



Mortality situation in selected HIV / AIDS seroprevalence regime clusters of Kenya

Tom Omondi Owuor

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**Mortality Situation in Selected HIV/AIDS
Seroprevalence Regime Clusters of Kenya**


By Owuor Tom Omondi

**'MORTALITY SITUATION IN SELECTED HIV/AIDS
SEROPREVALENCE REGIME CLUSTERS OF KENYA'.**

OWUOR TOM OMONDI.

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF THE DEGREE OF MASTERS
OF ARTS IN POPULATION STUDIES AND DEVELOPMENT AT THE POPULATION
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No. d'inventaire
Date 18/12/99
Cote KE/OMO T 312.2604

IFRA003059

P.O. Box 5648
Nairobi Kenya

DECLARATION.

This Thesis is my original work and has not been presented for a degree in any other University to the best of my knowledge.

P. A. R. A.,
P.O. Box 5648
Nairobi Kenya

OWUOR TOM OMONDI.
CANDIDATE

This Thesis has been submitted for examination with my Knowledge as the University supervisor.

1. Prof. A.B.C. OCHOLLA-AYAYO

2. Dr. BONIFACE K'OYUGI

DEDICATION.

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Lots of debts was accrued in the course of research and the writing up of this thesis. In an attempt to pay part of this debt, I wish to thank the following persons whose help was indispensable in the completion of this study.

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However, I alone bear the final responsibility of the contents and short comings of this study.

ABSTRACT.

This study attempts to determine the mortality differential by the prevailing HIV prevalence ^{regions} clusters. The sentinel surveillance results recorded for the last seven years, were averaged to determine the grouping of the clusters into high, mid and low HIV prevalence regimes (clusters), taking a recourse to determining the differentials in HIV/AIDS incidence and prevalence and the impact of HIV/AIDS on mortality.

The study used mainly the NASCOP and CBS data, the method used for analysis of infant and child mortality was Coale and Trussell version of the Brass method while for adult mortality the study used orphanhood method. Statistical methods used were, the chi-square, and analysis of variance.

At the household environmental level, the results of the indirect technique established that there was marked differentials in mortality by variable and by variable categories in each cluster. Children in households with modern toilet facilities, better quality housing, floor material type, clean drinking water and who reside in urban areas had generally lower risk of death than other variable categories. Cluster wise, the high HIV cluster generally had higher mortality levels, followed by the mid HIV cluster while the low HIV cluster had the lowest risk of death. This study data therefore supports the study objective that there is marked mortality differentials by household environmental factors. However, these factors associated with lower risk of child death were in fact impacted more by AIDS such that the percentage increment in child mortality by these factors was found to be higher.

The study also found that at the socio-economic level, higher level of mothers education, good employment opportunities, female children and those not married ^{or} and ~~those~~ in monogamous marriage have been associated with lower risk of death for child

and highest increase in child death attributable to AIDS. Cluster wise, the high HIV cluster experienced high child mortality followed by mid HIV regime cluster, while low HIV regime cluster experienced low child mortality.

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ACRONYMS AND ABBREVIATIONS.

AIDS Acquired immune deficiency syndrome

AIM Aids impact model.

HIV Human Immunodeficiency virus

NACP National Aids Control Programme.

NASCOP National AIDS/STD control programme

e₀ Life expectancy.

WHO/GPA World health Organization Global Programme for AIDS.

STD Sexually Transmitted Diseases.

KFS Kenya Fertility and Contraceptive survey

KDHS Kenya Demographic and Health Survey.

CHAPTER ONE GENERAL INTRODUCTION.

1.1 Introduction.

The United Nation lists Kenya among the 15 highest seroprevalence countries in Africa (UN, 1994:ix). Nationally, in Kenya, AIDS is expected to increase the death rates at all ages but the impact will be most severe among young adults and children under the age of five. For instance the annual number of deaths among young adults (aged 15-49) assuming a gradual decline in death rates from other causes, would increase slowly (because of growing population) from about 90,000 today to 100,000 by 2005. However AIDS will dramatically increase this number to 300,000 by 2005 (UN, 1994:58-9).

HIV/AIDS incidence and prevalence depend on the geography, the population affected, the prevalence of the risk behaviour, the practices and the timing of the introduction of the virus. However trying to explain the phenomenon by a single factor such as civil war, male circumcision, STDs or rate of partner change - is - simplistic. Instead it appears that the simultaneous occupance of several risk factors for HIV transmission determines how rapidly and to what level HIV spreads in among a population and who becomes infected.

Most AIDS experts further believe that vast majority if not all, of HIV infected persons will eventually develop AIDS and die from the disease (WHO, 1991) regardless of the length of the incubation. There are studies to suggest that HIV positive patients develop AIDS more quickly in Africa and that they die earlier once they have the disease as they have already been weakened by previous illness (Stato, 1991; Berkley, 1990 and Redfield and Burke, 1980:90-98) ie in Africa AIDS hasten death.

It therefore follows that the impact of HIV/AIDS (on mortality for instance) will depend on the incidence and prevalence of the disease, such that the impact will be more felt where the concentration of the virus is high and vis versa.

Evidence have accumulated that in Kenya, HIV/AIDS is distributed disproportionately based on the sentinel carried out in 13 sentinel centres across the country. The results of such sentinels have revealed that certain centres like Kisumu, the HIV prevalence is over 20 percent of the population; in other areas like mombasa for instance, the prevalence is between 10-20 percent; yet in other areas like Kisii, the prevalence is below 10 percent. From the available evidence arising from these centres overtime, the sentinel centres can usefully be divided into three broad HIV/AIDS regime clusters of high, mid and low HIV regime clusters.

As already been mentioned, the impact of HIV/AIDS depend on its incidence and prevalence in an area and since all HIV cases leads to AIDS and death in a relatively short period of time, high HIV cluster for instance would experience high mortality rate than mid HIV regime cluster and Mid than the low HIV regime cluster. However there is little knowledge on the mortality situation by the prevailing HIV/AIDS regime clusters. The need therefore to capture the mortality situation on the ground against the backdrop of the prevailing HIV prevalence^{regime} clusters taking a recourse to examining the observed HIV/AIDS differentials. It is hoped that the findings of this study will throw some light on the mortality situation in the HIV/AIDS seroprevalence clusters in Kenya.

1.2: Statement of the problem.

Available evidence indicate that mortality continued to decline sharply since 1962 in Kenya (CBS, 1979; Henin and Molt, 1979). Life expectancy at birth for both sexes rose from 40,5 years in 1962 to 55,7 in 1994. This makes Kenya the country with the highest life expectancy at birth and the lowest mortality rate in tropical Africa. However overtime despite any general decline in mortality, mortality differentials continue to persist. Moreover mortality levels in Kenya due to the large population growth rate is expected to rise because of the large number of population exposed to the risk of death, (UN, 1994). Often this mortality have been measured in terms of infant and child mortality, however evidence have accumulated that neither the level of nor the differentials in, child mortality is closely associated with that of adult mortality (Murray and others, 1992), yet overall mortality has attracted limited attention.

The estimation of adult mortality is further compounded by the differentials in the information given on death of parent by male and female respondents. With further evidence that mortality (both adult and child) mainly due to the rising HIV infection has stagnated at high levels, further suggest that adult mortality is not an issue to left to look after itself while attention is focused on child mortality. Besides the largest mortality impact are in otherwise relatively robust years of life, so that a person in the prime working and family care years is more likely to die.

With the advent of HIV/AIDS epidemic, most scholars predicted that the impact of HIV/AIDS will increase mortality so much that it would cause population decline (Stover, 1994:47) and by implication widen the already observed mortality differentials. However this impact has been documented to be more severe depending on the

prevalence and incidence of HIV/AIDS regime clusters (UN, 1994:1; ie high HIV regime areas will have high risk of infection); and the occurrence of the risk factors; like sex (ie girls are infected at an early age due to the imbalance of their age relative to their coitus partners and their wider biological susceptibility).

Moreover, evidence accruing from HIV/AIDS sentinel clusters reveal that HIV in Kenya is transmitted heterosexually (NASCO, 1995.1996) and since age at onset of sexuality is falling while age at onset of marriage is increasing (KDHS,1993), the net effect is the infection of HIV on couples in middle ages, the infection of which they may unwittingly pass to their unborn babies. These risk of infection is more pronounced in the urban areas than in the rural areas with the risk being more pronounced in areas with high HIV prevalence. Evidence further suggest that AIDS leads to death more rapidly in regions with a relatively high prevalence of malnutrition and infection of parasitic diseases (Katlama and Others, 1984; Pape et al. 1993) and the prevalence of HIV regime. AIDS hastens death such that place of birth plays a crucial role in ones infection.

However, demographers and epidemiologists have long been aware that chances of child surviving through infancy and early childhood are primarily determined by the resources available in the childs family, it has also been documented that HIV/AIDS is an opportunistic disease that depend among others on these factors to operate for instance, type of marriage determines the amount of resources shared among family members, such that those in polygamous union have to share the little family resources amongst themselves as opposed to those in monogamous marriage where the resources are shared amongst few members thereby bringing the observed child mortality differentials as children in monogamous families will be more provided for nutritionally than those in

polygamous union. On another level, those in a polygamous union are at more risk of HIV infection than those who are in monogamous marriage due to the multiple sexual partners involved. The net effect is that the children born to women in this marital categories with high risk of HIV infection would themselves be at risk of HIV infection and thereby further reducing their chances of survival and increasing the observed mortality rates and differentials.

The other co factor is the level of education of the mother. Education empowers the woman to make positive decisive decisions beneficial to child survival for instance an educated mother is more likely to question the traditional beliefs and be more hygienic and know the value of attending ante and post natal care than the uneducated mother and thereby reducing risk of death for child. On the other hand, since job opportunities are in towns, the educated migrate to towns to look for job opportunities often leaving their partners behind, they soon develop others sexual partners thus increasing their risk of HIV infection (depending on the prevailing HIV regime) and may pass the virus along to their partners and unborn babies.

Others are the hygienic and sanitational condition of the individual, the family and the immediate environment. For instance access to clean water and flush toilet reduce mortality especially that of infant and child through the reduction of Killer water born diseases and other infections such as bilharzia, cholera etc. The HIV/AIDS virus however complicate issues, the victim of AIDS is always susceptible to acute diarrhoea bouts which often gives the victim little time to run to the toilet, often the victim can also be very weak to walk (Owuor: This study). With lack of water and especially in poor housing toilet conditions, floor material type and availability of clean water, would play a major

role in the reduction of disease infection. For instance cemented floors could be easily cleaned up, while earth made floors would cause a problem often with the sand being sprinkled on the waste (Chepkon'ga, 1997:33), thereby increasing the chances of disease infection.

These factors have enormous potential to cause social strain and change. For instance concentration of HIV infection in the middle age range suggest that mortality due to AIDS will impact on patterns of care giving for both children and the elderly. Besides while families have traditionally provided care for large numbers of extended family members, (Ochalla-Ayayo, 1994) the nature of AIDS epidemic may severely test the conventional practices. Further more because HIV infection in these HIV regime areas is spread both horizontally and vertically potential care givers (spouses, other wives in polygamous households, older children) may also be sero positive severely reducing their life expectations.

At the household level, allocation of household resources will be altered as medical expenditure and funeral costs assume increased importance. Some families may not be able to afford even basic necessities, further increasing infant and child mortality; the children of whom might have been born sero positive in the first place, shattering the remarkable achievements so far made in this area.

As already mentioned these impacts depend on the prevalence of HIV/AIDS and its incidence in a given area. However there is little knowledge as regards the prevailing mortality situation on the ground against the prevailing HIV sero prevalence regions. This study is therefore set to examine the mortality levels and differentials by the prevailing HIV prevalence in Kenya, taking a recourse to examining the HIV/AIDS differentials of the

same.

1.3. Objectives of the study.

This study is therefore set to establish the observed mortality differentials by socio-economic and household environmental factors on child and adult mortality by the prevailing HIV regime clusters of high, mid and low; estimate the relative impact of HIV/AIDS on mortality by these factors taking a recourse to examine the observed differentials in the incidence and prevalence of HIV/AIDS. In more specific terms the study intends to establish:

- 1.3.1: Child mortality differentials by sex of child, level of education of the mother, her type of marriage, and her occupation by the prevailing HIV regime clusters.
- 1.3.2: Child mortality differentials by water source, floor material type, sewage disposal and place of residence by the prevailing HIV regime clusters.
- 1.3.3: Adult mortality differentials by the characteristics of the child and HIV regime clusters.
- 1.3.4: The relative contribution of HIV regime on child and adult mortality by the above mentioned factors.
- 1.3.5: The differentials in HIV/AIDS incidence and prevalence by age, sex, place of birth, and rural-urban and by HIV regime clusters.

1.4. Justification.

The fact that HIV/AIDs will impact mortality is given (though disproportionately), the need therefore arises to capture the mortality situation at an earlier stage before it is seriously impacted by HIV/AIDS. This study therefore attempts to examine the mortality situation in each cluster of HIV/AIDS.

Since HIV/AIDS increases mortality. High level of mortality indicate that those who survive childhood mortality die before they reach old age. Such deaths have a particular high social and economic cost in terms of breakup of families, rise in orphanhood/infant and child deaths, increased funeral expenses which is becoming costly to the families; loss of bread winner and care giver to children and the elderly. Besides where formal social welfare system is poorly developed, this death affects albeit indirectly the quality of health and life of the respective family. Thus the need to examine the mortality situation by the clusters of HIV regime.

Given the rising level of HIV infection which start at onset of sexuality which is a function of age. Implicit from this is the fact that HIV/AIDS occurs disproportionately to those in most economically productive years of life. This death is more painfully because it is at this age that the returns in education begins to pay off. Thus the need to focus on mortality situation by HIV regime clusters more so given the fact that the family members and the victims themselves are stigmatised, fostering in them a feeling of guilt and shame and therefore altering the relation among family members and with the neighbours.

A study of HIV/AIDS differentials is likely to throw some light and act as first step towards a more comprehensive study of the effect of HIV/AIDS and the formulation of

policy to minimise this effect.

Mortality studies of sentinel areas by prevailing HIV regime cluster are of interest as they provide indicators of health differentials that may result from differences in life style and risk factor exposures. Thus the findings of this study may be of use to policy makers in their attempt to curb regional variations in deaths.

From the academic point of view, the findings of this study is expected to contribute towards a better understanding of the prevailing mortality differentials emerging and the factors identified to associate strongly with child and adult mortality in the HIV sentinel regime clusters of Kenya, and those that are impacted more by HIV/AIDS induced deaths.

1.5. Scope and limitation of the study.

The study limits its scope to the estimation of adult/child mortality and differentials, the impact of HIV/AIDS on mortality and differentials in incidence and prevalence of HIV/AIDS in selected HIV seroprevalence regime clusters of Kenya and the calculation of HIV prevalence rates.

In trying to estimate differentials in mortality the study will only consider the HIV sentinel centres representing high, middle and low HIV prevalence rate.

Only the differentials in mortality by socio-economic variables (for instance: mothers education level, marital status, occupation, age, and sex of child); and household environmental factors (for instance: water supply, floor material type, rural-urban, and sewage disposal) will be considered.

Several limitations will hinder the easy collection and accuracy of data analysis. For instance the fact that recent data to the specific purposes of the study are not available makes us use data which is some what out dated. The mortality situation based on the 1989 census would reflect the mortality situation less accurately than would have been otherwise if there was a possibility of employing recent data, an attempt will be made at remedying this by time locating the computed mortality values.

The fact that census data in Kenya is normally coupled with massive under reporting of deaths, misreporting of age, adoption effect on orphanhood data and migration, memory lapse and cultural beliefs is likely to affect the accuracy of results obtained. Besides the sentinel data, the basis for calculation of HIV rates, is only based on women attending antenatal clinics, adjustment factors will be used to derive a more representative value. All these affect the accuracy of data obtained.

Further, the tape for Nyanza Province and Western Province in the CBS was contaminated and therefore data for Kisumu, Busia, Kakamega and Kisii districts were omitted in situations where there was no back up files.

Good data on AIDS related mortality are not available for most developing countries, Kenya included. In Kenya It has been estimated that about 30 percent of the actual deaths may be reported. This can happen for several reasons, some people never seek hospital care for AIDS, some doctors may not want to report a diagnosis of AIDS because of the stigma attached to AIDS, some people return to their villages to die, and their deaths may never be reported. Therefore, reported AIDS deaths cannot be used to estimate the extent of AIDS related mortality. As a result of the limitation of reported AIDS mortality, most estimates of AIDS mortality are based on the estimation of HIV

infection. Data from the sentinel surveillance of pregnant women are particularly useful for the estimating HIV prevalence among National population group. However this data have limitations, for instance it is based on women alone, and women of reproductive age group, who are no doubt sexually active. Adjustment for these factors will no doubt affect the quality of the study data.

CHAPTER TWO

LITERATURE REVIEW AND THEORETICAL FRAMEWORK.

2.1. Introduction.

AIDS results in a gradual and progressive impairment of the human immune system and the consequent emergence of recurrence of a variety of infections and neoplasms. HIV, the causative agent of AIDS, is a virus belonging to the family of retro-viruses. The virus was first identified independently in 1983-1984 by team of researchers in France and United States Of America. It was called lymphadenopathy-associated virus (LAV) by the French team and Human T-lymphotropic virus III (HTLV-III) by the team in USA. This term are now absolute and the Virus is now referred to as HIV-I or simply HIV (Gello and Wung-Steel; 1985, Galo and Montalgner; 1988).

In 1985, another retrovirus that could cause AIDS like illness was identified, at first called LAV-2 by the researchers in France and HTLV-IV by those in the USA, the virus is now referred to as HIV-2. The genetic material of HIV-2 is very similar to that of HIV-1, although HIV-2 has been Isolated from the body fluids of patients with AIDS, the clinical manifestation of infection with this new virus are not yet completely known (UN 1989:21). In Kenya HIV was first diagnosed in 1983.

The mode of transmission for HIV-1 and HIV-2 are fairly limited. These modes include virginal and anal intercourse with infected partners, infections and intravenous inoculation with contaminated needles or syringes, administration of infected blood or blood products though transfusion and perinatal transmission from mother to child (Curran, 1985:656; Friedland and Klein, 1987:1125-1135). If the efficiency of HIV transmission is measured by the probability of HIV infection following a single exposure

of a susceptible person to the infected substance (such as blood, semen or vaginal fluids), blood transfusion would rank most effective, followed by perinatal transfusion and infection with contaminated needles as a less effective mode.

After entering the human host, HIV attack certain cells of the immune system. In particular a subset of a white blood cell called T4 lymphocytes (also known as helper T cells) that play a crucial role in the coordination of immune response. T4 lymphocytes cells surface contains a molecule (CD4 Molecule or CD4 Antigen) through which HIV gains entry to the cell. The CD4 molecules are predominantly found in the T4 lymphocytes and these lymphocytes are therefore the prime target of HIV (Haseltine and Wong-Staal, 1988:52-62; Weber and Weiss, 1988:100-109).

As the immune system is increasingly disabled the outcome is an increased vulnerability to a variety of opportunistic infections caused by certain viruses, bacteria, fungi or parasites that would not normally cause disease in healthy persons. Evidence indicate that once AIDS occurs the disease is almost always fatal within a relatively short period of time (Redfield and Burke 1988:90-98).

The global HIV/AIDS epidemic consists of many separate individual epidemics, each with its own distinct characteristic that depend on geography, the population affected, the prevalence of risk behaviour and practices and the timing of the introduction of the Virus. No single factor, biological, or behavioral determines the epidemiologic pattern of HIV infection. Instead a complex interaction among several variables determines how and where HIV spreads in a population. However biological factors do influence the spread of the epidemic by increasing or decreasing susceptibility to the virus, altering the infectiousness of those with HIV and hastening the progression of infection to disease and

death. Such biological factors include the presence of classical STDs, male circumcision and the viral characteristics of both HIV-1 and HIV-2 and their multiple genetic strains (Cohen & Trussell (ed), 1986:4). However trying to explain the phenomenon by a single factor such as civil war, male circumcision, STD or rate of partner change - is - simplistic. Instead it appears that the simultaneous occurrence of several risk factors for HIV transmission determines how rapidly and to what level HIV spreads in among a population and who becomes infected. It should be noted here that the population of Kenya is predominantly young in sharp contrast to developed countries, and many of the behavioral factors associated with HIV transmission are common among young people.

The first AIDS cases was diagnosed in Kenya in 1984 and by August of 1993, the National AIDS Control programme (NACP) had reported 39,000 cases of AIDS. The reported cases only represent the visible part of the disease. It is believed that the true number of AIDS cases is three times the reported cases. If current trends continue the AIDS related deaths will rise from 20,000 in 1990 to 86,000 in 1996.

Concerning the subject matter of this study, articles extracted from journals, literature from empirical researches, theoretical exposition and official documents provide the basis upon which the literature review is based. That literature touches upon two major themes around which the discussion of this chapter revolves.

2.2. Mortality levels and differentials.

Although inequalities in mortality in developed countries have been a major fact of research, very little is known about the size of such differentials in developing countries populations. The study of differentials in mortality requires accurate measurements of levels in sub-populations to determine differentials and trends as well as careful detection of the variables closely correlated to mortality. An accurate measurement requires a strict matching between deaths and population at risk. Various scholars have studied this topic using a number of variables which will be reviewed below:.

All societies categories their people into dominant and marginal groups, these classification are frequently based on class, ethnicity, age and gender. These marginal groups, suffer social discrimination underpinned by discriminatory practices which work toward making these people economically disadvantaged among others bringing about mortality variations.

Das Gupta, (1989:349-65); for instance reviewed the evidence in health status and mortality and found that the differentials in health status and mortality suggest that the results from societies lesser degree of interest in ensuring the welfare and survival of its more marginal members as compared with its more dominant one.

Chen (1982:163-190), took the argument to the community level arguing that substantial mortality differentials may exist among different geographical, ecological, and economic regions within a country with disadvantaged regions exhibiting higher mortality than the economically advantaged ones.

Studies done in Sri Lanka, Mauritius, Taiwan and Cuba have confirmed Gupta's

finding but emphasize the importance of not only medical technology but also equitable distribution and accessibility to the people (Iusley Raymond and Others, 1993:444-9; Preston, 1979) especially mother and child health services (Neg, 1985:67; UN, 1985:281; Caldwell, 1975:263-267; Ewbank 1993:64-72). For instance, the 1985 UN comparative study of 'effect of Socio-economic factors on child Mortality', indicated that in Nigeria and Peru, both access to and utilization of health services were positively related to Child Survival (UN,1985:281).

However, in Kenya, availability of health services measured by the number of hospitals and dispensary beds per capita at district level was found not to be significant determinant of Infant and Child mortality rate in Kenya between 1969 and 1979 (UN, 1986:53; Anker and Knowles, 1977:24).

It is often surmised that education imparts knowledge of the association between; hygiene, cleanliness, and health protection -"the germ theory of disease causation". That Education fosters upward social mobility, behaviour and identification with the modern world and its values, such as: a clean house, neatness, and better personal hygiene. Such behavioral pattern not only serve to convey higher social status but also generate beneficial health resulting in better nutrition and care and lower mortality.

Hugo, (1978) for instance contend that maternal education is a proxy of the general level of living rather than a factor of its own. However, Caldwell disagrees, maintaining that the mothers education is not proxy nor a reflection of a household standard of living but has an important independent effect. He suggest that education enhances a mothers ability to provide adequate Child care by challenging her traditional

notion of disease and by altering family relationships such that an educated mother has more weight in the decision making process relating to the care of her Children. She (an educated mother) is said to be less fatalistic, better able to deal with the modern world and more aware of simple hygienic measures (Caldwell, 1979:408-413; 1989:396).

In an attempt to justify the above contentions, Cochrane and her colleagues, (quoted in Veland and Ginneken, 1989:79-100) based on several national studies, estimate that for every additional year of maternal education schooling the child mortality rate fall by approximately six per thousand live births, whereas for every paternal schooling, its reduced by 3/1000.

However there is a salient emerging dissent to this view, for instance Lindershaun, study based in a rural area of Bangladesh found contradicting results. The study found that both illiterate women, and majority with some education except for a few with more than primary education tended to share traditional beliefs about the causation of illness; neither did they differ in attitude towards the appropriateness of various types of interventions.

She maintain further, that the only difference between educated and uneducated mothers was that educated Mothers emphasized cleanliness an attribute which they acquired while at school (Lindershaun, 1990:423). However Cleland (1990:400-420) discounts this, arguing that it (Lindershauns study) lacks substantive empirical evidence; He maintains that maternal education enhances knowledge about effective ways to prevent, recognize and treat Childhood diseases.

Several studies have supported these views, Casterline, et al (1989: 25-29), study in Egypt, Majumber, (1989: 109-11), study in Bangladesh and Garsey, (1992:4)

study in Turkey, all of which found maternal education to have little or no significant effect on infant and child mortality. The analysis of the 1988 Uganda Demographic and Health Survey for instance showed that maternal education had no significant effect on Infant Mortality (Ebony, 1993:1).

Studies done in Kenya however supports the former view, (see: Ewbank, 1986:48; K'Oyugi, 1982; Ondimu, 1988; Kibet, 1989; Kichamu, 1988; Muganzi, 1984; Ondimu, 1988; Eubank, 1986:48). K'Oyugi, (1992) for instance looked at the impact of household and community level environmental factors on Infant and Child mortality and found that maternal education of under 9 years has a significant protective effect on infant and child mortality. However K'Oyugi's study was based in rural Kenya.

Moreover, in developing countries, recent studies have suggested that infant and child mortality is strongly associated with maternal education than with any other socio-economic variables, (Behm, 1980; Caldwell, 1979:396; 1984:108 Cochrane,1980; Caldwell and Meegama,1981:75-76; Mosley 1989:274). The same argument hold for adult mortality, for instance, Doornbar and Kromhout studied a cohort of men born in 1932, examined for military services, using life table analysis. They found that there is an inverse relation between educational level and survival.

United Nations, (1973); Batim, (1983) and Kaala, (1989); contend that currently married women's children and currently married spouse have a better mortality status than any other marital category. Batim, (1983), for instance reports in a study based in Bangladesh, that the mortality of children of widowed or divorced mothers was higher than currently married ones. A symmetric relationship holds true for adults, for instance

Kaala, (1989); studied marital status for the republic of Germany for the period 1961-86 and concluded that death rates of married persons persist to be lowest whereas that of once married persons remain the highest.

The possible reason for this could be due to the stress associated with widowhood and divorced, which in turn affects their health, and that of their children. Affects their financial situation (Tanbamen and Rosen, 1981:11), or are culturally mediated. Mosley and his associates, (1988:274) for example reported that newly separated or widowed women among the Imensi of Nigeria ventured with the children to her parents family and remained there until, she remarried which gives her child higher probability of survival.

Findings based on different Kenyan data confirm the aforementioned differentials (see, for example: Kichamu, 1981, K'Oyugi, 1982; 1992; Ouma, 1991; Ayehu, 1993). Kichamu, (1981:49-51); for instance found that the widowed had the highest mortality followed by the divorced and the married, then the single registered the lowest, while K'Oyugi (1992:v) found polygamous marriage unions to have a significant negative impact on Infant and Child mortality.

Nyamwange, (1982:1-90); in his study on infant and Child mortality differentials in Nairobi found that migration status (place of residence) of the mother in general plays an important role in determining the mortality levels among a community at the place of destination. Those migrating from high Infant and Child mortality areas induce higher rates in the destination where they settle than those from lower mortality areas.

The effects of occupation on mortality has also been documented (UN, 1986:50).

Martikunen, (1990:407-411), examined the relationship between unemployment and mortality among Finish men, controlling for background variables including age, Socio-economic status, and health. The study found unemployment to have an independent causal effect on male mortality. While, Blanc, (1990:429-32) found same evidence to exist in Britain. The possible reason for this could be that the fathers occupation could reflect Income differentials and possible association with socio-economic and cultural factors complicate the determination of the Independent effect on child mortality.

