Orphans and "Grandorphans" in Sub-Saharan Africa: The Consequences of Dependent Mortality

James Holland Jones* †
Center for Studies in Demography and Ecology
Center for Statistics and the Social Sciences
Center for AIDS and Sexually Transmitted Diseases

Martina Morris
Center for Studies in Demography and Ecology
Department of Sociology
Center for AIDS and Sexually Transmitted Diseases
University of Washington, Seattle, WA

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Abstract

Because AIDS is a sexually-transmitted disease (STD), most prevalent in the prime childbearing years, and because the epidemic has become generalized throughout Sub-Saharan Africa, it is generally recognized that many orphans will be generated by the AIDS epidemic. The outcome of children orphaned by AIDS need not be negative as research on fostering by extended family has shown. However, such positive outcomes for children orphaned by AIDS are largely predicated on the existence of extended family, and under a generalized epidemic, this very family structure is likely to be strained. Because HIV can be transmitted sexually, the infection of one parent is likely to be followed by the subsequent infection of the second parent, producing mortality correlations within households. In this paper, we present a stylized demographic model which incorporates parental AIDS mortality correlations. This model allows us to explore the effects of correlated mortality on the structure of the orphaned pool of children. As would be expected, within-household correlations increase the fraction of double orphans, however, they also induce structural changes on households across generations that have profound implications on the care of subsequent generations of children at risk for orphaning. Children born into new households formed by double-orphans lack grandparents, making them vulnerable to abandonment should their parents contract HIV.

*Corresponding Author: Center for Statistics and the Social Sciences, Box 354320, Seattle, WA 98195-4320, email: jameshj@stat.washington.edu, Phone: 206-221-6874, Fax: 206-221-6873

†Address as of 1 July 2003: Department of Anthropological Sciences, Stanford University, Building 360, Stanford, CA 94305-2117, email: jhj1@stanford.edu

1 Introduction

UNAIDS estimates that the generalized AIDS epidemic in Africa has led to 28.1 million adults and children living with HIV/AIDS. An estimated 2.4 million adults died from AIDS in 2000. AIDS is rapidly emerging as the leading cause of death throughout Sub-Saharan Africa (Gregson et al., 1994b). In the hardest hit countries of central, eastern and southern Africa AIDS accounts for anywhere from 50-80% of adult mortality (Mulder et al., 1994a,b; Sewankambo et al., 2000; Urassa et al., 2001) and has increased adult mortality rates from 2-3 times those of prior to the epidemic (Timæus, 1998; Boerma et al., 1998). The generalized epidemic is predicted to generate many orphans (Gregson et al., 1994a; Hunter and Williamson, 1997), creating both a humanitarian tragedy and tremendous social and economic instability.

Mortality due to infectious disease is likely to show correlations at different spatial and social scales (Becker and Dietz, 1995; Becker and Starczak, 1997). Indeed, it is these very correlations which allow for the science of epidemiology (Snow, 1860). Correlations within households are likely to be particularly strong for sexually-transmitted agents such as HIV, as the seroconversion of one parent is almost certain to lead to the infection of the second. This within-household correlation has been largely ignored in analyses of the social consequences of the generalized AIDS epidemic in Sub-Saharan Africa. In particular, the fraction of double orphans will be much higher than expected under a model of independent mortality. Foster (2002) notes that the number of children in Sub-Saharan Africa who will have lost both parents is projected to rise 1600 per cent between 1990 and 2010. Mortality data from the 2000-2001 Uganda DHS (Uganda Bureau of Statistics, 2001) indicate that 3% of children in the sample are maternal orphans and that 8% of the children are paternal orphans. Assuming independent mortality, then the expected fraction of double orphans should be 0.24%. However, DHS data indicate that 3% of the children in the sample are double orphans, an increase of 12.5-fold.

Orphanhood is an important issue for the future of Sub-Saharan Africa. 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). Not only does the magnitude of the problem define a humanitarian crisis, but it 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 less likely to be enrolled in school, have lower age at sexual debut, have greater morbidity, have greater delinquency rates, greater illiteracy and unemployment when adults (Foster and Germann, 2002). In addition, AIDS mortality among parents frequently leads to the dissolution of households, fostering social and geographic instability. Urassa et al. (2001), for example, found that 44% of households in which the head had died of AIDS dissolved.

While the life of children orphaned by AIDS is never likely to be easy, there are social arrangements which can substantially alleviate the negative social outcomes associated with orphanhood. In particular, when orphaned children are taken into the homes of both lineal and collateral kin, their outcomes can often be much more like children with living parents (Foster et al., 1995; Kamali et al., 1996; Urassa et al., 1997). Kin based fostering is generally considered the optimal living arrangement for children who have lost both parents Foster et al. (1995); Foster (2002); Foster and Germann (2002).

