.96]$) between the two HIV regions could be explained by the explanatory variables we considered in this study.

When additional explanatory variables were included as shown in model II in Table 7.2 above, the likelihood ratio test ($LR\chi^2$) met the $p < 0.05$ condition for both infant and child mortality components which implies that the explanatory power of the additional variables more than compensated for the loss of degree of freedom.

7.4 Decomposition analysis

This section assesses for each early childhood mortality component, the extent to which the change in each of the selected explanatory variables contributed to the upturn in mortality. This is shown in Tables 7.3 and 7.4 below. In these tables, the first two columns show the means of the explanatory variables in the two HIV regions and the third column shows the differences in the means ($\bar{u}-\bar{u}$). These means were also shown in

Table 7.1 above. This is followed by a computation of the hazard model coefficients (β) for each variable per HIV region and thereafter the product of these coefficients and the respective difference in the means. The computed product $\beta (\bar{u}-\bar{u})$ which is shown in column (g) is the contribution of the change in the two HIV regions for each explanatory variable to the difference in the mortality hazards between the two HIV regions.

Once this is done, the next column shows the summation of the amount that can be explained by the sets of variables. Where the sum for any set of our explanatory variables was negative, it implied that the changes between the two HIV regions for this particular set of variables reduced the mortality hazard by that amount (Hale *et al.*, 2009:11). On the contrary, if the sum for any set of explanatory variables was positive, it implied that *ceteris paribus*, the changes in the values for that set of variables between the two HIV regions resulted in an increase in mortality. Finally, the last column show the percentage of the change in the mortality rate between the two HIV regions that can be explained by each set of explanatory variables.

Results as shown in Table 7.3 below reveal that socio-economic factors accounted for the largest contribution to the upsurge in infant mortality (59 per cent) between the two HIV regions. This exceeded the total contribution of 56 percent upsurge in infant mortality that was observed between the two HIV regions as shown in Table 7.2 above.

Table 7.3: Decomposition of infant mortality differences between the high and low HIV regions into those due to particular explanatory variables

	(a)	(b)	(a)-(b)	(d)	(e)	(d).(e)	(f). (c)	h	% explained
Maternal factors								0.005	-10.2
Maternal age at child birth (D)	1.693	1.661	0.032	0.588	-0.111	0.019	0.001		
Baby size at birth (D)	0.810	0.832	-0.022	-0.182	0.072	0.004	0.000		
Duration of breastfeeding (D)	0.887	0.846	0.041	1.735**	1.444**	0.071	0.003		
Preceding birth interval (D)	0.756	0.693	0.063	0.368	-0.441*	0.023	0.001		
Socio-economic factors								-0.029	59.1
Paternal education (years)	5.884	5.373	0.511	-0.025	0.001	-0.013	-0.007		
Source of water (D)	0.336	0.262	0.074	0.238	-0.750*	0.018	0.001		
Type of Toilet Facility (D)	0.821	0.716	0.105	0.122	-0.582*	0.013	0.001		
Maternal education (Years)	5.447	5.303	0.144	-0.100	0.003	-0.014	-0.002		
Household wealth index (D)	0.655	0.501	0.154	-0.946*	-0.300	-0.146	-0.022		
Mother's health behaviour								-0.003	5.6
Place of delivery (D)	0.541	0.655	-0.114	-0.209	0.238	0.024	-0.003		
Tetanus Toxoid (D)	0.868	0.871	-0.003	0.922	-0.105	-0.003	0.000		
Sum								-0.026	

Notes: D =dichotomous variable. **p<0.001, *p<0.05; a & b=Means of covariates for Low and high HIV regions respectively; d & e = coefficients of covariates the Low and high HIV regions; % explained is the per cent of the sum for each set of covariates in column h over the overall sum (0.060) multiply by the total percentage change shown in Table 7.2 above (56%). Malaria endemicity was iteratively removed from analysis due to model omission

Source: Primary analysis of the merged KDHS dataset 1988/89 and 1998/99

The second most important set of explanatory variables during infancy were maternal factors. Unlike other factors, maternal factors accounted for 10.2 percent of the decline in the hazard of infant mortality between the two regions. Duration of breastfeeding and preceding birth interval are the two maternal factors that appeared to play a significant role in infant mortality decline between the two HIV regions ($p\text{-value} < 0.05$). Maternal health seeking behaviour accounted for an approximate 6 percent of the observed upsurge in infant mortality rate between the high and low HIV prevalence regions in this study.

An analysis of explanatory variables behind the observed child mortality differentials between the high and low HIV prevalence revealed that maternal and socio-economic factors accounted for the largest proportion of the observed upsurge in child mortality. For instance, Table 7.4 shows that maternal factors alone explained 20 percent of the observed rise in child mortality between the two HIV regions during the study period.

On the other hand, socio-economic factors accounted for almost 14 percent of the upsurge in child mortality between the two HIV regions considered in this study. However, an analysis of maternal health seeking factors revealed that far from the other factors that seemed to contribute to the upsurge, these factors accounted for 73 percentage decline in child mortality between the HIV regions.