Analysis of mortality rates by fathers occupation using data from Kenya Fertility Survey found that those with highest mortality risk were the children of agricultural workers who worked mainly at home, their reported child mortality rate was about 57 percent higher than that of professional cadre of workers even after controlling for maternal education. Also there was insignificant differences between child mortality rates between professionals and clerical cadre occupational categories.

It is generally evidenced that females have better survival status than men. In Africa less is known about gender differentials in adult mortality. Given the range of different data sources and methods used to make these estimates the results suggest that female have better survivorship in most sub-saharan Africa (Timeaus 1993). This is similar to those found in other parts of the developing world (UN, 1982a) and general families of princeton model life-tables (Coale and Demeny, 1982). Timeaus for instance found malawi and mali to have excess female mortality while the situation is reverse in South Africa (ie men have excess Mortality) (Timeaus, 1993:220).

In Kenya, Blacker argues that variation exist between districts within many

provinces, regionally all Districts in Nyanza province for instance show a higher female advantage in e_{20} than is found in other districts at similar level of Mortality. At a value of e_{20} of 42 years which is typical of Nyanza, shows a female advantage of 3.9 years. However this could be due to data errors, cultural practices and aggravation of sex differentials by environmental factors. Ronoh (1982) on the other hand found that female life expectancy at most ages were highest in Nairobi and Central province and lowest in Coast and North Eastern provinces. As regards infant and child mortality, male children tend to have high mortality than female children (KDHS, 1983).

Residence in rural areas in developing countries has been associated with higher infant and child mortality risk than residence in Urban areas (Gaisie, 1980:24; Trussell and Hammerslough, 1983:14-17; Caldwell and Ruzicka, 1985:200). For instance within countries, Timeaus (1993:218ff) found that mortality is lower in Urban Africa than in the countryside with the differentials being larger in the countryside indicative that the advantage of Urban areas is eroded as overall mortality declines.

Gaisie (1980:24) attempts to explain this, arguing that rural-Urban mortality differentials in Developing Countries are attributable to distribution of health technology, sluggish economic growth, poverty, malnutrition, poor housing, unhealthy environment and low levels of education that disadvantage rural population. Perhaps Behm and Vallim, (1980:29), summarizes this well, contending that, the population per-se does not explain rural-urban mortality differentials rather the differentials are a reflection of the differences in Socio-economic standards of the population in question.

In Kenya, (Muganzi, 1984:41; Ewbank et al, 1986:52), found Rural Infant mortality

to be slightly higher than urban Infant mortality, despite the fact that rural-urban survival rates were more or about equal (34.2 and 34.3, for rural and Urban areas respectively). However, Ewbank et al (1986:52) analysis showed that the rural-urban differentials in infant and child mortality disappeared in the presence of controls for a variety of factors.

The same argument holds true for adult mortality In Kenya, Blacker for instance argues on the basis of information collected using orphanhood data in 1969 and 1979 that, variations in the level of adult mortality is not restricted to the provincial level, that wide variation exist between districts within many provinces.

The importance of water supply and sanitation as the determinant of differentials in mortality has been documented in developing countries such as in Pakistan (Federal Pakistan Bureau of Statistics, 1986:30); In Brazil (Merrick, 1985); In latin American cities (Puffer and Serramo, 1973); and in Sri Lanka (Meegama, 1980). Meegama, (1980), for instance concluded that public health measures such as improved sanitation were important household factors associated with child mortality and gives example of Sri Lanka. Da Vanzo (1983) quoted in Ikimari, (1996:17) in their study in Malaysia, indicated that the infant mortality of infants born into households with piped water was better than those obtained from other sources such as wells, rivers or canals. However, Haines and Antery (1982) disagrees contending that sanitation has little effect on infant and child mortality, giving documentary evidence of Mexico, Puerto Rico and Costa Rica. Further, Shamba (1995:144-5) supports Haines and Ankley contention, in a study in Zimbabwe he found weak negative association between under five mortality and sanitation.

In Kenya, K'Oyugi, (1992:iv); in a study in rural Kenya found that less

contaminated water have statistically significant negative association with the risk of infant and child mortality. However K'Oyugi's study was biased towards rural areas of Kenya while Meegama's study underscores the importance of variation within cities.

Crowding as a household and community environmental factor has been found to be important in the spread of infectious diseases. There is a strong likelihood that Crowding within households may constitute a risk factor in diarrhoeal and some respiratory disease mortality since all the bacterial and viral agents of these diseases can be transmitted by person to person contact. Aaby, (1987), for instance concludes that crowding within households resulted in extensive exposure and larger dose of infective virus which in turn was the major determinant of measles severity and mortality. In Pakistan, the principle causes of Infant deaths after the first month of life are acute respiratory infections gastroenteritis, other communicable diseases and malnutrition (Awan, 1986:188). In Kenya K'Oyugi argues that transmission of the diseases such as influenza for instance can occur when sleeping arrangements of children are poor (K'Oyugi, 1992:10).

There are studies that have argued that malarial control has significant reduction effect on infant mortality. Malineaux, & Grammica, (1980-quoted in K'Oyugi 1992:20) for instance reported that the Garki District in Northern Nigeria, infant mortality rate declined from 245 per thousand live birth before malaria intervention project to 55 in intervening years.

However Garnham, (1935), using data from Kenya, the Blackbok and Gordon,

(1935) study using data from Sierra Leone and the recent study by Mc Gregory et al (1983) using data from the Gambia, have disagreed with this contention arguing that the difference between mortality in malaria and non-malarial zones are relatively small due to malaria immunity among children in malarial zones. Graham and Blackbok, and Gordon study are old while the Gabons study by Mc Gregory and associates suffer from problem of variability.

In Kenya, the areas in the Coastal belts and Lake Victoria basin are malaria endemic and are also high mortality areas (Kibet 1981; K'Oyugi 1982; 1992). K'Oyugi (1992:v) for instance concludes that malaria had a significant negative effect on child survival rates, while Anker and Knowles (1980:182), contend that in Kenya children born to families residing in malaria areas had a lower (by about 7 years) life expectancy at birth than children born to families residing in areas where malaria was less common or non existent.

Available evidence mainly from case study projects undertaken in developing countries show that a considerable proportion of deaths, especially those of children, are attributed to immunizable diseases such as tetanus, measles and pertussis (Voorhoeve and associates, 1978:125-139; Nag, 1985:67; UN, 1985:281, Ewbank, 1993:64-72; Handayan et al.,1983:88-92). This is also true of Kenya for instance in the Machakos project, a rural population of 2,400 were followed prospectively for three years and pertussis was found to account for 4.1 percent of all deaths in the 0-15 years of age (Voorhoeve:1978:35).

The effects of toilet facility on mortality levels in a given household has been documented. Such that Martin et al (1983) argue that access to toilet facilities has been one of the factors that contribute to the decline in Infant mortality in Philippines (Martin et al.,1983, the case has also been documented in Malaysia (Da Vanzo and Hobacht,1986). For instance, in India, Katiyer, reports that morbidity was higher among children where Families defected in the fields (Katiyer, 1981:545-550),

2.3 HIV/AIDS Prevalence and differentials.

The official number of Acquired deficiency syndrome (AIDS) cases worldwide since the start of the epidemic passed the one million mark near the end of 1994 (Cohen and Trussell, 1996; UN, 1996). This number however don't include millions of people with the human immune deficiency virus (HIV) but have yet to develop symptoms of AIDS. The world health organizations global programme on AIDS (WHO/GPA) for instance estimate that by mid 1995, there were more than 20 million infected with the virus Worldwide. As noted by Chen, (1991), however, HIV infection are not randomly distributed in any human population: Africa's infection accounts for a disproportionate share of the World total. Followed far behind are the Americas; south and south-east Asia (Primarily India, and Thailand); and western Europe. As of late 1992, other areas of the world are believed to have low rate of HIV infections.

By January,1992, nearly 2.5 million people had died of AIDS, including 1.9 million adults and more than 550,000 Children. Of these three fourth have been in Africa and nearly 20 percent in the Americas.

However, sub-saharan Africa is geographically, demographically, socially and

culturally heterogeneous and the extent and spread of HIV infection and AIDS, have accordingly been heterogeneous as well. This makes it difficult to generalize about the AIDS epidemic in the region. Fieldman (1991) for instance contend that the values, attitudes, beliefs and sexual practices within sub-saharan Africa are both complex and heterogeneous, which suggest that differences between the groups are likely to be important determinant of the size and the sustainability of the epidemic.

The most afflicted countries are geographically concentrated; Other than Cote d'Ivoire in the west Africa, they lie in a region of east and southern africa that stretches from Uganda and Kenya southwards to include Rwanda; Burundi; Tanzania; Malawi; Zambia; Zimbabwe and Botswana (Trussell and Cohen, 1996).

Among the salient factors that affect the size, shape and variation of the HIV/AIDS epidemic in sub-Saharan Africa are the age and gender composition of the population; the pattern of sex roles and expectation within Society, inequalities in gender roles and power, sexual access to young girls and the widespread differentials in the life of sexual partners; rapid Urbanization, under the condition of high unemployment, poverty, considerable transactional sex fostered by limited earning opportunities for women, and lack of access to health care, particularly treatment for various STDs. These factors are often exacerbated by social upheavals related to economic distress, political conflicts and wars.

Various researchers have studied the aforementioned factors using different data in various regions some of which will be reviewed below:

Virtually all AIDS cases reported in Africa have been in heterosexual who are not IV drug users (Quinn and others, 1996). Consequently the male to female ratio of AIDS cases in Africa as of 1, January 1992, has been close to unity, whereas in northern America and Western Europe that ratio has varied within a range of from, 14:1 to 20:1. In the caribbean and in parts of South America, the profile of reported AIDS cases is somewhere between that of North America and that of Africa. The profile in Asia and Eastern Europe are less well known, but they are likely to be a combination of those observed in Africa and North America (Mann et al., 1996:312-3; UN, 1994:10).

However, inspite of the fact that the findings of the empirical research on the matter are far from being definite, there are some evidence supporting the idea that, *ceteris paribus*, the probability of male - to - female transmission is higher than the probability of female - to - male transmission. This inequality has been largely attributed to differences in the rate of exchange of fluids and to the sheer volume of the virus that can be hosted in the semen in contrast to the Virginal fluids. In the absence of offsetting factors this difference alone could account for a heavier presence of HIV among females in some age groups (QUIN and Others, 1986:955-963; Palloni and Ju Lee, 1992:72).

In Kenya, the National AIDS Control Programme reports, (1996:17) that the ratio is close to Unity. The possible explanation for this could be that; while males may be more likely to engaged in casual sexual contact than females, the risk of HIV infection for women, depend not on their own sexual practice but on those of their partners as well, further more females have higher susceptibility to infection.

Within urban areas, AIDS has been explained in terms of loosening of norms

governing pre-marital sex, lack of relative negative sanction against promiscuity and the numerous opportunities for making contact with potential sexual partners within a setting of anonymity and freedom to misbehave (Barkley, 1992; Killewo and others 1990; Ochola-Ayayo; 1990;1996). Killewo and Others, (1990) for instance maintain that the level of HIV infection is higher in Urban areas and particularly more so in the Capital Cities (Bongaarts and Way, (1989:374). Chin, (1991); estimates that HIV seroprevalence among sexually active adults in Urban areas in some parts of sub-saharan Africa is 20-30 percent about 10 times that of the rural areas. Notable levels of HIV infection have also been chronicled in rural areas serviced by heavily travelled roads or with high level of rural- Urban migration (Ocholla-Ayayo, 1996).

In Kenya, sentinel done in 1995, found that in some densely populated districts, rural prevalence rates may approach, or even exceed the Urban prevalence rates. In other less densely populated districts, the rural prevalence rates may be only one third or less of the urban rates. The urban rates is estimated at 13 to 14 percent although there are differentials, Nakuru for instance had prevalence rate of 30 per cent in 1994 while, Thika had 19.6 per-cent (an increase from 9.9 and 2.5 in 1990 respectively). Rural sites however on average have prevalence rate of about 5 to 6 per cent with rates much higher in densely populated areas (NAS COP, 1995:5-8).

Although the empirical evidence is murky at best and the direction of the causality is somewhat unclear, It is widely believed that sexually transmitted diseases exacerbates and enhances infectivity (Plummer and Others, 1987: abstracts s.9.3; Kress and Meheus,

1988:1-2). Thus we would expect that in areas with high STD prevalence, the HIV prevalence exceeds levels observed in areas that are STD free (Palloni, 1992:72). Berezin, (1992); further observes that "the relationship between HIV and other sexually transmitted diseases appear to be both highly dynamic and synergistic. Persons who have histories of STDs are at increased risk of acquiring HIV, while HIV infected persons are likely to have greater susceptibility to infection with other STDs and, if co-infected, may experience them in an unusually severe and protracted course".

Similar evidence has been documented in Kenya, largely those that produce genital ulcers with chancroid probably playing a key role (Cameron et al., 1989:403-7; Plummer et al., 1991; Piot and Holmes, 1990:894-900). Further more, Undugu society randomly tested 22 female street children in their early teens for HIV. The study found that 21 of the 22 girls had more than one STD, including gonorrhoea, syphilis, and candida. There ^{was} corresponding findings for boys. When tested for HIV however, more than a quarter of these teenagers tested HIV positive (Mutemi and Saoke, 1995:47).

There is also considerable evidence that differentials in levels of infertility follows variations in prevalence of sexually transmitted diseases, ^{for} for instance Frank (1987) found highest rates of infertility in Central Africa and parts of eastern Africa, where HIV-seroprevalence levels are also generally very high. In an attempt to explain this, Ocholla-Ayayo (1996), maintain that in most African societies, if one spouse is infertile, the partner is encouraged to try outside, (mostly with in-laws), this invariably exposes the former to various infection which could include HIV virus.

Lack of male circumcision also appear to be positively related to increased risk of HIV infection (Diallo et al., 1992; Serwadda et al., 1992; Bongaarts and Way, 1989:373-9). This could be due to the increased viral survival under the foreskin and the more frequent occurrence of balanitis (Inflammation of the glans penis) (Piot and others, 1988:573-9; Fink, 1986). Ezel agrees with this but adds another dimension, He argues that female circumcision is also a contributing factor (Ezel, 1990), however Chin discounts both views, maintaining that the practice is not widespread in the areas where infection is most prevalent (Chin 1991).

Bongaarts and his colleagues, (1989:373-7) goes further to give the statistical evidence, they report an extremely high correlation of 0.9 between male circumcision status of sub saharan African ethnic groups and HIV-1 rates in Capital cities of the Countries where each ethnic group lived (see also Moses et al., 1990:693-7). However Vincenzi; (quoted in Caldwell and Caldwell, 1993:3) disagrees with this view arguing that proof can only be established by studying otherwise homogenous populations, where by chance some are circumcised and others are not. Besides, Bongaarts and his colleagues used Murdock's (1967) ethnographic atlas to base their study, the atlas of which is a bit old, the question also arises on how pervasive is the practice of non-circumcision among the ethnic groups in which it is a tradition.

In Kenya, research has also been done in this area, for instance in mid 1980s, the researchers became aware that the uncircumcised men from Western Kenya mostly Luos were more likely to be sero-positive than the circumcised Men from central and eastern Kenya (Plummer, Moses and Ndinya-Achola, 1991, Quoted in Anarfi, 1994:5).

Most scholars believe that HIV prevalence begins with the onset of sexual activities but mostly disagrees on the peak ages, for instance, Quinn and Others, (1996) contend that when HIV is transmitted mainly through Heterosexual contact, the maximum levels of sero-prevalence are generally found among young adults and very young children. Some researchers further argue that in the absence of countervailing influences the consequent mortality increase will invariably affect those same groups but at slightly older ages (Palloni and Ju Lee, 1992; UN, 1994; 1996). Rwandan sero prevalence study group (1984) reports the peak near age 35; Killewo and others, (1990) discounts this, He observes that the highest age specific seroprevalence is in age group 25-34 for both sexes, while Berkely, (1992:22-30) comes with the synthesis of the two, arguing that the rates peak in age group 20-24 for women and age groups 25-29 for men. Such evidence also exists in Kenya, (Nascop, 1994, 95, 96). Nascop rates however embraces all the three contentions, they report that young adults and children under five (1996:14, NASCOP, 1995:24).

It has been evidenced that shorter latency period is observed in infant, with most HIV-Infected infants developing AIDS before age five. Further cohort studies of HIV-infected adults in the developed countries estimate that 60-90 percent of those infected could develop AIDS in 10-15 years. (Carealand Piot, 1990; Rutherford at al., 1990). In Africa it has been argued that latency period between exposure to HIV and development of AIDS and between manifestation of AIDS and death is shorter because of prevalence of other infectious agents (Stoto, 1991; Berkley, 1990:20-30). Most AIDS experts believe that vast majority if not all, of HIV-infected persons will eventually develop AIDS.

and die from the disease (WHO, 1991) regardless of the duration of incubation (Chin, 1992).

However there are serious under reporting of AIDS cases and deaths. Particular characteristic that contribute to under recognition, under diagnosis and under reporting of AIDS cases include among others: the fact that AIDS is a new disease with a wide variety of manifestations, the diagnosis of AIDS further keep on changing as more is learnt about the disease, further the laboratory ~~the laboratory~~ facilities to carry out required diagnosis test are not available in many developing countries. Lastly because of the stigma associated with the disease there may be reluctance on the part of the physician to make a diagnosis of AIDS, and also the reluctance on some government, family members etc to acknowledge officially that AIDS existed in their countries or families (WHO,1988).

In Kenya NASCOP, gives the reasons to be: some people never seek hospital care for AIDS, doctors and health workers may not want to record a diagnosis of AIDS because of the stigma attached to the disease, people with HIV infection may die of other infections before they are ever diagnosed as having AIDS; and some health care facilities, especially in rural areas, may not have the capability to test HIV infection (Baltazar et al., 1996 in Forsythe & Rau (eds), 1996:16)

2.4. Summary of literature review.

From the foregoing two themes in literature review, it is evident that Demographers and epidemiologist have long been aware that chances of child surviving through infancy and early childhood are primarily determined by the resources available in the childs'

family. In a nutshell the pattern of mortality differentials is determined by the interaction of the disease ecology with the Physical environment on the one hand and the broad aspects of culture and social structure on the other. Indeed, Eubank, Henin and Kekovole (1985:33-86) argue that determinants of mortality treads and differentials can be usefully separated into social and economic factors viz income, employment etc., demographic (eg; age, sex, marital union etc), and health services. Although these are mainly interrelated they are often associated with different strategies for reducing mortality.

Various studies have been conducted in these areas, in developing studies, these studies have emphasized the inverse association between mortality and various socio-economic factors and underscored the significance of these factors in mortality levels existing in different segment of the population (Preston, 1980; Cochrane, 1980; Palloni, 1985; Caldwell, 1979). In Kenya such relationships have also been emphasized (see: Anker and Knowles, 1978:59-88; Mosley, 1983; Munganzi, 1984; Ouma, 1991; K'Oyugi, 1992; Ayehu, 1993). Mosley, 1983, for instance argues that much if not all of the geographical differences in mortality in Kenya are as a result of regional differences in the level of economic development. while Kizito (1985) and Nyokangi (1984), are of the view that environmental, Demographic and Socio-economic factors are likely to affect the chances of death at any age in a given District/province.

As regards HIV/AIDS, the main issues arising from the literature review are: The global HIV/AIDS epidemic consists of many separate individual epidemics, each with its own distinct characteristic that depend on geography, the population affected, the prevalence of risk behaviour and practices and the timing of the introduction of the Virus.

No single factor, biological, or behavioral determines the epidemiologic pattern of HIV infection. Instead a complex interaction among several variables determines how and where HIV spreads in a population. However biological factors do influence the spread of the epidemic by increasing or decreasing susceptibility to the virus, altering the infectiousness of those with HIV and hastening the progression of infection to disease and death. Such biological factors include the presence of classical STDs, male circumcision and the viral characteristics of both HIV-1 and HIV-2 and their multiple genetic strains (Cohen & Trussell (ed), 1986:4). However trying to explain the phenomenon by a single factor such as civil war, male circumcision, STD or rate of partner change - is - simplistic. Instead it appears that the simultaneous occurrence of several risk factors for HIV transmission determines how rapidly and to what level HIV spreads in among a population and who becomes infected.

Among the salient factors that affect the size, shape and variation of the HIV/AIDS epidemic in sub-saharan Africa are the age and gender composition of the population, the pattern of sex roles and expectation within society, inequality in gender roles and power, sexual access to young girls and widespread differentials in life of sexual partners, rapid urbanization under the condition of high unemployment, poverty, considerable transactional sex fostered by limited earning opportunities for women and lack of access to health care particularly treatment for various STDs.

2.5: Theoretical Framework

Against this backdrop therefore, the partial task of this study was to come up with a theoretical structure that would capture the mortality situation by the various HIV

regime clusters in terms of mortality levels, differentials, the impact of HIV/AIDS on mortality, and the differentials in HIV/AIDS incidence and prevalence and to assist in the discussion of the held hypotheses.

Typically, the conceptual models developed by demographers place great emphasis on demographic factors (age of the mother and her parity, for instance) and on social and economic variables. In some instances, however, the role of institutional developments is also recognised.

One of such model which this study borrows from is the conceptual framework developed by Pool (1982) which he used in the analysis of non Maori mortality trends in New Zealand. Pool discerned three types of variables which he assumed played a role in mortality transition in that population: The macro level variable; the micro level variable and the intermediate variables. His basic proposition was that ultimately, the differentials [in mortality] are explained by status level of individual or differences in the socio-economic level of the societies. The same assumption holds for other basic needs such as lifestyles, access to medical care and housing etc (Pool, 1982, in Ruzicka, 1989:5-6).

2.5.1: POOLS CONCEPTUAL MODEL.

The examinatory variables included in Pools model at the micro-level are: Health care infrastructure and technology (hospitals, clinic, ambulance services; high-cost technology: X-rays, clinical and surgical equipment; low cost technology: chemotherapeutics, vaccines, Insecticides); Health administration and health regulation (organization and delivery of health care, bureaucratic supporting structures; acts and regulations relating to sanitation); Environment; Socio-economic and Socio-Cultural

organization and change (socio cultural is in pools study primarily related to ethnicity but could encompass religion and fertility behaviour; 'socio-economic' includes; social organization and change, wealth, income, education, economic change loosely defined, the system of production and distribution of commodities, especially those which meet the "basic needs, political change); Sanitation (sewerage, water purification and distribution, and so on, and the construction and implementation of sanitation).

The micro-level variables include: Medical practice; Hygiene (personal; in the household; in the immediate environment); Personal life style; individual socio-economic status; socio-cultural and biological characteristics ("bio-social" being defined as age, gender and parity); genetic factors.

The intermediate variables in the model are: preventive medicine and health measures; curative medicine and health measures; disease mechanisms.

The system however appear to give little explicit role to biological factors; thus for instance nutrition~~al~~ does not appear on the list of bio-social explanatory variables. Further the model does not spell out the causal pathways of the effects of the variables.

On another level few attempts have been made to model the impact of HIV/AIDS on demographic factors. Most of the already formulated models have been based and/or solely focused on the determinants of HIV/AIDS such as behavioral, socio-economic and cultural factors like condom use, prostitution, rate of partner change, wife inheritance etc. One of the model on HIV/AIDS which focuses on the impact of HIV/AIDS on demographic parameters and which this study borrows from is that developed by Ocholla-Ayayo (1995).

The model assumes that social order forms the background variable which affects the intermediate variables (viz: social factors, demographic factors, cultural factors and economic factors) through proximate determinant (ie the timing of biological factors and psychological exposures) to bring about: fertility levels and differentials, mortality levels and differentials and migration volume in the impact area.

2.5.2. ABCs CONCEPTUAL MODEL.

In ABCs model, among the variables included in ~~ABCs-model~~ included in the intermediate variable include: social setting, social opportunities and education as social factors; sex, age, parity and marital status as demographic factors; others are age at first marriage, marital status, ideology of gender issue of children, ideologue of family size, family unit and kinship and family stability as cultural factors while poverty, bride wealth, commercial sex, market dependency and income (purchasing power) as economic factors. Among the variables included in the proximate determinants are the timing of the cycle, coital frequency, sex, age, nutritional status and health status and the psychological exposures.

The model however does not include the household environmental and sanitation factors which have been documented to influence demographic factors and which happens also to be the co factors for HIV/AIDS impact. For instance, lack of water and proper sewage disposal could trigger infection on an HIV sero positive individual, this could lead to the development of AIDS and death or to a fatal infection to the rest of the family (for instance in the case of an outbreak in cholera).

In order to achieve the objectives of this study, Pools model and ABCs model will be synthesized and slightly modified in view of the ease of the availability of data and the factors outlined in the literature review. Thus in Pools' model health administration and health regulation have been left out altogether because of data problems and the environmental factors have been merged with sanitation factors and hygienic factors to come up with household environmental factors, while socio-economic and socio-cultural factors have been merged and renamed Socio-economic factors. However the left out variables was used in the interpretation of the results.

Further in ABCs model, HIV clusters was taken as a measure of social structure and therefore forms the study's background variable, the intermediate variables in ABCs model have been merged together in view of the availability of the data and have been renamed broadly: Socio-economic factors, while fertility levels and differentials and migration volume outcome variables have been left out altogether because they are out of the scope of this study. Lastly, the reported HIV/AIDS incidence and prevalence was incorporated in the model, as a new set of variable.

The argument of the synthesized model, therefore was that: The prevailing HIV regime cluster mediated by proximate determinants will influence the impact of AIDS on mortality, the observed mortality differentials and the incidence and prevalence of HIV/AIDS differentials, through household environmental factors, and socio-economic factors.

From the above the following proposition could be further formulated:

1. That household environmental factors are likely to affect the risk of death at any age disproportionately by HIV regime clusters.
2. That socio-economic factors are likely to affect the chances of death at any age disproportionately by HIV regime clusters.
3. That HIV/AIDS regime cluster is likely to affect the observed differentials in the incidence and prevalence of reported HIV/AIDs disproportionately.
4. That HIV/AIDS regime cluster will influence the impact of AIDS on mortality disproportionately by each HIV regime cluster.

The paradyns arising from the conceptual hypotheses is as follows:

- * HIV clusters, Household environmental factors and death differentials
- * HIV clusters, socio-economic factors and death differentials
- * HIV clusters, the incidence and prevalence of reported HIV/AIDS and HIV/AIDS differentials.
- * HIV clusters and the impact of HIV/AIDS on mortality.

These factors are however high order concepts that cannot be subjected to empirical investigation. In an attempt to achieve the objective of this study an attempt was made to formulate the empirical model (see: 2.5) based on the theoretical structure described above.

2.6. The empirical model.

There are basically two different approaches to the study of mortality differentials. The first approach relates District estimates of mortality to other characteristics of the district. This approach has been applied to Infant Mortality estimates by K'Oyugi, 1992; and Ikimari, 1996 using 1989 KDHS and to general mortality by Eubank et al., 1986 using 1979 data.

The second approach relates data on the survival of mothers' Children to their Characteristics and that of the Mothers' household. This second approach has been applied to 1989 KDHS data by K'Oyugi (1992), Ikimari (1996), and by Eubank and Others (1986), using 1989 Census data. Due to the availability of data, this study used the second approach using the 1989 Census data.

The selection of variables included in the empirical model used in this study was based on the important factors already reviewed in the literature and the availability of data.

As already mentioned, personal life style, individual socio-economic status, socio-cultural, and bio-social characteristics variables in Pools model was renamed broadly: socio-economic factors as did the intermediate variables in ABCs model. Variables considered are: ~~surviving partners~~ mothers level of education; marital union status, sex, ^{of child} occupation and age.

The household environmental variables considered in the empirical model were: place of residence, place of birth, sewage disposal, floor material type, water supply, and toilet facility type.