Irrespective of the generalized AIDS epidemic, orphaning and foster care are both common-

place in Sub-Saharan Africa. In a household survey conducted in Tanzania, Urassa et al. (1997) found that 40% of the households contained children who were neither natal nor orphaned. Hunter et al. (1997) report that 23% of all households in Tanzania contain fostered children and that 34% of the children in Tanzania may be fostered. The historically high frequency of foster-age arises from at least three factors common throughout the developing world, but particularly acute in Sub-Saharan Africa:

1. High adult mortality rates.
2. High total fertility.
3. Hypergynous marriage practices and polygyny.

As a result of these factors, social institutions designed to provide foster care for orphaned children exist throughout Sub-Saharan Africa. Such institutions include descent and inheritance systems which provide for children who have lost parents (e.g., the avunculate, in which mothers' brothers have economic and social obligations toward their sisters' children), land tenure systems which keep kin in close geographic proximity to each other, as well as social norms that encourage flexible household structure. These institutions can be particularly elaborate in cultures where the expected age difference between husbands and wives is high or where extensive polygyny is practiced. As is the case for people throughout the world and throughout time, the primary caretakers for orphaned children throughout Sub-Saharan Africa are either lineal or collateral kin (Silk, 1990).

A second important consequence of correlated mortality is the creation of a vicious cycle of orphanhood vulnerability. Throughout eastern and southern Africa, orphaned children are frequently fostered in the households of their grandparents (Foster et al., 1995). Children who have lost both parents as well as their grandparents therefore define a class of particularly vulnerable orphan. Households formed by adults who were themselves double orphans are households with no grandparents to look after children should the parents die.

What fraction of children orphaned by AIDS get fostered by grandparents varies with region, ethnic group, and the household status of the orphaned child. While no double orphans were under the care of grandparents in a study in northern Uganda (Ntozi et al., 1999), 70% of AIDS orphans in peri-urban Mutare, Zimbabwe were fostered by grandparents (Foster et al., 1995), and 54% were in rural Tanzania (Urassa et al., 1997).

In a study undertaken in Kinshasa, Ryder et al. (1994) found that children under foster care by kin were indistinguishable from natal children economically, physically, and socially. Castle (1996) found that the anthropometric status of fostered children in rural Mali was related to the circumstances of being fostered, rather than by fostering *per se*.

The question now is whether these different social systems, however well adapted historically for the care of orphaned children, have the capacity to absorb the large number of additional children orphaned by the AIDS epidemic. The generalized nature of the epidemic, and the fact that it tends to strike down segments of the population in their prime for both reproductive and economic productivity, will place a severe strain on the care networks traditionally afforded by children's kindreds (Rutayuga, 1992; Drew et al., 1998; Foster, 2002).

Here we will explore one aspect of potential kindred-based care networks. Specifically, we will present a highly simplified model to assess the impact of correlated AIDS mortality on the fraction of children who have lost both their parents and grandparents in a unilineal descent system.

1.1 A Note on Terminology

The terminology associated with orphan studies can be confusing. Accordingly, we provide the following definitions. We consider "children" to be all children and adolescents prior to age at marriage (or age at first birth). While it is often conventional to define an orphan as any person under the age of 15 to have lost a parent, we believe that the potential negative social consequences of parental loss between the ages of 15 and 18 to be substantial enough to warrant consideration. Furthermore, defining children as those who have not yet married simplifies the model specification, and means fewer parameters are required to estimate. An "orphan" is a dependent child who has lost one or both parents, and is therefore the most inclusive of the categories. A "double-orphan" is a dependent child who has lost both parents. A "grand-orphan" is a child who has lost both parents and one set of grandparents (see section 2.2 below).

2 Methods

2.1 Data

Our analysis starts with a Ugandan life table. We used a life table derived from the 1995 DHS Uganda. This life table is taken from Lopez et al. (2000) and was calculated in Timæus (1998). The life table is graduated using standard DHS methods (Henry, 1960; Preston et al., 2001). The life table is presented in table 1.

Fertility schedules for Uganda were taken from the national average calculated DHS-III Uganda (Uganda Bureau of Statistics, 1995). The TFR for the Ugandan DHS sample in 1995 was 6.6.

2.2 The Model

We chose to model the population as a stage-classified projection matrix in which parental status and life cycle stage were the i-state variables (Caswell, 2000). The two life-cycle stages modeled were child and parent. Child stages were indexed by the status of parents and grandparents, whereas parental stages were indexed by the status of parents. We assumed a unilineal kin-reckoning system, and therefore only keep track of one set of grandparents (e.g., paternal). Figure 1 presents the life cycle diagram corresponding to the projection matrix.

The stage-based model of figure 1 represents a system of linear difference equations describing the dynamics of the structured population. The stage-based linear difference equation model has a number of distinct advantages. These include:

1. High interpretability of the model outputs (e.g., annual rate of increase, stage structure of the population).

2. A powerful analytic toolkit for exploring model implications and calculating probabilities of events, etc.

3. The models relate to classical formal demography. In particular, the predictions of the stage composition of the model can be converted to age structure, which is relatively easy to check empirically.

4. The relatively low demand on parameter estimates is a good match to data availability.

5. All the equations are linear allowing the analytic calculation of many quantities of interest.

In effect, the life-cycle model that we have employed represents a stylized multi-state life table (Schoen, 1975; Schoen and Land, 1979), with constant mortality and transition probabilities within life-cycle stages.

The projection interval of the model is five years. Since the major phenomenon that we are exploring in this paper is the impact of correlated mortality on orphanhood, this interval affords for a substantial fraction of the within-household sero-conversion and ultimate mortality to happen within one project interval. In Sub-Saharan Africa, the time from infection with HIV to death from AIDS is somewhat shorter than the developed world, and all-cause mortality of HIV seropositive people is higher than in the developed world (Morgan et al., 1997) In a rural cohort study in Uganda, Morgan et al. (1997) found that the five-year cumulative mortality for incident cases was 17% and 54% for prevalent cases. Okongo et al. (1998) found that the six-year mortality of incident and prevalent cases was 9% and 54% respectively.

