The Probability of Maternal Orphanhood Under a Generalized AIDS Epidemic[1]

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Abstract

The generalized HIV/AIDS epidemic is devastating African families. Understanding how epidemiological patterns are translated into aggregate demographic outcomes is an important task for addressing this crisis of care. Using methods from stable population theory and AIDS-decremented model life tables for Africa, I analyze the effect of different mortality and fertility schedules on the probability of a child losing her mother. In high mortality environments, the probability that a girl will die before reaching some age a is non-negligible. I develop a method to estimate the probability of orphanhood conditional on surviving to a based on the analysis of first passage times of a Markov chain. Mortality decrements in the characteristic AIDS shape have a large impact on the probability of maternal orphanhood. Fertility (both overall level and parity-specific subfertility), in contrast, has a small effect on orphanhood probability. Despite the severe limitations of a one-sex model using stable population identities, this analysis provides important qualitative insights into the demographic processes that link HIV disease and aggregate demographic outcomes. I suggest possibilities for future work in which the assumptions of the present analysis are relaxed.

1 Introduction

The generalized HIV/AIDS epidemic in Sub-Saharan Africa has led to a an orphanhood problem of crisis proportions. According to the recent UNICEF report on the state of the orphanhood crisis in Africa, HIV prevalence among adults ages 15-49 is currently 7.3% in eastern and 19.2% in southern Africa, with corresponding orphanhood prevalence of 12% and 17% of all children 0-14 respectively. AIDS claimed an estimated 2.2-2.4 million deaths in 2003. Despite some progress toward control of the epidemic (Stoneburner and Low Beer 2004), the year 2003 nonetheless witnessed an estimated 3 million new HIV infections in Sub-Saharan Africa.

Caring for Africa's orphaned children represents a major policy challenge for the 21st century (Preble 1990). Demographic projections suggest that by 2010, from 20 to 37 per cent of children under the age of 15 in Sub-Saharan Africa will have lost at least one parent (Foster 2002). In addition to the humanitarian crisis, the generation of so many orphans creates substantial negative structural consequences for the countries of Sub-Saharan Africa. Orphanhood carries a multitude of negative social outcomes. Children orphaned by AIDS are often less likely to be enrolled in school, have lower age at sexual debut, greater morbidity, higher delinquency rates, greater illiteracy and higher unemployment as adults (Walraven et al. 1996; Foster and Germann 2002; Bicego et al. 2003). In addition, AIDS mortality among parents frequently leads to the dissolution of households, creating social and geographic instability (Urassa et al. 2001).

Two important questions that arise in the context of the current orphan crisis in Sub-Saharan Africa. First, what is the probability is that a child will be orphaned at a given age? Second, what is the probability that a child will become an orphan before reaching adulthood? Empirical data on orphanhood have recently become available through

the Demographic and Health Surveys, providing estimates of the number of orphans for various ages. While these data are of vital importance, understanding the social and demographic processes surrounding orphanhood requires a model relating orphanhood to schedules of demographic events and the social, epidemiological, ecological, and economic forces that drive them. On the recent decline of the use of models in demographic research even as high quality data become more abundant Coale and Trussell (1996, 484) write, "while the trend toward the use of sophisticated statistical models appropriate for the problem being analyzed and the data that are available is healthy, the trend toward accepting demographic survey data at face value is not."

While the consequences of orphanhood are felt biosocially and economically, the generation of orphans is fundamentally a demographic process. As originally elucidated by Lotka (1931), the probability that a child will have a living parent is a function of the prevailing age-specific schedules of mortality and reproduction. This suggests that substantial insight into the orphanhood problem can be gained by analyzing – both analytically and through simulation – the consequences for orphanhood of changing demographic schedules. In this paper, I use the methods of the demography of kinship to explore the effect of AIDS mortality in particular on the probability of maternal orphanhood under a generalized AIDS epidemic.

2 Methods

2.1 Data

Mortality I used INDEPTH HIV-decremented model life tables to explore the effects of AIDS-related mortality on the probability of orphanhood (INDEPTH Network 2002). The INDEPTH model life tables provide the most complete and accurate record of the

mortality experience in Africa currently available.

The AIDS-decremented model life tables were constructed using component mortality models typically employed in the construction of model life tables (e.g., Coale et al. 1983). Let $\mathbf{C}$ be a $k \times l+1$ matrix of loadings of the k ages on the first l components of a principle components analysis, with a leading column of ones. Define $\mathbf{m} = \text{logit}({}_nq_x)$ for $x \in 0 \ldots k$. A model life table is defined by

$$\mathbf{m} = \mathbf{Ca}$$

where $\mathbf{a}$ is an $l + 1 \times 1$ vector of coefficients.

In general, the coefficients are estimated by regressing empirically-derived values of $\text{logit}({}_nq_x)$ on $\mathbf{C}$.

The INDEPTH model life tables depict seven general mortality patterns. For the present paper, the most relevant are patterns 1 and 5. Pattern 1 resembles the Coale-Demeny North model life table in that it is characterized by high infant and childhood mortality. The populations that primarily contribute to this pattern are from West Africa, where endemic malaria is a significant source of under-10 mortality. The INDEPTH Pattern 5 life tables correspond to regions in eastern and southern Africa with high HIV/AIDS prevalence, and are characterized by a substantial mortality bulge beginning in early adulthood.

The AIDS-decremented life tables are generated by creating a model life table with an underlying pattern similar to the pattern 1 model. Excess mortality due to AIDS is added by regressing the difference of model 5 and model 1 life tables for the same life expectancy at birth ($\mathring{e}_0$)against the first 15 principal components from the PCA. Let the coefficients of this regression be $\mathbf{d}'$. The AIDS-decremented life tables are then constructed by:

$$\mathbf{m} = \mathbf{C}(\mathbf{a} + \alpha\mathbf{d}),$$

where α is a scale parameter which determines the extent to which excess AIDS mortality is added.

Figure 4 plots the life table survivorship function $l(x)$ against age for two AIDS-decremented mortality families, $\mathring{e}_0$= 45 and $\mathring{e}_0$= 60, the lowest and highest life expectancy families of the published model life tables (INDEPTH Network 2002).

Fertility As a baseline for comparison, I use the synthetic natural fertility schedule of Coale and Trussell (1978), ultimately derived from Henry (1961). Using the parametric model fertility schedules of Coale and Trussell (1974), this pattern of age-specific fertility can be manipulated to achieve both different levels of overall fertility and deviations from natural fertility, measured as the extent of parity-specific subfertility. The Coale-Trussell model expresses the realized age-specific fertility rates as a function of a synthetic natural fertility schedule and two parameters M and m. These parameters characterize the overall level of marital fertility and the degree of departure from natural fertility respectively. The realized age-specific fertility for each age a between ages 20 and 44 is

$$r_a = n_a M e^{m v_a} \tag{1}$$

where v_a is a set of empirically derived deviations presented in Coale and Trussell (1974) and updated in Coale and Trussell (1978), and n_a is the synthetic natural fertility schedule derived by Coale and Trussell from sources in Henry (1961).

The values of M and m are typically estimated by regressing $\log(r_a/n_a)$ against v_a.

$\log(M)$ and and m are the intercept and slope, respectively, of this regression. Fertility rates below age 20 and above 44 are interpolated between the zero and the values for these these ages. This method, while somewhat ad hoc, typically produces sensible values (Coale and Trussell 1974, 1978). Alternatively, the parameters can be estimated using Poisson regression (Bronström 1985), where the number of births per age group is regressed against v_a using $\log(n_a E_a)$ as an offset (where E_a is the number exposed in age-class a). For the present study, variations on fertility schedules can then be produced by systematically varying M and m.

For this analysis, I focus on values of M approximately equal to 0.5. This choice reflects empirical patterns of fertility derived from DHS surveys of Sub-Saharan Africa from 1991-2002. The ASFR schedules from 43 DHS surveys are plotted in figure 1, along the the Coale-Trussell synthetic natural fertility schedule and the mean of the 43 schedules. The estimated value of M for the mean using the OLS method described in Coale and Trussell (1974) is 0.49. Because of the likely effects of HIV disease on the m parameter in the Coale-Trussell model (see Discussion), I focus particularly on the effect of variation in parity-specific subfertility on the probability of orphanhood.

I restrict attention to the female segment of the population. This decision reflects the general lack of model schedules of male age-specific fertility and the technical difficulties associated with the two-sex problem for kinship studies (Reeves 1987). I will take up the limitations imposed by the assumption of female demographic dominance in the Discussion and suggest some possible solutions.

2.2 Mathematical Demography of Kinship

Let $l(x)$ represent life-table survivorship – the probability that a girl will survive to exact age x, $l(x) = \exp(-\int_0^x \mu(a)da)$, where $\mu(a)$ is the instantaneous mortality rate at age a.