Various scholars have used these variables using different sets of data to analyze mortality situations in Kenya (for instance K'Oyugi, 1992, using 1989 KDHS; Eubank and others, 1986 sing 1979 census; Ikimari, 1996, using 1989 KDHS and Muganzi, 1984). This study used some of these variables, applying them to investigate the mortality situation in the HIV sentinel clusters of Kenya using 1989 census.

The variables considered in the incidence and prevalence of HIV/AIDS are age, sex, place of birth, and rural urban.

Fig 1: Empirical Model.

Assuming that:

1. Household environmental factors and socio-economic factors cause mortality through out the life span.
2. That although the risk of death at some ages in some clusters may be similar, the risk of death through out all ages are peculiar for each cluster.
3. That the transmission of HIV is mainly through heterosexual contact and that there is no marked variation in the reportage of the incidence and prevalence of the HIV/AIDS, such that the observed HIV/AIDS incidence and prevalence are peculiar for each cluster.
4. That the incidence and prevalence of HIV/AIDS disease determines its effectiveness and therefore the observed mortality and mortality differentials, and observed HIV/AIDS cases differentials.
5. Finally, that the outcome variable: differentials in risk of death and observed differentials in HIV/AIDS cases and the impact of AIDS on mortality is the cumulative consequences of disease mechanism, preventive and curative medicine and health measures.

From the above assumptions, it can be hypothesised generally that:

That at each variable and variable category, the risk of death and observed HIV/AIDS incidence will be less in low HIV cluster and more in the high HIV cluster.

For infant and child mortality, the following expectations holds true:

That differential in socio-economic factors will cause differentials in the risk of death for child by prevailing HIV/AIDS regimes.

That differential household environmental factors will spell differential in risk of death for child by prevailing HIV/AIDS regime clusters.

For adult mortality the expectation of the study was that:

That there will be observed differentials in adult mortality by characteristic of child.

The final expectation of the study was that:

That the differentials in prevailing HIV/AIDS regimes will spell differentials in the impact of HIV/AIDS on mortality by the above mentioned factors.

2.6. DEFINITION OF VARIABLES IN THE EMPIRICAL MODEL AND THEIR HYPOTHESIZED RELATIONSHIP WITH OUTCOME VARIABLE.

This section discusses the variables used and how they were measured stating the expected relationships between the dependent variable and the independent variable (see: ^{2.1}fig.1).

The framework (^{2.1}fig.1) consists of three proximate determinants: The preventive medicine and health measures; curative medicine and health measures and disease mechanism which operate with HIV/AIDS regime clusters through Household environmental factors, socio-economic factors and incidence and prevalence of reported HIV/AIDS to bring about mortality (HIV/AIDS) differentials and the observed differentials in the impact of AIDS on mortality.

2.6.1. THE DEPENDENT VARIABLE.

The dependent variable for each of the variables categories was defined in this study as the risk of death in each age segment taking into account the total life exposure (man years lived) in each age segments and the prevailing HIV clusters. It was measured

by the following variable indicator:

Probability of dying (q_x): regarded as the probability of dying at exact age x and/or $x + n$.

2.6.2. HOUSEHOLD ENVIRONMENTAL FACTORS.

In this category, ^{six}~~four~~ variables will be considered. The selection of variables was based on the review of the literature and the availability of data. The overall expectation of the study was that at each variable and variable category, those staying in high HIV clusters will experience high mortality than those in mid and mid than those in low HIV clusters.

Sewage disposal variable and Floor material type variable was intended to act as proxy to sanitation. The floor material type refers to the type of material used to make the floor while sewage disposal refers to how the human waste are disposed. Sewage disposal was categorized in this study as: main sewer, bush, pit latrine, and other while floor material type was categorized as cement, earth, tiles and other. The expected relationship is that the better the two variables the better the sanitation and therefore the lower the risk of death. The variation in the two (sewage and toilet) will spell variation in mortality and the impact of AIDS on mortality.

Residence: Refers to the usual place of abode of the respondent as per the administrative boundaries. The variable is intended to act as proxy for access to the available social amenities; urban area have been associated with better services and amenities conducive for survival as compared to rural areas but have high prevalence of HIV/AIDS. It is the expectation of this study that other things being constant, respondents

residing in urban areas have relatively lower risk of death than those in rural areas, while urban areas will experience high incidence and prevalence of HIV/AIDS and ^{therefore} high impact of AIDS on mortality.

Water supply: Refers to the means from which the family gets its drinking water and water for domestic use. This variable indicator was intended to capture the risk of morbidity arising from the use of contaminated water. The assumption here was that, the type of water has a direct relationship with morbidity risk which have a direct relationship with the risk of death. Therefore the variation in mortality will be determined by type of water supply. It is categorized as Piped if drawn from pipes, and others if drawn from wells/boreholes/Streams/rivers/ponds/dam and lakes. The expected relationship is that those households which use piped water have lower risk of death than those who use other sources of water, while the impact of AIDS on mortality will be more for those using other type of water.

Place of birth was considered as the place where the victim of AIDS was born. The assumption here was that the place of birth determines ones infection and disease accruing to him/her' which have a telling effect on HIV/AIDS infection. Those persons born in high HIV/AIDS cluster will no doubt have higher risk of HIV/AIDS infection relative to other clusters.

Incidence and prevalence of HIV/AIDS: The incidence of AIDS refers to the cumulative reoccurrence of estimated/reported AIDS cases from the initial reportage of the disease to the time of the study. This variable was intended to capture the number of persons with the disease in a given HIV regime cluster and the incubation period from HIV to AIDS. Prevalence of HIV on the other hand refers to the percentage of the total

population with the HIV virus. It will be measured in urban and rural areas. It was variable was intended to capture the percentage of the population with HIV virus and by extension the prevalence of risk behaviours. The assumption here was that the risk behaviour is less tolerated in the rural areas than in the urban areas bringing about the differentials in HIV/AIDS cases. The expected relation here is that the incidence and prevalence of HIV/AIDS will be high in the high HIV cluster and lower in the low HIV cluster.

2.6.3.: SOCIO-ECONOMIC VARIABLES.

Four variables will be considered for inclusion in the empirical model, the variables considered with the help of literature review and availability of data are: the mothers: education, occupation, and marital union and Sex. As already mentioned, the overall expectation of the study was that at each variable and variable category, the risk of death will be lower for low HIV cluster and higher in the High HIV cluster.

Mothers education: refers to the level of formal education a mother (surviving spouse) have achieved. It is intended to capture the knowledge and/or level of child or one's own care. The underlying assumption is that the higher the level of education attainment, the better the child's or ones' own care and also the higher the education, the higher the risk of HIV infection. The hypothesized relationship relating these educational [categorized into Never, Primary and Secondary plus] and outcome variables is that child mortality and impact of HIV on mortality is expected to vary with increase of the level of formal schooling of the mother.

The mother union status: Refers to conjugal relationship between couples of the

opposite sex. It is categorized as polygamous, monogamous, divorced & separated and single. This variable indicator was intended to act as control for the effect of the allocation of family level resources for child and ones own care as well as capturing the effect of cultural influences on risk of death. Monogamous families were assumed to have the complete access to all the resources that a family could devote for general care, (both child and oneself), while those in polygamous union and the unmarried were assumed to have only partial access, (based on assumption that family resources would be split among the wives of the household) while the widowed and the polygamous marriages exposes one to higher risk of HIV/AIDS based on assumption that one of the factors might be tempted to have extra marital affair and widowed may be sero positive or vulnerable to sexual manipulation. The expected relationship is that the risk of death and impact of AIDS on mortality will be lower for those in monogamous marriage than in other categories, while at each variable category, the probability of death will be lower for the low HIV cluster and higher in high HIV cluster.

Mothers occupation: It is regarded as the main economic activity which the respondent derives his/her own income, this variable acts as proxy for differences in the level of living in the household. The initial categorization of household occupations limited to three categories: all agriculture (ie agriculture, manufacturing & forestry); sales and services (ie sales/services/parking/storage & transport); professional and clerical (ie professional/managerial and clerical)]. The basic premise is that professional and clerical occupation is associated with higher standard of living and hence were expected to have relatively lower risk of death bringing about mortality variation relative to other categories mentioned above, on another level, they are more exposed to risk of HIV/AIDS as they

often work in towns and can afford the luxuries of life and vices of wealth such as such as sleeping with multiple sexual partners, thus may be expected to have high impact of AIDS on their children, while at each variable category, the risk of death will be lower for the low HIV cluster and higher in high HIV cluster.

The variable sex: Categorized into males and females was intended to capture gender based variation in mortality, *and information provided by the same.* The assumption here was that females have better survival chances than men due to their resistance to infectious diseases. The expected relationship therefore was that females had lower probability of death and lower impact of AIDS on child death than males but higher risk of HIV infection due to their wider biological susceptibility. While at each variable and variable category, low HIV clusters will have lower risk of death while high HIV cluster will have higher risk of death. On the other hand the expectation of the study was that females generally provide more accurate information on death than males.

Age of respondent was considered in this study as the number of years a respondent as lived. *It was intended to capture the differentials in HIV/AIDS incidence & prevalence* It was considered in complete years and categorized in five year age groups. The assumption here was that young children under age five and middle aged persons will be at more risk of HIV/AIDS infections than other variable categories, the former due to transmission of the virus from the mother to the child while the latter due to their higher coital frequencies and incubation period relative to other ages.

Further female respondents are thought to be more accurate in their reportage of mortality than their reportage of mortality than their male counterparts, such that *there* ~~their~~ will be observed mortality differentials by sex *of respondents* ~~and the characteristic of child. adult.~~

Of the five variable, mothers education is taken as the Key Variable.

CHAPTER THREE:

SOURCES OF DATA AND METHODS OF DATA ANALYSIS.

Introduction.

This section gives a brief description of the sources of data used in this study, their limitations and adjustments and methods employed in the analysis in order to achieve the set out objectives of the study. The chapter is divided into four main parts. The first section has the classification of the sentinel centres, the second part has the sources of data, third section have the evaluation of study data and adjustment and the last section have methods of data analysis.

3.1. Classification of the study clusters.

The sampling frame of the study was all the Districts with HIV surveillance system. These districts were categorized into high, mid and low HIV clusters based on the percentages of the observed HIV prevalence rates. Centres having 20 percent and above of the population with HIV virus, was designated to fall in the high HIV regime cluster, those having between 10-20 per-cent were considered to be in the mid HIV regime cluster while those below 10 percent were considered to be falling in the low HIV regimes cluster. Against this background therefore, the percentage recording of HIV sero-positive results for each centre (Urban), for the last seven years was averaged and noted, the result was then used to lump up the individual centres into HIV clusters (see: Table 3.3).

The basis for using only the urban rates was that, based on the review of studies conducted in other countries, it has been estimated that the rural HIV prevalence were between one-half and two-thirds of the urban HIV prevalence rate in each district (Baltazar

and others in Forsythe and Rau (eds), 1996:14). Further transmission pattern of HIV, is still largely from urban to rural areas in Kenya, with the exemption of Busia district where the transmission rate is predominantly within the rural environment (Saoke and others in Forsythe and Rau (eds), 1996:47). Besides rural surveillance system is not complete and covers fewer districts, the present study considered it more advantageous to use the urban surveillance systems.

Table 3.1: Percent of pregnant Women testing positive for HIV virus by year of testing.

SENTINEL SITE.	YEAR OF TESTING							AVERAGE 1990-1996
	1990	1991	1992	1993	1994	1995	1996	
Nakuru	10.0	13.2	12.5	22.5	58.7	-	11.0	21.32
Busia	17.1	10.5	29.7	22.2	22.9	-	-	20.48
Kisumu	19.2	18.6	-	19.6	30.4	25.3	27.4	23.42
Kiambu	2.5	9.6	2.6	27.5	39.6	-	12.9	15.78
Kakamega	5.3	12.8	15.1	8.6	13.7	-	10.5	11.00
Mombasa	10.2	16.7	11.1	16.5	11.0	15.8	12.0	13.30
Kisii	1.6	4.4	-	2.5	9.0	-	15.8	6.66
Garissa	5.9	-	-	7.8	12.9	-	-	8.87
T.Nzoia	3.5	5.6	6.6	7.5	11.1	-	12.1	7.73
Nairobi	-	-	14.4	17.1	22.0	24.6	24.6	20.5
Nyeri	2.9	3.7	7.6	5.4	5.6	9.6	9.2	6.29
Meru	2.7	0.0	0.0	2.3	11.2	-	15.5	5.28
Kitui	1.0	4.8	2.0	-	20.4	-	4.5	6.54

Source: The present study.

Note: * Value for Thika for 1992 is average of 1991 and 1993 due to the high value for 1992. Values for Nairobi was taken from USAID.

Thus Nakuru, Busia, Kisumu and Nairobi formed the high HIV^{regime} cluster; Thika, Kakamega and Mombasa formed the Mid HIV cluster while Kisii, Nyeri, Meru, Kitui, Garissa and Trans Nzoia formed the low HIV^{regime} clusters.

Against this background, the study sample was found to be 758,019; 639,023, and 878,699 Women in the High, Mid and Low HIV regime, of which 1,257; 886 and 1,796 in the high mid and Low HIV regimes did not state their ages.

Further 116,555; 106,420 and 137,901 women in the three respective HIV regimes of High, Mid and Low did not state their parity while 470; 448 and 615 Women did not declare the mortality status of their Children (see: Table 3.2). Further the total number of respondents aged 15-49 who declared the survivorship status of their parents was 7,859,566, this accounted for about 2,118,981, 2,462,410 and 3,278,175 children in ^{the} he high, mid and low HIV clusters respectively.

TABLE 3.2: DISTRIBUTION OF WOMEN AGED 15-49 BY FIVE YEAR AGE GROUP OF MOTHER BY CHILDREN EVER BORN AND THOSE DEAD BY DIFFERENT HIV CLUSTERS

HIV CLUSTER	WOMEN	CEB	CD
HIGH	758,019 (2.7)	2,057,038 6.7*	305,210 0.148**
MID	639,023 (3.0)	1,894,882 7.2*	262,719 0.139**
LOW	878,699 (3.2)	2,795,323 9.1*	306,033 0.109**
SENTINEL	2,275,741 (3.0)	6,747,243 7.7*	873,962 0.130**

Source: Computed from the present study data.

Note:

- (i) CEB denotes children everborn while CD denotes Children dead.
- (i) Figures in parentheses are average Parities which is just CEB/FPOP.
- (ii) A starred figure in Column 3 are proportion of Children Surviving which is just CEB/CD.
- (iii) A two starred figure are proportion of Children dead, which is just CD/CEB for Women age 15-49

Of these women, for the high HIV regime cluster, on average women in reproductive ages (15-49) had about 2.7 children borne to them per woman of which 6.7 survived and 0.1 died on average.

In the mid HIV regime cluster on average, about 3 children were borne to women in the reproductive ages. The proportion of the children surviving in this cluster

was 7.2 while, the proportion dead of the children ever borne to these women was 0.1.

The scenario is almost similar in the low HIV regime cluster where on average a woman had 3.2 children ever borne to her. The proportion surviving in this regime was about 9.1, while the proportion of children dead was 0.1 (table 3.2).

The obvious empirical question that follows is whether there is a statistical relation between the clusters by different key variable inputs mentioned above. For instance the information on child mortality was provided by women, this study therefore felt that it was mandatory to test whether these women were drawn from the same cluster or whether they are actually from different clusters. To achieve this, analysis of variance was employed to women aged 15-49 in the high, mid and low HIV clusters, ie are these clusters different with respect to their women. Table 3.3 gives the results of the analysis of variance of women by HIV clusters.

Table 3.3 Analysis of variance by women aged 15-49 and HIV clusters

SOURCES OF VARIANCE	DEGREES OF FREEDOM	F DISTRIBUTION
BETWEEN SAMPLES	2	95,103,050,006
WITHIN SAMPLES	18	3,712,967,362
TOTAL	18	25.614

Source: The present study.
 Note: $F_{(0.05)} = 3.55$ and $F_{(0.01)} = 6.01$.

Thus the null hypothesis that these women were drawn from the same population (cluster) ^{regime} was rejected both at an alpha level of 0.05 and 0.01 with 2 and

18 degrees of freedom. The alternative hypotheses was then accepted ie these women were drawn from different clusters. This study therefore concludes that the information given by these women were peculiar for each cluster. Further the information used to estimate adult mortality was provided by children who declared the survivorship status of their parents for various clusters. To test therefore the validity that these information were not actually from the same cluster ie that there exist differentials by HIV clusters by the information on parents provided by their children, this study applied the analysis of variance to test whether there was statistical difference between the HIV clusters and those who declared the survivorship^{status} of their parents (Table 3.4).

Table 3.4: Analysis of variance for children who declared the survival status of parent by HIV clusters.

SOURCES OF VARIANCE	DEGREES OF FREEDOM	F DISTRIBUTION
BETWEEN SAMPLES	2	1,413,783,126,103
WITHIN SAMPLES	18	51,042,839,644
TOTAL	18	27.698

Source: The present study.

Note: $F_{(0.05)} = 3.55$ and $F_{(0.01)} = 6.01$.

~~delete~~
Ho: That the children who declared the survivorship of parent were drawn from the same cluster.

H1: That the children who declared the survivorship of parent were drawn from different HIV clusters.

The null hypothesis was then rejected while the alternative hypotheses was accepted, both at alpha levels 0.05 and 0.01. Thus the study concludes that these

clusters were statistically different from each other.

3.2: Sources of data.

Data used in this study were drawn from ^{two} three sources; the first source which provided data on individual and household characteristics of the population was 1989 census, conducted on the night of August 25/26. The focus of the study were women and men aged 15 plus and/or their children. This data will hence forth be called CBS data.

The second source of data was data from National AIDS and Control Programme (NASCOP). This will provide data on HIV/AIDS cases. The surveillance of HIV is mainly done in Ante Natal Clinic (ANC) attendees and for patients with sexually transmitted diseases (STDs). The STD data are primarily designed to represent high risk populations while the ANC data represent the general population. For the purposes of this study, only the ANC data were used. Every quarter of the year, women attending antenatal clinic for the first time with the current pregnancy are tested for HIV in 13 urban sites and six rural sites (see: Table 3.1), the results are then forwarded to NASCOP together with confirmed AIDS cases in all the major hospitals (Government or private). This data will henceforth be called NASCOP data. It should be noted here that the assumption made here was that the transmission of HIV is mainly through heterosexual relationship and two, the rate of migration of expectant women from rural to Urban to attend ANC clinic is minimal.

3.3. QUALITY OF THE STUDYS' DATA AND DATA ADJUSTMENT.

The present study felt it would be useful to assess ^{the} ~~he~~ quality of the data to be used in the study and/or make same adjustments to the data where it is deemed appropriate. These were done both before ^{during} and/or after the analysis.

3.3.1: Checking of the quality of the study data

The quality of the data used was assessed using both the internal and the external checking procedures, such as examining the sex ratio and the average parities per Woman by five year age group. The external check were done by comparing some of the rates obtained using the study data and those obtained in the relevant published documents on mortality situation in Kenya.

The most used and recommended way of evaluating the quality of study data is to look at the reported sex ratios of males to females (see: Table ^{3.5} 3.7). This sex ratio has to range between 1.01 to 1.05, Any value above or below this range depicts marked variations in the data. For instance, United Nation estimates that Countries in Africa have a sex ratio closer to 1.03, (UN 1983:77).

TABLE 3.5: DISTRIBUTION OF CHILDREN EVER BORN BY SEX AND FIVE YEAR AGE GROUP OF MOTHER AND AVERAGE PARITY.

AGE GROUP OF MOTHER	TOTAL FEMALE POPULATION	CHILDREN EVERBORN		SEX RATIO OF CHILDREN EVERBORN	AVERAGE PARITY	
		MALES	FEMALES		MALES	FEMALES
HIGH HIV REGIME CLUSTER						
15-19	175878	25293	24742	1.02	0.144	0.141
20-24	183959	128252	128018	1.00	0.682	0.696
25-29	151033	216321	213681	1.01	1.432	1.415
30-34	92698	199232	197772	1.01	2.149	2.134
35-39	68793	186858	182219	1.03	2.716	2.349
40-44	48573	141941	149072	0.95	2.922	3.069
45-49	37090	127938	123400	1.04	3.449	3.327
TOTAL	758024	1025835	1018904	1.01	1.353	1.344
MID HIV REGIME CLUSTER						
15-19	162814	19170	18696	1.03	0.118	0.115
20-24	140352	101446	100138	1.01	0.723	0.714
25-29	112920	172425	171098	1.01	1.527	1.515
30-34	77155	180585	179785	1.00	2.341	2.330
35-39	60487	176072	175116	1.01	2.911	2.895
40-44	46250	157604	156713	1.01	3.408	3.388
45-49	39045	143652	142382	1.01	3.697	3.647
TOTAL	639023	950954	943928	1.01	1.488	1.477
LOW HIV REGIME CLUSTER						
15-19	233415	26394	26107	1.01	0.113	0.112
20-24	182438	141514	138628	1.02	0.776	0.760
25-29	154573	263671	256908	1.03	1.706	1.662
30-34	100905	260952	255164	1.02	2.586	2.509
35-39	82980	264256	259753	1.02	3.185	3.130
40-44	69680	251041	245049	1.02	3.603	3.517
45-49	54708	205576	200510	1.03	3.758	3.665
TOTAL	878699	1413424	1382119	1.02	1.609	1.573

Source: Computed from the present studys' data.

Sex ratio given in column 5 of table 3.5 fluctuate somewhat by age of mother but show no significant trend for the high, mid and low HIV cluster. Further more the overall sex ratio for the high HIV cluster (1.01), mid HIV cluster (1.01) and low HIV regime (1.02) is acceptably within the accepted range of 1.00 to 1.05 for developing countries and that of Africa.

Another way to assess the quality of the data on Children ever born is by

examining the behaviour of average parities reported by Women of each age group. Unless fertility rose at some time in the past, average parities should increase with age up to age group 45-49. It is important to do this because omission of CEB might be made up disproportionately of dead children (UN, 1983:81). Columns 6 and 7 of the same table reveal that the average Parity is the average number of children borne per Woman in each age group tend to rise by age of the mother for both sexes, an indication of the validity of the data. This study therefore can safely conclude that there is no marked clear deficiency in the data.

Table 3.6 then gives the distribution of women who did not declare the mortality and parity status of their children.

Table 3.6: Distribution of women who did not state the mortality status of their children and their parity by HIV Clusters.

AGE GROUP OF MOTHER	HIGH HIV REGIME		MID HIV REGIME		LOW HIV REGIME	
	PARITY NOT STATED	MORTALITY NOT STATED	PARITY NOT STATED	MORTALITY NOT STATED	PARITY NOT STATED	MORTALITY NOT STATED
15-19	58191	58997	83935	84645	59425	59909
20-24	26215	28732	28589	30214	23531	24584
25-29	11411	13212	11262	12574	10177	11090
30-34	5333	6440	5233	6191	5259	5815
35-39	3344	4114	3569	4304	3519	3911
40-44	2499	3010	3030	3604	2483	2766
45-49	1786	2050	2284	2755	2026	2245
Age NS	460	470	599	615	444	448
Total	165622	177359	229785	238281	168835	173007

Source: Computed from the present Studys data.

A look at table 3.6, reveal that the mid HIV cluster had the highest cases of women who did not declare the mortality status of their children (238,281) while the high HIV cluster had the lowest (177,359). The same scenario applies for the Parity

not stated. In all the stratus of the HIV cluster, women in the category of mortality and the parity not stated in relation to their children declined steadily by additional increase of age of mother. The probable reason for this is that younger women tend to shy away from stating their parity or the mortality status of their Children.

3.3.2: Adjustment of the study's data.

To explore the consistency of mortality estimates obtained, the estimated values was converted into mortality levels in the Coale-Demeny system (in line with the recommendation of most scholars that it is best suited to Kenya) in order to compare the age pattern of the estimates obtained with that of the models.

To be able to facilitate comparison both within and between data sets, each estimated $q(x)$ was converted to a single measure. The common index used in this study was $q(5)$, the probability of dying by age 5 (${}_5q_0$) expressed per thousand live births. This index was chosen because 'it captures almost all the mortality risk prior to adulthood, is less affected by the age profile of child rearing practice like weaning and is not particularly sensitive to the pattern of mortality than Infant mortality rate' (Eubank and others 1993:13). Further the computed $q^c(x)$ values were time located to give; on average an indication of the time the computed $q(x)$ rates refer to, the resultant values were then plotted on a line graph.

↑ The required conversion was carried out using the $q(x)$ values corresponding to North Model of Coale Demeny Family of Life tables. The table in annex b, contains the necessary values of $q(x)$ ordered by mortality level and expectation of life for males,

Females and both sexes separately. The actual conversion is carried out by interpolation between tabulated values.

The present study used orphanhood approach to estimate adult mortality. However, there are basically two methods that have been developed on adjustment of basic demographic parameters before the formulae used on stability are used. The two techniques for such adjustment are: the synthetic or hypothetical approach and the age specific growth rate technique.

The synthetic approach uses two sets of data which are either 5 years or 10 years apart to synthesize a third set. Age specific Growth Rate Technique also uses two sets of data to obtain the adjusted proportions. However now, the two consecutive Censuses can be any number of years apart (See: Mudaki, 1986:30-36). Both methods were not attempted since neither the 1979 nor the 1969 Censuses were available from the CBS. For the calculation of each, please see the relevant sections.

As already mentioned in the relevant sections (Sec 3.2) however, this study used ANC data in the analysis of HIV cases. Although, the ANC clients are generally representative of the general population, there are differences between the two groups, for instance the HIV test involves only women but for pregnancy to occur the men too have to be invariably involved but each (males and females) have different infection rate, the study adjusted the data to include men. Secondly for a woman to be attending ANC she has to have been sexually active but there are those women in

this group who are not sexually active and are therefore not exposed to the risk of HIV infection, this study adjusted the data to cater for this group. Third, sentinel sites are only in few sites mostly in urban centres. Results then had to be weighted by size of adult population in each site in order to come up with representative figure. Fourth, the age distribution of pregnant women is younger than the age distribution of all sexually active women but HIV prevalence is higher in younger ages this called for adjustment for this disparity. Fifth the sites are located in urban centres only and does not cover rural population. Lastly, the pregnant women are mostly in age range of 15-49 but infections can also occur to those above that age. The data was adjusted to include the population 50 plus.

Lastly the children ever born to each woman in each variable category for the calculation of child mortality was truncated at 20 children and above. ie those women who had twenty children and above were excluded from the analysis simply because it was not feasible. This percentage was however very negligible to bring about any major impact on the proportion of children dead and average parity per woman. However they were included in the cross tabulation to help the researcher notice figures omitted in the analysis.

The present study also encountered the case of those women who did not declare the parity of their children. El-Bandry formulated a formula for correcting the number of Women who did not state their parity. However the use of El-Bandry is recommended when the relationship mentioned is linear in nature. Otherwise, it is

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recommended that women whose parity is not stated should be included in the denominator when calculating the average parities UN, Manual X:230ff. This study did not use El Bandry method but rather added those women who did not declare their parity in the denominator.

3.4: METHODS OF DATA ANALYSIS.

This section covers the methods used in the analysis of data, in an attempt to meet the outlined objectives of the study.

3.4.1: Demographic Techniques.

Most recent effort to estimate mortality differentials are based on two basic approaches. The first approach involves direct methods which are based on reported births and deaths of individuals.

The alternative approach is to use Indirect methods which are based on data that do not provide for tabulation of deaths and persons years of risk by age. The most common is the Brass Child survival approach for Infant and child (I & C) and orphanhood approach for adult mortality. Several variations of these methods are described in detail in United Nations (1983), Manual X:73ff.

The present study employed Indirect techniques as basis for analysis of mortality levels.

Step 1: Calculation of average parities for women.

Average parities $P(1)$, $P(2)$ and $P(3)$ are calculated by dividing the number of children everborn of each sex appearing in column (3) and (5) of table 3.7) by the total number of women (column 2 of the same table). For Instance, the mean number of male children overborne by women aged 15-19 denoted by $P_m(1)$ is:

$$P_m(1) = 70857/572107 = 0.1238527 \approx 0.124. \quad (2)$$

The complete set of average parities for males $P_m(i)$ and that for females $P_f(i)$ values shown in columns (3) and (4) of table 3.8.