The arcs of the life cycle graph presented in figure 1 fall into four general classes:

1. Within-stage survival (self-loops)

2. Transitions between (grand-) parental status.

3. Recruitment into adult (i.e., breeding) population.

4. Births of new children

Stage-specific survival probabilities were estimated as the nth root of the value $l_i - l_f$, where l_i is the value of the survivorship schedule at the start of the class, l_f is the value at the expected end, and n is the expected number of years in the stage. Annual fertility rates are taken from DHS III, Uganda, and represent the TFR for Ugandan women divided by the length of the adult period.

The probability of a child moving from child class C_{ij} to C_{kl} was a function of two mortality rates. The transition from state $i \rightarrow k$ is simply the sum of the baseline parental death rate μ_p and the parental AIDS death rate μ_a, while the transition from state $j \rightarrow l$ is the grandparental death rate μ_g. Following Timæus (1998), we added AIDS mortality to the baseline in increments of two- and three-fold.

There are few data on mortality correlations within couples, so we varied these from 0-1. Correlated mortality[1] entered the model in the transitions from single orphanhood to double orphanhood. The transition probability can be conceptualized as

$$(\mu_p + \mu_a) + \rho\mu_a. \tag{1}$$

However, this particular parameterization creates a potentially serious problem with the model. Should the correlated mortality within a household be modeled as extra mortality, or is it simply a reflection of the underlying population mortality schedule? The question amounts to what fraction of HIV infections occur outside of household-defining partnerships? To account for this potential problem with the model specification, we included an additional parameter, π, which reflects the fraction of cases that are "new" in the sense of coming from outside the household system being modeled. The final parameterization for the correlated mortality thus becomes: $\pi\mu_a + (1-\pi)\rho\mu_a$.

The difficulty with employing an additional parameter is estimating the appropriate value to include. We used serodiscordance rates to estimate π. In a cohort study of couples in Zambia, Trask et al. (2002) found, using molecular epidemiological techniques, that 87% of observed transmissions of HIV could be confidently assigned to the partnership. We thus employ an initial value of $\pi = 0.13$.

Children could occupy one of nine states before maturing into parents or dying (3 parental states $\times$ 3 grandparental states). Stages 4-6 were the single orphan states, while stages 7-9 were double orphan states. Stage 9 represents children missing both parents and grandparents (i.e., grand-orphans). Transitions to parenthood occurred as the inverse of the mean age of first reproduction, α^{-1}. Table 2 lists the parameters defining the model and their values.

From the projection matrix, we calculated standard demographic quantities such as the annual rate of increase, $\lambda = e^r$ (where r is the Euler-Lotka intrinsic rate of increase), and the stable stage distribution, $\mathbf{w}$, the dominant eigenvalue and right eigenvector of the projection matrix respectively. The stable stage distribution was scaled so that $\sum_i w_i = 1$, and from this scaled distribution, we calculated the fraction of orphans in different classes. The fraction of orphans was calculated relative to the total pool of children (i.e., the sum of stages 1-9).

2.3 Markov Model

By converting the demographic projection matrix specified by the life-cycle diagram of figure 1 into a discrete-state Markov model (see e.g., Caswell, 2000), we were able to calculate the probability of becoming a double- or grand-orphan conditional on surviving. The details of the calculation are presented in the Appendix.

[1] We should note that the mortality phenomenon we envision here is not strictly a correlation, rather it is the probability that the partner of an individual who dies of AIDS will also die within the projection interval. As a probability, $1 \geq \rho \geq 0$. This probability of AIDS mortality in excess of the baseline will generate mortality correlations at the population level.

3 Results

For this analysis, we are primarily interested in the double orphans and, particularly the grand-orphans. The results therefore focus on the fraction of the total number of children (i.e., classes 1-9) in the final three stages, 7-9. Stages 7-9 represent the double orphans in the life cycle graph of figure 1 and stage 9 represents the grand-orphans.

3.1 Model Output

We converted the stable stage to the stable age distribution, and plotted the age pyramid of the model with no correlation in AIDS mortality in figure 2. The model output is plotted with the observed age structure of households from the 1995 DHS Uganda. The model clearly has relatively too many older individuals and too few very young. These shortcomings are, of course, related since the age distribution is normed. The excess of old in the model population is due to the constant within-stage mortality assumption. Length of life is thus exponentially distributed, with a very long right tail. Despite these weaknesses, the model output is qualitatively quite similar to the observed age structure.

3.2 Fraction of Orphans

With independent mortality, and a fraction of new cases set to $\pi = 0.13$, the fraction of orphans (relative to all children) is 6.06%, while the fraction of double orphans is 0.37% and the fraction of grand-orphans is 0.02%. The fraction of orphans is clearly somewhat low relative to the Uganda Bureau of Statistics (2001) data, and the fraction double orphans is extremely low. However, the mortality schedule that we used dates from 1995 demographic data, so it should not be too surprising that there are differences.