From this elementary life table function, the probability that a girl age a will have a living mother is simply $l(x+a)/l(x)$, where x is the age of the mother when the girl was born. To get the probability that a randomly-selected girl age a will have a living mother, we must integrate $l(x+a)/l(x)$ across all possible ages of mothers. This integral is weighted by the fraction of births attributable to mothers of each age, $W(x)$. Following Goodman et al. (1974), I refer to the probability that a girl age a's mother being alive as $M_1(a)$:

$$M_1(a) = \int_{\alpha}^{\omega} \frac{l(x+a)}{l(x)} W(x|t-a)dx, \tag{2}$$

where $W(x|t-a)$ is the age structure of mothers at time $t-a$.

Under the stable population assumptions of constant demographic schedules and zero net migration, the fraction of mothers age x ceases to depend on $t-a$, and the fraction of births attributable to women age x is:

$$W(x) = e^{-rx} l(x) m(x), \tag{3}$$

where $m(x)$ is the fertility rate at age x, and r is the instantaneous rate of increase, which is given by the unique positive real root of the Euler-Lotka solution to the renewal equation,

$$1 = \int_{\alpha}^{\omega} e^{-rx} l(x) m(x) dx. \tag{4}$$

Substituting the stable population weights for the distribution of mothers into equation 2, we get

$$M_1(a) = \int_{\alpha}^{\omega} e^{-rx} l(x+a) m(x) dx. \tag{5}$$

This result was first derived by Lotka (1931) and was expanded upon by Goodman et al. (1974) to examine other kinship relationships as a function of demographic schedules.

2.3 Numerical Methods

While the mathematical theory of kinship (Goodman et al. 1974; Keyfitz 1985) is developed in a continuous-time framework, demographic data typically come from abridged life tables with discrete age intervals. I graduated the five-year interval demographic data using interpolating splines, and solved the Euler-Lotka equation for r using numerical minimization of the squared error. I then numerically integrated equation 5 using the graduated data by means of adaptive quadrature.

Software to estimate the probability of having a living mother given schedules of age-specific mortality and fertility was originally written by Mark Handcock in R and is available on request.

2.4 Conditional Orphanhood Probability

The probability of maternal orphanhood calculated through equation 3 represents the unconditional probability of a girl having a living mother. In high-mortality environments, there is a non-negligible probability that a girl will die before reaching age a, regardless of the particular value of a. The calculation of the conditional probability that a newborn child will become orphaned before attaining adulthood requires the combination of three related, but usually distinct domains of demographic theory: (1) projection of structured population, (2) analysis of Markov-chains, and (3) stable population theory and the mathematical demography of kinship, discussed above.

2.4.1 Projection of Structured Population

I model the female component of a general population divided into k five-year age-classes. The projection matrix, $\mathbf{A}$, is a $k \times k$ matrix with survival probabilities $P_x = {}_5L_{x+5}/{}_5L_x$ along the sub-diagonal, age-specific effective fertilities along the first row, and zeros elsewhere. To study the dynamics of orphanhood, however, we only need the survival component of the Leslie matrix (i.e., the subdaigonal).

2.4.2 Markov Chain

The projection matrix resembles discrete-time Markov transition matrix (Norris 1997), and is easily converted to one. Following Caswell (2000), the population projection matrix can be usefully decomposed into its fertility and survival components:

$$\mathbf{A} = \mathbf{T} + \mathbf{F} \tag{6}$$

In the terminology of Markov chains, all of the states of a demographic projection matrix are "transient" ones. The transformation of the standard projection matrix to a Markov transition matrix involves adding absorbing states and the transition probabilities thereto. These absorbing states typically include death, but may also include some other demographic outcomes of interest.

I consider $m = 3$ absorbing states: orphanhood, death, and attaining adulthood. For simplicity and compatibility with standard abridged life tables, I define "adulthood" as a girl reaching her 20th birthday. Define a new, $k + m \times k + m$ matrix $\mathbf{P}$

$$\mathbf{P} = \left[\begin{array}{c|c} \mathbf{T} & \mathbf{0} \\ \hline \mathbf{M} & \mathbf{I} \end{array}\right], \tag{7}$$

where $\mathbf{T}$ is the $k \times k$ survival component of the original projection matrix, $\mathbf{M}$ is an $m \times k$ submatrix containing the transition probabilities into the absorbing states, $\mathbf{0}$ is a $k \times m$ matrix of zeros, and $\mathbf{I}$ is an $m \times m$ identity matrix. This new matrix is column stochastic with $\sum_j p_{ij} = 1$, where p_{ij} is the ijth element of matrix $\mathbf{P}$.

A vector of age-specific conditional probabilities of absorption in the mth absorbing state is given by the mth row of the $m \times k$ matrix:

$$\mathbf{B} = \mathbf{M}(\mathbf{I} - \mathbf{T})^{-1}. \tag{8}$$

where this time $\mathbf{I}$ is a $k \times k$ identity matrix.

In general, a child within each age class i can do one of three things: (1) survive into the next stage (p_i), (2) die within the age class (μ_i), or become orphaned within the age class (ω_i). These three transitions uniquely define the possible events that can happen to a child. Thus, $p_i + \mu_i + \omega_i = 1$. p_i is a standard life table quantity (i.e., $l(i+1)/l(i)$), and μ_i follows from p_i and ω_i. What remains is to determine the values of ω_i. Equation 5 provides the probability that a girl age a will have a living mother. Its complement therefore provides precisely the transition probability needed to complete the Markov transition matrix $\omega_i = 1 - M_1(i + 2.5)$, the complement of equation 5 centered on the age class.

3 Results

Analytical Observations As noted by Keyfitz (1985), a constant addition k to the age-specific mortality rate will increase the logarithm of the probability of orphanhood in a linear fashion since addition of a constant decrement k to the force of mortality $\mu(x)$ between ages x and $x + a$ reduces $M_1(a)$ by the fraction e^{-ka}.

The main impact of fertility on the probability of orphanhood is through the net maternity function. Keyfitz (1985) notes that when the net maternity function is concentrated around the mean age of childbearing κ, $M_1(a) \approx l(\kappa + a)/l(\kappa)$.

Taking logarithms of both sides of this expression and differentiating with respect to x yields

$$\frac{1}{M_1(a)} \frac{dM_1(a)}{d\kappa} = -\left[\mu(\kappa + a) - \mu(a)\right]. \tag{9}$$

The effect of a change in the mean of the net maternity function is most clearly seen by re-writing this expression in discrete terms.

$$\frac{\Delta M_1(a)}{M_1(a)} \approx -\left[\mu(\kappa + a) - \mu(\kappa)\right] \Delta\kappa.$$

If the mortality rate increases between the mean age of the net maternity function κ and $\kappa + a$, the proportionate change in $M_1(a)$ will be negative. Furthermore, increases in κ will be negatively related to the proportionate change in $M_1(a)$.

In general, $l(\kappa + a)/l(\kappa)$ overestimates the value of $M_1(a)$ calculated more precisely from equation 5 because of the concavity of the survivorship function. Analysis of this discrepancy yields considerable insight into understanding how changes in net maternity affect the probability of orphanhood. Expand $l(x + a)/l(x)$ around κ using a Taylor polynomial. Denote the derivative of $l(\kappa + a)/l(x)$ at $x = \kappa$ by $[l(\kappa + a)/l(\kappa)]'$, and the variance of κ as σ^2. Keyfitz (1985) shows

$$M_1(a) \approx \frac{l(\kappa + a)}{l(\kappa)} + \frac{\sigma^2}{2} \left[\frac{l(\kappa + a)}{l(\kappa)}\right]''. \tag{10}$$

The probability that a girl will have a living mother is thus strongly influenced by

three factors. First is the mean age of childbearing. Higher κ will mean higher probability of orphanhood. Second is the degree of concavity of the survivorship function. Keyfitz (1985) suggests that in most cases $d^2l(x)/dx^2 < 0$ for almost the entire range of x, making the second term less than one. The more negatively concave this curve is, the higher the probability of orphanhood and the more the actual value of $M_1(a)$ is reduced from $l(\kappa + a)/l(\kappa)$. The effect of increasingly severe AIDS-decrements to the model life tables makes the second derivative of $l(x)$ more negative. The degree of negative concavity is related to the base $\mathring{e}_0$, with lower life expectancies having more negatively concave shapes through early adulthood (and more positively concave shapes in later adulthood). Third, the greater the variance in the net maternity function, the lower the value of $M_1(a)$ relative to $l(\kappa + a)/l(\kappa)$.

While the effect of the latter two factors is typically small (Keyfitz 1985), when $l(x)$ changes very rapidly in the prime childbearing years, the effects will be considerably larger. This is exactly what happens with the addition of a major mortality decrement incident in childbearing years.

Probability of Maternal Orphanhood Under Model Schedules Figure 6 presents the probability of a girl being a maternal orphan at age a under two $\mathring{e}_0$ model life tables, with the four values of parity-specific subfertility depicted in figure 5. The two model life tables are for $\mathring{e}_0$= 45 and $\mathring{e}_0$= 60, with no AIDS decrement. The effect of varying parity-specific subfertility is relatively small. Figure 7 presents the effect on the probability of orphanhood of adding AIDS-characteristic mortality to the population. In the most severe mortality scenario ($\mathring{e}_0$= 45 with 20-year AIDS decrement), the probability of orphanhood rises rapidly so that a young woman of 20 has less than a one-in-five chance of having a living mother.