Table 7.4: Decomposition of child mortality differences between the high and low HIV regions into those due to particular explanatory variables

	(a)	(b)	(c)	(d)	(e)	(f)	(g)	h	% explained
	Means HREG	Means LREG	(a)-(b)	β LREG	β HREG	(d)*(e)	(f)*(c)	0.031	20.4
Maternal factors									
Maternal age at child birth (D)	1.671	1.699	-0.028	1.213	-0.407	-0.493	0.014		
Baby size at birth (D)	0.874	0.849	0.025	0.754	0.840	0.633	0.016		
Duration of breastfeeding (D)	0.721	0.717	0.004	0.932	0.405	0.377	0.002		
Preceding birth interval (D)	0.116	0.117	-0.001	0.554	-0.586	-0.325	0.000		
Socio-economic factors								0.020	13.8
Paternal education (years)	5.629	5.688	-0.059	-0.145	0.013	-0.002	0.000		
Source of water (D)	0.288	0.334	-0.046	0.021	0.480	0.010	0.000		
Type of Toilet Facility (D)	0.717	0.84	-0.123	-0.833	-0.136	0.113	-0.014		
Maternal education (Years)	5.182	5.478	-0.296	-0.019	-0.051	0.001	0.000		
Household wealth index (D)	0.507	0.666	-0.159	0.439	-0.515	-0.226	0.036		
Maternal health behaviour								-0.113	-72.8
Place of delivery (D)	0.627	0.501	0.126	0.933	0.756	0.705	0.089		
Tetanus Toxoid (D)	0.903	0.913	-0.01	35.239	0.572	20.144	-0.201		
Overall Sum								-0.060	

Notes: LREG= Low HIV region; HREG=High HIV region; (c) is the difference in the mean values of explanatory variables between the two HIV regions; f = Is the product of the coefficient of each explanatory variable in both HIV regions (i.e. d & e); g is the product of columns (f) and c; h is the sum of values for each of the three sets of explanatory variables. % explained is computed as the per cent of the sum for each set of covariates as shown in column h over the overall sum (0.060) multiply by the total percentage change shown in Table 7.2a above (i.e. 39%).

Source: Primary analysis of the pooled KDHS dataset

7.5 Concluding remarks

The purpose of this chapter was to examine the factors that accounted for infant and child mortality upsurge in the high and low HIV prevalence zones in Kenya. Results revealed that higher infant and child mortality rates were observed in the high HIV regions as compared to the rates observed in the low HIV regions. The difference in infant mortality between the two HIV prevalence regions was 9.2 deaths per 1000 live births. On the other hand, child mortality variation between the two HIV regions was only 2.3 deaths per 1000 live births.

The preceding birth interval, paternal education, source of drinking water, type of toilet facility, household wealth index and place of delivery appeared to be important explanatory variables behind the infant mortality differentials between the low and high HIV prevalence regions ($t\text{-statistic} > 1.96$). On the other hand, maternal age at child birth, size of the baby at birth, source of drinking water, type of toilet facility, maternal education, household wealth index and place of delivery appeared to be important explanatory variables behind child mortality differentials between the low and high HIV prevalence regions ($t\text{-statistic} > 1.96$).

Generally, socio-economic factors appeared to play the greatest role in the observed infant mortality differentials between the high and low HIV prevalence zones while maternal factors exhibited the highest contribution to the observed child mortality differentials between the two HIV regions under this study. For instance, during infancy, socio-economic factors alone contributed to 59 percent increase in infant mortality

between the low and high HIV prevalence regions which was more than the total amount of the upsurge that was explained by the model during infancy (about 56 percent).

Likewise, during childhood, maternal factors alone accounted for 10 percent decline in mortality between the two HIV regions. Maternal health seeking behaviour accounted for almost 6 percent of the upsurge in infant mortality between the two HIV regions. However, these factors were responsible for 73 percentage decline in child mortality between the high and low HIV prevalence regions in this study.

CHAPTER 8: SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

8.1 Introduction

After enjoying a tremendous decline in infant and child mortality since the 1960s, Kenya witnessed an upsurge in early childhood mortality the late 1980s throughout the 2003 period. The factors underlying the upsurge in early childhood mortality in Kenya have remained poorly understood. The main purpose of this thesis was to investigate the factors behind the upsurge in early childhood mortality in Kenya during the 1988-2003 period. Three specific objectives were pursued: firstly, the study established whether the observed upsurge in early childhood mortality was indeed real and if so, the extent to which it increased over the study period; secondly, it made bold attempts to establish some of the factors associated with the upturn in infant and child mortality in Kenya between 1988 and 2003. Finally, it examined the factors behind the upsurge in infant and child mortality in the high and low HIV prevalence zones in Kenya.

A number of steps were undertaken to address the main aim of the study. Firstly, literature review was done to identify potential factors that could be responsible for an upsurge in infant and child mortality in Kenya. The Mosley and Chen (1984) theoretical framework was used to guide the specification of variables for inclusion in this study.

Findings showed that the upsurge in infant and child mortality was not a consequence of the deterioration in data quality or measurement error. An assessment of data quality for early childhood mortality estimation found that the completeness of age reporting for children was almost universal in Kenya. Myer's index revealed that in general, age heaping was low with an index of overall 86 percent of women aged 20-49 years

reporting their ages within the correct terminal digits. Age reporting for children revealed some heaping at months 12, 18, 24 and 36. However the effect of age heaping at month 12 could only have led to an understatement of the level of infant mortality and thus it could not be responsible for its upsurge. Further, while there was heaping at months 12, 18, 24 and 36, the effect of this bias on child mortality estimation is negligible since all these ages are conventionally part of child mortality estimation.

Likewise, parity increased with the age of the mother as is conventionally expected - a pointer to the high quality of mortality data used in this study. Further, as expected, an analysis of age ratios revealed that in general, there were little perturbations in the calculated age ratios from 100 - a further pointer to the fact that the upsurge in infant and child mortality is unlikely to have been a consequence of poor data quality. Age heaping on terminal digit 0 and 5 was also low as shown by the computed Myer's Indices. The framing of questions remained basically the same in all the three rounds of KDHS and thus the upsurge could not have been a product of measurement errors. Earlier studies also failed to confirm the assertion that poor data quality explained the upturn in childhood mortality in Kenya (Hill *et al.*, 2004). Consequently, the upturn in early childhood mortality must have been genuine arising due to changes in some of the proximate determinants on early childhood mortality in Kenya.