Validating this figure with pre-AIDS era orphan numbers is a difficult task, both because of the general lack of such detailed data, but also because of the ubiquitous fosterage in Sub-Saharan Africa (Urassa et al., 1997; Goody, 1982). Data gathered for the 1991 Tanzania DHS compiled in Hunter et al. (1997) for eight study regions averaged 5.31% of children orphaned, a figure more in the range of the estimate from our model. The total number of orphans from the 1995 Ugandan DHS is 8.4%. There are six DHS country surveys from southern and eastern Sub-Saharan Africa in the early 1990s that include information on orphans: Rwanda, Malawi, Zimbabwe, Tanzania, Uganda, and Zambia. The mean total number of orphans for these surveys is 5.98%. The mortality figures from the DHS country surveys of the early nineties, of course, include a substantial contribution from AIDS, but it is before AIDS became the leading cause of death in the countries of the African AIDS belt Gregson et al. (1994b).

The effect of mortality correlations on the fraction of orphans in the stable stage distribution is presented in tables 3 and 4. Using population attributable mortality estimates of Mulder et al. (1994b), we compared the effect of ρ under two mortality scenarios: (1) 50% of deaths attributable to AIDS, and (2) 75% of deaths attributable to AIDS. Overall, mortality correlations have the effect of substantially increasing the fraction of double- and grand-orphans. For $\rho = 0.5$, the increase in grand-orphans is nearly 70% under the low mortality scenario. Under the high mortality scenario the relative risk nearly doubles.

The relative fraction of grand-orphans increases more under correlated parental mortality than the other two double-orphan classes. This results from the fact that there are three routes for correlated mortality to produce grand-orphans. In the life-cycle model of figure 1, the general flow is from top to bottom and left to right, making naturally more routes for the creation of grand-orphans from the other orphan states.

3.3 Conditional Probability of Grand-Orphanhood

The conditional probabilities of grandorphanhood represent the probability, for living children, of entering stage 9 before recruitment into the adult pool. The baseline conditional probability of becoming a grand-orphan by starting stage is presented in figure 3. With a zero correlation in AIDS mortality, the conditional probability of becoming a grand-orphan varies between 0.0005 and 0.102. With AIDS mortality correlations, these probabilities increase. The relative risks (i.e., relative to the baseline of $\rho = 0$) of grand-orphanhood are presented in tables 5 and 6. Under the low AIDS mortality scenario of Mulder et al. (1994a), and with a probability of a second parental AIDS death within the projection interval of $\rho = 0.5$, the relative risk of grand-orphanhood is approximately 1.7 for all initial classes. Under the high mortality scenario, the risk is essentially doubled at $\rho = 0.5$.

3.4 Sensitivity

The relative risk of grandorphanhood increases approximately linearly with both ρ and π, and considerably faster with π than with ρ (figure 4). This result highlights the well-known feature of AIDS epidemiology which is confining sexual behavior to a more limited network (e.g., through reduced partner number or concurrency of partnerships) will have the effect of increasing the equilibrium fraction infected in the subnetwork, but decrease the overall fraction infected (Anderson and May, 1991; Morris, 1997).

Clearly, the adult mortality rate and the total fertility of the population will have an impact on the number of orphans generated under a generalized ADIS epidemic. To assess the impact of adult mortality and fertility, we varied the estimated parental AIDS mortality (μ_A) and the interval fertility (β). For this analysis, we held the parental AIDS mortality correlation constant at $\rho = 0.5$ and the fraction of new cases to $\pi = 0.13$.

The apparently paradoxical results are plotted in figure 5. Not surprisingly, the fraction of double-orphans increases with increasing AIDS mortality. However, the fraction of double orphans decreases with increased fertility. This result is easily explicable by the assumption we used that all children are born with both parents alive. Increasing the flow of children into the first row of the life cycle will naturally reduce the relative fraction of double orphans.

4 Discussion

Our model results demonstrate the potential importance of AIDS mortality correlations within households on the welfare of children in Sub-Saharan Africa. Correlated mortality has two important effects on the composition of AIDS orphans. First, it dramatically increases the fraction of all orphans who have lost both parents. Second, it has multi-generational effects,

ultimately reducing the fraction of children who could be fostered by grandparents should they become orphaned. These effects are magnified as the fraction of all deaths in the population due to AIDS increases.

It is important to note that the model we have presented has no pretense at realism or precision. Most notably, our results are predicated on constant demographic rates, and asymptotic measures of population structure. In his classic paper, Levins (1966) notes the inherent trade-off between realism, generality, and precision in modeling studies. Our model uses a generic formulation to illustrate the potential impact of a feature of the natural history of HIV on the demographic landscape. While we acknowledge the variety of ways that it is insufficient as a demographic model, it succeeds in a number of the goals of modeling exercises enumerated by Levins. Specifically, it has helped us to clarify our verbal argument, allowed us to highlight the critical assumptions that will be weaknesses in a variety of demographic approaches to the problem, and allowed us to explore a potentially biologically and socially significant variables (i.e., ρ, μ_A).

The fraction of grand-orphans under any combination of parameters is always quite small. However, given the extreme vulnerability of these children, we consider it of vital importance to account for their needs in any intervention program. Foster care of AIDS orphans is clearly best provided by kin (Foster, 2002). This point is particularly true given the long history of foster care, even of non-orphans, in Sub-Saharan Africa (Urassa et al., 1997). Hunter and Williamson (1997) also noted that the expense of western-style institutional care, coupled with the scale of the orphan problem in Sub-Saharan Africa mean that the care of children orphaned by the AIDS epidemic is best undertaken in the context of family and community.