The effects of the overall level of fertility (M in the Coale-Trussell model schedule) are small. Figure 8 shows the results of changing the level of fertility to $M = 0.33$ from $M = 0.5$, again with the five levels of the parameter m. The two different levels of fertility yield essentially indistinguishable curves in monochrome. Color plots reveal very small differences in the $M_1(a)$ curves against daughter's age. The probability of orphanhood changes little with the overall level of fertility because the shape of the net maternity function remains constant for different values of M and the mean of the net maternity function κ does not change with a change in M. Reduced level of fertility increases the unconditional probability of orphanhood at all ages since it raises the mean age of childbearing slightly.

Note that all these probabilities are unconditional and do not account for the very real possibility of the girl's death before age a.

Conditional Probability of Orphanhood Figure 9 presents the baseline conditional probabilities of orphanhood from the $\mathring{e}_0$= 45 and $\mathring{e}_0$= 60 model life tables with no AIDS decrements. Figure 10a displays the striking impact the AIDS mortality pattern has on the conditional probability. The decrement of five years in the AIDS pattern from the $\mathring{e}_0$= 45 model life table nearly doubles the conditional probability of orphanhood at all ages. The conditional probability that a girl who survives to age a will have lost her mother by age 20 is approximately 25% for the highest mortality model life table (i.e., $\mathring{e}_0$= 45) in the absence of additional AIDS mortality. With the subtraction of five years in the AIDS pattern from $\mathring{e}_0$, this probability jumps to 42%.

Figure 10 presents the effect of the various AIDS-related decrements. As the AIDS mortality grows more severe, the conditional probability increases though a girl's tenth birthday.

As with the unconditional probabilities, the effects of fertility differentials are small compared to the effects of mortality. The overall level of fertility has little impact on the conditional probability of orphanhood. The exercise of parity-specific control (or simply parity-specific subfertility) similarly has little impact on the probability – conditional or unconditional – of orphanhood. This contrasts to analyses looking in the other direction with respect to the life cycle. The probability that a parent will have a living child in a population characterized by high AIDS mortality is strongly influenced by parity.

AIDS Mortality and Orphanhood Figure 11 plots the effect of AIDS mortality on the unconditional probability of orphanhood for two model life tables with equal life expectancy at birth. The solid line represents the unconditional probability of orphanhood for a $\mathring{e}_0 = 45$ model life table with no AIDS mortality decrement, while the dotted line plots the same probability for $\mathring{e}_0 = 60$ with a 15 year AIDS-decrement. The lines rapidly diverge through childhood, reaching a maximum difference in early adulthood. The probability that a 50 year-old woman with a living mother will experience the loss of her mother, not surprisingly, is not affected by AIDS mortality.

In figure 12, the same two model life tables are input into equation 8. The remarkable implication of this plot is that AIDS mortality more than doubles the conditional probability that a girl will lose her mother before reaching her 20th birthday (Jones 2004).

4 Discussion

AIDS-induced mortality has a substantial impact on the probability that a girl will become orphaned. This is not surprising since the preponderance of AIDS mortality strikes during the prime reproductive years. In contrast, the effects of fertility on the probability of orphanhood are modest. In high mortality environments, the probability of a girl dying

before reaching any given birthday is not ignorable. By analyzing the process in terms of the first passage time of a Markov chain, I have shown how to calculate the probability of orphanhood before adulthood conditional on surviving to a. Analysis of the conditional probabilities of orphanhood paints a grim picture for children in Sub-Saharan Africa.

Cumulative survivorship enters the expressions for the probability of orphanhood (equations 5 and 8). However, two life tables with exactly the same life expectancy at birth – one with the AIDS pattern of mortality and one without it – produce very different age profiles of orphanhood. These differences are particularly acute for the conditional probability of orphanhood (figure 12), in which the conditional probability for the AIDS-decremented model life table is more than double that of the non-decremented life table with the same $\mathring{e}_0$. Not surprisingly, AIDS generates more orphans. What is perhaps surprising is the magnitude of the impact the AIDS-pattern of excess mortality has when compared to an equally high-mortality model life table with effectively no AIDS mortality (Jones 2004).

While the Lotka equation for orphanhood probability (equation 5) is an extremely valuable tool, the probabilities it generates must be taken in context. The risk at birth of orphanhood must account for the fact that many children – particularly in the highest mortality populations – will die before reaching maturity. The methods described here to calculate conditional probabilities from the first passage of the Markov chain are therefore an important contribution to understanding the social impacts of the generalized AIDS epidemic in Africa.

In addition to the many obvious negative consequences for children of being orphaned, the extremely high probabilities of orphanhood have downstream social consequences as well. The more children without parents in the $f1$ generation, the more children will lack grandparents in $f2$ generation (Jones and Morris 2004). As grandparents are the

primary caregivers for orphans in many parts of Sub-Saharan Africa, a general lack of grandparents could potentially leave a care vacuum for future generations of children at risk of orphanhood due to the generalized AIDS epidemic.

The two limitations of the analyses presented in this paper are: (1) the one-sex model, and (2) the use of stable population identities in calculating frequencies of kinship. One obvious solution to these problems is demographic microsimulation (Wachter 1987). Microsimulation should be a particularly valuable tool for looking at collateral kin relations (e.g., aunts, uncles, cousins). Keyfitz (1985) notes that equations for lineal kin frequencies (e.g., equation 5) are quite robust to violations of their underlying assumptions, while those for collateral relatives are not. Collateral kin are of central importance in many kinship and inheritance systems. For example, paternal aunts are the preferred caregiver for orphans in Zimbabwe (Foster et al. 1995).

Microsimulation should facilitate the calculation of a wide variety of important living-kin frequencies, particularly those that cross lineages (i.e., from mother's to father's line and vice-versa), however, the general absence of male fertility schedules will still present challenges.

The value and limitations of the stable population model have been widely discussed. The stable population relationships that I employ in this paper do not represent measurements per se. Rather, they elucidate the relationship between demographic schedules and a variety of outcomes. The great value of closed-form analytic expressions such as those of the stable population model is that they provide qualitative insight from analysis. Complex behavior can be simulated, but insight can elude the investigator. While the restrictive assumptions that are typically required to produce an analytical model hinder their realism, the payoff in terms of understanding makes the development and use of such models well the effort (Keyfitz 1975; Coale and Trussell 1996; Lee 2001). Similarly,

empirical data are of fundamental importance for demography, and science in general. However, data collected without a model to support them can be difficult to interpret and, more problematic, misleading (Keyfitz 1975; Coale and Trussell 1996). As Keyfitz (1985: 409) succinctly described it: "no model, no understanding."

A problem with the stable population model is the assumption of constant demographic schedules. Patterns of age-specific mortality and fertility are clearly changing in Africa. The analysis I have presented here suggests the likely effect of different types of demographic change. For the foreseeable future, AIDS mortality is likely to get worse in much of Southern Africa (Zimbabwe, Zambia, Botswana, South Africa, Namibia, Mozambique, and Malawi), while countries in eastern Africa, notably Uganda, have shown small improvement (Stoneburner and Low Beer 2004). Mortality, for the most part, has an unambiguous effect on orphanhood. Increases in mortality after age at first parturition will increase the number of orphans. Mortality correlations are likely to arise due to HIV-infection. More infected mothers means more infected children due to vertical transmission as well as more deaths because infected mothers are unlikely to be able to fully provide the quality of care required to successfully raise a child in an already high-mortality environment (Lindblade et al. 2003). However, since mortality enters equation 5 only through the $l(x)$ function, increased mortality at any age will decrease the fraction of girls in the stable population with living mothers. These considerations suggest that the orphanhood probabilities based on stable population identities I have presented in this paper may represent best-case estimates.

The effects of changing fertility are less clear cut. Gregson et al. (1999) have presented evidence for fertility reduction with HIV infection. In terms of the Coale-Trussell model used in this paper to model fertility, such changes are most likely to be seen in the second parameter, m, which models the effect of parity-specific fertility control. One criticism

of the Coale-Trussell model fertility schedule has been that m, in addition to its lack of ready interpretability, can be confounded by factors other than parity-specific control. Of particular interest is the onset of permanent sterility. The effect on fertility of sexually-transmitted infections resembles that of parity-specific control (Harpending and Draper 1990).

HIV disease potentially affects fertility through several pathways. Women screened in ante-natal clinics have substantially lower HIV prevalence than the general population (Gray et al. 1998; Zaba et al. 2000). Increased rates of fetal loss are seen beginning with HIV seroconversion (Zaba and Gregson 1998; Ross et al. 2004), with the rate progressing with the disease. With progression to AIDS, sexual activity is greatly reduced and fertility is further curtailed (Ross et al. 2004). There is also a potential of behavioral selection effects on low fertility, as low gravidity is strongly associated with subsequent low fertility with HIV disease (Ross et al. 1999).