The synthetic life table cohort approach revealed that there was an upsurge in infant mortality of 61 deaths per 1000 live births during the five years preceding the 1993 KDHS and 70 and 80 deaths per 1000 live births during the 1993-98 and 1999-2003

period respectively. This rise accounted for a 30 percent upturn in infant mortality during the 1988-2003 study period. The observed upturn in infant mortality compares well with the 32 percent increase that was recorded in standard KDHS reports (CBS, *et al.*, 2004). On the contrary, child mortality rose from 42.6 deaths per 1000 live births in the 1988-92 to 46 deaths per 1000 live births in the 1998-2003 time periods which was a 9 percent increase for the 1988-2003 period. The study also found that infant and child mortality increased when assessed against most of the covariates that were measured in this study.

Chapter Five apart from presenting the factors associated with infant and child mortality also revealed that the rise in early childhood mortality components was not by chance. The results from the Weibull analysis in the accelerated failure framework revealed that the ancillary parameter (p) for infant mortality was >1 which implies that the risk of dying in infancy increased during the 1988-2003 period. On the overall, the explanatory variables accounted for about 55 percent increase in infant mortality which implies an average annual upsurge of about 3.7 percent during the study period. Further, the positive time dependence as shown by the Weibull AFT ancillary parameter (p) indicates that child mortality increased by 62 percent during the 1988-2003 study period.

Findings from the regression decomposition techniques revealed that duration of breastfeeding, maternal education, regional malaria endemicity and regional HIV prevalence were important explanatory variables in accounting for the mean differences in infant mortality between the 1988-95 and 1996-2003 period. Secondly, apart from maternal age at child birth, tetanus toxoid, and household wealth index, the effect of the

rest of the explanatory variables considered in this study were significantly important in accounting for the period differences in mortality during infancy.

When all explanatory variables were added to the time variable (1988-1995 to 1996-2003 study period), the likelihood ratio test satisfied $p < 0.001$ for all the models which implies that the explanatory power of the additional variables more than compensated for the loss in degrees of freedom. The differences in the values of explanatory variables, taken together, during the two periods contributed to differences in the observed infant mortality. Assessed on the overall, the variables considered in this study accounted for 38 percent of the observed upsurge during infant mortality.

The duration of breastfeeding and regional HIV prevalence accounted for the largest portion of infant mortality increase during the 1988-1995 and 1996-2003 period. On the other hand, regional malaria prevalence accounted for the greatest portion of the infant mortality decline during the study period. Counterfactual analysis revealed that had all the infants belonging to the 1996-2003 period experienced the same mean values of all explanatory variables as those belonging to the 1988-1995 period, then infant mortality could have increased by 14 deaths/1000 live births. However, had all the infants belonging to the 1988-1995 period experienced the same mean values of all explanatory variables as those belonging to the 1996-2003 time period, then the rise in infant mortality could have been negligible (less than 1 death per 1000 live births).

On the other hand, preceding birth interval, place of delivery, whether or not the mother had received a tetanus toxoid during pregnancy, source of water supply, type of toilet facility, maternal education and malaria endemicity though lacked significance in the mean differences in child mortality in the two periods were all statistically significant at 5 percent level.

In general, maternal and paternal education both had a negative effect on child mortality in both periods and appeared to explain a large portion of the observed differences in child mortality rates in the two periods. Other factors that were significantly associated with child mortality in the two periods were maternal age at child birth and type of place of delivery. Had the children belonging to the 1988-1995 period experienced the same mean values of all explanatory variables as those of children belonging to the 1996-2003 period, then child mortality would have increased by 6 deaths per 1000 live births. However, had all the children belonging to the 1996-2003 period experienced the same mean values of all explanatory variables as those of children belonging to the 1988-1995 period, child mortality could have reduced by almost 2 deaths per 1000 live births.

The results presented in this chapter did not only confirm the existence of the upturn in early childhood mortality components but also provided the much needed answers regarding the explanatory factors that played a critical role in the observed upsurge. When the high and low HIV prevalence regions were examined, results revealed that higher mortality rates were observed in the high HIV mortality regions as compared to the rates observed in the low HIV mortality rates. The difference in infant mortality

between the two HIV prevalence regions was 9.2 deaths per 1000 live births. On the other hand, child mortality variation between the two HIV regions was only 2.3 deaths per 1000 live births.

The preceding birth interval, paternal education, source of water supply, type of toilet facility, household wealth index and place of delivery appeared to be important explanatory variables behind the infant mortality differentials between the low and high HIV prevalence regions ($p < 0.05$). On the other hand, maternal age at child birth, size of the baby at birth, source of drinking water, type of toilet facility, maternal education, household wealth index and place of delivery appeared to be important explanatory variables behind child mortality differentials between the low and high HIV prevalence regions ($p < 0.05$).

Generally, socio-economic factors appeared to play the greatest role in the observed infant mortality differentials between the high and low HIV prevalence zones while maternal factors exhibited the highest contribution to the observed child mortality differentials between the two HIV regions. During infancy, socio-economic factors alone contributed to 59 percent increase in infant mortality between the low and high HIV prevalence regions which was more than the total amount of the infant mortality upsurge that was explained by the model during infancy (about 56 percent).

During childhood, maternal factors alone accounted for 20 percentage increase in mortality between the two HIV regions. Maternal health seeking behaviour accounted for

almost 6 percent increase in infant mortality between the two HIV regions. However, these factors were responsible for 73 percent reduction in child mortality differences between the high and low HIV prevalence regions in this study.

The study found that while a number of factors were responsible for early childhood mortality changes, not all factors examined in this study were responsible for the upsurge. Generally, during infancy, maternal factors appeared to explain much of the upsurge in infant mortality. However during childhood, socio-economic factors appeared to explain much of the upsurge in mortality. When child mortality was analysed by HIV status, socio-economic factors appeared to explain much of the upsurge in mortality. Maternal health seeking factors appeared to have contributed largely to the decline in infant and child mortality during the study period.