Consequently planning for the care provision of the most vulnerable of AIDS orphans, children lacking close kin, becomes a central priority for intervention programs.

The model we have presented here does not address the issue of collateral kin. This is a weakness given the potential importance of parents' siblings for providing care to orphaned children. While the multi-state lifetable approach to orphan family dynamics could, in theory, be extended to other kin categories, we suspect that the ultimate utility of this approach will be limited by the paucity of data required to construct and validate such models.

The HIV epidemic is changing the demographic landscape of Sub-Saharan Africa. Clearly, there are substantial increases in mortality Timæus (1998). However, fertility is also affected by HIV/AIDS Zaba and Gregson (1998); Gregson et al. (1999). Fertility decline results from increased fetal loss of HIV-positive women, and possibly, reduced sexual activity of people sick with AIDS as well as changes endogenous fertility preferences (Baylies, 2000). These demographic changes, coupled with the poor vital registration infrastructure of Sub-Saharan Africa generally, make the prospects for calculating multi-state lifetables, which lack the unrealistic assumptions we have employed here, unlikely.

These observations apply equally to the various analytic methods for calculating the frequencies of specified kin or household composition (Goodman et al., 1974; Pullum, 1982; Bongaarts, 1987; Pullum and Wolf, 1991). The methods of Goodman and colleagues rely on strong stable population assumptions which clearly are not appropriate for Sub-Saharan Africa under a generalized AIDS epidemic. The branching process approach of Pullam, while not requiring a stable population, requires mortality to be low, making it inapplicable to the present case.

These observations suggest that the best approach to modeling the kinship resources of

children at-risk of orphanhood in Sub-Saharan Africa is through demographic microsimulation (Wachter, 1987). Simulation approaches to kin resource studies are fairly common in the context of planning for the care needs of the elderly in an aging, low-mortality population (Wachter, 1997; Suzuki, 2001), but such work has not yet been undertaken in the context of the high mortality, high fertility demographic regimes characteristic of Sub-Saharan Africa. This is an important avenue for further demographic research into the AIDS orphan problem in Sub-Saharan Africa.

Using the right eigenvectors of the projection matrix to calculate the fraction of different classes of orphans, of course, has limitations. Specifically, the values of the dominant eigenvector represent only the asymptotic stage structure. However, the projection matrix which defines our model has very high entropy (Tuljapurkar, 1993), indicating rapid mixing to the stable population structure.

The model, while tracking both parents is still essentially a one-sex model. There is the potential for problems here. A satisfactory solution to the two-sex problem is still wanting Reeves (1987). Again, we suspect that the best solution to this shortcoming lies not in further refinement of the analytical model, but in simulation.

In conclusion, we have demonstrated here, using a stylized demographic model, the potential importance of within-household AIDS mortality correlations on the structure of the orphan population in Sub-Saharan Africa. We hope that through these initial explorations we have begun to lay the foundations for further investigation into the dynamics of kindreds under a generalized AIDS epidemic, and their consequences for children's welfare.

Appendix: Markov Model

The dynamics described by demographic project on matrices of the Leslie-Lefkovitch type are formally equivalent to discrete-state Markov chains (Tuljapurkar, 1993). To calculate the probability of reaching the grand-orphan state (i.e., state 9) before maturity, and conditional on surviving, the 9×9, $\mathbf{A}'$, submatrix of the original projection matrix, $\mathbf{A}$, was converted into a discrete-time Markov transition matrix, $\mathbf{T}$, by first decomposing the projection matrix as $\mathbf{A}' = (\mathbf{P} + \mathbf{F})$, where $\mathbf{P}$ represents all the between-stage transitions and $\mathbf{F}$ represents the fertilities,[2] and then adding two absorbing states, death and grandorphan, to the matrix $\mathbf{T}$ in addition to the existing transient states (Caswell, 2000). The structure of the Markov transition matrix is given in equation 2:

$$\mathbf{T} = \left[\begin{array}{c|c} \mathbf{P} & \mathbf{0} \\ \hline \mathbf{Q} & \mathbf{I} \end{array}\right], \tag{2}$$

where $\mathbf{0}$ is a $k \times 2$ submatrix of zeros, $\mathbf{Q}$ is a $2 \times k$ submatrix containing the probabilities of mortality in the first row and the probabilities of transition to grandorphanhood in the second, and $\mathbf{I}$ is a 2×2 identity matrix.

From the Markov transition matrix $\mathbf{T}$, the expected conditional waiting time to absorption (i.e., the fundamental matrix $\mathbf{N}$ of the Markov matrix (Iosifescu, 1980)) can be calculated and the conditional probability of absorption in any particular absorbing state can be calculated as $\mathbf{M} = (\mathbf{T} - \mathbf{I})^{-1}$.

[2] which, because we are dealing only with the pre-adult submatrix, are all zero.

In addition to the full children's submatrix, it will also be of interest to use smaller submatrices to calculate, for example, the probability of losing *at least* one parent for children in the population governed by those particular demographic schedules.