The classic pattern of subfertility induced by sexually transmitted infections (STI) in non-contracepting populations resembles the exercise of parity-specific control. As a woman ages, both her parity and her cumulative hazard of experiencing secondary sterility increase. While there are significant differences – both in terms of epidemiology and pathogenesis – between HIV disease and the classic sterilizing STIs (e.g., gonorrhea, syphilis), the cumulative nature of the hazard should nonetheless exert the parity-specific control mimicry that makes identifying the onset of contraception using the Coale-Trussell model problematic (Ewbank 1993; Okun 1994; Okun et al. 1996).

Somewhat unlike the classic sterilizing STIs, HIV disease will also exert an impact on the level of fertility. One of the largest risk factors for HIV positivity in ante-natal clinics is age at sexual debut. If the fertility-reducing effects of increased fetal loss occur from the outset of HIV infection, as suggested by (Ross et al. 2004), then HIV should have a

real impact on level of fertility.

The analysis I have presented here indicates that the both parameters M and m in the Coale-Trussell model have negligible impact on the probability of orphanhood when compared to $\mu(x)$, the age-specific force of mortality. Thus, although HIV disease has large effects on fertility, unless these effects specifically change the shape of the net maternity function (and, in particular, its mean, κ), they will have little impact on the process of orphanhood when compared to the effect of changing mortality rates.

The probability of orphanhood will increase with κ. The location of the net maternity distribution can be changed fundamentally in two ways: (1) through survivorship and (2) through fertility. Improving mortality conditions will weight the net maternity distribution more toward younger ages, reducing the probability of orphanhood in daughters. In contrast, delaying reproduction – without substantially improving AIDS mortality – will shift net maternity to older ages, increasing the probability of orphanhood. Though typically associated with later net maternity, delayed age at sexual debut, because it is associated with lowering risk of HIV infection (Ferry et al. 2001; Drain et al. 2004), will in all likelihood reduce the probability of orphanhood.

Children frequently experience differential outcomes depending upon whether they are maternal, paternal, or double orphans. Loss of a father has been documented to impart greater economic hardships in some areas (Lindblade et al. 2003). Very young children typically do poorly when they lose their mother (Masmas et al. 2004).

The unconditional and conditional probabilities of orphanhood have different uses in application to the orphan crisis in Sub-Saharan Africa. In terms of planning for capacity-related responses, the unconditional probability of orphanhood is key since this represents a snap-shot of the number of orphans of a given age at a given time. In terms of thinking about the social impact of the HIV/AIDS epidemic, the conditional probability is perhaps

more useful.

Understanding the aggregate effects of HIV disease on social outcomes is essential for formulating sound policy to help children affected by the generalized HIV/AIDS epidemic in Sub-Saharan Africa. While empirical studies of the frequency of orphanhood are of immense value, well-informed policy requires the understanding of process that models provide. The models I have presented in this paper represent a first step toward an understanding of the interaction of epidemiology, demographic processes, and social outcomes that characterize the generalized HIV/AIDS epidemic of Sub-Saharan Africa.

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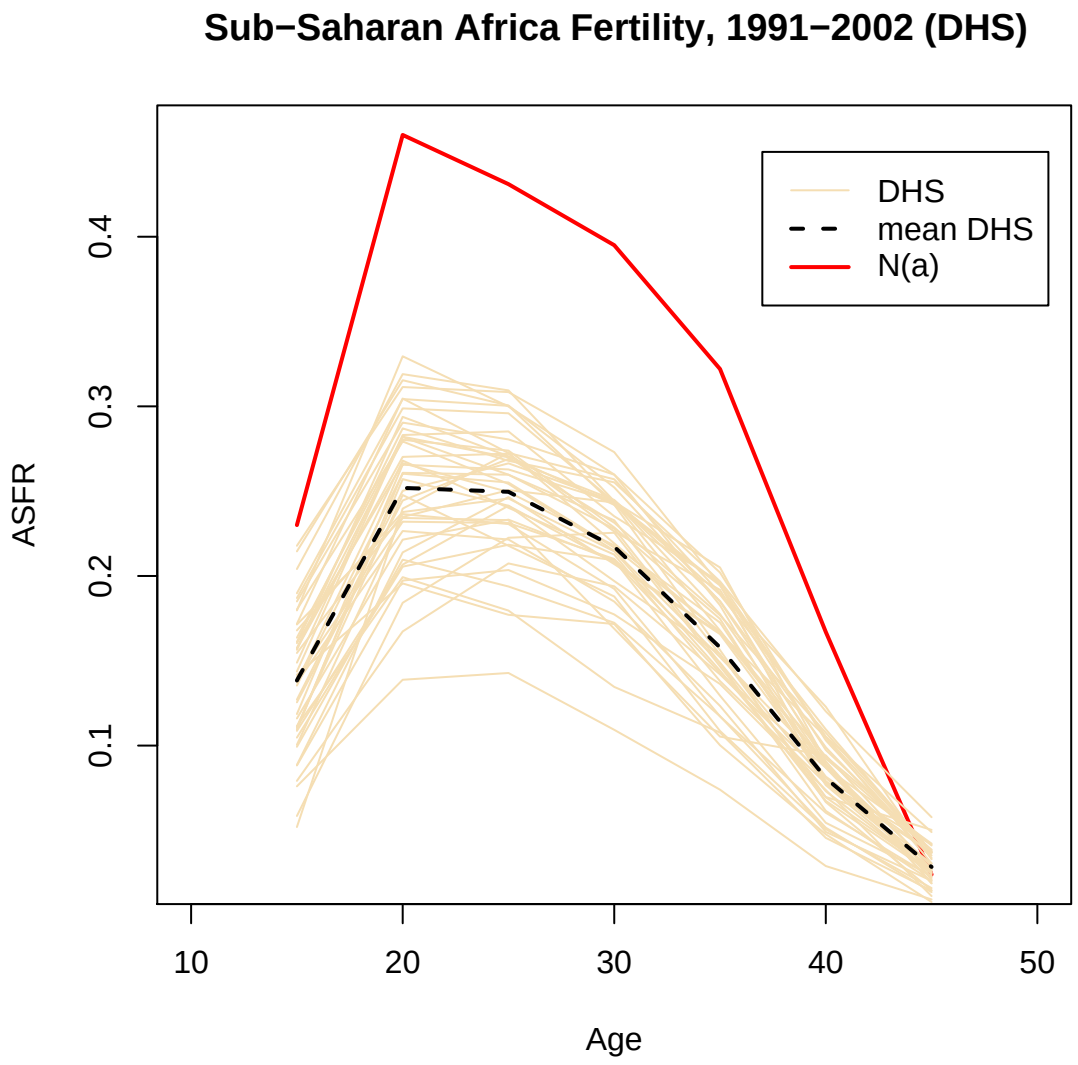

Figure 1: Age-specific fertility rates for 43 DHS surveys in Sub-Saharan Africa, 1991-2002. The synthetic natural fertility schedule of Henry (1961) and Coale & Trussell (1974, 1978) is indicated in red.