8.2 Policy Implications

Given the above findings, a number of policy recommendations are proposed for strengthening and accelerating child survival options if the country has to meet national and international public health goals such as reducing the 1990 level of infant and child mortality rates by two-thirds by 2015 as articulated in the Millennium Development Goal number four.

The main results revealed that maternal factors (which are basically biological) played a critical role in the upsurge in infant and child mortality in this study. The maternal factors in this study included maternal age at child birth, size of the baby at birth, duration of

breastfeeding and the preceding birth interval. Therefore, this study calls on policy makers to promote the uptake of family planning program in order to reduce short birth intervals as well as prevent high risk births which tend to be associated with a heightened risk of early childhood deaths.

Further in a generalized HIV setting like Kenya, there is need for the government with the assistance of developing partners to strengthen the prevention of mother to child (PMTCT) programs in order to minimise new paediatric infections. This may entail encouragement of intensive breastfeeding for the first six months among HIV positive mothers and weaning of infants thereafter by introducing alternative feeding options as well as encouraging the use of non hormonal contraceptives among HIV positive mothers to postpone or limit births. Through this, fewer HIV positive children will be born but we shall also have fewer babies born with small size which tend to have a heightened risk of dying.

It is not a surprise that the role of socio-economic factors played an increasing role to the upturn in mortality. This study speculates that the downturn in Kenya's economy during the study period with some years recording a negative growth in the GDP must have depressed the household level spending on health. This was further worsened by the introduction of the structural adjustment programs (SAPs) in the late 1980s which among other things introduced cost sharing in health and education leading to retrenchment of excess government staff (Bedi *et al.*, 2002a, Bedi *et al.*, 2002b). Efforts to revitalise the economy and reduce the rampant poverty and establish an affordable social health

insurance scheme for the poor should be enacted and implemented in government planning and expenditure. There is need for an increase in budgetary allocation to meet if not surpass the target of 15 percent annual budget allocation on health as passed in the Abuja Declaration and plan of action of 2000. Such an increase in budgetary allocation will cover substantially the health provision to the majority poor who currently cannot afford any form of healthcare.

This study also pointed out that malaria prevalence played a pivotal role in the observed upturn in infant and child mortality. The increased contribution of Malaria was expected owing to the fact that malaria resistance which was first documented in the early 1980s peaked by 1997 (when resistance to chloroquine ranged from 66-87 percent) even though it was only then that the government of Kenya changed the first line treatment of malaria from chloroquine to artemisinin combination therapy (ACT) for treatment of uncomplicated malaria (WHO *et al.*, 2003; IAEA, 2007, Houwelling *et al.*, 2006). While earlier rounds of KDHS before 2003 did not ask questions on household net ownership, anecdotal evidence shows that there was low ownership of insecticide treated mosquito nets prior to the social marketing campaigns on ITN ownership that were initiated in early 2000s (World Bank 2012).

Additionally, programs aimed at intensifying the distribution of Insecticide treated mosquito nets, increasing community awareness on the danger signs and early treatment of malaria, in house spraying and other vector control strategies in high Malaria prone

regions as well as biomedical research for malaria vaccine development need renewed emphasis in Kenya's health programming.

Owing to the fact that Malaria endemicity differs from one region to another, the Division of Malaria control in its technical role of implementing the national malaria strategic plan should work with other stakeholders in health in ensuring high impact Malaria interventions in middle and high malaria endemic zones. This among other things should be accompanied by culturally sensitive behaviour change and communication campaigns to address cultural barriers associated with ITN utilization.

8.3 Future research

Like Barbieri (1994), I argue that an exhaustive review of the causes of early childhood mortality upturn in Kenya during the 1988-2003 period cannot be adequately addressed by use of KDHS data alone. This study proposes that future research should entail triangulation of both quantitative and qualitative techniques. Alongside KDHS, other types of data sets for a similar study should use the Housing and Population Census as well as the multiple cluster indicator surveys and the Kenya integrated household budget surveys. The use of qualitative techniques should also be explored especially in delving information on household level factors that could have accounted for mortality changes.

Further the DHS module on the household questionnaire should be revised to include additional information. For instance, while information on source of drinking water supply was important in this study, information on whether the water supply was treated

or not was not included in the survey modules yet it is critical in assessing the influence of this variable on early childhood mortality. This information needs to be included in future modules of KDHS owing to the increasing use of water purification techniques among low income households in Kenya's high mortality regions (Makutsa *et al.*, 2001).

Additionally future studies should also investigate the sources of differences in infant and child mortality between time periods or between the different HIV prevalence regions across the baseline hazard. One potential source of investigation is censoring. In addition, there is need for use of analytic models that encompass interactions on variables such as duration of breastfeeding and birth interval in assessing interaction effects on the observed mortality changes.

The use of longitudinal data in future research on this topic will serve to provide even further insights on this topic. The possible such sources include the ongoing demographic surveillance surveys in selected sites within the country that are being coordinated by the INDEPTH Network. The advantage with such datasets is the availability of large datasets than can allow for more analysis of the different components of early childhood mortality beyond infant and child mortality without any loss in the statistical power in the results obtained.

The study proposes that future DHS should routinely collect data on HIV testing and Malaria to enable further analysis on this topic. In this study for instance, it was not possible for a direct analysis of the effect of HIV and malaria on early childhood

mortality since not all the KDHS datasets used in this study collected information on these two diseases. While a regional analysis of HIV which entailed grouping of provinces into whether they had high or low HIV provinces was used, there is a possibility that this categorization in itself could have been biased due to differential response rates between provinces. Future studies ought to assess the extent of participant non response on HIV prevalence and if necessary re-assess the covariate effects on the upsurge in infant and child mortality.