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	${}_nM_x$	${}_nq_x$	l_x	$\mathring{e}_x$
0	0.083	0.079	1.000	42.410
1	0.021	0.081	0.921	45.010
5	0.006	0.031	0.847	44.820
10	0.004	0.020	0.821	41.150
15	0.007	0.034	0.805	36.950
20	0.014	0.067	0.777	33.170
25	0.022	0.105	0.725	30.370
30	0.026	0.123	0.649	28.640
35	0.024	0.112	0.569	27.290
40	0.022	0.106	0.506	25.420
45	0.020	0.095	0.452	23.130
50	0.020	0.094	0.409	20.290
55	0.024	0.115	0.371	17.130
60	0.033	0.154	0.328	14.030
65	0.051	0.226	0.278	11.130
70	0.079	0.328	0.215	8.670
75	0.116	0.443	0.144	6.730
80	0.163	0.566	0.080	5.220
85	0.248	1.000	0.035	4.000

Table 1: Uganda life table for women, 1995

parameter	description	value	notes
μ_p	parental mortality rate	variable	
μ_c	child mortality	0.0611	
μ_a	AIDS mortality	variable	
μ_g	grandparental mortality	0.0476	
ρ	mortality correlation	0-1	
π	fraction new cases	0-1	
β	fertility rate	1.25	
γ_a	parental probability	0.25	
γ_g	post-reproductive recruitment	0.25	

Table 2: Parameters used to construct stage-based projection matrix.

ρ	C7	C8	C9
0	1.000	1.000	1.000
0.1	1.008	1.137	1.138
0.2	1.016	1.273	1.276
0.3	1.024	1.409	1.413
0.4	1.032	1.545	1.551
0.5	1.040	1.680	1.688
0.6	1.048	1.816	1.826
0.7	1.056	1.950	1.963
0.8	1.064	2.085	2.101
0.9	1.072	2.219	2.238
1.0	1.080	2.353	2.375

Table 3: Relative risk of entering the three double orphan states under the low mortality scenario (i.e., 50% of deaths attributable to AIDS) as a function of the parental mortality correlation, ρ, using the value of $\pi = 0.13$.

ρ	C7	C8	C9
0	1.000	1.000	1.000
0.1	1.006	1.187	1.186
0.2	1.013	1.374	1.373
0.3	1.019	1.560	1.558
0.4	1.025	1.747	1.744
0.5	1.031	1.933	1.928
0.6	1.038	2.119	2.113
0.7	1.044	2.304	2.297
0.8	1.050	2.490	2.481
0.9	1.056	2.675	2.664
1.0	1.062	2.860	2.847

Table 4: Relative risk of entering the three double orphan states under the high AIDS mortality scenario (i.e., 75% of deaths attributable to AIDS) as a function of the parental mortality correlation, ρ, using the value of $\pi = 0.13$.

ρ	1	2	3	4	5	6
0	1.000	1.000	1.000	1.000	1.000	1.000
0.1	1.133	1.136	1.138	1.131	1.134	1.138
0.2	1.265	1.271	1.276	1.260	1.268	1.276
0.3	1.396	1.405	1.414	1.387	1.400	1.414
0.4	1.525	1.539	1.552	1.512	1.532	1.552
0.5	1.654	1.672	1.690	1.636	1.662	1.690
0.6	1.781	1.804	1.829	1.758	1.792	1.829
0.7	1.907	1.936	1.967	1.879	1.921	1.967
0.8	2.032	2.067	2.105	1.997	2.048	2.105
0.9	2.155	2.198	2.243	2.114	2.175	2.243
1.0	2.278	2.328	2.381	2.230	2.301	2.381

Table 5: Effect of parental AIDS mortality correlation, ρ, on the conditional probability of becoming a grand-orphan, low population attributable risk of AIDS mortality.

ρ	C1	C2	C3	C4	C5	C6
0	1.000	1.000	1.000	1.000	1.000	1.000
0.1	1.184	1.186	1.188	1.182	1.185	1.188
0.2	1.366	1.371	1.376	1.362	1.369	1.376
0.3	1.548	1.556	1.563	1.540	1.551	1.563
0.4	1.728	1.739	1.751	1.717	1.733	1.751
0.5	1.907	1.923	1.939	1.891	1.914	1.939
0.6	2.085	2.105	2.127	2.064	2.094	2.127
0.7	2.261	2.287	2.314	2.235	2.273	2.314
0.8	2.436	2.469	2.502	2.404	2.451	2.502
0.9	2.611	2.650	2.690	2.572	2.629	2.690
1.0	2.783	2.830	2.878	2.737	2.805	2.878

Table 6: Effect of parental AIDS mortality correlation, ρ, on the conditional probability of becoming a grand-orphan, high population attributable risk of AIDS mortality.