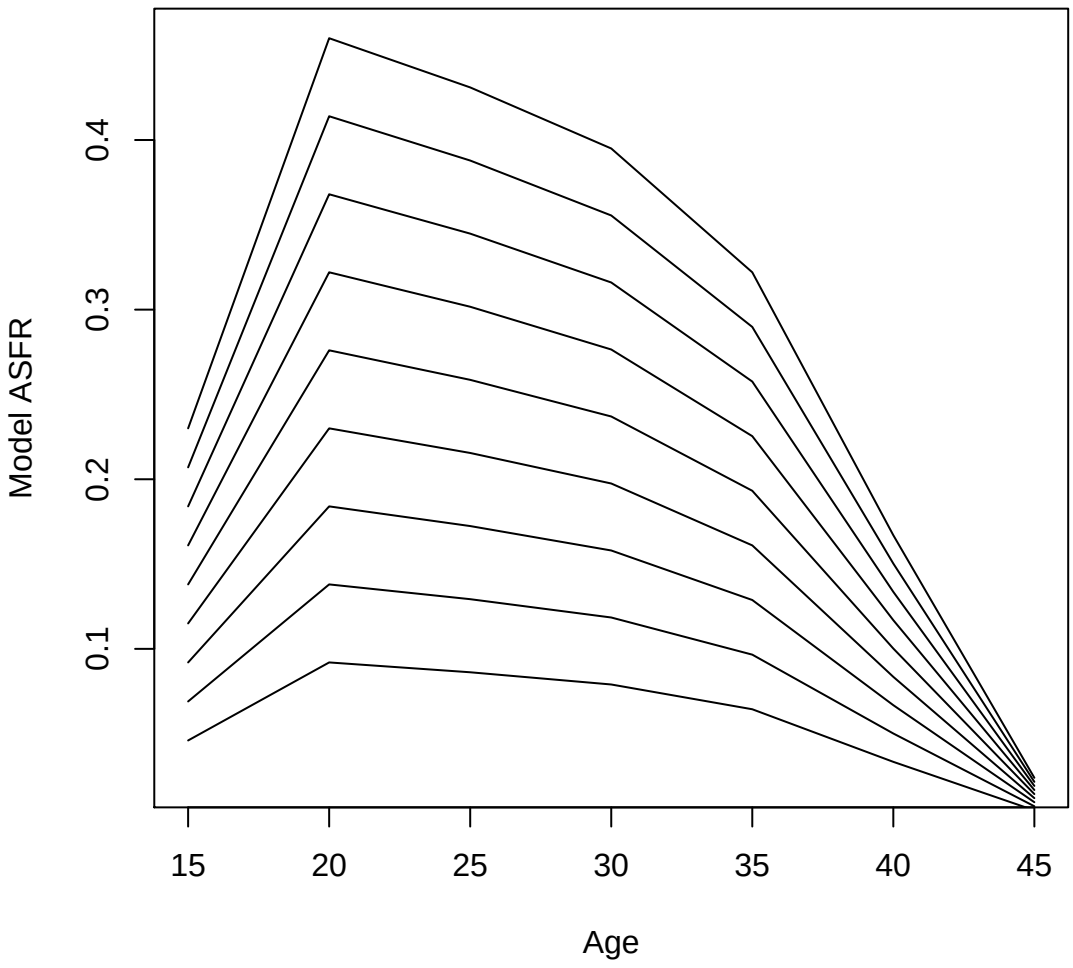

Figure 2: Model Age-specific fertility rates for nine different levels of natural fertility (no parity-specific control).

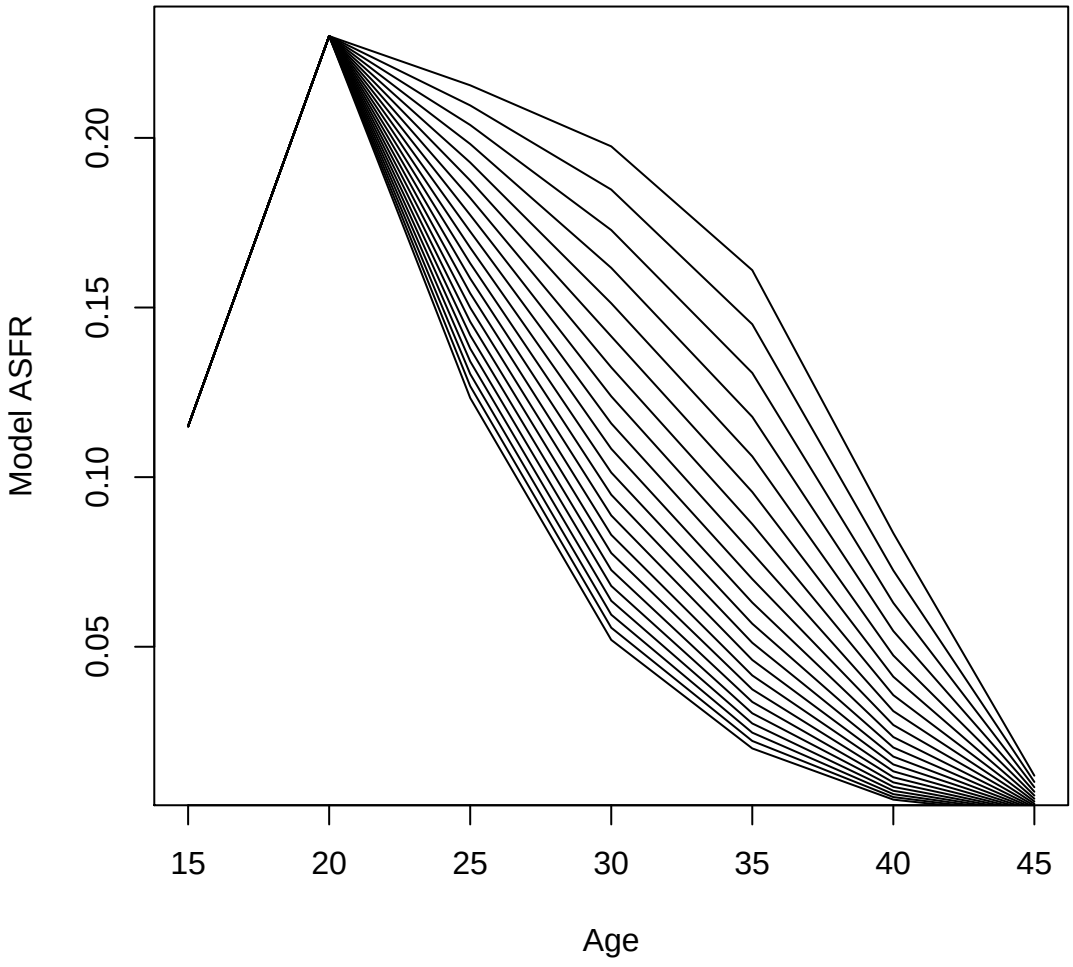

Figure 3: Model Age-specific fertility rates for twenty different patterns of deviation from natural fertility at one overall fertility level, $M = 0.5$.

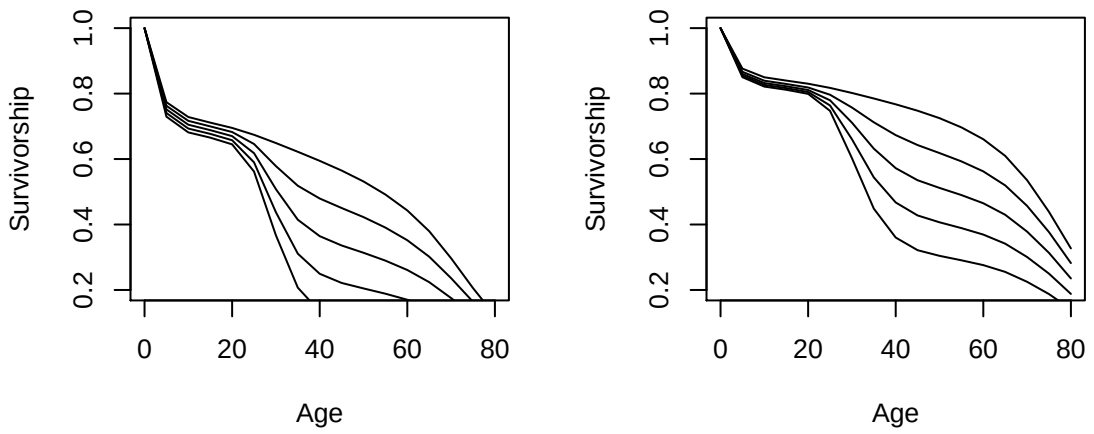

Figure 4: AIDS decremented model survivorship schedules. Left panel: INDEPTH $\mathring{e}_0$= 45 AIDS-decremented model life tables with decrements, 0, 5, 10, 15, and 20 years from $\mathring{e}_0$. Right panel: INDEPTH $\mathring{e}_0$= 60 with same AIDS mortality decrements

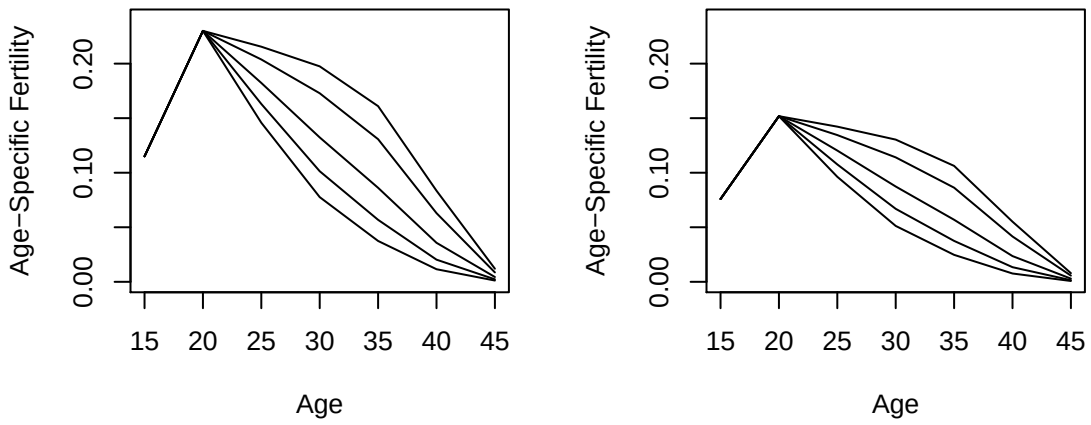

Figure 5: Model age-specific fertility rates used. Left panel: Coale-Trussell model fertility schedule with $M = 0.5$ with parity-specific control $m = (0, 0.2, 0.6, 1.0, 1.4)$. Right panel: $M = 0.33$, same values of m