This study also proposes that future studies should consider using alternative methodologies such as lagged models to confirm whether results from such methodologies could be comparable to these study findings. The use of lagged models underscore the fact that if infant and child mortality indeed changed, it must have done so only after some lag since some studies have pointed out that health status is a 'stock' that normally provides a margin of health reserves above a critical threshold in any economy (Gakusi and Garenne 2007:413).

Kenya as a country has rich demographic data and one critical issue that the researcher came across was that while the used datasets in this study were collected during an era of an upsurge in early childhood mortality, the later datasets – specifically the KDHS 2008/09 shows that the trends in early childhood mortality have since reversed and on the downward trends. It is therefore imperative for future studies to focus on the contextual and individual factors that explain the reversals in early child mortality trends after 2003.

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Method

In this study, Stata version 11 was used to compute early childhood mortality estimates. This can also be done in Microsoft Excel. The first step entails determining all live births within the five years preceding each survey of study in months. In DHS, the century month code (CMC) can be used to convert the child's age in years to the number of months lived by the time of interview for living children. The survey also has data on age at death in months and where data are missing on either age at death or birth, imputed variables of the same are computed. Owing to the fact that multiple births have a

Appendix 1: A technical note on Direct estimation of Early Childhood Mortality Estimation using the synthetic life table approach

Rationale

In this study, the computation of early childhood mortality is restricted to a period not exceeding five years prior to each selected survey for several reasons: firstly, to ensure that the background variables tie closely as possible to the timing of early childhood mortality. Secondly, restricting mortality estimates to the most recent period limits recall biases on births and deaths (Omariba and Boyle 2007). Thirdly, in generalized HIV epidemic regions such as Kenya, such restrictions serve to avoid biases associated with mothers' death, which leads to exclusion of children of such mothers in the child mortality estimates (Mahy 2001; Zaba *et al.*, 2004). Finally, restricting births to a period not exceeding five years prior to survey ensures that children of older women in relation to those of younger women are not disproportionately represented in the sample, which could lead to biased estimates against the latter (Omariba & Boyle 2007).

Method

In this study, Stata version 11 was used to compute early childhood mortality estimates. This can also be done in Microsoft Excel. The first step entails determining all live births within the five years preceding each survey of study in months. In DHS, the century month code (CMC) can be used to convert the child's age in years to the number of months lived by the time of interview for living children. The survey also has data on age at death in months and where data are missing on either age at death or birth, imputed variables of the same are computed. Owing to the fact that multiple births have a

heightened risk of death as compared to singletons, this study adjusted early mortality estimates to include only singletons. One further adjustment specific for KDHS 2003 which is used here as an illustration was the exclusion of North Eastern Province for comparability with initial surveys where the region was not sampled.

The second step after tabulating all births for each survey data will entail constructing a life table. To do this, it is prudent to get the total live births disregarding all those cases that have no information. Thus children with missing information on date of birth will have to be imputed. These total live births form the number at start. The next columns of the table will be the number alive at interview tabulated by months since births (i.e. from 0-59 months). Next will be the number who died during any age interval (measured in months from 0-59 months).

The next step entails computing the number of live births exposed to the risk of dying.

This method, assumes that any child who dies after birth does so halfway through the interval from age x to age $x+1$. With these data, one is able to compute the probability of dying (${}_nq_0$) by simply dividing those who died within each age interval by the population of live births who were directly exposed to the risk of dying. It thus becomes easier to compute the rest of the columns of the life table. One can run this procedure by using *ltable* command in stata. The command used in this illustration is as shown below:-

ltable age dead if brec2==3, survival intervals(brec2). This commands generated a table shown below: -

An illustration of Early Childhood Mortality Estimates: KDHS 1999-2003

Interval	Beg.		Std.		Survival	Error	[95% Conf. Int.]
	Total	Deaths	Lost				
0	1	4851	140	62	0.971	0.0024	0.9658 - 0.9753
1	2	4649	17	99	0.9674	0.0026	0.9619 - 0.9720
2	3	4533	17	109	0.9637	0.0027	0.9580 - 0.9686
3	4	4407	16	99	0.9602	0.0028	0.9542 - 0.9653
4	5	4292	28	75	0.9538	0.0031	0.9475 - 0.9595
5	6	4189	19	104	0.9495	0.0032	0.9428 - 0.9554
6	7	4066	11	89	0.9469	0.0033	0.9400 - 0.9529
7	8	3966	17	93	0.9428	0.0034	0.9356 - 0.9491
8	9	3856	13	90	0.9395	0.0035	0.9322 - 0.9461
9	10	3753	14	98	0.936	0.0036	0.9285 - 0.9427
10	11	3641	9	95	0.9336	0.0037	0.9260 - 0.9405
11	12	3537	6	100	0.932	0.0038	0.9243 - 0.9390
12	13	3431	15	100	0.9279	0.0039	0.9199 - 0.9352
13	14	3316	5	89	0.9265	0.0039	0.9184 - 0.9338
14	15	3222	4	91	0.9253	0.004	0.9171 - 0.9327
15	16	3127	2	80	0.9247	0.004	0.9165 - 0.9322
16	17	3045	1	86	0.9244	0.004	0.9161 - 0.9319
17	18	2958	1	90	0.9241	0.004	0.9158 - 0.9316
18	19	2867	10	75	0.9208	0.0041	0.9123 - 0.9285
19	20	2782	2	64	0.9202	0.0042	0.9116 - 0.9279
20	21	2716	0	86	0.9202	0.0042	0.9116 - 0.9279
21	22	2630	0	79	0.9202	0.0042	0.9116 - 0.9279
22	23	2551	0	93	0.9202	0.0042	0.9116 - 0.9279
23	24	2458	1	65	0.9198	0.0042	0.9112 - 0.9276
24	25	2392	17	88	0.9131	0.0044	0.9040 - 0.9214
25	26	2287	0	84	0.9131	0.0044	0.9040 - 0.9214
26	27	2203	1	62	0.9127	0.0045	0.9035 - 0.9210
27	28	2140	0	66	0.9127	0.0045	0.9035 - 0.9210
28	29	2074	1	78	0.9122	0.0045	0.9030 - 0.9206
29	30	1995	1	86	0.9118	0.0045	0.9025 - 0.9202
30	31	1908	0	64	0.9118	0.0045	0.9025 - 0.9202
31	32	1844	0	66	0.9118	0.0045	0.9025 - 0.9202
32	33	1778	0	84	0.9118	0.0045	0.9025 - 0.9202
33	34	1694	0	68	0.9118	0.0045	0.9025 - 0.9202
34	35	1626	0	82	0.9118	0.0045	0.9025 - 0.9202
35	36	1544	0	85	0.9118	0.0045	0.9025 - 0.9202
36	37	1459	8	107	0.9066	0.0048	0.8966 - 0.9156
37	38	1344	0	91	0.9066	0.0048	0.8966 - 0.9156