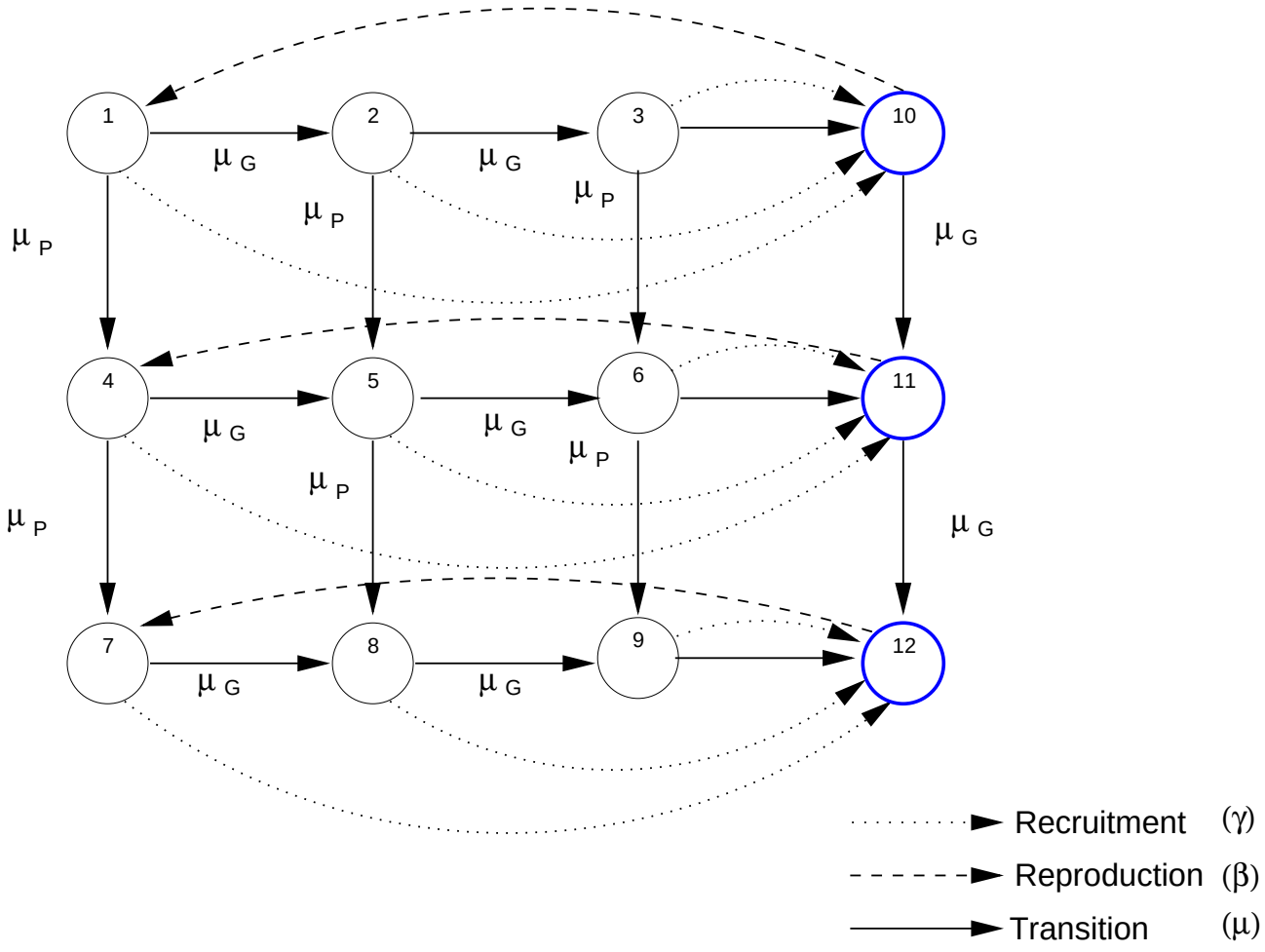

Figure 1: Life cycle diagram for the structured population model. Stages 1-9 represent children and stages 10-12 represent parents. Children lose grandparents moving to the right and lose parents going down the figure. Stages 7-9 are double orphans, and stage 9 represents grandorphans.

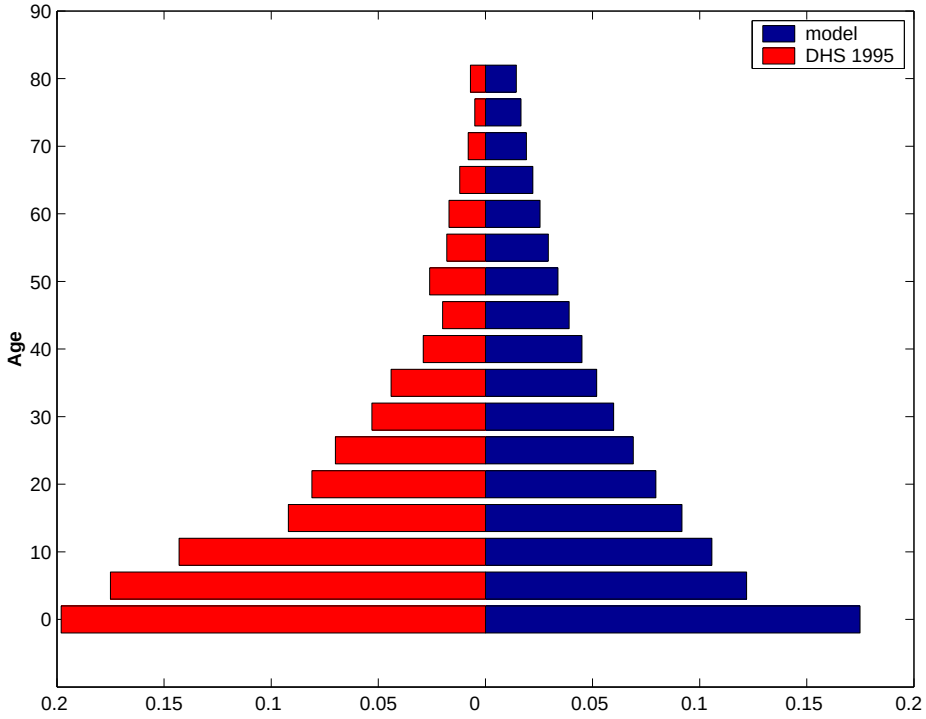

Figure 2: Comparison of age structure from the model with the household age structure of the 1995 DHS Uganda sample.