Table 3.8: Average parity per woman by sex of child and age of mother, HIV Sentinel cluster.

AGE GROUP OF WOMEN	INDEX (i)	AVERAGE PARITY PER WOMAN		
		MALES $P_m(i)$ 3	FEMALES $P_f(i)$ 4	BOTH SEXES $P(i)$ 5
15-19	1	0.1238527	0.1215594	0.2454121
20-24	2	0.7325362	0.7237981	1.4563344
25-29	3	1.5588446	1.533207	3.0920516
30-34	4	2.3665746	2.3368506	4.7034252
35-39	5	2.9548007	2.907227	5.8620277
40-44	6	3.3469663	3.3484739	6.7702048
45-49	7	3.3230054	3.563752	7.2090826
P1/P2		0.1691	0.1679	0.1685135
P2/P3		0.4699	1.4721	0.4709929

Source: The present study - Calculated From CBS 1989 Census.

Step 2: Calculation of Proportion of Children dead $D(i)$ for each age group of Mother.

The values of this proportion $D(i)$ are computed from table 3.7 by dividing the number of children dead of each sex, given in

column (4) and (6) by the children everborn of the corresponding sex, shown in columns (3) and (5). Thus $D_m(1)$, the proportion of male children dead among those everborn to women aged 15-19 is:

$$D_m(1) = 7,518/70,857 = 0.106101 \dots \dots \dots (3)$$

and for both sexes, the deaths have to be added and then divided by the total number of children everborn (sum of males and females). Table 3.9 shows a complete set of the proportion of children dead.

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Table 3.9: Proportion of children dead by sex of children and age of mother, HIV sentinel cluster.

AGE GROUP OF WOMEN	INDEX (i)	PROPORTION OF CHILDREN DEAD		
		MALES $D_m(i)$ 3	FEMALES $D_f(i)$ 4	BOTH SEXES $D_b(i)$ 5
15-19	1	0.106101	0.1079301	0.1069927
20-24	2	0.1020198	0.0973324	0.0996902
25-29	3	0.1058402	0.0990778	0.1024879
30-34	4	0.1222609	0.1133327	0.117825
35-39	5	0.1354287	0.1242448	0.1298821
40-44	6	0.166123	0.1489123	0.1557762
45-49	7	0.2048726	0.169664	0.1776714

Source: The present study - Calculated From CBS 1989 Census.

However, this proportion of children dead is affected by age of mother. Brass formulated adjustment factor $K(i)$ to carter for this disparity in age pattern for child bearing. The adjustment of proportion of children dead was achieved by calculating the multipliers $K(i)$, and using it to adjust the age disparity (see: step 3).

Step 3: Calculation of Multipliers

The calculation of the multiplier is needed to adjust for the reported proportion dead $D(i)$, for the effect of the age pattern of child bearing. Thus taking north family of model life tables to be adequate representation of mortality in Kenya, further given the values of P_1 , P_2 and P_3 , shown in Table 3.8, values of $K(i)$ are calculated for each and both sexes as shown below using male children for illustration.

$$K(i) = a(i) + [b(i) (P_1/P_2)] + [c(i) P_2/P_3] \dots (4)$$

substituting the values then:

$$K_m(1) = 1.1119 + ((-2.9287) * (0.1238527) / 0.7325362) + 0.8507 (0.7325362 / 1.5588446) = 1.0164 \dots (5)$$

ie the multiplier for male Children of women aged between ages 15 and 19 ($i=1$) is 1.0164. The complete set of $K(i)$ values are shown in table 3.10 (The coefficients $a(i)$, $b(i)$ and $c(i)$ were derived from table A.1 in ~~annex A~~ ^{UN (1983), Manual X pages 77}).

Table 3.10: Trussell multipliers for child mortality estimation, north model, HIV sentinel cluster.

AGE GROUP OF WOMEN	INDEX (i)	MULTIPLIERS $K(i)$ FOR		
		MALES $K_m(i)$ 3	FEMALES $K_f(i)$ 4	BOTH SEXES $K_b(i)$ 5
15-19	1	1.0164	1.0218	1.0191
20-24	2	0.9539	0.9941	0.9940
25-29	3	0.9532	0.9521	0.9526
30-34	4	0.9902	0.9886	0.9894
35-39	5	1.0531	1.0513	1.0522
40-44	6	1.0390	1.0373	1.0382
45-49	7	1.0189	1.0174	1.0181

Source: The present study.

Step 4: Calculation of probability of dying $q(x)$.

The estimated value of probability of dying $q(x)$ are found by multiplying the $K(i)$, values appearing in table 3.10 by the corresponding proportion dead $D(i)$ given in table 3.9. For instance, the value of $q_m(1)$ is obtained as follows:

$$q_m(1) = K_m(1)D_m(1) = (1.0164)*(0.1061) = 0.1078 \approx 0.1086$$

A complete set of $q(x)$ values are displayed in column 2 of table 3.11.

Since every $q(x)$ values is the probabilistic complement of the probability of surviving, $l(x)$, the latter value can be obtained by subtracting the former from 1.0, thus, the probability of surviving for males born to women aged 15-19 ie $l_m(1)$ was derived as:

$$l_m(1) = 1.0 - 0.1078 = 0.892. \dots \dots \dots (7)$$

In Table 3.11, every $q(x)$ value (appearing in column 2) is accompanied by its corresponding $l(x)$ value (appearing in column 6).

Step 5: Calculation of Reference Period $t(x)$.

It is obvious that due to various factors outlined in chapter 2 subsection 2.2 among others, that mortality in Kenya was not likely to have remained constant until 1989 or there after, therefore the present study felt that it was useful to know the reference period $t(x)$ of each $q(x)$ estimate. Using the values of $P1/P2$ and $P2/P3$, already computed (see:step 3) and assuming north model mortality pattern of the coale and Demeny

regional life tables, the value of $t(x)$ for male children of women age 15-19 was calculated using the following equation:

$$t(x) = a(i) + [b(i)P1/P2] + [c(i)P2/P3] \dots (8)$$

→ The coefficients $a(i)$, $b(i)$ and $c(i)$ in this equation was taken from table ~~A.2 in annex B~~ ^{*48 in UN Manual X (1983) Page 78*}. Substituting the values in the equation above:

$$t_m(1) = 1.0921 + 5.4732(0.1691) + (-1.9672)(0.4699) = 1.0932 \approx 1.1 \dots (9)$$

This imply that the estimated $q_m(1)$ value of 0.108 is similar to that corresponding to the period life table in operation 1.1 years before the date of the survey. Since the interview took place in August 1989, the surveys reference date can be taken to be 1989.7 ie August corresponds to month eight in the year, which when divided by 12 months in a year is $8/12=0.66$, which is roughly 0.7 (when rounded in decimal terms). Hence the rough terms of $q_m(1)$ refer to $1989.7 - 1.1 = 1988.6$ or towards the end of calender year 1988. The complete set of $t(x)$ and the reference dates calculated from them are presented in table 3.11, columns 3 and 4.

Step 6: Conversion to mortality levels in the Coale-Demeny System.

In order to compare the age pattern of the estimates of $q(x)$ and that of the model life tables and to explore the consistency of the mortality estimates calculated, the calculated $q(x)$ values were converted to mortality levels by sex (ie mortality levels

associated for each estimate $q(x)$ for each sex and for both sexes) of Coalle-Demeny regional life tables. Table 3.10 column 8 shows the levels as derived from the north model life tables by interpolating between the values of Table A.9, A.10 and A.11 of annex A. Levels for both sexes are not shown since being effectively averages of the levels for the two sexes individually they would not contribute any more information.

According to table 3.10, $q_m(1)$ is equal to 0.1078 and the corresponding $l(1)$ is 0.8922. Using $l(x)$, values appearing in Annex A, table A.10 (Males North Model), for interpolation purposes; in columns labeled ' l_1 ' in table A.10, the two values that enclose the observed $l(1)$ are $l_{14}(1) = 0.8877$ and $l_{15}(1) = 0.8993$ (the $l(1)$ values at levels 14 and 15 respectively). Therefore the interpolation θ is:

$$\theta = (\text{Central value} - \text{lowest value}) / (\text{highest value} - \text{lowest value}) \quad (10)$$

therefore:

$$\theta = (0.8922 - 0.8877) / (0.8993 - 0.8877) = 0.3879. \quad (11)$$

and because the distance between levels 14 and 15 is just one, the level consistent with $q_m(1)$ is 14.3879 or roughly 14.39. The rest of the values in column 7 of table 3.10 are arrived at in similar manner.

Step 6: Conversion into a Common Index

This was done as already mentioned to facilitate comparison both within and between data sets. $q(5)$ was chosen as a common

index mainly because it is not sensitive to the pattern of mortality. Therefore each estimated $q(x)$ obtained in step 4 was converted into a single measure $q(5)$. The conversion is carried out by interpolating between the $q(x)$ values of the model life table presented in annex A, table A.4, A.7, and A.8.

For instance, the conversion of estimated $q_m^e(2)$ to $q_m^e(5)$, according to North model was derived as follows: the estimated value of $q_m^e(2)$ is 0.1014. According to table A.1, this value falls between the $q_m^e(2)$ of level 16, $q^{16}(2) = 0.10995$ whose $q^{16}(5)$ equivalent is 0.14031 and that of level 17, $q^{17}(2) = 0.09527$, whose $q^{17}(5)$ equivalent is 0.12145.

Thus the interpolation factor was obtained as follows:

$$h = q^e(x) - q^j(x) / q^{j+1}(x) - q^j(x) \quad (12)$$

Substituting these values the study got:

$$h = 0.1014 - 0.10995 / 0.09527 - 0.10995 = 0.5824. \quad (13)$$

The $q^e(5)$ equivalent for the estimated $q^e(2) = 0.1014$ is then derived using question as follows:

$$q^e(5) = (1.0 - h)q^j(5) + hq^{j+1}(5) \quad (14)$$

where $q^j(5)$ and $hq^{j+1}(5)$ are model values for levels j and $j+1$ respectively (ie enclosing the estimated value $q^e(x)$, and h is the interpolation factor. Substituting these values then:

$$q^e(5) = (1.0 - 0.58243)(0.14031) + (0.58243)(0.12145) = 0.12933 \approx 0.129. \quad (15)$$

That is the model life table for males, the $q^e(5)$ corresponding to $q^e(2)$ of 0.1014 is 0.129. The complete set of $q^e(5)$ values equivalent to the estimated $q(x)$ values are shown in

column 6 of table 3.10.

The rest of the Infant and Child mortality rates are derived in similar manner following the above 6 steps.

However, the QFIVE package developed by UNFPA population division of United Nation, has helped in the task of converting the proportions of children dead by age group of mother, into values of $q(x)$ matching them against model life tables systems to determine equivalent values of $q(1)$ or $q(5)$ and, the calculation of the time locations. This package produces equivalent of $q(x)$ given by Trussell regression equation based on four families of the Coale-Demeny life tables, and those from the Palloni-Helgman equations using the five families of the new UN models, together with equivalent values of $q(1)$ and $q(5)$ and time allocations of these estimates. This package was employed in this study to help ease the computation of the Infant and child mortality rates for the rest of the variable categories.

Table 3.10: Estimation of early age mortality for males, HIV sentinel cluster.

AGE X	PROBABILITY OF DYING $q(x)$	TIME REFERENCE $t(x)$	REFERENCE DATE	COMMON INDEX $q^*(x)$	PROBABILITY OF SURVIVING $l(x)$	NORTH MORTALITY LEVEL
1	2	3	4	5	6	7
1	0.1078	1.09	1988.6	0.173	0.8922	14.39
2	0.1014	2.33	1987.4	0.129	0.8986	16.58
3	0.1008	4.10	1985.6	0.115	0.8992	17.35
5	0.1211	6.20	1983.5	0.121	0.8789	17.02
10	0.1426	8.55	1981.1	0.124	0.8574	16.93
15	0.1726	11.12	1978.6	0.139	0.8274	16.05
20	0.2088	13.99	1975.7	0.156	0.7912	15.22

Source: The present study.

From Table 3.10, the $t(x)$ values imply that the estimates of $q(1)$, $q(2)$ and $q(5)$ obtained refer to mortality conditions prevalent approximately one year, two years, four years and six years before the survey respectively, therefore the estimated value of $t(x)$ increased by some 2-3 years per age group.

Looking at the value of $q(2)$ which correspond to women aged 20-24, who are recent in childbearing, it follows that their $q(2)$ value should be fairly recent which is depicted in the table (about two years prior to the survey). This study concludes that the plausibility and consistency of the values of $t(x)$ are okay.

Ignoring age groups 15-19, the mortality levels for males in the sentinel cluster declined steadily by each additional age group of the mother, implying that the mortality levels for males declined gradually from 1975 to 1985. The high levels of mortality associated with women in age group 20-24 is expected since they are recent in their child rearing and are associated with high risk factors for their children.

35.10 An examination of the levels displayed in column 7 of table 5.5 shows that estimates of $q(1)$ imply relatively high mortality (they are associated with relatively low levels in the Coale - Demeny Models) and should not be considered. Making some allowance for the random variation inherent in any measure derived from probability samples, the remaining estimates of levels decline steadily by age of mother, strongly suggesting that child mortality has been falling. These mortality estimates in conjunction with reference dates estimates does not reveal any

obvious problems.

After using QFive application package, the resultant probabilities of dying by age x for each HIV cluster by various variables are presented in annex B.

3.4.1.2: Estimation of Adult Mortality.

To estimate Adult mortality; the study used conditional probability using Brass and Hill method (1973) applied to estimate male(female) mortality.

In this method the conditional probability of a Father(Mother) surviving from age x to age $x+n$ was measured using the following input data:

Proportion of respondents with surviving Father(Mother) in each five year age group from n to $n+4$. This proportion is denoted by $S(n)$. The set of $S(n)$ can be calculated when any two of the following are present (a) the number of respondents with Father(Mother) alive, or dead and (b) Total number of respondents whose paternal survival status is known.

The other input data used was births in the last year classified by five year age group of mother. This approximation is needed in the calculation of the average age at which target population begin their exposure to risk of dying.

Thus the conditional probability of a father surviving from age 32.5 to age $35+n$ is given by the formula:

$$l_{(35+n)} / l_{(32.5)} = W_{(n)} S_{(n-5)} + [1 - W_{(n)}] S_{(n)} \quad \dots \dots \dots (16)$$

Where, $S_{(n)}$ is the proportion of the respondents aged between

n and $n+5$, $W_{(n)}$ is the weighting factors employed to make allowance for the typical age pattern of fertility and mortality, while, $S_{(n-5)}$ is the proportion in the age group of respondents in age group n to $n-5$ with surviving Father(Mother). For further arguments, see: UN, manual x, 1983:100-106.

The same principle applies to females though the base now changes from a value of 32.5 to 25. This is principally because females are generally younger by the birth of their children.

For instance the following steps were followed to derive adult mortality rate. The data shown in table 3.12 and 3.13 were used to illustrate the steps followed in an attempt to estimate adult male mortality. Table 3.12 shows the raw data gathered by the 1989 census and the proportion of respondents whose father was alive at the time of the interview. In calculating this proportion, however the number of respondents who were classified in the category of 'unknown paternal orphanhood status' were ignored, this was mainly because the study felt that the levels of non response might have occurred. As an example, $S_{(20)}$ (the proportion of males with surviving father) was computed as follows:

$$S_{(20)} = 624055 / (624055 + 165532) = 0.07904 \quad (17).$$

Given the above information, the following steps were followed to derive adult mortality rate. For further details, see UN (1983), Manual X:pages 102-107.

Table 3.12: Distribution of respondents (both sexes) who declared the survival status of their father. Sentinel cluster.

AGE OF RESPONDENTS	NUMBER OF RESPONDENTS WITH			PROPORTION WITH FATHER ALIVE
	FATHER ALIVE	FATHER DEAD	UNKNOWN PATERNAL ORPHANHOOD STATUS	
15-19	895988	146093	10969	0.85981
20-24	624055	165532	5791	0.79036
26-29	472902	194520	3305	0.70855
30-34	296770	191746	2333	0.80749
35-39	191466	191547	1857	0.49989
40-44	115957	191874	1673	0.37669
45-49	68535	174091	1403	0.28247
50-54	38615	164515	1143	0.19010

Source: The present study.

Step 1: Calculation of mean age at Paternity.

The mean age Paternity is given by the formula

$$M(\text{Paternity}) = M(\text{Maternity}) + \frac{Md(\text{Males}) - Md(\text{Female})}{18}$$

The first step then is to calculate the mean age at maternity. As already stated, the mean age at maternity is calculated as follows:

$$M = \frac{\sum a(i)B(i)}{\sum B(i)} \quad \dots \dots \dots (19)$$

Where $a(i)$ is the mid-point of age group i and $B(i)$ is the number of births to women in age group i . However the question asked at the time of the census relates to the ages of woman at the time of the interview and not at the time of the birth of their Children. In view of the foregoing some allowances was made for this fact. The simplest strategy was to assume that births are uniformly distributed in time and with respect to the mothers age (UN, 1982:105).

Hence on average, women are six months younger at the time

of the Interview, so that for example, age 15-20 age span may be considered to span exact ages from 14.5 to 19.5 at the time the birth occurred. Therefore the Central $a(i)$ value to be used in applying equation 19 in this study are 17 instead of 17.5, 22 instead of 22.5 etc as shown in column 3 of table 5.13.

Table 3.13: ¹² CHILDREN BORN DURING THE LAST 12 MONTHS BY AGE OF THE MOTHER AT THE TIME OF THE CENSUS, KENYA 1989. SENTINEL CLUSTER.

AGE GROUP OF MOTHER	INDEX	MID POINT OF AGE GROUP (adjusted by six months)	NUMBER OF CHILDREN BORN IN PAST YEAR
15-19	1	17	4850
20-24	2	22	22498
25-29	3	27	7134
30-34	4	32	1128
35-39	5	37	298
40-44	6	42	82
45-49	7	47	44
TOTAL			36034

Source: The present study data.

Then $M(\text{Maternity})$ is calculated as follows:

$$\begin{aligned}\sum a(i)B(i) &= 17(29,945) + 22(31,525) + \dots + 47(67) \\ &= 822,658. \dots \dots \dots (20)\end{aligned}$$

and

$$\sum B(i) = 36,034$$

so

$$M(\text{Maternity}) = 822,658/36,034 = 22.8 \dots \dots \dots (21)$$

The next step involves the calculation of median age at marriage for both males and Females. Table 3.14 ¹³ gives the distribution of married population by sex used to compute median age at marriage.

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Table 3.14: Distribution of respondents by Marital status and cumulative frequencies: HIV Sentinel Cluster.

AGE GROUP	MARRIED		CUMULATIVE FREQUENCY	
	MALES	FEMALES	MALES	FEMALES
12-14	1191	2689	1191	2689
15-19	2330	25029	3521	27718
20-24	24770	82585	28291	110303
25-29	65936	87446	94227	197749
30-34	68359	63794	162586	261543
35-39	56847	50404	210485	311947
40-44	44847	37810	264332	349757
45-49	33588	31533	297920	381290
50-54	27782	24390	325702	405680
55-59	21263	18515	346965	424195
60-64	16050	13033	363015	437228
65+	128022	84773	491037	522001
TOTAL	491037	522001		

Source: Computed from the present study data.

To calculate the median age at marriage, the study used the formula:

$$Md = L + (N/2+C)/f \quad (22)$$

Where, 'Md' refers to median value, 'L' refers to lowest limit of median class, 'f' is the frequency of the median class and 'c' is the cumulative frequency up to and including the class preceding that which the median lies, N is the total frequency.

For instance, for males $N/2 = 491037/2 = 245,519$.

From this value, (245,519), the other values of the formula can be derived (from table 3.13) as $L = 40$, $C = 264332$ and $f = 44847$.

Then median age at marriage for males was calculated as follows:

$$Md_{(males)} = 40 + ((245519 - 264332)/44847) = 39.6. \quad . (23)$$

For females, the median age at marriage was derived in similar manner thus:

$$30 + (522001/2) - 261543)/63794 = 30. \quad (24)$$

Therefore mean age at Paternity was derived using equation

18 as:

$$M_{(\text{Paternity})} = 22.8 + 39.6 - 30 = 32.4. \quad \dots \quad (44)$$

Step 2: Calculation of weighting factors.

The appropriate weights $W(n)$ were obtained by interpolating linearly between the values given in table ~~A.6~~ ^{87 in UN Manual X pg 103.} in the Annex A. The values of 'M' was 32.4 years, so the weights in columns labelled '32' and '33' were used as extremes in the interpolation. For all values of n , the interpolation factor ' θ ' was:

$$\theta = (33 - 32.4) / (33 - 32) = 0.6. \quad \dots \quad (25)$$

Therefore to obtain $W(30)$ for instance, the values for $n = 30$ appearing under the columns for M equals 32 and 33 of table ~~A.6~~ ⁸⁷ are weighted by θ and $(1 - \theta)$, respectively yielding

$$W(30) = 0.6(0.319) + 0.4(0.475) = 0.3814. \quad \dots \quad (26)$$

The complete set of $W(n)$ values of $M = 32.4$ are shown in column (2) of table 3.1~~A~~.

Step 3: Calculation of Survivorship Probabilities.

Given the weighting factors, the values of $l_m(35+n)/l_m(32.5)$ are computed as indicated by equation ¹⁶ **. Table 3.1~~A~~ gives their values. The table also shows the mortality levels in the Coale-Demeny North family of model life tables associated with each survivorship ratio. These levels were calculated by interpolating linearly between the values given in table ~~A.13~~ ²¹ (see: annex A). These values provide a useful index for

in UN Manual X

examining the consistency of the survivorship ratios themselves.

As an example, the calculation of the level consistent with the estimated $lm(65)/lm(32.5)$, according to table 3.15 is 0.770. The column labelled '65' of table ^{211 of UN (1983) Manual K} ~~A.13~~ in annex A is then used to locate the two values that bracket the estimated value. They are 0.76702 corresponding to levels 21 and 0.78883 corresponding to level 22. Hence the interpolation factor θ is:

$$\theta = (0.770 - 0.767)/(0.78883 - 0.76702) = 0.14 \quad (27)$$

and the estimation equation is:

$$(0.14)(22) + (0.86)(21) = 21.14 \quad (28)$$

The complete set of mortality levels is shown in column 7 of table 3.15.

Table 3.15: Estimation of adult male mortality using Orphanhood approach (Brass Method). HIV sentinel cluster.

AGE X n (1)	Weighting factors W(n) (2)	Proportion with Father surviving S(n-5) (3)	Complement of Weighting Factor (1-W(n)) (4)	Proportion with Father surviving S(n) (5)	Female adult survivorship probability lf(35+n)/lf32.5 (6)	North Mortality level (7)
55	0.569	0.8598	0.431	0.7904	0.830	17.25
25	0.529	0.7904	0.471	0.7086	0.757	16.90
30	0.381	0.7086	0.619	0.8075	0.770	21.14
35	0.173	0.8075	0.827	0.4999	0.553	16.93
40	-0.135	0.4999	1.135	0.3767	0.360	14.95
45	-0.433	0.3767	1.433	0.2825	0.242	

Source: Computed from the present studys' data.

Step 4: Calculation of the number of years before the survey to which the survivorship estimates refer.

The reference period of each estimate derived in previous step, is calculated from $_{10}S_{(n-5)}$, the proportion of respondents aged

from $n-5$ to $n+4$ whose father was alive at the time of the interview. These are derived by dividing the number of respondents with surviving father in the two adjacent age groups from $n-5$ to $n-1$ and from n to $n+4$ by the sum of the number of respondents in both age groups who provided information on fathers' survival. For instance:

$${}_{10}S_{20} = (624055 + 472902) / (624055 + 472902 + 165532 + 194520) = 0.7529. \quad (29)$$

The complete set of ${}_{10}S_{(n-5)}$ values are shown in column 2 of table 3.16.

Since 'M' was found to be 32.4, this means that the values of $x = M + n$ range from 52.4 to 77.4 increasing in steps of five years. Standard function, $Z(x)$ was obtained by linear interpolation¹, for instance:

$$\begin{aligned} Z(62.4) &= 0.6Z(62) + 0.4Z(63) = 0.6(0.374) + 0.4(0.431) \\ &= 0.397. \end{aligned} \quad (30)$$

All $Z(x)$ values are given in column (4) of table 3.16. Then $U(n)$, the correction function is derived as:

$$U(30) = 0.3333 \ln {}_{10}S_{25} + Z(60.2) + 0.0037(27-M+0.75). \quad (31)$$

which is:

$$\begin{aligned} U(30) &= 0.3333 \ln(0.56021) + 0.381 + 0.0037(27-32.4 + \\ &0.75) = 0.07021. \end{aligned} \quad (32)$$

Then $t(n)$ is calculated as follows for each case, using the same illustration of $n = 30$.

¹ The Z values were obtained from the Table A,3 in annex B.

$t(n) = (n - 0.75)(1.0 - U(n))/2$, and substituting the values, the study got:

$$t(30) = (30 + 0.75)(1.0 - 0.07021)/2 = 14.3. \quad \dots (33)$$

This implies that mortality estimates at age 65 of (0.770) refers to mortality in operation some 14.3 years prior to the survey. As already mentioned the Kenya 1989 Census was carried out in August 25/26, changing the months in decimal terms, this refer to about 1989.7, so when 14.3, was subtracted from 1989.7 gives the $lm(65)/lm(32.5)$ estimates referring to mortality estimates prevailing to approximately to mid 1975. The complete set of reference dates and the North mortality levels associated with them are shown in columns (7) and (8), respectively, of table 3.16.

Table 3.16: Estimation of time reference period for male survivorship probability derived from paternal mortality data. HIV Sentinel cluster.

AGE n (1)	PROPORTIO N WITH FATHER SURVIVING $10S_{N-5}$ (2)	LEVEL OF EXPOSURE M+n (3)	STD FUNCTION Z(M+n) (4)	CORRECTIO N FUNCTION U(n) (5)	REFERENCE PERIOD t(N) (6)	REFERENCE DATE (7)	NORTH MORTALIT Y LEVEL (8)
20	0.8299	52.4	0.223	0.14365	8.9	1980.8	17.3
25	0.7529	57.4	0.295	0.11106	11.4	1978.3	16.9
30	0.6658	62.4	0.381	0.07021	14.3	1975.4	21.1
35	0.5602	67.4	0.482	0.01266	17.6	1972.1	16.9
40	0.4450	72.4	0.606	-0.06408	21.7	1968.0	15.0

Source: The present study.

The estimates obtained from orphanhood data for sentinel cluster refer, to periods between 8 and 21 years prior to the census. The estimates imply that male adult mortality levels

fluctuated somewhat between ages 60 and 70. The slightly high mortality associated with n of 30 is suspect because it does not conform with the trend of the rest. It is possible that the survivorship ratio for n of 40 is distorted both by over reporting of age and by misreporting of paternal survival.

The pace of mortality change implied by these estimates between 1978 to 1980 seems implausibly rapid. It seems that mortality of adults males in sentinel cluster seemed to be on the increase, increasing rapidly by about one mortality level bi annually.

From the foregoing statement, it transpires that the data for Sentinel Cluster may be slightly not as consistent as one would desire and that further analysis and additional evidence may be necessary to establish with greater degree of certainty the mortality levels to which the male population in HIV sentinel cluster has been subject. As already been mentioned the basic methods for adjusting orphanhood data depends on availability of two surveys which in the Kenyan situation is not feasible as the CBS does not have the previous Censuses.

The same procedure was used to derive the rest of the adult mortality estimates. However minor adjustments was employed to derive female adult mortality as outlined in chapter three subsection 3.4.1.2. The complete results are outlined in annex B. These results are then used to discuss the prevailing adult mortality differentials displayed in the chapter five, section 5.2.