38	39	1253	0	80	0.9066	0.0048	0.8966 - 0.9156
39	40	1173	0	73	0.9066	0.0048	0.8966 - 0.9156
40	41	1100	0	66	0.9066	0.0048	0.8966 - 0.9156
41	42	1034	0	80	0.9066	0.0048	0.8966 - 0.9156
42	43	954	0	75	0.9066	0.0048	0.8966 - 0.9156
43	44	879	0	73	0.9066	0.0048	0.8966 - 0.9156
44	45	806	0	84	0.9066	0.0048	0.8966 - 0.9156
45	46	722	0	94	0.9066	0.0048	0.8966 - 0.9156
46	47	628	0	64	0.9066	0.0048	0.8966 - 0.9156
47	48	564	0	85	0.9066	0.0048	0.8966 - 0.9156
48	49	479	2	65	0.9025	0.0056	0.8909 - 0.9129
49	50	412	0	88	0.9025	0.0056	0.8909 - 0.9129
50	51	324	0	98	0.9025	0.0056	0.8909 - 0.9129
51	52	226	0	84	0.9025	0.0056	0.8909 - 0.9129
52	53	142	0	71	0.9025	0.0056	0.8909 - 0.9129
53	54	71	0	49	0.9025	0.0056	0.8909 - 0.9129
54	55	22	0	20	0.9025	0.0056	0.8909 - 0.9129
55	56	2	0	1	0.9025	0.0056	0.8909 - 0.9129
56	57	1	0	1	0.9025	0.0056	0.8909 - 0.9129

Notes: The calculated rates are not exact as those recorded in KDHS 2003 because the data used here were merged and relate to the period of 1999-2003. Further, KDHS calculates infant mortality by including month 12 not 11 as is conventionally recommended hence the KDHS rates are slightly higher for infant mortality.

From the above table, one can compute the following key early childhood mortality rates:-

Neonatal mortality (q0): the probability of dying within the first month of live. From the table above, this rate is computed as follows: $(1-0.971) \times 1000 = 29$ **deaths/1000 live births**. Thus for every 1000 live births, about 29 infants die prior to attaining their first month of life.

Infant Mortality (0q1): the probability of dying between 0-11 months. From the table, this is computed by subtracting 1 from the cumulated probability of surviving at month 11 and then multiplying by 1000 i.e. $(1-0.932) \times 1000 = 68$ **deaths per 1000 live births**.

Perinatal Mortality which is the difference between infant and neonatal mortality i.e.

$68-29 = 39$ deaths per 1000 live births.

Child Mortality (1q4): this is the probability of dying between the first and fourth birthday i.e. the difference between under-five mortality and infant mortality i.e.

$(98-68) = 30$ deaths per 1000 children.

Under-five Mortality (0q4): the probability of dying between 0-59 months. From the table, this is computed by subtracting 1 from the cumulated probability of surviving at month 59 (l_{59}) and then multiplying by 1000 i.e. $(1-0.99541)*1000=98$ deaths/1000 live births.

	Infant mortality	Child mortality	Prob>chi2	rho	chi2	Prob>chi2
Maternal age						
20-34 years						
<20 years	-0.024	0.05	0.818	-0.019	0.01	0.923
35-49 years						
Baby Size at birth						
Very low						
Small	0.079	0.9	0.479	-0.076	0.15	0.696
Pre-eclampsia						
37 months+						
<24 months	-0.143	1.87	0.171	0.218	1.42	0.234
20-34 months	-0.062	0.32	0.572	-0.017	0.01	0.923
Breastfeeding						
>20+ months						
<10 months						
10-19 months	-0.202	0.7	0.07	-0.18	1.69	0.194
Place of birth						
Government facility						
Home	0.042	0.14	0.708	0.245	1.77	0.184
Private Health Facility	0.059	0.32	0.574	0.149	0.65	0.419
Had Tetanus Injection						
No						
Yes	-0.045	0.2	0.652	0.000		
Maternal Education						
Sec+						
None	0.023	0.05	0.822	0.128	0.44	0.506
Primary	0.129	1.34	0.247	0.276	1.64	0.201
Paternal Education						
Sec+						
None	-0.087	0.56	0.418	0.212	1.97	0.160
Primary	-0.038	0.13	0.714	0.250	1.33	0.250
HH Wealth Index						
Upper						
Lower	0.084	0.62	0.432	-0.078	0.15	0.696
Middle	-0.012	0.01	0.915	-0.126	0.45	0.502
Study Period						
1988-92						
1993-98	0.021	0.01	0.859	-0.048	0.06	0.809
1999-03	0.128	1.31	0.253	-0.043	0.05	0.826
Global test	χ^2 test = 26.77; df = 26; p = 0.449			χ^2 test = 23.74; df = 26; p = 0.522		