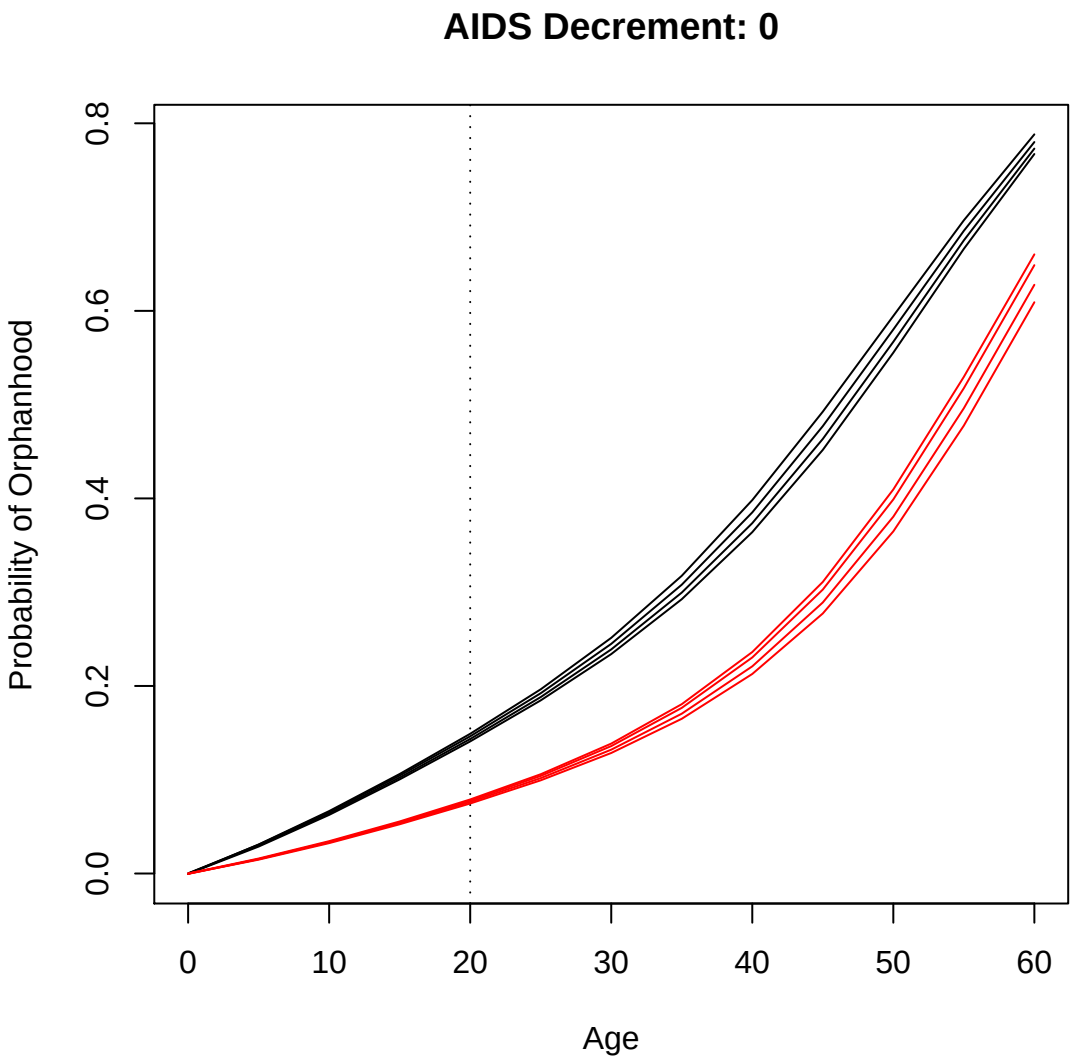

Figure 6: Orphanhood probabilities for baseline mortality, $\mathring{e}_0 = 45$.

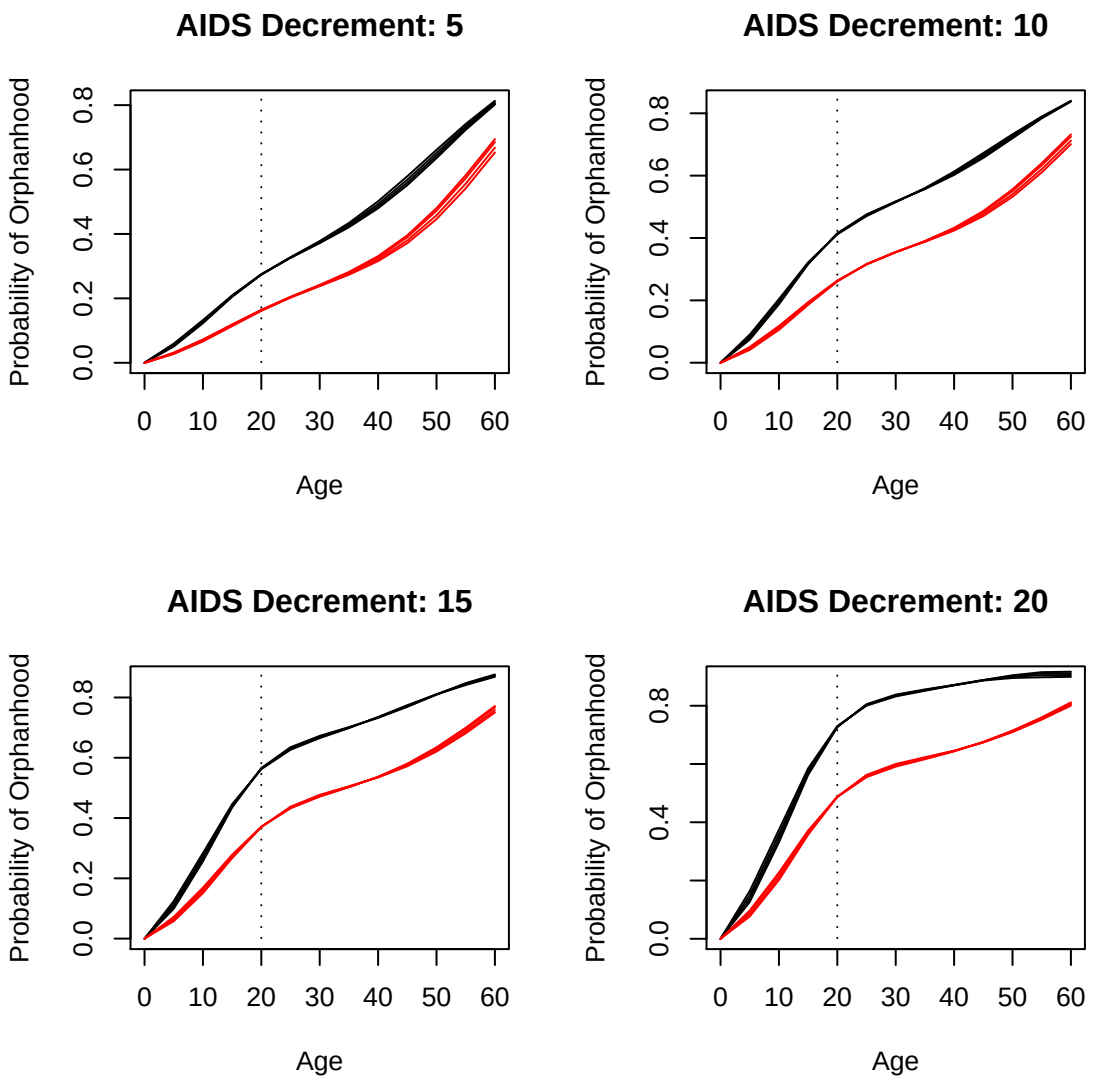

Figure 7: Orphanhood probabilities for AIDS mortality decrements, of 5, 10, 15 and 20 years from $\mathring{e}_0 = 45$ and fertility schedules with level $M = 0.5$.