3.4.3: CALCULATION OF THE LIFE TABLE.

A life table is the demographers way of representing the effects of mortality. A complete life table consists of several functions or sets of numbers, each representing a different aspect of the impact of mortality (UN,1990:3; Shyrock 1973, Vol.2: 429-461). The core of the life table is the set of the $l(x)$ values. Letting x denote age, the $l(x)$ values represent the number of survivors by age out of an initial number of births (100,000 in this study).

The probability of surviving was derived as already mentioned in the relevant sections by subtracting one from the computed probability of dying (q_x) for child and for adults. The study then focused on linking the two estimates given that their computations required different formulae. Taking the computed values of l_x from Child mortality estimates and those from adult mortality estimates. The computed q_2 , q_3 and q_5 mortality levels was averaged to give the mean mortality levels for the infant and child for both males and females. For adult female mortality, the mean mortality levels was derived from $l(45)$, $l(50)$ and $l(55)$ while for males the mean mortality level was derived from $l(55)$, $l(60)$ and $l(65)$. Thus for females the study took the values of l_x , from $x = 1, 2, 3, 5, 10, 15, \dots$, up to 40, then from 45 onwards the calculated values from adult mortality estimates from orphanhood method were taken. For males the study took the values of l_x from $x = 1, 2, 3, 5, 10, 15, \dots$, up to 50, then from 55 onwards, values of l_x from calculated adult mortality

estimates were used.

After computing the probability of survival $l(x)$, this is then multiplied by the radix l_0 to obtain the number of survivors at age x , $l(x)$.

If you add those who have survived between age x and $x+n$, the resulting figure is the probability of survival nPx . ie

$$nPx = l(x+n)/l(x) \quad (34)$$

The proportion of the cohort (nqx), who were alive at the age interval x and who will die at the end of the age interval $x+n$, is then derived as:

$$_nq_x = 1 - nPx \quad (35)$$

The number of persons who die between age x and $x+n$ out of the original cohort, is then derived as $ndx = l(x) - l(x+n)$. (36)

The number of person years lived between age x and $x+n$ (ie the number of persons in the stationery population in the age interval is given by:

$$ndx = n/2 [(l(x)+l(x+n))] \quad (37)$$

However for age groups 0-1, the formula is slightly adjusted to:

$$_1L_0 = 0.3L_0 + 0.7l_1 \quad (38)$$

and

$$_4L_1 = 1.3l_1 + 2.7l_5 \quad (39)$$

for age groups 1 to 5.

And $L(x+)$ is given by:

$$L(x+) = L(x) . Log L(x) \quad (40)$$

for last age group.

$T(x)$ is the total number of population from age x , it shows

the total number of persons in the stationery population in that age group and the subsequent age intervals

$$T(x) = T(x+n) + nLx \dots \dots \dots (41)$$

And finally $e(x)$, which is the expectation of life at age x or the average number of years remaining to be lived by those surviving to age x , on the basis of the prevailing mortality.

This is given by:

$$e_x = Tx/lx \dots \dots \dots (42)$$

ESTIMATION OF

3.5: HIV/AIDS PREVALENCE RATES.

There are basically two methods for calculation of prevalence rate: Point prevalence and period prevalence. Point prevalence of a disease is its frequency at a given moment in time. It is the measure of what prevails or exists and corresponds to current bank balance. Expressed as a rate for a specified population, point prevalence is the proportion of that population who exhibit the disease at a particular instant. The numerator includes all people having the disease at the given moment, irrespective of the length of time elapsed from the beginning of the illness to the time when the point is measured. The denominator is the population among whom the disease is ascertained.

Period prevalence on the other hand is the number of cases that were observed during (rather than at) a specified time, for example during a year. Period prevalence therefore, consist of the point prevalence at the beginning of a specified period of

time plus all cases that arise during the period. Period prevalence is a particularly complex measure, since both point prevalence and incidence are incorporated in it. In this study, for HIV point prevalence was used, while period prevalence was used for AIDs. The prevalence rate is therefore given by:

$$\frac{\text{Disease}}{\text{Time (Period)}} \times \frac{10^2}{\text{Total Population}} \dots \dots \dots (43)$$

The following steps were followed to estimate HIV prevalence rate. As already mentioned, the data used to calculate HIV prevalence in this study ^{was} is the ANC data. This data as already pointed out have limitations. Based on these limitations the ^{based on assumption} following adjustment factors were derived to smoothened the observed HIV results.

Factor 1. That 10 percent of the reported AIDS cases are over the age of 50. Further, the female population aged 15-49 is 40.2 of and the population 15+ males is 15.6 percent all females giving the adjustment factor of 0.86.

According to 1993 KDHS, 85 percent of females 15-49 reported to be sexually active. This gives the second adjustment factor.

The third adjustment factor was on the basis that since the surveillance data refer only to Women, and looking at the ratios of reported AIDS cases for both Males and Females, this ratio has declined from 2:4 to 1:1 in 1996. It is expected to be 1.0 in the next few years (USAID, 1998). This study used the adjustment factor of 1.0.

Further the present study adds a fourth adjustment factor, that the general population aged 15-49 has a 20 percent higher prevalence than the ANC. This is mainly because they are likely to have multiple partners, further it has been documented to be the case in Uganda (USAID, 1996).

Therefore the adjustment factors used in this study was 0.877. The NASCOP's adjustment factor of 0.731 was also employed for comparison.

3.5.1: Urban prevalence rate, differentials.

Thus using the high HIV cluster for illustration, the HIV prevalence and incidence was arrived at as follows:

The total number of women who visited the District hospitals in the high HIV clusters in 1996 for instance for the first time with the current pregnancy was 661 out of which 539 women tested negative, while 122 tested positive. Thus using formula ⁴²***. The HIV prevalence was derived as follows:

$$(122/539) * 100 = 22.634. (44)$$

This prevalence rate imply that about 22 of 100 women are HIV positive. As already stated, this prevalence rate have limitations, for instance, it covers only women and only women who are sexually active, these women are further mostly in age groups 15-49 etc. Adjusting for the factors above using the studys adjustment factor of 0.877, the study got:

$$22.634 * 0.877 = 19.850 (45)$$

Table 3.1⁶ displays the adjusted values of HIV prevalence

The 1994-95 urban prevalence rates for all the clusters were derived in similar manner.

for all the three clusters. The same table also displays the prevalence rates adjusted by NASCOP adjustment factors.

Table 3.1^b: Distribution of adjusted urban HIV prevalence by HIV clusters.

HIV CLUSTERS	YEAR OF TESTING						
	1990	1991	1992	1993	1994	1995	1996
HIGH	10.078 8.400*	12.176 10.149*	19.996 16.667*	18.803 15.673*	31.738 26.454*	22.144 18.458*	16.187 13.492*
MID	4.899 4.084*	11.073 9.229*	11.515 9.613*	12.942 10.787*	14.915 12.432*	13.816 11.516*	10.066 8.390*
LOW	1.666 1.389*	4.345 3.622*	8.951 7.461*	4.913 4.095*	9.379 7.818*	11.477 9.566*	8.073 10.628*
SENTINEL	4.892 4.077*	9.577 7.983*	12.504 10.422*	11.012 9.179*	17.141 14.288*	15.167 12.648*	10.628 8.859*

Source: The present study.

Note: Starred figures were derived using NASCOP adjustment factors.

The results of the table 3.1^b reveal that there was a high HIV prevalence in 1994 than in the proceeding and succeeding years in both the high and the mid HIV clusters while the low HIV cluster, the peak was in 1995.

The table also reveal that in the high HIV cluster about ten percent of the population aged fifteen plus were infected with the HIV virus in 1990. In 1996 however there were about 31 percent of the adult population aged 15 plus who were HIV positive, thereafter the prevalence declined by six percent to stand at 16 percent in 1996.

An examination of table 3.1^b also reveal the same pattern to occur in the mid HIV cluster, however, in this cluster the change

was more gradual increasing from four percent in 1990 to peak at fourteen percent in 1994 thereby declining gradually to stand at ten percent in 1996.

In the low HIV cluster only one percent of the adult population was infected with the virus, however the figure of 1992 is suspect since it does not conform with the trend of the rest, further in the low HIV cluster, the peak is at 1995 (eleven percent), before stabilising.

The implication of the above is that in the high and mid HIV clusters, the prevalence has reached saturation levels and therefore stabilising while in the low HIV cluster, it is just picking up.

The prevalence prevailing as at 1996, show that high HIV cluster was about twice that of the low HIV cluster and about five percent more than that prevailing in the mid HIV cluster. At 13 percent, high HIV cluster prevalence rate was above the sentinel rate of eight percent. However the mid and the low HIV cluster compare favourably with the sentinel cluster (see:Table 3.16 3.17).

3.5.2: Rural HIV Prevalence Rate, ~~Differentials~~

Most scholars in the field of HIV/AIDS believe that in Africa and in Kenya in particular the rural prevalence rate approaches half to two thirds that of urban areas depending on the urban HIV prevalence rate. Thus high prevalence rate areas, it is estimated that the rural-urban prevalence is 0.8 in the

high prevalence areas, 0.7 in the mid prevalence areas and 0.5 in the low prevalence areas (NASCOP, 1996; USAID, 1996). Using these adjustment rates, the rural prevalence rates were computed as follows (still using the high HIV cluster for illustration).

The urban HIV prevalence rate for high HIV cluster in 1996 from table 4.5 was 16.187, therefore the rural prevalence rate in high HIV cluster was computed as follows:

$$\text{Rural Prevalence} = \text{Urban prevalence} * \text{adjustment factor} \quad (46)$$

Substituting the values:

$$\text{Rural Prevalence} = 16.187 * 0.8 = 12.950 \quad (18)$$

That is, the HIV prevalence rate in rural areas of high HIV cluster was 12.950 percent of the total adult population in that cluster. Table 3.18 has the computed rural prevalence by HIV clusters, also displayed are the rates derived using the NASCOP adjustment factors.

Table 3.18: Distribution of adjusted rural HIV prevalence by HIV cluster.

HIV CLUSTERS	YEAR OF TESTING						
	1990	1991	1992	1993	1994	1995	1996
HIGH	8.062 6.720*	10.208 8.119*	15.997 13.334*	15.042 12.538*	25.390 21.163*	17.715 14.766*	12.950 10.794*
MID	3.429 2.859*	7.751 6.460*	8.061 6.729*	9.059 7.551*	10.441 8.704*	9.671 8.061*	7.046 5.874*
LOW	0.833 0.695*	2.345 1.811*	4.476 3.731*	2.457 2.048*	4.690 3.909*	5.739 4.783*	4.037 3.365*
SENTINEL	2.446 2.039*	4.789 3.992	6.252 5.211*	5.506 4.590*	8.571 7.144*	7.584 6.324*	5.314 4.430*

Source: The present study.

Note: Starred figures were derive using NASCOP adjustment factors .

The prevalence of HIV virus in the rural areas of the various clusters are displayed in table 3.18. These rates displays similar pattern as the urban prevalence rates. The peak years in the rural areas in 1994 for both the high, the mid and the sentinel cluster, while for the low HIV cluster, the peak was in 1995.

The high HIV cluster in 1996, had a high prevalence rate (12.950) than other clusters and above the sentinel average of five percent. Mid HIV cluster had the second highest prevalence rate (seven percent) followed by the low HIV cluster (four percent). However the low HIV cluster prevalence unlike the mid and the high HIV cluster were below the sentinel average of five percent (see: column 8 of table 3.18).

The computed prevalence rates of this study was about one percent more than the prevalence rates of NASCOP. These rates are further surmise to be about thirty percent of the actual rates.)

3.6 ESTIMATION OF HIV/MID IMPACT ON CHILD MORTALITY
~~3.6.3~~ According to the IMPACT model developed by 1995 (Stover, 1996),
 The under five mortality rate due to AIDS can be calculated

from the adult prevalence of HIV, the perinatal transmission rate and the proportion of HIV infected children who die before reaching age five:

$$U5MRA = PrevWRA * PTR * PD5 * 104 \dots \dots \dots (47)$$

$$U5MR = U5MRWA + U5MRA * (1-U5MRWA/1000) \dots \dots \dots (48)$$

Where U5MRA is the under five mortality rate due to AIDS;
 U5MR is the under five mortality rate; PrevWRA is the adult HIV

prevalence; PTR is the perinatal transmission rate, PD5 is the proportion of HIV infected children dying before age five. For the purposes of this study, the following epidemiologies have been selected based on the studies done by the USAID, The future group and the research triangle: the proportion of HIV infected infants dying before age five was ninety percent, perinatal transmission rate (PTR) was taken to be forty percent, the adult prevalence rate was taken from the computations done in ^{section 3.5} ~~chapter~~ four.

Using the total HIV sentinel clusters for illustration, the impact of HIV/AIDS on child mortality can be estimated as follows:

The under five mortality rate for total sentinel HIV regime cluster was 0.177 per thousand, the adult prevalence rate (as at 1989) was 3 percent, the PTR was forty percent and the proportion of HIV infected infants dying before age 5 was ninety percent, then the under five mortality rate to AIDS is estimated using equation 47 as:

$$U5MRA = 3\% * 40\% * 90\% * 10 = 0.108 \dots \dots \dots (49) \quad 41$$

Therefore the under five mortality rate will be given employing equation 48 as:

$$U5MR = 117 + 10.8 * (1 - 117/1000) = 126 \dots \dots \dots (50) \quad 42$$

This gives a ratio of 1.08 (ie 126/117). The results reveal that with the prevalence prevailing in the three HIV clusters, the under five mortality rate increased by eight percent. The rest of the impact of HIV/AIDS on child mortality was derived in

similar manner. Table 5.5 and Table 5.6 of chapter five gives the impact of HIV/AIDS on underfive mortality by socio - economic and household environmental factors respectively.

3.7: STATISTICAL METHODS.

As already mentioned in the preceding sections, this study used indirect techniques to estimate mortality levels and the impact of HIV/AIDS on mortality. The use of indirect techniques are complete in themselves and do not need the statistical analysis to support it. However, this study categorized the prevailing observed HIV/AIDS into regime clusters. The present study felt that it would be appropriate to establish whether the categorization of the clusters was statistically meaningful. This was done by use of analysis of variance.

One way analysis of variance.

Analysis of variance test is used to test whether sub-samples may be regarded as homogenous or alternatively whether there is some indication that the sub-samples were drawn from different populations (Yule and Kendall, 1968:503).

It is used to test whether the observed difference in more than two means can be attributed to chance or whether they are indicative of the actual differences among the means of the corresponding populations (Freud, 1974:334). The formula which form the basis of one-way-analysis of variance is given by:

$$ANOVA = n\sum (x_i - \bar{x})^2 + \sum \sum (x_{ij} - \bar{x}_i)^2 \quad (51) 43$$

Where, x_j is the j th observation of the i th sample ($i=1,2,\dots,k$) and $j = 1,2,\dots,n$) and \bar{x} is the grand mean ie means of all the n th observation and \bar{x}_i is the mean of the i th sample.

CHAPTER FOUR.

HIV/AIDS DIFFERENTIALS

This section serves as a bench mark for the discussion of the study results, the presentation in this section is through tables which illustrates the percentages, sex ratios and the incidence of AIDS per ten thousand of the population. The first section involves AIDS differentials whilst the second part encompass the computation of HIV prevalence/incidence and differentials

4.1: Reported AIDS differentials.

Kenya has a sentinel surveillance system which provides the basis for estimating the extent of HIV/AIDS infection. It is the policy of the ministry of Health that requires all major hospitals in the country (private or Government) to report the number of AIDS cases diagnosed in their hospitals to the NASCOP monthly.

Most AIDS experts believe that vast majority if not all of HIV-infected persons will eventually develop AIDS and die from the disease (WHO, 1991:) regardless of the length of the incubation (Chin, 1992). Evidence further suggest that HIV leads to AIDS more rapidly in regions with relatively high prevalence of malnutrition and parasitic diseases (Katlama and others, 1994; Piot and others 1984).

The progression of the disease to AIDS depend on other co-factors (~~Chapter 2~~ ^{Subsection 2.3, in Chapter 2} ~~Theme II~~) which do vary from region to region. A

look at the reported AIDS cases by the various HIV prevalence clusters revealed the following (Table 4.1).

Table 4.1: Distribution of reported AIDS cases by HIV surveillance Clusters 1990-1997.

HIV SENTINEL CLUSTER	REPORTED AIDS CASES
HIGH HIV CLUSTER	19,863 (42.8)
MID HIV CLUSTER	16,496 (32.2)
LOW HIV CLUSTER	10,001 (24.9)
SENTINEL	46,360 (100)

Source: Nascop. Kenya.

Note:

- Figure in parentheses are column percentages (Sentinel).
- Missing cases 107.

A look at the reported AIDS cases by HIV surveillance ^{regime} clusters of High, Mid and Low revealed that the high HIV cluster had the highest percentage of reported AIDS cases (42.8 percent), this is about twice that of the low HIV ^{regime} cluster (21.6 percent). ^{regime} Mid HIV cluster had about 35.6 percent.

The probable reason for this is that the high HIV cluster is mainly situated in the major international transportational routes, these routes are plied by heavy trucks whose drivers are in the high risk category.

The other reason for the observed AIDS distribution revealed in the table ^{4.1} ~~6.1~~ could be for the fact that high HIV cluster is mainly urban based while the low HIV cluster is mainly rural based and in Kenya, the prevalence of AIDS is more in urban oriented areas than rural. The other factor could be that, given

the fact that most people still do not know how best to protect themselves, those areas where the prevalence of HIV is high will also have high AIDS cases due to the possibility of multiple viral infection. Some people for instance wear the condom on their thumbs while engaging in sexual intercourse in line with how the distributors of condoms demonstrated to them how to put them on, others even wash and peg the condoms out to dry after use ^{for latter use} and ~~use them latter~~, yet others wear the condoms on their way to meet a date (see: Ochola-Ayayo, 1996). In areas where the virus is concentrated in a population the net effect is that these people will be more at risk of multiple viral infection which are observed in the AIDS distribution.

The other reason could be the removal of sexuality from the control of the community resulting in individual decision about whom, where, with whom and for what purpose to have sexual intercourse. This results in indiscriminate sex which exposes one to the risk of AIDS. This is exacerbated by the fact that the age at marriage increases while the age at onset of sexual intercourse declines (see: NCPD 1995, Ocholla-Ayayo, 1992) exposing one to longer duration of age at sexuality, often with potential changing of sexual partners and therefore the risk of HIV infection which is observed in the AIDS distribution.

It has been documented that the women are more at risk of HIV infection than men mainly due to among others the difference in the rate of exchange of fluids and to the sheer volume of

virus that can be hosted in the semen in contrast to the vaginal fluids. In the absence of offsetting factors, this difference alone could account for a heavier presence of HIV among females. The above table (Table 4.1) however camouflage the actual difference inherent in the AIDS infection in terms of gender. A desegregation of the reported AIDS cases by sex revealed the following (Table 4.2).

Table 4.2: Distribution of reported AIDS cases by sex, and sex ratio. HIV Surveillance regime clusters, 1990-1997.

HIV SURVEILLANCE CLUSTER	MALES	FEMALES	SEX RATIO
HIGH HIV CLUSTER	10257 (41.1)	8606 (39.5)	1:19
MID HIV CLUSTER	9215 (36.9)	7108 (32.7)	1:30
LOW HIV CLUSTER	5501 (22.0)	6055 (27.8)	0:90
SENTINEL TOTAL	24973(100.0)	21809	1:15

Source: NASCOP.

Note:

- figures in the parentheses are Column percentages (Sentinel).
- Missing cases 170.
- X^2 Tabulated = 5.991, X^2 Calculated 224.

Table 4.2 reveals that the overall male-female ratio was 1:15 in sentinel, with the value ranging from 0:90 in the low HIV cluster to 1:30 in mid HIV cluster. In a nutshell, there are more males than females with AIDS in high and mid HIV clusters (1:19 and 1:30 respectively). While in the low HIV cluster, the opposite is the case. This is mainly because all AIDS cases reported in Africa have been in heterosexuals who are not IV drug users, consequently the male to female ratio of AIDS cases in

Africa has been close to unity.

A further look at table 4.2 reveal that of all the reported male cases, High HIV cluster at 41.1 percent had the largest share of males reported to have AIDS followed by Mid HIV cluster (36.9 percent), the lowest reported male cases are to be found in Low HIV cluster (22 percent). Interestingly the number of AIDS cases amongst males tend to follow the clusters with the big towns, these are the towns that attract male migrants.

Column 3 of table 4.2 also reveal that the reported number of females with AIDS follow similar pattern to that of males with High HIV cluster 39.5 percent, mid HIV cluster 32.7 percent. High HIV cluster had the highest AIDS cases reported, while Low HIV cluster at 27.8 percent had the lowest number of reported females with AIDS.

The probable reason for the high number of males than females with AIDS in the high and mid HIV clusters is that these areas are in-migration regions, it is likely therefore to find single males in excess of females. These men often liberated from the mandate of familial roles and traditional culture for the first time, may engage in sexual behaviour that might not have been tolerated in the rural community. Such behaviours might include unprotected sex with multiple sexual partners, pre marital or extramarital casual sexual relationship or sexual practices with commercial sex workers (who typically have HIV prevalence level far in excess of the National average).

The other reason could be due to the level of the

concentration of commercial sex workers² in the big towns to be found in these clusters, and also the fact that these areas fall within the major transportational routes, (Track drivers are in high risk group and are believed to play a major role in the transmission of HIV).

Further
Lastly, while males may be more likely to engage in casual sexual contacts than females, the risk of HIV infection for Women depend not on their own sexual partners but on those of their partners as well, furthermore because females appear to have a higher biological susceptibility to infection, the overall results of these countervailing trends is a sex ratio of HIV infected persons that approaches unity at the population level.

Ochola-Ayayo, (1992) contend that the family of the AIDS victim do not give up on their sick member and if other treatment fails, they transport them back 'home' (home being the ancestral lands) to try traditional medicine. It has also been surmised that those who develop aids often tend to go back to their home districts to await their death if all the medications have failed in attempt to reduce the funeral expenses. Since most of the AIDS cases occurred to older people who might have migrated to towns, an attempt was made to capture the reported AIDS cases by district of birth (Table 4.3). Further as already mentioned by

²Commercial sex workers have typically a high viral transmission to their clients well in excess of the National average.

Berkeley (1990:20-30) prevalence of infectious diseases especially those that afflicted the victim when the victim was in his/her early years greatly influences the progression of the HIV virus to the disease. A look at the percentage distribution of the reported AIDS cases by district of birth therefore revealed the following (Table 4.3).

Table 4.3: Percent distribution of reported AIDS cases by District of birth, HIV clusters.

HIV CLUSTER	1989	1990	1991	1992	1993	1994
HIGH	2051 (44)	1823 (44)	1931 (34)	1676 (26)	1805 (26)	1420 (35)
MID	1253 (27)	917 (22)	927 (16)	947 (15)	1313 (19)	677 (17)
LOW	723 (15)	784 (19)	1365(24)	1738 (27)	1937 (28)	1671 (41)
UNKNOWN	359 (8)	466 (11)	1188 (21)	1910 (30)	1878 (27)	235 (6)
N/A	303 (6)	178 (4)	222 (4)	160 (2)	96 (1)	43 (1)
SENTINEL	4689 (100)	4168 (100)	5633 (100)	6431 (100)	7029 (100)	4046 (100)

Source: Nascop

Note: Figure in Parentheses are column percentages (Sentinel).

Table 4.3, reveal an interesting finding in that, from 1989 when the AIDS impact were just beginning to be felt, the high HIV regime cluster had the highest number of diagnosed AIDS cases born in the cluster (44 percent). This was about double the nearest cluster (mid HIV regime cluster at 27 percent), and about three times the lowest cluster (low HIV regime cluster at 15 percent). From 1992, the pattern changed, for instance, in 1994, the low HIV cluster had the highest number of AIDS cases born to the cluster (41 percent) and mid HIV cluster at 17 percent had the lowest.

The probable reason for this is that those with AIDS tend to die more quickly, and the development of HIV to AIDS is equally faster, such that when one gets HIV it develops to AIDS more quickly in high HIV clusters (Owuor: This study), than in the other clusters, such that with the passage of years, the low HIV cluster will have a slightly more cases of AIDS than the other clusters because of the twin factors of gradual development of HIV to AIDS and the latter to the disease, the net effect is more cases of AIDS. Besides, where the prevalence is high, the AIDS cases tend to stabilize and even decline whereas where it is low, it tends to pick up.

Quinn and others contend that when HIV is transmitted through heterosexual contacts, the maximum level of seroprevalence are generally found among young adults and very young children. Berkeley puts the peak ages at ages 20-24 for Women and 25-29 for men (Berkeley, 1992:22-32). Killewo and others (1990) on the other hand puts it at age 25-34 for both sexes, Rwandan seroprevalence study group (1984) puts it at near age 35. To get a clear picture of the Kenyan situation, the reported AIDS cases was looked at vis avis age of the victim (Table 4.4).

Table 4.4: Distribution of reported AIDS cases by age and sex of Patient. HIV clusters.

HIV CLUSTER		AGE GROUP OF PATIENT								TOTAL
		0-4	5-14	15-19	20-24	25-34	35-44	45+	NS	
HIGH CLUSTER	M	921 (9)	126 (1.2)	137 (1.3)	630 (6.1)	3227 (31.5)	2071 (20.4)	1062 (10.4)	2073 (20.2)	10257
	F	771 (9)	128 (1.5)	456 (5.3)	1425 (16.6)	2802 (32.6)	967 (11.2)	322 (3.7)	1635 (19)	8606
MID CLUSTER	M	1152 (13.5)	49 (0.6)	90 (1.1)	519 (6.1)	2676 (31.3)	1742 (20.4)	879 (10.3)	1451 (17)	8554
	F	724 (11.3)	41 (0.6)	277 (4.3)	1026 (1.6)	2337 (36.5)	777 (12.1)	333 (8.2)	936 (14.6)	6396
LOW HIV CLUSTER	M	567 (9.0)	63 (1.0)	78 (1.3)	443 (7.4)	2241 (37.3)	1474 (24.5)	649 (10.8)	498 (8.3)	6013
	F	240 (3.9)	58 (1.0)	349 (5.7)	1241 (20.4)	2492 (40.9)	786 (12.9)	257 (4.2)	632 (10.4)	6087
SENTINEL TOTAL	M	2640 (10.6)	238 (1.0)	305 (1.2)	1592 (6.4)	8144 (32.8)	5287 (21.3)	2590 (10.4)	4022 (16.2)	24824
	F	1735 (8.2)	227 (1.1)	1082 (5.1)	3692 (17.5)	7731 (36.7)	2530 (12)	912 (4.3)	3203 (15.1)	21089

Source: NASCOP

Missing cases 107.

Note: ~ Figures in parentheses are row percentages.

~ M = Males while F = Females.

It is inherent in our structure that some social positions carry with them differential access to desirable scarce resources including sexual partners. It happens that younger men who have economic power are in this position are therefore in high risk of HIV infection.

Table 4.4 reveal that in Kenya the peak age for AIDS cases for both sexes is ages 25-34. The cases increases gradually from age groups 15-19 to peak at age group 25-34 before gradually declining. There is also an observed high number of cases in ages 0-4, in all the clusters with more males ^{children} infected than

females. The probable reason for the high number of AIDS cases in this age group (0-4) is the probable transmission of the virus from the mother to the child either/or during pregnancy, birth, or breast feeding (or in the cutting of the umbilical cord). While the observed peak at later years is because, of the latency period of HIV to AIDS which is put at between 5-10 years in Africa (Berkeley, 1990:20-30).

This is attributed to the prevalence of other infectious agents further since prevalence starts with onset of sexuality which in Kenya is very low, (Ocholla-Ayayo, 1996) the net effect is the observed AIDS cases at higher ages, with men being more affected because of their migratory nature in search of jobs (ABC 1996), live a bachelors life and are more likely to have multiple casual sexual partners among others. All these make them vulnerable to HIV infections and as already mentioned virtually all HIV infections eventually leads to AIDS. A look at the HIV prevalence therefore reveal the following (section 4.2).