Source: Author's computation from DHS (1991-2003)

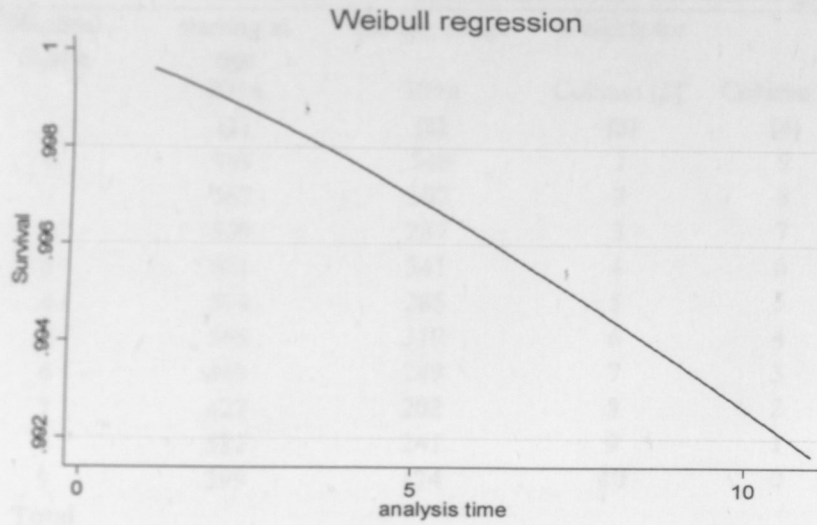
Appendix II: Testing the Proportionality Assumption: A full Model

	Infant mortality			Child mortality		
	rho	chi2	Prob>chi2	rho	chi2	Prob>chi2
Maternal age						
20-34 years						
<20 years	-0.024	0.05	0.818	-0.019	0.01	0.923
35-49 years	-0.075	0.45	0.505	-0.274	2.14	0.143
Baby Size at birth						
Av./large						
Small	-0.079	0.5	0.479	-0.076	0.15	0.696
Prec. Birth Interval						
37 months+						
<24 months	-0.145	1.87	0.171	0.218	1.42	0.233
20-34 months	-0.062	0.32	0.572	-0.017	0.01	0.923
Breastfeeding period						
>20+ months						
<10 months	-0.244	4.83	0.028**	0.116	0.41	0.523
10-19 months	-0.262	5.7	0.017**	0.218	1.69	0.194
Place of Delivery						
Government facility						
Home	-0.042	0.14	0.708	0.245	1.77	0.184
Private Health Facility	0.059	0.32	0.574	0.149	0.65	0.419
Had Tetanus Injection						
No						
Yes	-0.046	0.2	0.652	0.000		
Maternal Education						
Sec+						
None	0.023	0.05	0.822	0.128	0.44	0.506
Primary	0.129	1.34	0.247	0.276	1.64	0.201
Paternal Education						
Sec+						
None	-0.087	0.66	0.418	0.212	1.97	0.160
Primary	-0.038	0.13	0.714	0.250	1.33	0.250
HH Wealth Index						
Upper						
Lower	0.084	0.62	0.432	-0.078	0.15	0.696
Middle	-0.012	0.01	0.915	0.126	0.45	0.502
Study Period						
1988-92						
1993-98	0.021	0.03	0.859	-0.048	0.06	0.809
1999-03	0.126	1.31	0.253	-0.043	0.05	0.826
Global test	χ^2 test = 26.27; df=26; p = 0.449			χ^2 test = 28.74; df = 26 p = 0.323		

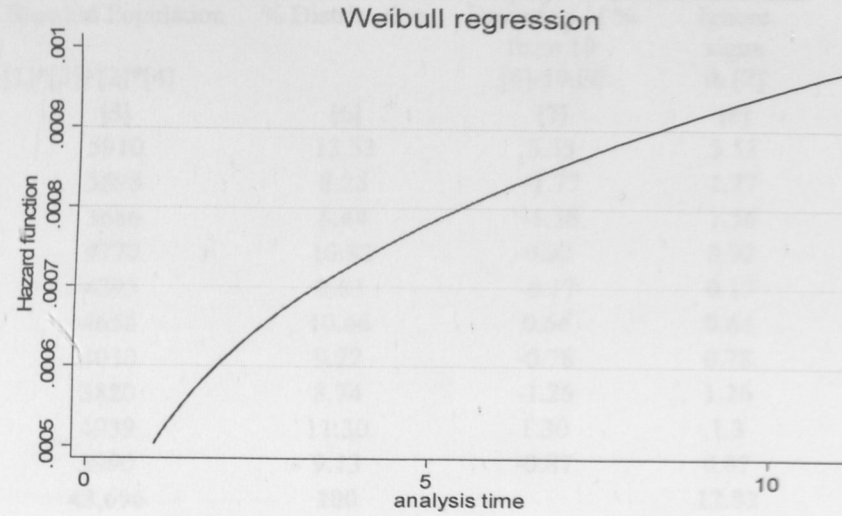
Source: Author's computation from KDHS 1993-2003