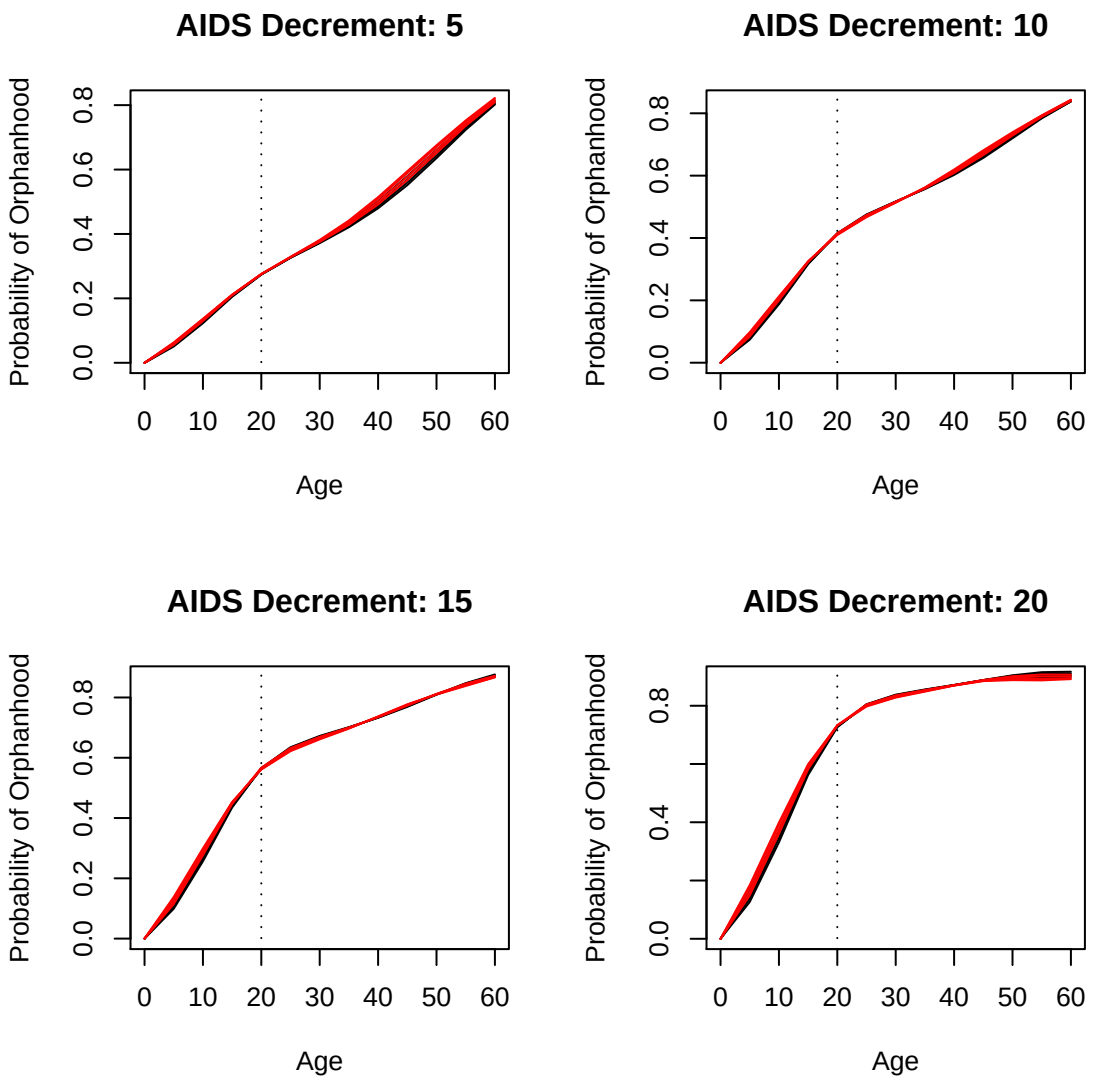

Figure 8: Orphanhood probabilities for AIDS mortality decrements, of 5, 10, 15 and 20 years from $\mathring{e}_0 = 45$ and fertility schedules with level $M = 0.33$.

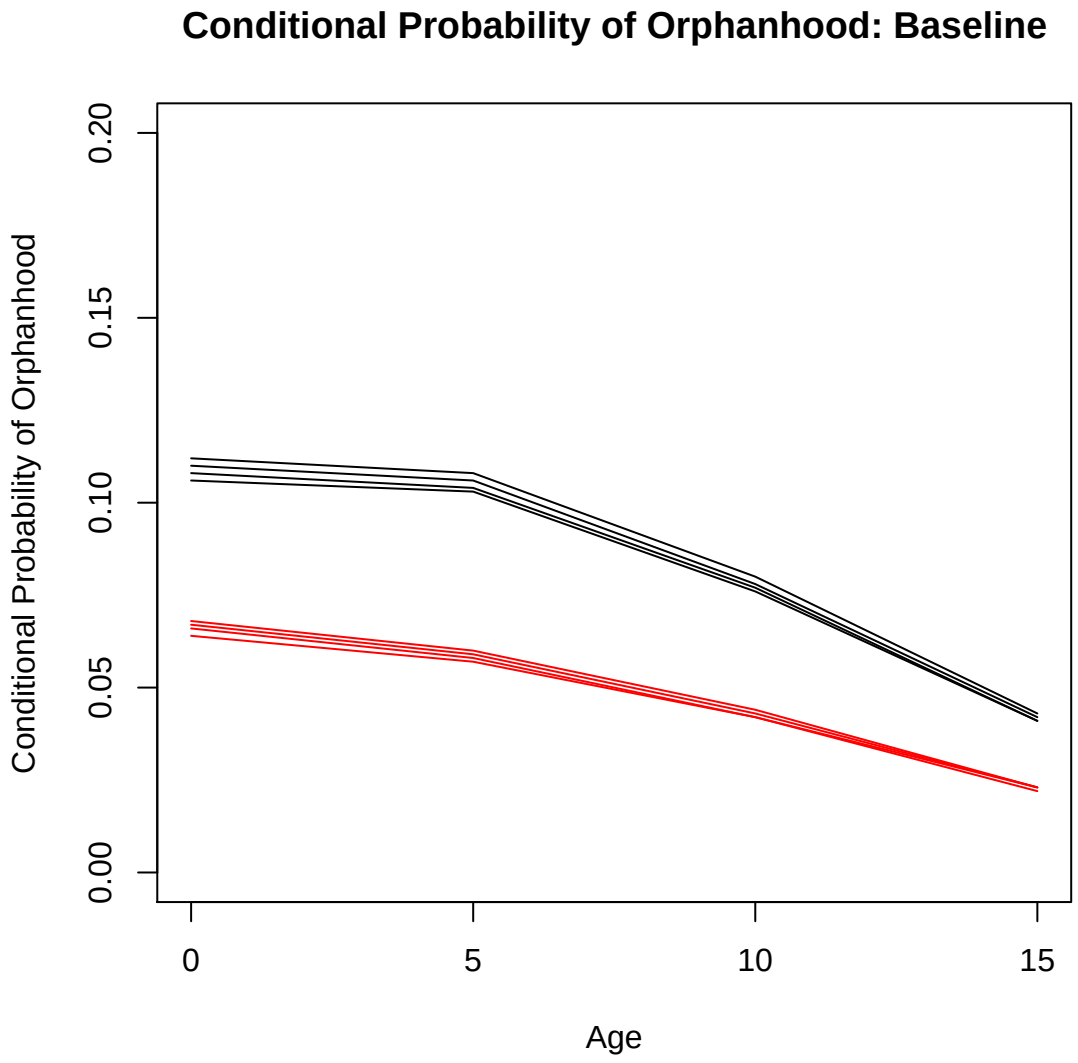

Figure 9: Conditional orphanhood probabilities for baseline mortality, $\mathring{e}_0 = 45$ (upper curves) and $\mathring{e}_0 = 60$ (lower curves).