4.2.: Differentials by HIV Incidence

The number of adults at time 't' that matches the assumed prevalence levels is the number of adults multiplied by the assumed prevalence, it is given by:

$$\text{Target HIV}(t) = \text{Adults}(t) * \text{Prevalence}(t) \quad . . . \quad (19)$$

Using the high HIV cluster for illustration, the projected urban adult population in this cluster was 1293440, using the prevalence rates appearing in table (**), the incidence of HIV

cases was derived as follows.

$$\text{HIV Incidence (t)} = 1,293,440 * 16.187 = 209,369 \dots ()$$

This implies that 209,369 adults out of 1,293,440 adults in the urban high HIV cluster had HIV virus. More significantly this translates to about 13,492 adults per every 100,000 adult persons in this cluster urban areas had HIV virus. The full set of HIV incidence is displayed in table 4.7.

Table 4.7: Distribution of HIV incidence rate by HIV cluster.

HIV CLUSTERS	PARAMETERS			
	POPULATION 15+	HIV PREVALENCE	HIV CASES	HIV INCIDENCE RATE PER 100,000
URBAN AREAS				
HIGH HIV CLUSTER	1,293,440	16.187 (13.492)	209,369 (174,511)	16,187 (13,492)
MID HIV CLUSTER	459,709	10.066 (8.390)	46,274 (38,570)	10,066 (8,390)
LOW HIV CLUSTER	228,328	8.073 (6.729)	18,433 (15,364)	8,073 (6,729)
SENTINEL CLUSTER	1,981,417	10.628 (8.859)	210,585 (175,534)	10,628 (8,859)
RURAL AREAS				
HIGH HIV CLUSTER	977,290	12.950 (10.794)	126,559 (105,489)	12,950 (10,794)
MID HIV CLUSTER	1,398,464	7.046 (5.874)	98,536 (82,146)	7,046 (5,874)
LOW HIV CLUSTER	2,445,337	4.037 (3.365)	98,718 (82,286)	4,037 (3,365)
SENTINEL CLUSTER	4,821,091	7.584 (4.430)	365,632 (213,574)	7,584 (4,430)

Source: The present study.

Note: Figures in Parentheses were derived using are NASCOP adjustment factors.

The table (Table 4.5) reveal that high HIV cluster had about 209,369 persons in the urban areas with HIV virus. This translates to about 16,187 persons per 100,000 of the population being HIV sero positive. This rate was way above the sentinel rate of 10,628 per 100,000 persons.

The mid HIV cluster closely follows the high HIV cluster, with about 10,066 persons per 100,000 of the adult population being HIV sero positive. This compares favourably with the Sentinel rate of 10,628 HIV sero positive individuals per 100,000 of the adult population.

The low HIV cluster came a distant third, with about half the High HIV cluster HIV incidence. This cluster at 8,073 persons per 100,000 of the adult population, was below the sentinel rate of 10,628 persons per 100,000 persons of the population.

The possible reason for the observed incidence rates is possibly due to the timing of the entry of the virus in these clusters. High HIV clusters being mainly situated along major international transportational routes is more likely to have been introduced to the virus much earlier than the low HIV cluster which is mainly rural based, while the mid HIV cluster is mainly peri-urban.

Table 4.5 also reveal that in the rural areas, the high HIV cluster had about 12,950 persons per 100,000 who were HIV sero positive. This is above the sentinel rate of 7,584 HIV persons per 100,000 of the adult population.

The mid HIV cluster had a rural HIV incidence rate of 7,046 HIV sero positive individuals per 100,000 adult population, ie about 98,536 persons positive out of the total adult population of 13,98,464. This incidence rate however is slightly below the sentinel average.

The low HIV cluster the same table reveal had about 4,037 persons HIV sero positive per every 100,000 of the population residing in the rural areas. This compares favourably to the sentinel rate of 7,584 persons per 100.000 of the adult population.

Based on these computed, the average HIV prevalence and incidence was computed. To derive the overall prevalence in the clusters, the computed HIV incidence for both urban and rural were summed up and generated backwards to give the prevailing HIV prevalence and incidence in the clusters. See table 4.5.

For instance, deriving the HIV prevalence we use the formula 16. The number of HIV cases in the high HIV cluster was 335,928 (ie 209,369 persons in urban and 126,559 persons in rural areas) out of the total adult population of 2,270,730 in the high HIV cluster. Substituting these values in the formula, the study got:

$$HIV\ Prevalence = (335,928/2,270,730) * 10^2 = 14.794\ (20)$$
and HIV incidence was derived as:

$$(335,928/2,270,730) * 10^6 = 14794 \dots\dots\dots (21)$$

This implies that about 14,794 persons for every 100,000 adult population was HIV positive. This against the HIV prevalence rate of 14.794.

CHAPTER FIVE.

MORTALITY DIFFERENTIALS.

Introduction.

This section encompass mortality differentials by the variables outlined in the empirical model (see: sub section 2.5 page **) using the methodologies outlined in chapter three subsection 3.4. The first section discusses child mortality differentials, the second part discusses adult mortality differentials while the third part has the impact of HIV on mortality.

The picture painted of a Kenyan mortality situation often is that of marked variations, variation in relation to such factors as Occupation, Gender, Marital Status, level of education, Sewage disposal of the household, Floor material type etc.

These factors also, has already been seen in the literature review, also happen to be the co-factors for the HIV/AIDS Infection, Such that introduction of the HIV virus and/or the type of HIV regime prevailing in the region will exacerbate the already existing mortality differentials. As already mentioned the study aimed at capturing the existing mortality differentials on the ground, for High, Mid and Low HIV regimes clusters.

5.1: Child mortality differentials by HIV regime clusters.

This section covers, the Child mortality differentials using the computed rates appearing in annex b. The parameter used to

estimate and discuss child mortality differentials was the probability of dying by age five (q5).

The basis for using this index was that it captures almost all the mortality risk prior to adulthood and is less affected by the age profile of child rearing practices such as weaning, than the infant mortality rate (Eubank and others 1993:13). Tables 5.1 and 5.2 gives the computed $q(x)$ values by the variables outlined in the empirical model, also displayed are the mortality levels and the reference periods the estimates refer.

Table 5.1: Child mortality differentials by socio-economic factors. HIV clusters.

SOCIO ECONOMIC FACTORS	HIV PREVALENCE REGIMES			
	HIGH	MID	LOW	SENTINEL TOTAL
BY SEX OF CHILD				
MALES	0.138 (16.1) 6.3*	0.132 (16.4) 6.2*	0.101 (18.1) 6.1*	0.121 (16.7) 6.2*
FEMALES	0.126 (15.8) 6.3*	0.124 (15.9) 6.2*	0.092 (17.8) 6.1*	0.112 (16.6) 6.2*
BY MARITAL STATUS OF MOTHER				
Single	0.077 (19.1) 7.8*	0.079 (19.1) 8.6*	0.058 (20.5) 10.0*	0.071 (19.6) 8.7*
Monogamous	0.080 (19.0) 6.0*	0.083 (18.8) 5.9*	0.088 (18.5) 5.8*	0.152 (14.9) 5.8*
Polygamous	0.121 (16.6) 5.4*	0.154 (14.9) 5.2*	0.134 (15.4) 5.0*	0.130 (16.1) 5.6*
Widowed	0.150 (15.1) 3.6*	0.149 (15.1) 3.5*	0.134 (15.4) 4.1*	0.142 (15.5) 3.8*
Divorced/separated	0.129 (16.2) 4.6*	0.128 (16.2) 5.1*	0.124 (16.4) 6.1*	0.127 (16.3) 5.2*
BY EDUCATION OF THE MOTHER				
No education	0.139 (15.6) 4.7*	0.149 (15.1) 4.8*	0.135 (15.8) 4.7*	0.139 (15.6) 4.7*
Primary	0.086 (18.4) 7.0*	0.079 (19.1) 6.9*	0.064 (20.1) 7.2*	0.063 (20.1) 9.5*
Secondary +	0.047 (21.2) 5.7*	0.053 (20.8) 5.8*	0.045 (21.4) 8.2*	0.051 (21.0) 4.6*
BY Occupation of the mother				
Professional	0.053 (20.4) 3.9*	0.066 (19.9) 3.9*	0.047 (21.2) 4.1*	0.055 (20.7) 3.9*
sales/services	0.135 (15.8) 6.5*	0.142 (15.5) 5.7*	0.096 (18.0) 6.1*	0.144 (15.4) 5.5*
all agriculture	0.181 (13.5) 5.7*	0.141 (15.5) 5.9*	0.105 (17.3) 5.9*	0.130 (16.1) 5.9*

Source: The present study.

Note:

- i. Figures in parentheses are mortality levels, North model of Coale and Demeny regional life tables.
- ii. Starred figures are Time references to which each of the mortality estimates refers.

5.1.1: Child mortality differentials by Socio-economic factors and HIV regime clusters.

Studies done in developing countries have emphasized, the inverse association between mortality and various Socio-economic factors and underscored the significance of these factors on the mortality levels and differentials existing in a population.

To avoid missing out the extensive bases of bio-socio-economic factors therefore, factors which may be of crucial importance in the mortality differentials in the I & C, the present study adopts as its analytical variables: sex of the child, parental occupation, marital status of the mother and occupation of the mother. The discussion are in the proportion of ^{age five} children dead for each variable category.

A look at child mortality differentials by gender of child by different HIV regimes clusters revealed the following (5.1.1a).

5.1.1a: Child mortality differentials by sex.

Table 5.1 gives the mortality differentials by sex of child. Generally the study found female children to have lower mortality than their male counterparts in all HIV clusters. Further there was noticeable differentials in child mortality for both sexes by HIV clusters with high HIV clusters experiencing high child mortality followed by the mid HIV cluster and while low HIV regime cluster experienced low mortality rate.

In the high HIV cluster therefore, male children experienced high child mortality (0.138) than female children (q5 of 0.126).

These rates were further supported by the mortality levels in the Coale-Demeny North Model Life Table associated with them, the North Model mortality levels reveal that the females gained about one mortality level more than the male children. However these rates were in operation about the same time - six years prior to the survey.

In the mid HIV cluster, male children experienced high child mortality compared to female children (q5 of 0.132 and 0.124 respectively) slightly below that experienced in the high HIV clusters. In this cluster as in the high HIV cluster, the observed mortality differentials is further evidenced by the mortality levels associated with them, - female children gained about one mortality level with respect to male children. These rates were however both in operation some six years prior to the survey.

A closer look at Table 5.6 further reveal that mid HIV regime cluster experienced lower mortality for both male and female children than the high HIV regime cluster. This differential is such that for male children, the mid HIV regime cluster gained about 0.5 mortality levels more than high HIV regime cluster, while for female children the mid HIV cluster gained a paltry 0.1 mortality level more than high HIV implying that mid HIV cluster had lower mortality levels than high HIV cluster.

Similar mortality situation is witnessed in the low HIV cluster, however this cluster gained about two mortality levels

more than the mid and the high HIV clusters for both male and female children.

The probable reason for the observed sex differentials is probably the sex differences in the infectious disease mortality (females are more resistant) which is further influenced by cultural factors such as access of males and females to food and medical care which make females more resistant to diseases than males.

However, sex of child perse do not solely contribute to the observed differentials in the risk of death. The marital union^{WPE} of the child mother have also been documented to have a telling effect on infant and child mortality regardless of the sex of child. The mortality of children of widowed and divorced mother for instance has always been found to be higher than that of currently married one (Kichamu 1881: ; K'Oyugi, 1982:). This is mainly attributed to stress associated with widowed and the divorced and the withdrawal of financial support to these women which may force them to fend for themselves and their children further exposing them to risk factors such as AIDS, the widowed and divorced may develop new sexual partners further exposing themselves to the risk of HIV infection and passing the virus to their unborn children depending on the prevailing HIV regime. Consequently child mortality differentials was examined by mothers marital status by the prevailing HIV/AIDS regime cluster (5.1.1b).

5.1.1b: Child mortality differentials by marital status of the mother.

In this study the marital status of mother was categorised into five variable categories: single, monogamous, polygamous, widowed and divorced and separated. The results reveal that generally, the widowed experienced the highest child mortality while the never married experienced the lowest child mortality. Further, ignoring the monogamous and polygamous marital variable category, there was observed mortality differentials by HIV cluster with high HIV cluster experiencing the lowest child mortality.

In the high HIV cluster therefore, children of single women experienced the lowest child mortality (0.077), followed by those children of women in monogamous marriages (0.080). Children of women in polygamous union came a distant third (q5 of 0.121), closely followed by those of divorced and separated (q5 of 0.129) while children of the widowed had the highest child mortality (q5 of 0.150). These differential is further supported by the mortality levels and time period associated with each estimate (see table 5.1).

The mortality situation in the mid HIV cluster was slightly different from that prevailing in the high HIV cluster. In the mid HIV cluster, the single women had the lowest child mortality (q5 of 0.149) followed by those of women in monogamous union (q5 of 0.083). Children of divorced/separated came a distant third (q5 of 0.154). This mortality differentials is further supported

by the mortality levels in the Coale/Demeny regional life tables north model associated with each estimate. This translates to those children of single women at mortality level 19.1 being about 0.3 mortality level more than those of women in monogamous union, and about three, four and five mortality levels more than those of women in divorced and separated, widowed and polygamous unions respectively. However, these observed mortality estimates in the mid HIV cluster were more than those observed in the sentinel cluster.

In the low HIV regime cluster however children of single women experienced the lowest child mortality (q5 of 0.058), followed by those of women in monogamous union (q5 of 0.088). In the same cluster children of women in polygamous union and widowed experienced the highest child mortality (q5 of 0.134). This differential is further supported by the mortality levels and time references associated with each estimate.

The probable reason for this is that the widowed and the divorced are associated with substantial stress which in turn may affect child health. Further, polygamy may increase the length of the inter birth interval for women by delaying the resumption of intercourse after childbirth or by reducing the frequency of the intercourse. Secondly the men who take several wives in a polygamous society are generally wealthier, so that polygamy may be associated with increased resources for child rearing. On the other hand, these resources must be shared among more wives and more children, so that the predicted net effect is ambiguous.

A handwritten signature in dark ink, appearing to be 'J. A. T.', is written over a horizontal line.

Further those who maintain polygamy are to an extent adhering to the tradition as opposed to the modern practice. This traditionalism is likely to be associated with traditional health practices, which has been documented to be less suitable for child survival.

However, recent studies have suggested that Infant and Child mortality in developing countries is strongly associated with maternal education than with any other Socio-economic variable (Behm, 1980; Caldwell, 1979; Preston, 1989; Kibet, 1981; Muganzi, 1984; Ikamari, 1996; and K'Oyugi, 1992).

In certain societies however, maternal education may not be significantly related to child survival, such is the case in Egypt (Casterline, 1989:25-29), Turkey (Gursev, 1992:4), Bangladesh (Mafunder, 1989:109-11) and Uganda (Ebong, 1993:11).

To find out the extent to which the findings of this study reflects on either of the contention mentioned above; differentials in child mortality was looked by womens' educational levels against the prevailing HIV clusters, ~~(5.1.1c)~~

5.1.1c: Child mortality differentials by level of education of the mother.

Table 5.1 also reveal child mortality by level of education attained by the mother. Education level of the mother was categorized in this study into no education, primary education and secondary plus. The results reveal that children born to

women with no education experienced high child mortality than those of women who had at least primary level of education, while those in secondary level of education had their children experiencing the lowest child mortality. This is further supported by the mortality level associated with each q5 values. Those children born to women in primary level of education had about between 3-5 mortality level above children of women with no education and between one to three mortality level less than children of women with secondary level of education in all the HIV regime clusters.

In the high HIV cluster therefore, those children born to women with no education experienced the highest child mortality (q5 of 0.139), followed by children born to women with primary level of education (q5 of 0.086) while those children born to women with at least secondary plus level of education had the lowest child mortality (q5 of 0.047). These rates are further supported by the mortality levels in the Coale and demeny regional life tables north model. The observed mortality differentials translates into those children with primary level of education experienced about 3 mortality levels more than those of women with no education and about three mortality levels lower than the children of women with at least secondary plus level of education. Further these observed mortality levels were in operation some four, seven and five years prior to the survey for no, primary and secondary plus level of education respectively.

The table also reveal that the mortality situation in the

mid HIV cluster displayed similar results to that of high HIV cluster of a declining child mortality by level of education of the mother. This differential is such that children of women with primary education had lower mortality than those children of women with no education, but higher child mortality than those of women with secondary plus level of education (q5 of 0.079 corresponding to mortality levels 19.1; q5 of 0.149 corresponding to mortality level 15.1 and q5 of 0.053 corresponding to mortality level 20.8) for primary, no education and secondary plus level of education respectively.

These observed mortality differentials were in operation some 6, 4 and 5 years prior to the survey respectively. Further the observed mortality rates in the mid HIV cluster were lower than those in the high HIV cluster by about one mortality level except in the no level of education variable category where the observed rates were slightly high than those observed in the high HIV cluster (difference of about 0.5 mortality levels).

The low HIV cluster displayed similar patterns observed in the high and mid HIV cluster. In this cluster those children born to women with no education experienced high mortality than those born to women with primary education. While those children born to women with secondary plus level of education experienced the lower child mortality. This differential translates into those children born to women with no education experiencing about five mortality levels more than those children of women with primary level of education. Further the children of women with

primary level of education experienced about one mortality levels less than those children born to women with secondary plus level of education (see: Table 5.1).

These observed mortality levels in the low HIV cluster were lower than those observed both in the high and mid HIV cluster by about two and one mortality levels respectively for primary level variable category, while in the no education and secondary plus level of education variable category, the differential was about 0.2 mortality levels with respect to high HIV cluster and 0.7 to that of mid HIV cluster. These rates were however above those of the sentinel rates.

The probable reason for the above observed child mortality differentials by level of education of the mother is that an educated mother is in a better position to bargain for her children and challenge the traditional notion of disease causation, thus improving the survival chances of child.

Education further fosters upward social mobility, behaviour and identification with the western world and its values such as: a clean house, neatness and personal hygiene which generate beneficial health resulting in better nutrition and care and lower mortality. While at school, the mother most probably must have come to learn of most attributes conducive for child survival, such as cleanliness, the need to attend post and ante natal clinics and more aware of basic hygienic measures - all favourable to child survival.

In the primary level variable category however, the observed

variations could be that women with primary level of education are normally casual workers who are more often exposed to the risk of HIV infection both by their nature of work and by their husbands (often of higher education and migrants). Besides these women often give birth to higher number of children, are overworked thus giving birth to weak children whose probability of dying are high.

Education both facilitates participation in the modern sector and protects women and children from the gruelling agricultural work. If a girl is sent to school, she spends less time labouring in fields, may receive special treatment, thus educated girl may grow up to be better nourished and more likely to produce an infant of an adequate birth weight. Rejecting taboos, the educated women may be more inclined to eat high protein foods - such as chicken and eggs - which are often prohibited during pregnancy in traditional societies.

Often the case, the level of education determines the type of ones occupation: The mothers occupational status is believed to be indicative of the time spent in child rearing and domestic activities. Thus maternal participation in the labour market is expected to raise child mortality. A desegregation of the proportion of children dead by Occupation of the mother categorized into professional, semi-professional and all agriculture revealed marked variation as revealed in the table below (5.1.1d):

(d)
~~5.3.1d:~~ Child mortality differentials by occupation of the mother.

When examined by occupation of the mother, the expected pattern of child mortality was witnessed only to the mortality experienced by children of women in agricultural orientate activities, where the high HIV cluster witnessed a high child mortality followed by the Mid and lastly the low HIV cluster; falling by about two mortality levels by each level in the HIV cluster. However this observed child mortality in each cluster were in operation relatively around the same time - some five years prior to the survey, ie towards the end of 1984.

Generally children of women in professional occupation experienced the lowest child mortality (q5 of 0.053), followed by those of women in sales/services occupation (q5 of 0.135). While those children of women in agricultural oriented occupation experienced the highest child mortality (q5 of 0.181). This differential is further supported by mortality levels in the coale and Demeny regional life tables north model. The differentials is such that mortality of children of professional women at mortality levels 20.4 had about four mortality levels advantage over those of sales and services; who themselves had about two mortality levels advantage over children of women in agricultural activities.

The mortality situation in the mid HIV cluster mirrored that of the high HIV cluster except that there was no differentials between children of women in sales/services occupation and those

in agricultural occupation (ie both had about 15.5 mortality levels). However the mortality levels associated with children of women in sales and services occupation was in operation some two months prior to that of children of women in agricultural occupation.

The mortality situation prevailing in the low HIV cluster mirrored that witnessed in the high HIV cluster. In this cluster (Low), those children born to women in professional occupation had the lowest child mortality (~~q5-of-0.047~~), followed by those whose mothers were in sales and services occupation, while the children of women in agricultural occupation had the highest child mortality. This is further supported by the mortality levels associated with each estimate.

The observed mortality differentials is such that mortality of children born to women in professional occupation at mortality level 21.2 had about 3 mortality level advantage over those of women in sales and services and about four mortality levels advantage over those in agricultural activities. These rates were however below those observed in the sentinel. ^{10%} *24% + 10%*

The table also reveal that in the professional variable category, the mid HIV cluster had the highest child mortality, (mortality level 19.9). This was about 0.5 mortality level less than the high HIV cluster and about two mortality levels less than the low HIV cluster which had the lowest mortality (mortality levels 21.2). This against the sentinel mortality level of 20.7. For sales and services variable category, the mid

HIV cluster had the highest mortality (mortality level 15.5) which was about 0.3 mortality levels less than the next highest cluster - high HIV cluster. The low HIV cluster had about 3 mortality levels more than both that of mid and the high HIV cluster (ie low HIV cluster had the lowest mortality, this against the sentinel average of 15.4). For agricultural variable category, high HIV cluster had the highest mortality followed by mid while low HIV cluster had the lowest. This translates to low HIV cluster having about 2 mortality levels advantage over mid and four mortality level advantage over high HIV cluster. The sentinel cluster had mortality level 16.1

The probable reason for this is that those in professional occupation are better paid and therefore can afford to give their children favourable services and materials beneficial to survival of the child, for instance, better housing, food, health, and additional help to take care of their children while those in agricultural occupation often have low returns and therefore have the opposite of the above. Further, most agricultural activities take place in rural areas and these areas have poor child survival factors.

The probable reason for this is that those in professional occupation have better pay which they can channel to the general upkeep of their families, often are employed in better working environments, have access to better health care facilities and are often well educated thus are literate on child survival techniques.

5.1.2: Child mortality differentials by environmental factors and HIV regime clusters .

Thus far, the focus has been on the child mortality differentials by Socio-economic factors, by the prevailing HIV regime clusters. However these factors do not work in isolation, they interact with both the physical and the environmental factors which further perpetuates mortality and bring about mortality differentials in the human population.

Table 5.2 gives child mortality differentials by environmental factors and the prevailing HIV clusters. *H/H*

Household environmental factors proxied by amenities such as: Availability of clean drinking water, floor material type and sewage disposal, have been documented to be having an Influence on mortality especially that of Infant and Child.

[Handwritten signature]

Table

Marketability differences by household
on various materials.
The HHS estimates
are provided by:
Sewage & gas material type.

Table 5.2: Child mortality differentials by environmental factors. HIV clusters.

ENVIRONMENTAL FACTORS	HIV PREVALENCE REGIMES			
	HIGH	MID	LOW	SENTINEL TOTAL
BY WATER SOURCE				
PIPED	0.085 (18.7) 6.2*	0.092 (18.1) 6.0*	0.060 (20.3) 6.0*	0.053 (20.8) 6.0*
OTHERS	0.082 (18.9) 5.9*	0.066 (19.9) 6.4*	0.103 (17.6) 6.0*	0.095 (19.1) 6.0*
BY PLACE OF RESIDENCE				
RURAL	0.194 (12.9) 6.6*	0.131 (16.1) 6.3*	0.097 (17.9) 6.1*	0.254 (10.3) 6.4*
URBAN	0.134 (15.9) 6.6*	0.114 (17.0) 6.0*	0.090 (18.4) 6.3*	0.117 (17.8) 6.3*
BY SEWAGE DISPOSAL				
MAIN SEWER	0.071 (19.6) 5.7*	0.094 (18.3) 5.0*	0.058 (20.5) 4.8*	0.073 (19.5) 5.6*
PIT LATRINE	0.157 (14.7) 6.4*	0.136 (15.8) 6.3*	0.095 (18.1) 6.0*	0.124 (16.4) 6.3*
BUSH	0.211 (12.2) 6.4*	0.230 (11.4) 6.4*	0.145 (15.3) 6.1*	0.172 (13.9) 6.2*
OTHERS	0.127 (16.3) 5.1*	0.091 (18.3) 6.0*	0.085 (17.1) 5.0*	0.101 (17.1) 6.1*
BY FLOOR MATERIAL TYPE				
CEMENT	0.077 (19.2) 6.3*	0.081 (18.9) 6.0*	0.059 (20.4) 5.7*	0.074 (19.4) 6.1*
EARTH	0.093 (18.2) 6.5*	0.082 (18.9) 6.6*	0.093 (18.2) 6.1*	0.092 (18.2) 6.3*
WOOD	0.064 (20.1) 5.5*	0.086 (18.6) 6.6*	0.072 (19.5) 6.3*	0.069 (20.3) 5.9*
TILES	0.046 (21.3) 4.9*	0.060 (20.3) 5.1*	0.069 (19.7) 6.3*	0.075 (19.3) 7.1*
OTHER	0.094 (15.1) 7.0*	0.096 (18.0) 7.0*	0.101 (17.7) 6.6*	0.093 (18.2) 7.1*

Source: The present study.

Key:

- i. Figures in parentheses are mortality levels, North model of Coale and Demeny regional life tables.
- ii. Starred figures are Time references to which each of the mortality estimates refers.

Mortality of Infants born in households with piped water for instance has been documented to be normally lower than that of Infants born in households where water was obtained from other sources, such as Wells, Rivers or Canals (Katiyer, 1981:545; Da Vanzo and Co. 1983:396). Consequently, to get a clearer picture by the prevailing HIV regimes, child mortality was looked at vis a vis the different HIV clusters, by sources of drinking water, (5.1.2a).

(a)
5.1.2a: **Child mortality differentials by water source.**

Table 5.2 also displays child mortality differentials by water source available in the household by HIV clusters. Water source available in the household was categorized into two variable category piped water and other type of water source other than piped. The results of the table reveal that generally those households with other sources of water had the lowest child mortality in the high and mid HIV cluster, while in the low HIV cluster, the opposite is the case.

There was no clear trend emerging from the table. Those children born in the households using piped water, had the mid HIV cluster having the highest child mortality (q5 of 0.092), followed by the high HIV cluster (q5 of 0.085) and lastly low HIV cluster had the lowest child mortality (q5 of 0.060). These rates were further supported by the mortality levels in the Coale and Demeny regional life tables - North model in operation some six years prior to the survey.

On the other hand in the households using other sources of water other than piped, the low HIV cluster witnessed the highest child mortality (q5 of 0.103, North mortality levels 17.6), followed by the high HIV cluster (q5 of 0.082 North mortality level 18.9) while the mid HIV cluster experienced the lowest child mortality (q5 of 0.066, North mortality levels 19.9). These rates were in operation between late 1983 to mid 1984.

Differentials in child mortality, when looked by source of water available in the family revealed that for the high HIV

cluster, those children born in households using water other than piped had a slight advantage over those using piped water (0.082 versus 0.085) respectively. This is further evidenced by the mortality levels in the Coale and Demeny regional life tables, north model; depicting a difference of 0.2 in favour of other sources of water), moreover, the former refers to mortality in operation in a more recent period, about 5 years prior to the survey.

In the mid and the low HIV cluster however, those children born in households with piped water had lower probability of a child dying by age 5 than those using other sources of water other than piped, with the differentials being more marked in the low HIV cluster (roughly about three mortality levels in favour of piped water) see: Table 5.2. These rates were however above those experienced in the sentinel cluster.