Appendix III: Graphical Evaluation of the Appropriateness of the Weibull Model

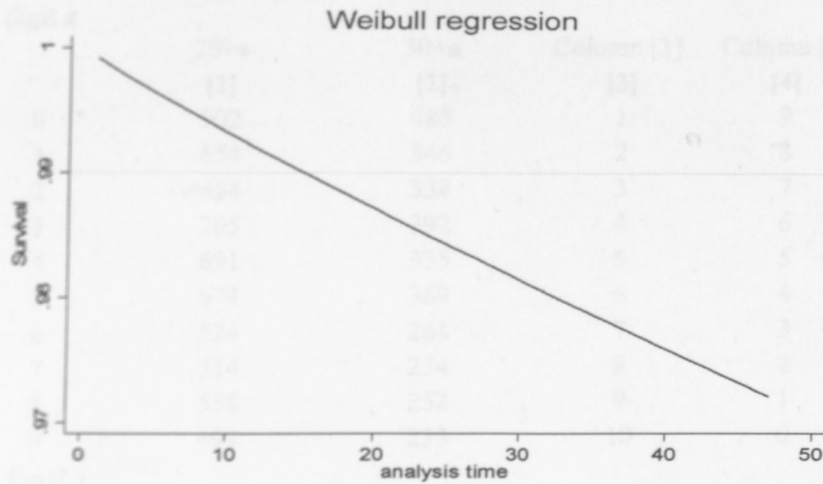
a) Log log survival curve- infant mortality



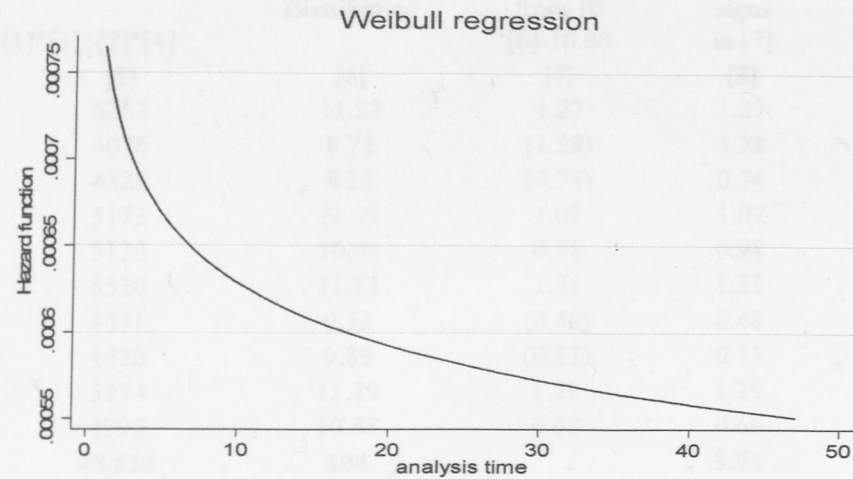
b) Hazard curve- child mortality



c) Log log survival curve - child mortality



d) Hazard curve- child mortality



Appendix IV: Computation of Myer's Index

Calculation of Preference Indexes for Terminal Digits by Myers' Blended Method for Kenya: 1993 KDHS

Terminal digit a	starting at age 20+a	starting at age 30+a	Weights for		Blended Population	% Distribution	Deviation of % from 10 [6]-10.00	Ignore signs in [7]
	[1]	[2]	Column [1] [3]	Column [2] [4]	[1]*[3]+[2]*[4] [5]	[6]	[7]	[8]
0	969	549	1	9	5910	13.53	3.53	3.53
1	587	303	2	8	3598	8.23	-1.77	1.77
2	559	287	3	7	3686	8.44	-1.56	1.56
3	681	341	4	6	4770	10.92	0.92	0.92
4	574	285	5	5	4295	9.83	-0.17	0.17
5	565	317	6	4	4658	10.66	0.66	0.66
6	469	249	7	3	4030	9.22	-0.78	0.78
7	427	202	8	2	3820	8.74	-1.26	1.26
8	522	241	9	1	4939	11.30	1.30	1.3
9	399	174	10	0	3990	9.13	-0.87	0.87
Total					43,696	100		12.82
Summary Index of age preference= [Total for Column I]/2								6.41

Calculation of Preference Indexes for Terminal Digits by Myers' Blended Method for Kenya: 2003 KDHS

Terminal digit a	starting at age 20+a	starting at age 30+a	Weights for		Blended Population	% Distribution	Deviation of % from 10 [6]-10.00	Ignore signs in [7]
	[1]	[2]	Column [1] [3]	Column [2] [4]	[1]*[3]+[2]*[4] [5]	[6]	[7]	[8]
0	902	485	1	9	5267	11.27	1.27	1.27
1	654	346	2	8	4076	8.72	(1.28)	1.28
2	654	338	3	7	4328	9.26	(0.74)	0.74
3	705	392	4	6	5172	11.07	1.07	1.07
4	691	335	5	5	5130	10.98	0.98	0.98
5	674	369	6	4	5520	11.81	1.81	1.81
6	524	261	7	3	4451	9.52	(0.48)	0.48
7	514	254	8	2	4620	9.89	(0.11)	0.11
8	558	252	9	1	5274	11.29	1.29	1.29
9	499	233	10	0	4990	10.68	0.68	0.68
Total					48,828	104		9.71
Summary Index of age preference= [Total for Column I]/2								4.86

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Calculation of Preference Indexes for Terminal Digits by Myers' Blended Method for Kenya: 1998 KDHS								
Terminal digit a	starting at age	starting at age	Weights for		Blended Population	% Distribution	Deviation of % from 10	Ignore signs in [7]
	20+a	30+a	Column [1]	Column [2]	[1]*[3]+[2]*[4]		[6]-10.00	
	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]
0	752	423	1	9	4559	9.76	(0.24)	0.24
1	581	294	2	8	3514	7.52	(2.48)	2.48
2	634	313	3	7	4093	8.76	(1.24)	1.24
3	614	295	4	6	4226	9.04	(0.96)	0.96
4	581	295	5	5	4380	9.37	(0.63)	0.63
5	727	419	6	4	6038	12.92	2.92	2.92
6	498	259	7	3	4263	9.12	(0.88)	0.88
7	473	245	8	2	4274	9.15	(0.85)	0.85
8	648	343	9	1	6175	13.21	3.21	3.21
9	521	257	10	0	5210	11.15	1.15	1.15
Total					46,732	100		14.56
Summary Index of age preference = [Total for Column I]/2								7.28