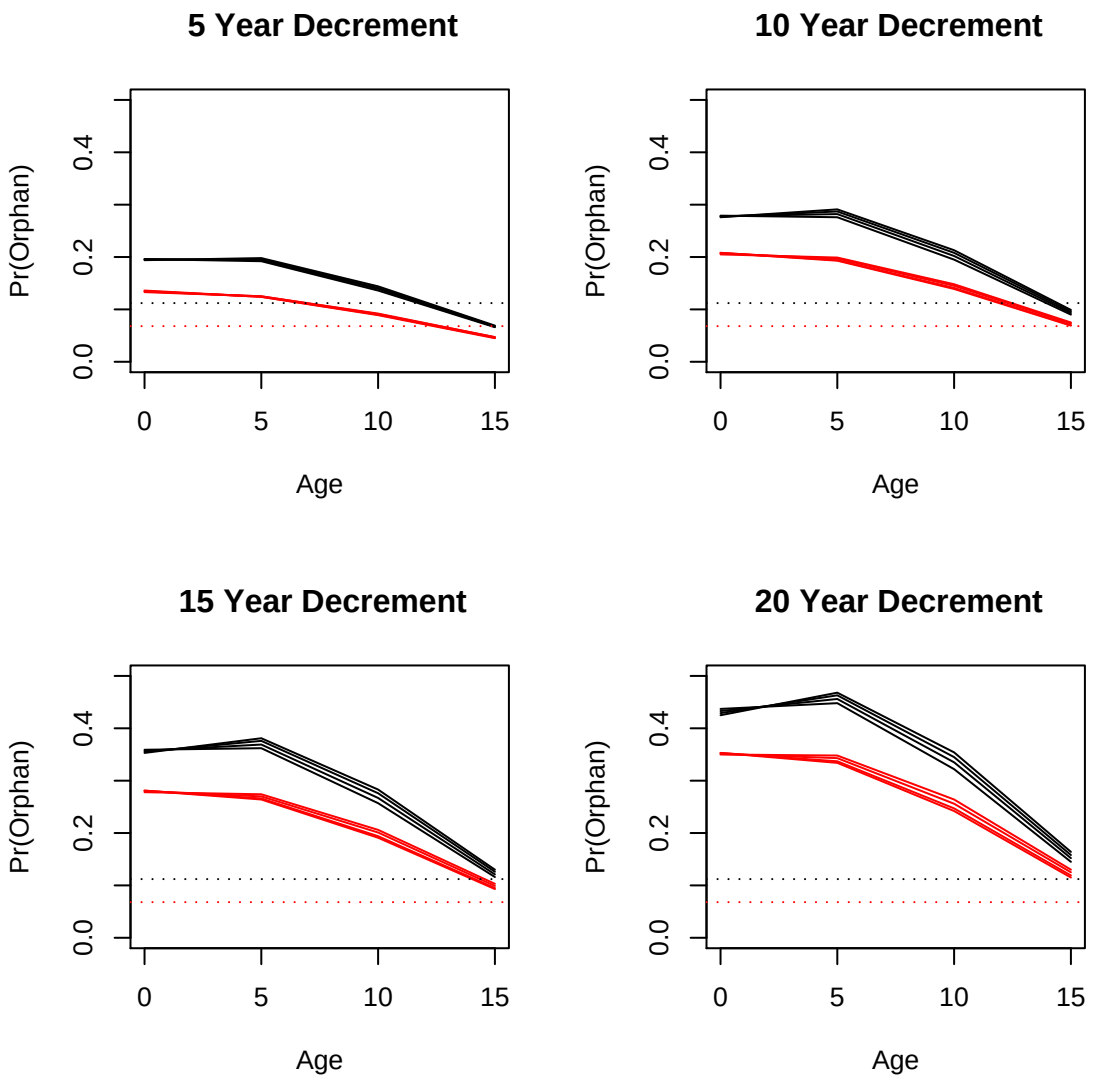

Figure 10: Conditional orphanhood probabilities for AIDS mortality decrements, of 5, 10, 15 and 20 years from $\mathring{e}_0 = 45$ (upper curves) and $\mathring{e}_0 = 60$ (lower curves).

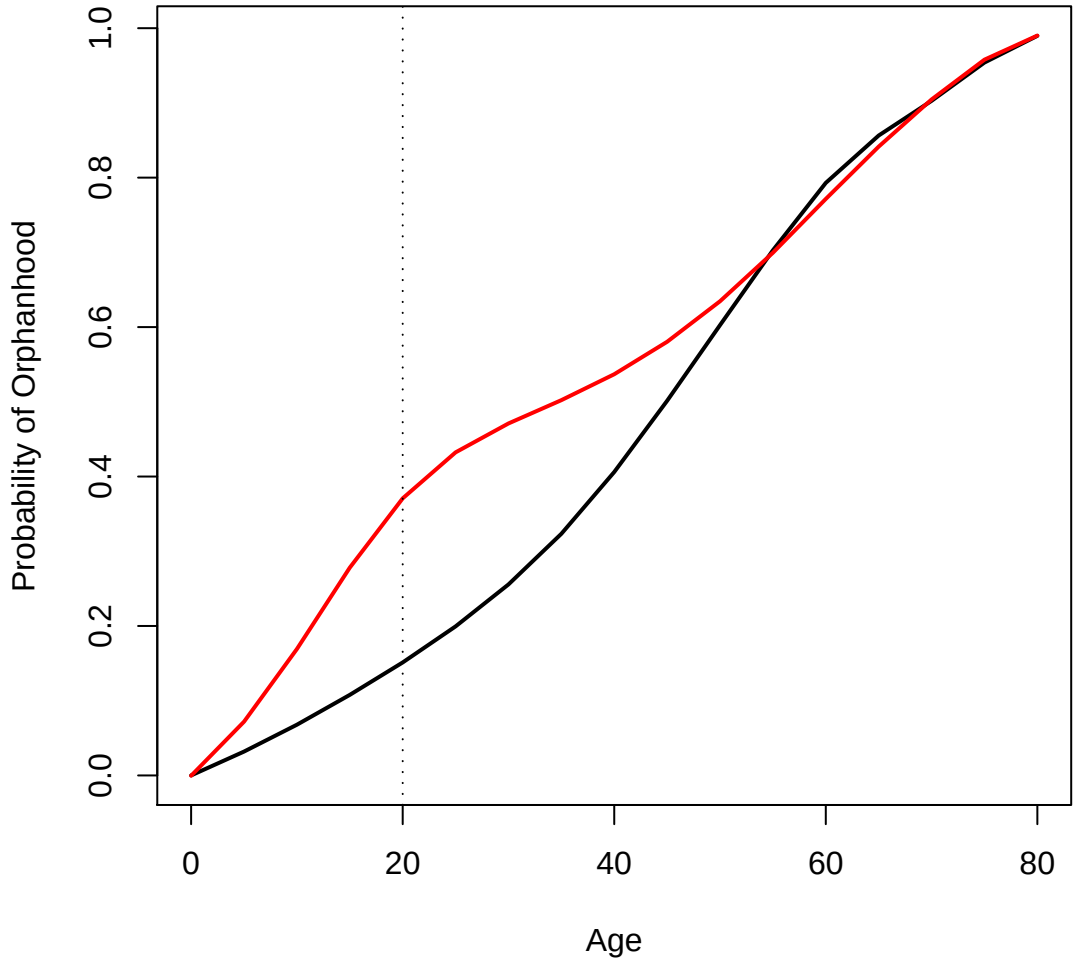

Figure 11: Impact of AIDS mortality on the unconditional probability of orphanhood. Both curves represent probabilities for a $\mathring{e}_0 = 45$. Solid curve: $\mathring{e}_0 = 45$ family model life table and no AIDS decrement, $\mathring{e}_0 = 60$ model life table with 15-year AIDS mortality decrement.

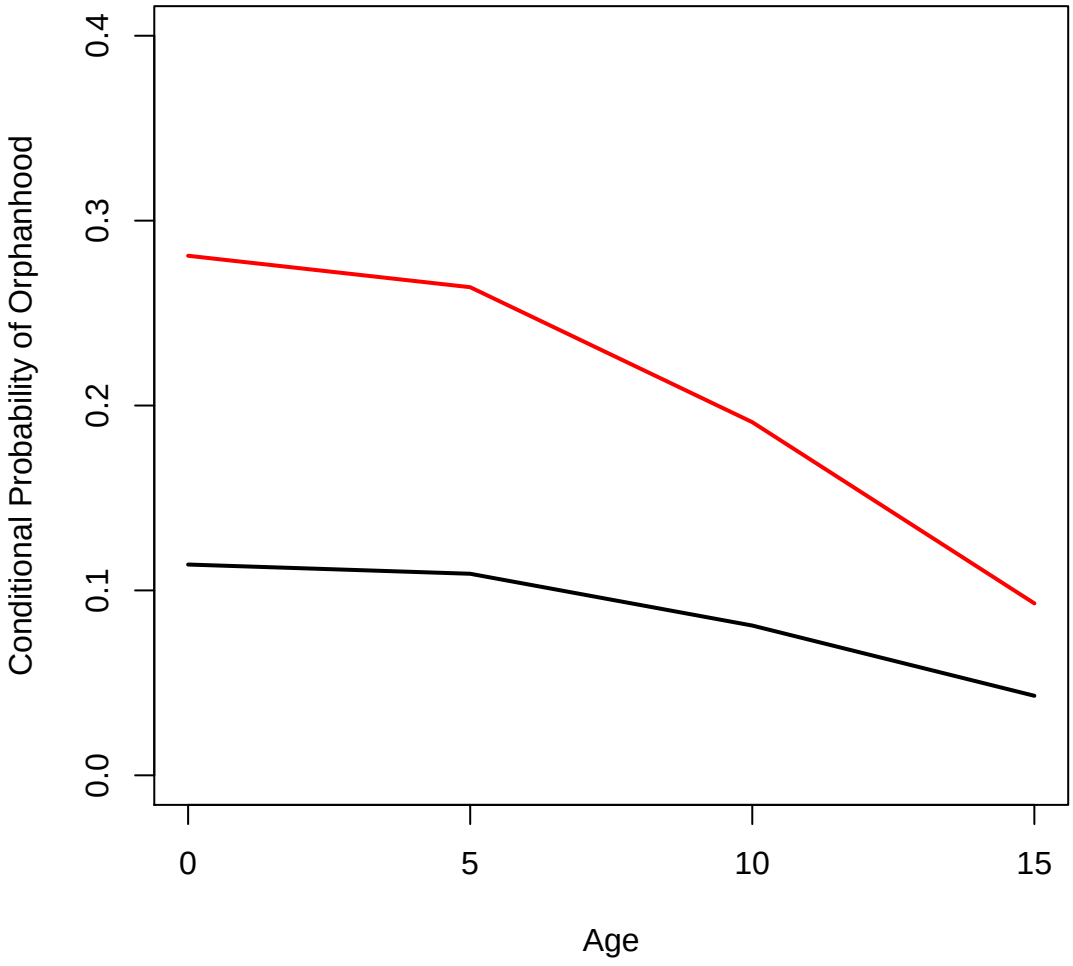

Figure 12: Impact of AIDS mortality on the probability of orphanhood conditioned on surviving to a. Both curves represent probabilities for a $\mathring{e}_0$= 45. Solid curve: $\mathring{e}_0$= 45 family model life table and no AIDS decrement, $\mathring{e}_0$= 60 model life table with 15-year AIDS mortality decrement.