A closer examination of the same table by the same variable and variable categories reveal that for those households using piped water, the mid HIV cluster experienced high child mortality followed by the high HIV cluster, while low HIV cluster experienced the lowest child mortality. However their^{re} was a small differential (of about mortality level 0.6) between the high and the mid HIV cluster and about 2 mortality levels between the low HIV cluster and the other clusters. These rates were however below those experienced in the sentinel cluster.

For other sources of water other than piped, the low HIV cluster experienced the highest child mortality, this was about

one mortality and two mortality levels above that of the high and mid HIV cluster respectively. This against the sentinel mortality level of 19.1.

The probable reason for this is that, the low HIV cluster is ^{regime} mainly in rural agricultural areas most of which have agrarian factories (like sugar mills, Tea factories) which pollute the streams and rivers that most villagers use for domestic purposes. Further those areas which do not have piped water take precautionary measures with the water they intend to consume more often than those using piped water which is often not treated. Thus frequent attacks of typhoid in a malarial infected areas could lead to more child death due to wrong diagnosis of typhoid than would have been otherwise. .

In trying to find out whether the prevailing HIV regime cluster differ significantly by type of housing, and whether this has an effect on survival status of Children, attempt was made to establish the proportion of children dead by floor material type for the various HIV regimes. (5.1.2b).

5.1.2b:— Child mortality differential by floor material type.

Floor material type in this study was categorized into five variable categories: Cement, Earth, wood, Tiles and Others. The results of the study found that those children in households where floor was made of cement and tiles generally had lower child mortality than those in households whose floor was made of

earth and other type of floor material other than cement, earth wood or tiles. The results also reveal that generally the mid HIV cluster experienced high child mortality, while the low HIV cluster experienced low child mortality.

In the high HIV regime cluster therefore, children in floor tiled households experienced the lowest child mortality (q5 of 0.046), followed by those made of wood (q5 of 0.064), while those households whose floor was made of other type of floor material type experienced the highest child mortality. This is further evidenced by the mortality levels and the time references associated with each estimate.

In the mid HIV cluster however, those households with floor made of tiles had the lowest child mortality (q5 of 0.060 corresponding to mortality levels 20.3 in operation some five years prior to the survey, followed by those made of cement (q5 of 0.081 corresponding to mortality level 18.9 in operation some six years prior to the survey). While those households whose floor was made of wood and other type of floor material experienced the highest child mortality (q5 of 0.099 and 0.086 corresponding to mortality levels 18.0 and 18.6 in operation some seven and six years prior to the survey) ^{res p. 4-5} for other type of floor material type and wood respectively. These rates compared favourably to the sentinel averages.

In the low HIV cluster, the same mortality differentials scenario is witnessed. However in this cluster, those households with cemented floors experienced lowest child mortality followed

by those in tiled floors (q5 of 0.059 and 0.069 corresponding to mortality levels 20.4 and 19.7 in operation some five and seven years prior to the survey respectively) for cemented and tiled floors respectively. Those households whose floors were made of other type of floor material and whose floors were made of earth experienced the highest child mortality (q5 of 0.101 corresponding to mortality levels 17.8 in operation some six years prior to the survey for the other type of floor material type and q5 of 0.093 corresponding to mortality levels 18.2 in operation some six years prior to the survey for earth. However these rates compared favourably to the sentinel rates.

A closer look at the same table reveal that for households with cemented floors, the mid HIV cluster at child mortality level 18.9 experienced about one child mortality levels less than high HIV cluster and about two child mortality levels less than the low HIV cluster; implying that the mid HIV cluster experienced high child mortality while low HIV cluster experienced the lowest child mortality.

The same differentials is witnessed for households with earth made floors, however in this variable category, high and low HIV clusters had the same mortality levels (18.2), this was about 0.6 mortality levels more than the mid HIV cluster, ie the mid HIV cluster experienced the highest child mortality.

For wood and tiles variable category, the high HIV cluster experienced high child mortality (mortality levels 20.1 and 21.3 for wood and tiles respectively), this declined by one mortality

level by each cluster. However for other variable category, the low HIV cluster experienced the highest child mortality (mortality levels 17.8, followed by mid HIV cluster (about one mortality level more than low HIV cluster) while high HIV cluster experienced the lowest child mortality (mortality levels 15.1).

As already mentioned , examining risk of death for child by floor material type may camouflage other vital factors, most important of which is the general sanitation in the household and the immediate environment. For instance, access to toilet facilities has been documented to be one of the factors that contribute to the decline in Infant and Child mortality rate. This has been confirmed in the Philippines (Martin and Others, 1993) and Malaysia (Peterson et al, 1986, Da Vanzo and Habacht, 1986), In Kenya K'Oyugi 1992 Ikamari, 1996 and Muganzi 1990).

Consequently variations in toilet facility was considered in this study as the basis for mortality differentials. A look at child mortality as per the prevailing HIV regimes by sewage disposals available in the household revealed the following (~~5.1.2c~~).

^(c)
5.1.2c: Child mortality differentials by sewage disposal.

Generally, Children born in households with main sewer as form of sewage disposal had the lowest probability of dying by age five in all the stratus of HIV regime (0.071, 0.094 and 0.058 in high, mid and low HIV cluster respectively); while those

children born in households disposing of their sewer in the bush had the highest child probability of dying by the age of five, (ie 0.211, 0.230 and 0.145 in the high mid and Low HIV/AIDS regime respectively). These results are further supported by the mortality levels in the Coale-Demeny regional life tables - North model and the time references associated by each (see: Table 5.2).

However no clear trend emerged when the variable was dissected to the 4 types of sewage disposal. Whereas Pit latrine and other sources of sewage disposal displayed the expected mortality pattern, ie of a declining mortality from high HIV cluster to mid HIV cluster to low HIV cluster. Those defecting in the Bush and those using the main sewer however had the mid HIV cluster experiencing high child mortality of about one mortality level less than high HIV cluster and two to four - mortality levels less than the low HIV clusters for main sewer and Bush respectively.

In the high HIV cluster therefore, those children born in households where people defect in the bush experienced the highest child mortality (q5 of 0.211), followed by those born in households using pit latrine (q5 of 0.157). Those children in households using main sewer had the lowest child mortality.

These differentials are further evidenced by the mortality levels associated with each estimate. Main sewer with the lowest child mortality at mortality level 19.6 was about 3 mortality levels more than the next variable category with the lowest child

mortality - other type of sewage disposal, and about five and seven mortality levels more than pit latrine and bush respectively. These rates were further in operation some five years (for main sewer and other) and six years (for pit) prior to the survey.

The mid HIV cluster displayed similar mortality situation to that experienced in the high HIV cluster. In this cluster, (mid), those households with main sewer experienced lowest child mortality, followed by other sources of sewage disposal then those in households using pit latrine, while those children in households disposing their sewer in the bush had the highest child mortality. This translates into the probability of a child dying of 0.094, 0.091, 0.136 and 0.230 respectively.

This observed mortality differential means that children in households with main sewer and other means of sewage disposal at mortality level 18.3 was about 3 and 7 mortality levels more than pit latrine and bush respectively. Further apart from the mortality rates for main sewer which was in operation some five years prior to the survey, the rest of the variable category mortality rates were in operation some six years prior to the survey.

The table also reveal that in the low HIV cluster, the same scenario is witnessed. The children born in households using main sewer experienced lowest child mortality, followed by those in households using other means of sewage disposal, while those in households using Bush to dispose their sewer had the highest

child mortality. This translates to children born in households using main sewer experiencing two mortality levels more than those in households using pit latrine and other means of sewage disposal and about five mortality levels more than those in households disposing of their sewage in the bush.

A closer look at the same table by the same variable reveal that for those households using main sewer, those in mid HIV cluster experienced the highest child mortality (q5 of 0.094 corresponding to mortality level 20.5 in operation some early parts of 1984) while low HIV cluster had the lowest child mortality (q5 of 0.58 corresponding to mortality level 20.5 in Coale and Demeny regional life table North model, in operation some four years prior to the survey). The same situation is mirrored for those households defecating in the bush. However for those households using pit latrine and other sources of sewage disposal, high HIV cluster experienced high child mortality followed by mid and lastly low HIV cluster.

The probable reason for the observed mortality differential is that main sewer is more hygienic than other means of sewage disposal and therefore reducing the chances of disease transmission such as cholera or bilharzia which induce mortality in younger ^{ages} children. Those who defecate in the bush often the waste is washed to the rivers that is more often used for domestic purposes. This spreads other water born diseases which affects the children bringing the observed high risk of death, to those born in households where members defecate in the Bush.

Kenya like many developing Countries is faced with the problem of unequal distribution of the benefits of progress, the case is such that Urban areas have been viewed to be endowed with better survival chances and therefore less risk exposures to death than the rural areas. Infant and child mortality has been found to vary according to the place of residence. Rural residence has been associated with higher risk of infant and child mortality than residence in urban areas (Gaisie, 1980:24; Caldwell and Ruzicka, 1985:20; Hull and Gabbaju, 1986:116). Rural - Urban mortality differentials have been seen as reflecting the differences in social and economic conditions between rural and urban areas (Behm and Valiin , 1980:29); and the prevailing HIV/AIDS. A closer examination of the proportion of children dead by rural-urban, revealed the following (5.1.2d).

(d)
5.1-2d: Child mortality differentials by place of residence.

The place of residence of the Childs family is perhaps the variable that best displays the expected mortality pattern and differential. The probability of a child dying by age five declined by HIV cluster from high to mid to low HIV cluster for both rural and urban place of residence. This is further evidenced by the mortality levels and the reference periods associated with them.

Generally those children residing in rural areas experienced high child mortality compared to those in urban areas.

In the high HIV cluster therefore, rural areas experienced the highest child mortality (q5 of 0.194), against the urban mortality rate of 0.134. This translates to the urban areas gaining about 3 mortality levels with respect to rural areas. However, these rates were in operation around the same time, some six years prior to the survey.

The same differential is mirrored for both the mid and the low HIV cluster. However in these clusters, the differential was about one mortality level in favour of urban areas. These observed mortality rates were in operation some six years prior to the survey.

A closer look at the same table by variable category and HIV cluster reveal that for rural areas variable category, the high HIV cluster at mortality level 12.9 was about 4 mortality levels less than mid HIV cluster and about seven mortality levels less than low HIV cluster, implying that high HIV cluster experienced high child mortality followed by mid HIV cluster, while low HIV cluster experienced low child mortality.

The same scenario is witnessed for urban areas variable category. However the differential between the high and mid HIV cluster was about one mortality level, while that between high and low HIV cluster was about two mortality levels. However the observed mortality levels were in operation some six years prior to the survey.

The probable reason for this could be due to better social amenities found in Urban areas which are conducive for child



survival. The results supports Mungazis' (1984:41) and Eubank and associates 1986:52 that rural Infant mortality was slightly higher than Urban Infant mortality, however rural and Urban survival rates were about equal (See table 5.2).

Urban areas usually have the largest share of the countries well educated, high income population and the best medical and health facilities, thus urban areas is expected to have lower infant and child mortality.

5.2: ADULT MORTALITY DIFFERENTIALS.

This section covers the adult mortality differentials using the computed rates (orphanhood approach using Brass method) appearing in appendix B. The parameters used to estimate and discuss adult mortality differentials was the probability of dying by age x (q_x). Also displayed are the mortality levels in the Coale and Demeny regional life tables north model and the time references each estimates refer. Table 5.3 and 5.4 have the adult mortality differentials by the characteristics of the child.

Table 5.3: Distribution of probability of dying for adult males using Brass method by HIV clusters.

AGE	HIV REGIME CLUSTERS			
	HIGH	MID	LOW	TOTAL SENTINEL
MALE RESPONDENTS				
55	0.180 (16.6) 9.5*	0.183 (16.4) 9.5*	0.148 (18.6) 9.1*	0.150 (18.5) 9.1*
60	0.250 (16.5) 11.4*	0.267 (15.7) 11.5*	0.221 (18.0) 10.8*	0.216 (18.2) 10.8*
65	0.366 (15.4) 13.3*	0.468 (13.5) 15.2*	0.324 (17.2) 12.5*	0.312 (17.7) 12.3*
70	0.508 (14.5) 15.1*	0.532 (12.9) 17.0*	0.451 (16.8) 14.0*	0.439 (17.2) 13.8*
FEMALE RESPONDENTS				
55	0.195 (15.7) 9.6*	0.188 (16.1) 9.5*	0.150 (18.5) 9.1*	0.157 (18.0) 9.1*
60	0.280 (15.0) 11.6*	0.275 (15.3) 11.5*	0.225 (17.8) 10.9*	0.239 (17.1) 10.8*
65	0.408 (13.5) 13.6*	0.396 (14.1) 13.4*	0.331 (16.9) 12.5*	0.353 (15.0) 12.5*
70	0.540 (13.1) 15.6*	0.541 (13.1) 15.3*	0.458 (16.5) 14.1*	0.489 (15.3) 14.0*

Source: The present study.

Note:

- (i) Figures in parentheses are mortality levels in the Coale and Demeny regional life tables North model.
- (ii) Starred figures are time references to which each estimate refer.

5.2.1: Adult male mortality differential by characteristic of child

Table 5.3 above displays male adult mortality derived by using orphanhood approach by the characteristic of the child. The characteristic of the child was categorized into males and females.

The results of the table reveal a marked adult male mortality differentials by the different HIV regime clusters, with the low HIV regime cluster experiencing low adult male mortality while mid HIV cluster experiencing high adult male mortality when the information was provided by male respondents.

→ However when the information was provided by female respondents,

the high HIV regime cluster experienced high adult male mortality while the low HIV cluster experienced low adult mortality.

In the high HIV cluster therefore, female respondents reported high adult male mortality of about one mortality levels than male respondents. These rates were however higher than those observed in the total sentinel cluster but were in operation around the same time.

The same mortality situation is witnessed in the mid HIV cluster, except that in this cluster (mid), the observed male mortality differentials is slight, (ie below one mortality level). These observed rates were in operation some nine to seventeen years prior to the survey.

The low HIV cluster, mirrors the mortality situation witnessed in the high HIV cluster. In this cluster (low), female respondents reported high adult male mortality of about mortality levels 0.1 to 0.3 mortality levels less than male respondents (ie they experienced higher adult male mortality than their male counterparts. These rates were generally below the rates experienced in the total sentinel regime cluster.

female.
5.2.2: Adult ^fmortality differentials by characteristics of child.

Thus far we have been examining adult male mortality differentials by the characteristics of the child. However it is not only males who are subjected to the risk of death, but both males and females too. A look at the adult female mortality

differentials by characteristics of child revealed the following (Table 5.4).

Table 5.4: Distribution of adult female probability of dying by characteristics of child and HIV regime clusters.

AGE	HIV CLUSTERS			
	HIGH	MID	LOW	SENTINEL
MALE MORTALITY <i>respondents</i>				
45	0.055 (20.5) 8.9*	0.051 (21.9) 8.9*	0.050 (20.9) 8.9*	0.052 (20.7) 8.9*
50	0.080 (20.2) 10.7*	0.091 (20.1) 10.7*	0.083 (20.0) 10.7*	0.091 (20.1) 10.7*
55	0.122 (19.5) 12.3*	0.127 (19.3) 12.3*	0.131 (19.0) 12.4*	0.127 (19.3) 12.3*
60	0.188 (18.5) 13.7*	0.191 (18.3) 13.7*	0.199 (17.9) 13.8*	0.193 (18.2) 13.7*
65	0.280 (17.2) 14.9*	0.285 (17.3) 14.8*	0.299 (16.8) 15.0*	0.292 (17.0) 14.9*
70	0.405 (16.7) 16.0*	0.398 (16.9) 16.0*	0.404 (16.4) 16.2*	0.417 (16.6) 16.0*
FEMALE MORTALITY <i>respondents</i>				
45	0.061 (19.9) 9.0*	0.056 (21.4) 8.9*	0.055 (20.5) 8.9*	0.057 (20.2) 8.9*
50	0.094 (18.2) 10.8*	0.089 (19.5) 10.8*	0.091 (19.4) 10.8*	0.092 (19.3) 10.8*
55	0.144 (18.3) 12.4*	0.137 (18.7) 12.4*	0.157 (18.1) 12.5*	0.143 (18.3) 12.4*
60	0.213 (17.3) 13.9*	0.203 (17.7) 13.8*	0.218 (17.0) 14.0*	0.212 (17.3) 13.9*
65	0.225 (15.7) 15.4*	0.310 (16.3) 15.2*	0.336 (15.2) 15.4*	0.326 (15.6) 15.4*
70	0.451 (15.0) 16.8*	0.433 (15.7) 16.4*	0.447 (15.1) 16.7*	0.445 (15.2) 16.7*

Source: The present study.

Note:

- (i) Figures in parentheses are mortality levels in the Coale and Demeny regional life tables North model.
- (ii) Starred figures are time references to which each estimate refer.

Generally children in mid HIV regime cluster experienced high female adult mortality closely followed by high hiv regime cluster, while low HIV cluster experienced low female adult mortality.

For the high HIV cluster therefore, there was high female ^{adult} mortality by information provided for by female respondents than those done by male respondents by about one to two mortality levels. These rates were however in operation around the same

time and compared favourably with those witnessed in the total sentinel regime cluster.

In the mid HIV cluster, the same mortality situation is witnessed, except the mortality differentials was slightly narrower (about mortality level 0.5 to one mortality level), in favour of male respondents ie there was lower observed mortality differentials when examined by information provided for by male children. Except for female mortality at ages 50-60, mid HIV cluster experienced lower mortality than high HIV cluster and those experienced in the low HIV regime cluster. These rates compared favourably to the total HIV sentinel rates.

Low HIV cluster mortality situation mirrored that of mid HIV regime cluster, these rates were however in operation around the same time and compared favourably with the sentinel cluster.

From table 5.3 and 5.4, it is evident that adult female in all the three HIV clusters experienced low mortality than males (about two mortality levels in favour of females) with the probability of females in mid HIV survival being higher than in the other HIV clusters.

However, unlike male counterparts, female respondents had more paternal deaths in the high HIV clusters followed by mid HIV clusters and lastly the low HIV clusters in line with the expectation of this study. This differential is more pronounced at ages 55 and 65. The observed differential is such that the mid HIV cluster is about one mortality level more than the high

HIV cluster and two mortality levels lower than the low HIV clusters. However the observed mortality rates in the low HIV cluster after age 60 was about one year earlier than those observed in the high and the mid HIV clusters.

However females in the mid HIV cluster experienced a slightly higher mortality rate closely followed by the high HIV cluster while the low HIV cluster come last. These observed mortality variations were further supported by the mortality levels and time references associated with each estimate. However these observed mortality differentials were very minimal (less than one mortality levels).

like Examined by the information provided by male respondents, females(mothers) had better survival rates than males in all the regimes of HIV clusters, in all the ages.

Examined by variable category however, low HIV cluster experienced high adult mortality which was about two mortality levels above the high HIV cluster and three mortality levels above that of the low HIV cluster (with exception of n of 20). These rates were in operation some nine to fifteen years prior to the survey. However, the rates in the low HIV cluster ignoring that at ages 55, were in operation about one year earlier than the mid and the high HIV clusters. The experienced mortality rates were favourably compared to the sentinel rates.

However female mortality as reported by male children (Table 5.3), depicted the expected pattern of mortality differential of

a declining mortality from high to mid to low HIV clusters but only up to age 60. From age 65 onwards, the mid HIV cluster experienced the highest mortality followed by the high HIV cluster while the low HIV cluster experienced the lowest mortality. These rates are further supported by the mortality levels associated with them and the time references to which each estimate refers.

A look at the adult mortality differentials by female respondents revealed that adult female mortality in all the three HIV clusters experienced low mortality than male (about two mortality levels in favour of females) with the probability of females in mid HIV survival being higher than in the other HIV clusters.

However, unlike male counterparts, female respondents had more paternal deaths in the high HIV clusters followed by mid HIV clusters and lastly the low HIV clusters in line with the expectation of this study. This differential is more pronounced at ages 55 and 65. The observed differential is such that the mid HIV cluster is about one mortality level more than the high HIV cluster and two mortality levels lower than the low HIV clusters. However the observed mortality rates in the low HIV cluster after age 60 was about one year earlier than those observed in the high and the mid HIV clusters.

However females in the mid HIV cluster experienced a slightly higher mortality rate closely followed by the high HIV cluster while the low HIV cluster come last. These observed

mortality variations were further supported by the mortality levels and time references associated with each estimate. However these observed mortality differentials were very minimal (less than one mortality levels).

5.3: THE IMPACT OF HIV/AIDS ON ^{CHILD} MORTALITY.

At the beginning of the AIDS epidemic some observers predicted that this disease would increase mortality so much that it would cause population decline in many countries. Others have said mortality impact of AIDS would not be large. The present study reviews the evidence available on the issue using the methodologies developed by the Future Group International in collaboration with the Research triangle Institute (RTI) and centre for development and population activities (CEDPA) (see Stover 1994:417). Several indicators of mortality have been used to assess the impact of HIV/AIDS on mortality. The present study used under five mortality and life expectancy at birth.

5.3.1: THE IMPACT OF HIV/AIDS ON CHILD MORTALITY BY SOCIO-ECONOMIC FACTORS.

Using the methodologies described ^{in chapter 3} ~~above~~, the impact of HIV/AIDS on child mortality by socio economic factors are described in the next sub sections.

Table 5.5: Estimation of impact of HIV/AIDS on underfive mortality by socio - economic factors.

SOCIO ECONOMIC FACTORS	HIV/AIDS REGIME CLUSTERS			
	HIGH	MID	LOW	TOTAL SENTINEL
SEX OF CHILD				
Males	138 (162) 1.17*	132 (144) 1.09*	101 (104) 1.03*	121 (130) 1.07*
Females	126 (151) 1.20*	124 (137) 1.10*	92 (95) 1.03*	112 (121) 1.08*
EDUCATION LEVEL OF MOTHER				
No education	139 (163) 1.17*	149 (169) 1.13*	135 (138) 1.02*	139 (148) 1.06*
Primary education	86 (112) 1.30*	79 (92) 1.16*	64 (67) 1.04*	86 (73) 1.16*
secondary plus	47 (74) 1.57*	53 (66) 1.24*	53 (56) 1.06*	47 (61) 1.20*
OCCUPATION OF MOTHER				
All Agriculture	53 (80) 1.51*	66 (79) 1.20*	47 (50) 1.06*	55 (65) 1.18*
Sales and Services	135 (159) 1.18*	142 (154) 1.08*	96 (99) 1.03*	135 (153) 1.06*
Professional	181 (204) 1.13*	141 (153) 1.09*	105 (108) 1.03*	181 (139) 1.07*
MARITAL STATUS OF MOTHER				
Single	77 (103) 1.34*	77 (103) 1.34*	77 (103) 1.34*	77 (103) 1.34*
Monogamous	80 (106) 1.33*	80 (106) 1.33*	80 (106) 1.33*	80 (106) 1.33*
Polygamous	121 (146) 1.21*	121 (146) 1.21*	121 (146) 1.21*	121 (146) 1.21*
Widowed	150 (174) 1.16*	150 (174) 1.16*	150 (174) 1.16*	150 (174) 1.16*
Divorced & Separated	129 (154) 1.19*	129 (154) 1.19*	129 (154) 1.19*	129 (154) 1.19*

Source: The present study.

Note:

- Figures in parentheses are child mortality with AIDS.
- Stared figures are the ratio of child mortality with AIDS to that of child mortality.

5.3.1a: The impact of HIV/AIDS on child mortality by sex of child.

The table reveal that female under ^{Child}five mortality was the hardest impacted by HIV/AIDS in all the three clusters of HIV regime. The impact of HIV/AIDS on child mortality was more pronounced in the high HIV regime cluster (17 and 20 percent for males and females under five mortality respectively). The mid HIV regime cluster came a close second with 9 and 10 percent ^{for males & females} respectively, while the low HIV regime cluster was the least

for both males & females
impacted at 3 percent. This against the total sentinel cluster total of 7 and 8 percent for males and females respectively.

5.3.1d: The impact of HIV/AIDS on child mortality by marital status of the mother.

Table 5.5 also reveals the impact of HIV/AIDS on child mortality by marital status of the mother. The results reveal that child mortality for single women was the hardest hit by ~~HIV/AIDS~~. Further the largest percentage of the impact of HIV/AIDS on mortality was large in the high HIV regime cluster ^{& low in the} while the low HIV regime ~~cluster was the lowest hit~~.

In the high HIV regime cluster therefore, HIV/AIDS increased child mortality of single women by about 34 percent, monogamous women by 33 percent, while the widowed, and the divorced and separated by 16 and 19 percent respectively.

The same scenario was witnessed in the mid HIV cluster, however in this cluster, child mortality of single and monogamous women increased by 16 percent due to ~~HIV/AIDS~~, while that for widowed and polygamous women increased by 8 percent.

In the low HIV cluster, HIV/AIDS increased child mortality for single women by five percent, monogamous women by three percent, and lastly those of polygamous and widowed women by two percent.

The observed differentials in the total HIV regime sentinel cluster however mirrored those of mid HIV regime cluster. Further, the ^{mid} mod HIV regime cluster were below those of the high ^{high}

HIV regime cluster but above those of the low HIV regime cluster.

The probable reason for the low HIV/AIDS impact on child mortality ^{of} ~~by~~ widowed and divorced ^{women} children is that majority of ~~these children~~ them die due to other causes disadvantageous to promotion of life (see: previous section). The observed high percentage increment of child mortality of single women was mainly because these women often have multiple partners, lead a wild life which exposes them to the risk of HIV/AIDS infections. besides the unmarried are more often in the high risk age group.

5.3.1c: Impact of HIV/AIDS on child mortality by education level of the mother.

The impact of HIV/AIDS on child mortality by education level of mother revealed that in the high HIV cluster the impact of HIV/AIDS was more for children of women with at least secondary level of education (57 percent), followed by Primary education (30 percent), then no education (17 percent). The largest differential in the impact of HIV/AIDS on under five mortality was witnessed between those of primary and secondary education.

In the mid and the low HIV cluster, the same scenario is also witnessed but the differential witnessed in the low HIV cluster between the variable categories was minimal.

Generally the impact of HIV/AIDS on child mortality by education of the mother was higher in the high HIV regime cluster and lower in the low HIV regime cluster for all variable categories.

The impact of HIV/AIDS on child mortality was high for women with secondary plus level of education because its these women who are more often in better paying occupation and were in single state for a longer duration thus were exposed to the risk of HIV infection for a longer time. Besides these womens children are well taken care of such that they are more unlikely to die from other causes than the children of women with no education for instance.

5.3.1d: Impact of HIV/AIDS on child mortality by occupation of the mother.

Table 5.5 reveal that the under five mortality for professional women was the hardest hit by HIV/AIDS, while children of women in sales and services occupation and those in all agricultural occupation was the least impacted.

The table further reveal that in all variable categories, the impact was more felt in the high HIV regime cluster than in the mid HIV regime cluster and the mid HIV cluster than the low HIV regime cluster.

In the high HIV cluster therefore, the impact of HIV/AIDS was more felt on children of women in professional occupation, followed by those of women in sales and services and lastly those women in all agricultural occupation.

In the mid HIV cluster, those children of women in professional occupation experienced the highest impact of HIV/AIDS in the mortality of their children under the age of

five, however in this cluster it was followed by those of women in all agricultural occupation and in sales and services (ie an increment of about 3 percent).

5.3.2: IMPACT OF HIV/AIDS ON CHILD MORTALITY BY ENVIRONMENTAL FACTORS.

It has often been thought that HIV/AIDS is an opportunistic disease that more often depend on the infection of bacterial, viral and other infections on the host human body to develop into AIDs and cause mortality. As it has often been documented that good sanitational observation of the individual, the household and the immediate environment more often than not keep that infection away. Source of clean drinking water, means of sewage disposal, the quality of housing and place of residence, have often been singled out as the key factor in the upkeep of a good sanitation both at the individual, the household and the immediate environment. Table 5.6 gives the estimated impact of HIV/AIDS on child mortality by these variables.

Table 5.6: Estimation of the impact of HIV/AIDS on child mortality by household environmental factors.

HOUSEHOLD ENVIRONMENTAL FACTORS	HIV REGIME CLUSTERS			
	HIGH	MID	LOW	TOTAL SENTINEL
WATER SOURCE				
PIPED	85 (111) 1.31*	92 (105) 1.14*	60 (63) 1.05*	53 (63) 1.19*
OTHERS	82 (108) 1.32*	66 (79) 1.20*	103 (106) 1.03*	95 (104) 1.09*
MEANS OF SEWAGE DISPOSAL				
MAIN SEWER	85 (111) 1.31*	94 (107) 1.14*	58 (61) 1.05*	73 (83) 1.17*
PIT LATRINE	82 (108) 1.32*	136 (148) 1.09*	95 (98) 1.03*	124 (127) 1.02*
BUSH	211 (233) 1.10*	230 (241) 1.05*	145 (148) 1.02*	172 (180) 1.05*
OTHERS	127 (152) 1.20*	91 (104) 1.14*	85 (88) 1.04*	101 (109) 1.08*
FLOOR MATERIAL TYPE				
CEMENT	77 (103) 1.34*	81 (94) 1.16*	59 (62) 1.56*	74 (84) 1.14*
EARTH	93 (119) 1.28*	82 (95) 1.16*	93 (96) 1.03*	92 (101) 1.11*
WOOD	64 (90) 1.41*	86 (99) 1.15*	72 (75) 1.04*	69 (79) 1.14*
TILES	46 (73) 1.59*	60 (73) 1.22*	69 (72) 1.33*	75 (84) 1.12*
OTHERS	94 (120) 1.28*	96 (109) 1.14*	101 (104) 1.03*	93 (102) 1.11*
RESIDENCE				
RURAL	194 (217) 1.19*	131 (143) 1.09*	97 (100) 1.03*	254 (262) 1.03*
URBAN	134 (158) 1.18*	114 (126) 1.11*	90 (93) 1.03*	117 (126) 1.08*

Source: The present study.

Note:

- Figures in parentheses are under five mortality rate with the impact of HIV/AIDS.
- Stared figures are the ratio.
- Plain figures are underfive mortality.

5.3.2a: Impact of HIV/AIDS on child mortality by source of water.

Examined by type of water source available in the household which in this study was categorized into piped water and others (ie other sources of water other than piped). The results reveal that the highest impact of AIDS on child mortality by source of water was highest to households using other sources of water other than piped for the high and the mid HIV clusters, while the opposite is the case for low HIV cluster and the total HIV regime

cluster.

In the high HIV cluster therefore, those in households using other sources of water other than piped had the highest percentage increase in child mortality attributed to AIDS (32 percent) while those of piped was increased by 31 percent.

In the mid HIV cluster, the differential is more pronounced, with HIV/AIDS increasing child mortality to those in households using piped water by 14 percent, while that for other sources of water by 20 percent.

The low HIV cluster experienced the lowest differential but the opposite of the first two HIV/AIDS regime clusters. In the low HIV/AIDS regime cluster, child mortality of piped water households was increased by five percent while those of children in households using other sources of water by three percent.

This differentials is such that high HIV regime cluster experienced the highest impact of HIV/AIDS on child mortality followed by mid HIV cluster, while the low HIV cluster experienced the least. The probable reason for this is that lack of access to piped water often spell lack of other amenities conducive for child survival, besides water plays a very important role in the general cleanliness of the household and therefore the risk of infection.

5.3.2b: Impact of HIV/AIDS on child mortality by sewage disposal.

The impact of HIV/AIDS on child mortality by sewage disposal available in the household was also examined (see: table 5.6). Sewage disposal available in the household was categorized into main sewer, pit latrine, bush, and others (ie other means of sewage disposal other than main sewer, pit latrine or bush).

From the table it is evident that HIV/AIDS will increase child mortality in all the variables categories of sewage disposal; with the highest impact on child mortality being witnessed to those in households using main sewer, while the lowest impact was to those in households where the members defecate in the bush. Overall high HIV cluster experienced the highest impact of HIV/AIDS on child mortality while the low HIV regime cluster experienced the lowest.

In the high HIV regime cluster therefore, HIV/AIDS increased child mortality by 37 percent for children in households using main sewer, 15 percent for those in household using pit latrine, while the increase of HIV/AIDS on child mortality was 10 and 20 percent for those households who defecate in the bush and other places.

In the mid HIV/AIDS cluster those households using main sewer and other means to dispose their sewer experienced the largest increment to child mortality due to HIV/AIDS (ie 14 percent), followed by households using pit latrine (p percent), while those households where members defecate in the bush, HIV/AIDS

increased child mortality by five percent.

In the low HIV cluster, the same situation is witnessed as that prevailing in the mid HIV cluster. In this cluster, child mortality of children born in households using main sewer and other means of sewage disposal had HIV/AIDS increasing child mortality by about five and four percent, respectively. The impact of HIV/AIDS on child mortality ~~is~~ in this HIV regime cluster is below those observed in the total sentinel regime cluster.

The probable reason for the observed high increment of child mortality by HIV/AIDS on households with main sewer was that as seen in others sections, households using main sewer are generally located in the urban areas or the surrounding environs where the concentration of HIV/AIDS is high.

5.3.2c: Impact of HIV/AIDS on child mortality by floor material type.

Table 5.16 also gives the impact of HIV/AIDS on child mortality by floor material type. The floor material type as already mentioned was categorized into cement, earth, wood, tiles, and others. The results reveal that HIV/AIDS increased child mortality more in households with cemented floors, while the lowest impact was to those in households with earth made floors.

From the table generally the results reveal that high HIV regime cluster experienced the highest impact of HIV/AIDS on

mortality while the low HIV/AIDS regime cluster experienced the lowest. However the observed impact in the low HIV regime cluster was below that observed in total HIV regime sentinel clusters. Those observed in the high and mid HIV regime cluster however were above those of the total HIV sentinel regime cluster.

In the high HIV regime cluster therefore, HIV/AIDS increased child mortality for those in households with tiled floors by the highest percentage (59 percent), followed by those in households with cemented floors (34 percent). The lowest impact was seen to those in households whose floor was made of earth and other materials (28 percent respectively).

In the mid HIV regime cluster, the same situation is seen to exist, however in this cluster, the lowest impact of HIV AIDS on child mortality was witnessed to those in households with other type of floor material type (14 percent) and those in households with wood floors (15 percent). The highest impact was to those on households with cement and earth made floors (16 percent respectively).

In the low HIV/AIDS regime cluster, the same differential is witnessed to that of high HIV cluster, but in this cluster, child mortality to those in households with cemented floors experienced the largest percentage increase (56 percent). The lowest increment ~~as~~^{is} in the other two HIV regime clusters was to child mortality of those in households whose floors was made of earth and other materials (3 percent respectively).

The probable reason for the observed differential is that cemented and tiled floors households are usually found in towns, near trading centres etc and HIV/AIDS is concentrated along this route and this probably explains the largest impact observed in the low HIV/AIDS regime cluster.

The largest impact of HIV/AIDS on child mortality in the high HIV cluster is probably due to the fact that HIV/AIDS is more concentrated in this cluster than in the other clusters and thus the possible high concentration of HIV/AIDS infection to men and women in this cluster which affect the children born to them.

5.3.2d: Impact of HIV/AIDS on child mortality by place of residence.

Unlike the other variable categories, the differential in impact of HIV/AIDS on child mortality was minimal when examined by place of residence. The results reveal that in the high HIV regime cluster, the impact was more pronounced in child mortality of those in rural areas, the mid HIV regime cluster, the opposite is the case, while in the low HIV regime cluster, the impact was uniform in the urban and rural areas. However the largest impact was witnessed in the high HIV regime cluster while the lowest impact was to those in the low HIV regime cluster.

In the high HIV cluster therefore, the HIV/AIDS increased child mortality by 19 percent in the rural areas and by about 18 percent in the urban areas. In the mid HIV regime cluster, HIV/AIDS increased child mortality by eleven percent in the urban

areas and about nine percent in the rural areas. These observed rates were however above the sentinel rates.

There was no observed differentials in the low HIV regime cluster, in this regime cluster, HIV/AIDS increased child mortality by 3 percent in both the rural and urban areas. This compared favourably with those of the sentinel areas.

The probable reason for this is that the migratory nature of those in high HIV cluster, thus spreading the virus in the rural areas coupled with poverty in these regions. In the urban areas the prevalence is just high.

REFERENCES.

- Aaby P.
(1987): 'Over crowding a major determinant of variations in measles mortality in Africa' IUSSP Séminar, Yaunde.
- Aaby, P.
(1989): 'Overcrowding a major determinant of variations in measles mortality in Africa' IUSSP seminar. Yaunde.
- Alberto Palloni and Yean Jul Lee
(1992): 'Some aspects of the Social context of HIV and its effect on Women, Children and Families', in population Bulletin 33, 1992.
- Alberto Palloni:
(1992): 'Some aspect of the social context of HIV and it's effects on children and families.' Population bulletin of the United Nation 33, New York.
- Anarfi J. K.
(1994): 'HIV/AIDS in Africa Sub-Saharan Africa: Its demographic and Socio-Economic implications'; African population and Environmental institute, Nairobi.
- Anker R & Knowles J.
(1980): 'An empirical analysis of Mortality in Kenya at the Macro & Micro levels economic development and cultural change', Population and employment working paper No. 60, Geneva. International labour office.
- Bailey, Norman T J
(1988a): 'Review of available and required data for applied modelling of HIV/AIDS with special reference to assisting public health decision making'. Geneva.
- Baltazar God fey, Hellen Ododo, John Stover, Allan Johnson and Tom Mboya Okeyo
(1996): 'AIDS in Kenya, Socio-economic impact and policy implications, NASCOP, Kenya.
- Barney Cohen and James Trussell (Eds)
(1996): 'Preventing and mitigating AIDS in Sub-Saharan Africa'. National academic press, Washington DC.
- Behm H
(1983): 'Socio-economic determinants of mortality in Latin America'. Population Bulletin of the United Nations, New York
- Berezin N.
(1992): 'HIV and other sexually transmitted diseases. In AIDS in the world: A global report. Cambridge massochuets and London, Havard University press.
- Berkely S.
(1992): 'HIV in Africa: what is the future?'. Annals of internal medicine Vol 116, No4 (15 February) (Philadelphia, Pennsylvania).
- Berkeley S.
(1990): 'Aids in sub saharan Africa;. An overview'. Progress report on health and development in southern Africa (Fall-Winter). Unpublished.
- United Nations
(1994): 'Aids and the demography of Africa'. New York.
- Berkley, S Naanara, W., Okware, G.
(1990): 'AIDS and HIV infection in Uganda - are more women infected than men?', AIDS, 4, 1237-42.
- Blanc, David; Smith George D; Berkley Mel
(1990): 'Social class differences in years of potential life lost; size, trend and principle causes', in British journal, Vol. 301, No.6749, Sept1, 1990. London England.
- Bongaarts J
(1987): 'Does family planning reduce infant mortality rate?'. Population and development review 13 (2): 323-334.
- Bongaarts, John Reining, Way P., and Conant F.
(1989): 'The relationship between Male circumcision and infection in African population': AIDS (3):373-7.
- Caldwell, J., C and Caldwell, P.
(1993): 'The nature and limits of the sub-saharan African AIDS epidermic: Evidence from geographical and other patterns'. Paper presented at the seminar on AIDS impact and prevention in the developing world': The contribution of Demography and Social Sciences, Annecy, France, 5-9 Dec.

- Caldwell J and Ruzicka L.T.
(1985): 'Determinants of mortality change in south Asia' in K. Srinivansan and S. Mukarji (eds), *Dynamics of population and family welfare*, Himalayan publishing house, Bombay.
- Caldwell J and Meegama S.A.
(1981): 'Influence of maternal education on Infant and child mortality'. *International population conference manila*, Vol. 2 Liege: International Union for the scientific study of population.
- Caldwell, J
(1979): 'Education as a factor in mortality decline', *population studies* 33 (3). New York.
- Caldwell, John C., Pat Caldwell and Pat Quiggin.
(1989): 'The social context of AIDS in sub saharan Africa'. *Population and Development review* vol.15 No.2 (june) (New York)
- Cameron, D William, Simonsen, J. N. and others.
(1989): 'Female to Male transmission of HIV Type 1: Risk factor for seroconversion in Men'. *Lancet*, London.
- Casterline, J.B, Cooksey E. C and A.F.E Ismail.
(1989): 'Household income and child survival in Egypt', *Demography*,
- Chen L.; C.E Hug and S. D'Souza.
(1982): 'Sex bias in family allocation of food and health care in rural Bangladesh; *Population and development review*, 7(1).
- Chin James
(1990): 'Current and future dimensions of HIV/AIDS pandemic in Women and Children'. Vol.336. No.6709 (28,July), *Lancet* (London).
- Chin James.
(1992) 'The multi sectorial impact of HIV/AIDS pandemic'. Unpublished manuscript.
- Chin James, S Lwanga and Jonathan Mann
(1989): 'The global epidemiology and projected short term demographic impact of AIDS'. *Population Bulletin of the United Nation*, No. 27. New York.
- Cleland, J and J. Van Gineken.
(1990): 'Maternal education and child survival in developing countries: The research for the pathways of influence', in Caldwell J. and G.M Santow (eds), *selected readings in cultural, social and Behaviourial determinants of Health: Health transition centre*. The Australian national University canberra.
- Cochrane, S,
(1980): 'The effects of education on health, washington DC. *World Bank staff working paper*. New York.
- Cohen Berney and Trussell James (Eds)
(1996): 'Preventing and mitigating AIDS in sub saharan Africa'. National academic Press, Washington DC.
- Curran James w
(1985): 'The epidemiology and the prevention of acquired immunodeficiency syndrome'. *Annals of internal medicine* (Philadelphia, Pennsylvania), vol 103, No.5 (Nov) pp 657-662.
- Das Gupta Monica
(1989): 'The effect of discrimination in health and mortality', in *international de la population*, New Delhi, Sept. 20-27, 1989, vol 3, pp 349-65, International Union for the scientific study of population.[IUSSP], Liege, Belgium.
- Davanzo, Julie and Jean-Pierre Habicht.
(1984): 'What accounts for the decline in infant mortality in peninsular malaysia, 1946 - 1975'. Paper presented at the PAA meeting, minneapolis, Minnesota.
- de Vincenzi Isabelle
(nd): 'Review of currently available evidence on lack of male circumcision. A risk factor for STD including HIV infection'. European centre for the epidemiological monitoring of AIDS, Paris.
- Diallo M, and Others
(1992): HIV-1 and HIV-2 infection in men attending sexually transmitted diseases clinics, cote d'ivore. *AIDS*, vol 6 No 6. Philidelphia, Pennsylvania.
- Doornhos, G; Kromhout,D.
(1990): 'Educational level and mortality in a 32 year follow up study of 18 year Old Men in Netherlands' in *International journal of Epidemiology*, Vol.19, No.2 june 1990. 374-9pp. Oxford, England.

- Ebony, G
(1993): 'Family formation patterns and child survival in Uganda, M.A project paper, National centre for development studies, the Australian National university, Canberra.
- Ewbank D., R. Renin & J. Kekovole
(1996): 'An investigation on Demographic Epidemiological Research on Mortality in Kenya' in U.N. Determinants of Mortality Change and differentials in Developing Countries. The full Country case project, New York
- Ezel, P
(1990): 'Nigeria: Female circumcision row'. New Africa. London
- Family Health International/AIDSCAP.
(1996): 'AIDS in Kenya: Socio-Economic impact and Policy implication'; USA, 1st Ed.
- Fauci Anthony S:
(1988): 'The human immunodeficiency virus, infectivity and mechanism of pathogenesis'. Science, (Washington DC). vol 239 No.4840 (5 feb) pp 617-622.
- Fieldman, D
(1991): 'The socio-cultural impact of AIDS in central and east Africa', in AIDS and the social sciences: common threads. Richard Wack and William F Skinner, (eds) Louisville, Kentucky University press.
- Fredrick T. Sai and Janet Massin
(1991): 'Diseases & Morbidity in sub-Saharan Africa', World Bank, Washington.
- Friedland, Gerald H and Robert S. Klein
(1987): 'Transmission of the human immunodeficiency virus'. The New England journal of Medicine (Waltham Massachusetts), vol. 317, No.18 (29, Oct) pp.1125-1135.
- Gaisie S.K.
(1980): 'Some aspects of socio-economic determinants of mortality in Tropical Africa'. Bulletin of the United Nations, No. 13, United Nations, New York.
- Gallo Robert C, and Flossia Wong Staal.
(1985): 'A human T. lymphotropic retro-virus (HTLV-II), as the cause of the acquired immuno deficiency Syndrome. Annals of internal medicine (Philadelphia Pennsylvania). Vol.103, No.5 (Nov) pp. 679-689.
- Gallo Robert C and Luc Montaignar:
(1988): 'Aids in 1988', Scientific America', (New York) Vol.259 No.4 (Oct) pp 40-48.
- Gasie, S.K
(1995) 'Demographic transition: The predicament of sub saharan Africa. Paper presented at the continuing Demographic transition. The John Caldwell seminar Aug 14-17, The Australian National University Camberre.
- Kibet M. K. I.
(1989): 'A study of mortality differentials in Kenya'. Msc Thesis, Population Studies and Research Institute, University of Nairobi.
- Haseltine, William A and Flossic Wong-Staal.
(1988) 'The molecular biology of the AIDS virus. Scientific American (New York), Vol.259, No.4 (Oct) pp 52-62.
- Ikamari L. D. E.
(1996): 'Factors affecting child survival', Phd Thesis, population studies and research institute, University of Nairobi.
- Iusley, Raymond, Le Curand, Juliana
(1993): Journal of epidemiology and community Health, Vol 47 No.6, Dec, 1993:444-9, London, England.
- Jonathan Mann, Daniel J M, Tarantala and Thomas W Netter (Eds)
(1992): 'AIDS mortality by Jan.1, 1992. Global report', Havard University Press.
- K'Oyugi B.O.
(1992): 'The impact of household and community level factors on Infant and child mortality in rural Kenya. Phd Thesis, population studies and research institute, University of Nairobi.
- K'OYUGI B. O
(1982): 'Mortality & Morbidity situation in Siaya', Msc Thesis, UON.

- Kenya Bureau of Statistics
(1996): 'Analytical reports on mortality, Ministry of planning, Nairobi.
- Kichamu G.A
(1988): 'Mortality estimation in Kenya with special case study of vital registration in central province, unpublished Msc Thesis, University of Nairobi.
- Killewo J, and Others.
(1990): 'Prevalence of HIV-1 infection in the Kagera region of Tanzania: a population based study. AIDS, Vol. 4 No 11. London.
- Kless, J. K.
(1986): 'AIDS virus infection in Nairobi prostitutes: spread of epidemic to East Africa'. New England journal of Medicine, 13th Feb, 414-418.
- Kotler, Pamela L.
(1989): 'Having it all: Multiple roles and Mortality. in Historical Demography, 1989, pp 223-7, Gerland publishing, New York.
- Kreiss J.M Careel and A Maheus:
(1988) 'Role of sexuality Trans diseases in transmitting of HIV. Genito
- Kwasi Anarfi John
(1994): 'HIV/AIDS In Sub saharan Africa: Demographic and Socio-Economic implication'. Africa population and Environmental Institute.
- Lado Ruzicka, Guillaume Wunsch and Penny Kane.
(1989): 'Differential mortality; Methodological issues and Bio-social factors', Clarendon press, Oxford, USA.
- Lema, V.M and Others.
(1990): 'A review of the medical aspects of adolescence fertility in Kenya'. Journal of obstetrics and gynaecology of eastern and central Africa. Nairobi.
- Lindashaun, S.
(1990): 'Maternal education and health care processes in Bangladesh: *The health and hygienic of the middle classes*' in Caldwell *et al* (eds), *Health transition series 2 (vol 1). What we know about health transition: The cultural, social and behavioural determinants of health*, Health transition centre, the Australian National University Canberra.
- Lyod B Potter
(1991): 'Socio-Economic determinants of white and black males life expectancy differentials' in Demography, Vol 28, 1991:304.
- Magadi Monica
(1990): 'Estimation of age distribution census coverage and death registration completeness in Kenya'. Msc Thesis PSRI,UON. Kenya.
- Majumber A.K .
(1989): 'Determinants of infant and child mortality in Bangladesh, unpublished Phd Thesis, The Australian National university, Canberra.
- Mann, J and Chin J.
(1986): 'AIDS: A global perspective'. New England journal of medicine, vol 319, pp 302-3. London.
- Martin L.G, Trussell, J Florentina, R.S and Nasra, M.S.
(1983): 'Co-variate of early child mortality in Philippines, Indonesia and Pakistan; an analysis based on hazard model' *Population studies* 37 (3).
- Meegama S.A
(1980): 'Socio-economic determinants of Infant and child mortality in Sri-Lanka; An analysis of post war experience' WFS scientific reports No. 8. Voolburg Netherlands, International statistics Institute.
- Merrick T.W
(1985): 'The effects of piped water on early childhood in urban Brazil, 1970-1976, Demography, 22 (1).
- Ministry of planning:
(1983): 'Demographic and Health survey' Government printers, Kenya.
- Mortikanen Pekka T.
(1990): 'Unemployment and mortality among Finish Men 1981-85', British Medical Journal, Vol.301, No.6749, Sept.1, 1990 pp 407-11, London, England.
- Moses, S and Bradley, J, E.
(1990): 'Geographical patterns of male circumcision practices in Africa: Association with HIV prevalence '. International journal of epidemiology. New York.

- Mosley W. H
(1990): 'Social determinants of Infant and Child mortality: Some consideration for an analytical framework'. Discussion paper presented at a conference on health and mortality in Infancy and early childhood, held in Cairo, 19-20, May 1980.
- Mulandi T.M.
(1984): A study of sexually transmitted diseases and the effect of these on cervical cytology in contraceptors antenatal and control population groups as a rural area in northern division of machakos district, kenya. Unpublished disatation University of nairobi.
- Munganzi Z.S
(1984): 'The effect of individual and contextual factors on infant mortality, unpublished Phd Thesis, The Florida state university, Tallahassee.
- Nag, M.
(1985): 'Impact of social and economic development on mortality: Comparative study of Kerela and West Bengal in S.B Halstead'; J.A.A Walsh and K.S Warren (1985). *Good health at low cost* proceeding of a conference held at the Bellagio conference centre, Bellagio, Italy.
- NASCOP
(1996): 'AIDS in Kenya, background infection, Impact and Intervention'. Third Edition, Ministry of Health, Kenya.
- United Nations
(1986b): 'Update. public health services workshop on human T-lymphotropic virus type III. Anti body testing - USA, Morbidity and mortality weekly report (Atlanta Georgia) Vol 34, No 31 (9 Aug) pp 477-478.
- Nyakangi, J
(1982): 'Unpublished thesis', PSRI, University of Nairobi.
- Ocholla - Ayayo A.B.C.
(1990): 'The risk of the spread of STDs and AIDS in Kenya. WHO and population studies and research institute, Nairobi.
- Ondimu K.N.
(1987): 'The influence of socio-economic factors on child mortality in Kenya; evidence from 1984 Kenya contraceptive survey'. Unpublished M.A Thesis, population studies and research Institute, University of Nairobi.
- Otieno J.A.M.
(1986): 'Estimation of mortality levels and Differentials in Kenya': paper presented in seminar on Socio-Economic aspects of Differential mortality between 9th and 12th Sept 1986 in Zamardi; Hungary.
- Palloni Alberto and Yean JU Lee.
(1992): 'Some aspects of the social context of HIV and its effect on women, children and families' in population Bulletin 33, 1992, New York.
- Piot, P and Mann, J.
(1987): 'AIDS in the tropics, Bai clinical Tropical medicine and communicable diseases, vol 2, pp 205-22.
- Piot, P and others
(1984): 'Heterosexual transmission of HIV. AIDS, Vol. 1, No. 4, Pennsylvania.
- Piot P, Plummer F., A.
(1987): 'Retrospective seropidemiology of AIDS virus infection in Nairobi. Population journal of infectious diseases, New York.
- Pool, I. D
(1982): 'Is new Zealand a Healthy country?', New Zealand population Review, 8(2)2-27.
- Preston, S. H.
(1979): 'Mortality. morbidity and development paper presented to the seminer on population and development in ECA region.
- Quinn T. C and Others:
(1986) 'Aids in Africa, an epidemiologic paradigm'. Science (Washington DC) Vol.234 No. 4,779 (Nov)
- Redfield Robert R and Donald J Burke
(1988): 'HIV infection the clinical picture'. Scientific American (New York) Vol. 259, No. 4 (Oct) pp 90-98.
- Ronoh, J., K
(1982): 'Unpublished Msc Thesis, PSRI, University of Nairobi, Kenya.

- Safian Abu J. M
(1989): 'Socio-economic correlates of life expectancy at birth; the case of developing countries in journal of population and Health studies Vol, 9 no.2 Dec 1989, pp 214-27.
- Stephen Forsyth and Bill Rau:
(1996): 'AIDS in Kenya. social economic impact and policy implications'. Family Health International/AIDSCAP. First edition USA.
- Stoto M. A.
(1991): 'Models of the demographic impact of AIDS in Africa. Draft prepared for the panel on the Demography of sub-Saharan Africa, committed on population, National research council (october 29). Unpublished.
- Thomas P. Pugh and Johnson Ipsen
(1960) 'Epidemiological methods', Havard school of public health.
- Timeaus, I. A. N:-
(1990) 'Advances in the methods of adult mortality from data on orphanhood. CPS Research paper No.90-1, June 1990 v.59. University of London, school of Hygiene and tropical medicine. Centre for population studies, London England.
- United Nations
(1983): 'Manual X', New York, Population Division.
- _____
(1994a): 'World population prospectus: the 1992 revision'; annex tables, New York, Population Division.
- _____ and World Health Organization.
(1989): 'The AIDS epidemic and its demographic proceedings of the UN/WHO workshop on modelling the Demographic impact on the AIDS epidemic in pattern II countries: New York, 13-15 Dec.
- _____
(1994a): 'World Population prospectus; The 1994 revision', annex tables, New York Population division, New York.
- _____
(1993): 'World Population prospectus, 1992 revision - New York, Population Division.
- _____
(1990a): 'World population Monitory 1969, Population studies No. 113, Pg. 68-107, New York.
- _____
(1986): 'An integration of Demographic and epidemiologic research on mortality in kenya' in *determinant of mortality change and differentials in developing countries*, New York.
- _____ and World Health Organization.
(1989): 'The AIDS epidemic and its demographic consequences preceding of the UN/WHO workshop on modelling the demographic impact on the AIDS epidemic in pattern II countries: Dec, 1989, 13-15, New York.
- _____
(1986): 'Determinants of mortality change and differentials in developing countries'. New York.
- _____
(1994) 'Step by step guide to the estimation of child mortality. New York.
- _____
(1995): 'Population Bulletin , Pop. Studies No. 39 New York.
- Voorhoeve A; M Muller A.S; Schullpen T. W. S; Manefjan W.T.; and Van Ras M.
(1978): 'Machakos project studies IV: Epidemiology of pertussis Tropical and geographical medicine 30:115-139.
- Way P.O and Stanecki K. A
(1993): 'An epidemiological Review of HIV/AIDS in sub Saharan Africa'. Centre for International Research, US Bureau of the Census.
- Weber Stanley and Weiss Robert A:
(1988) 'HIV Infection: The cellular picture'. Scientific American, vol. 259, No 4 (Oct) New York.
- William Brass and Coale L Jolly(Ed).
(1993): 'The population dynamics of Kenya'. National Academic press, Washington DC.
- World Health Organization
(1991): 'In point of fact, No 74. Geneva.