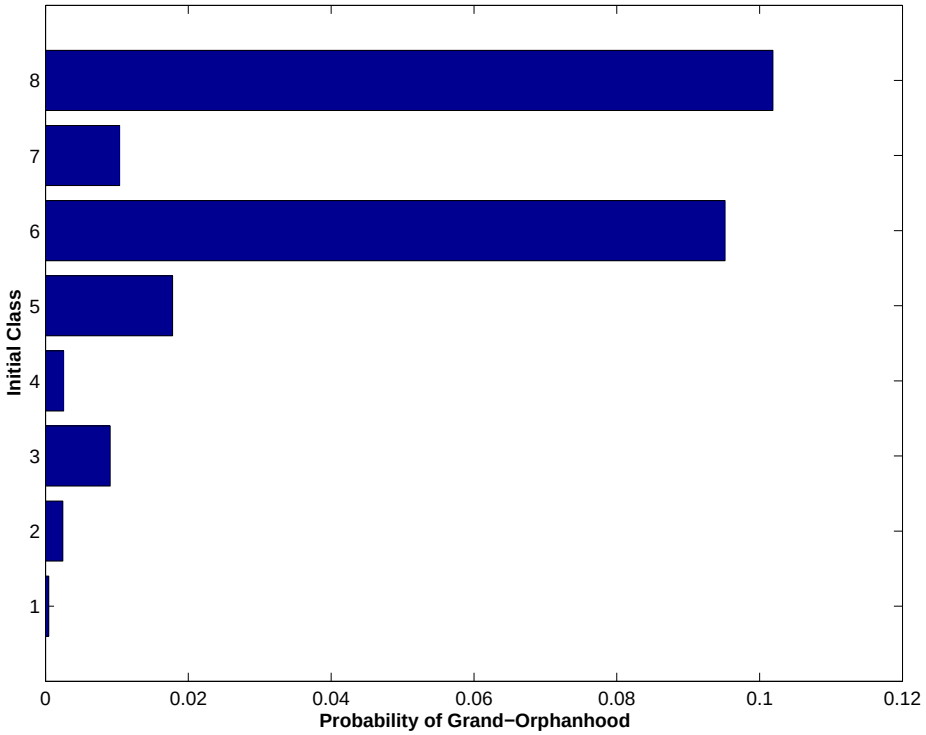

Figure 3: Baseline values of the conditional probability of becoming a grand-orphan by initial stage. This calculation assumes that a correlation of $\rho = 0$ in AIDS mortality.

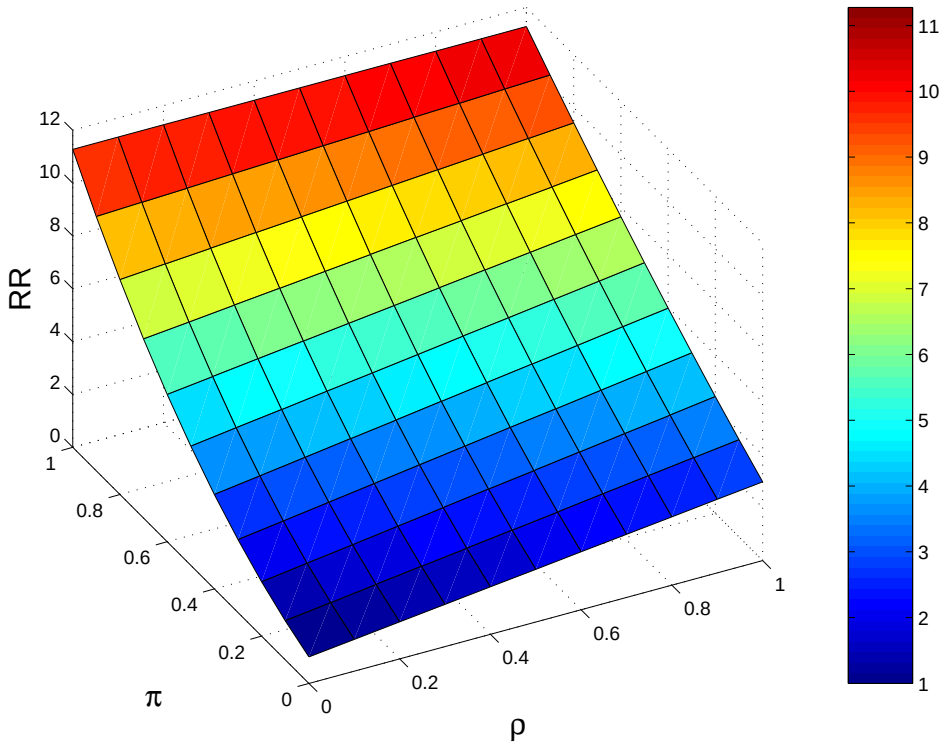

Figure 4: Surface plot of the relative risk of grand-orphanhood for combinations of the two parameters describing within-household correlations in AIDS mortality, ρ and π.

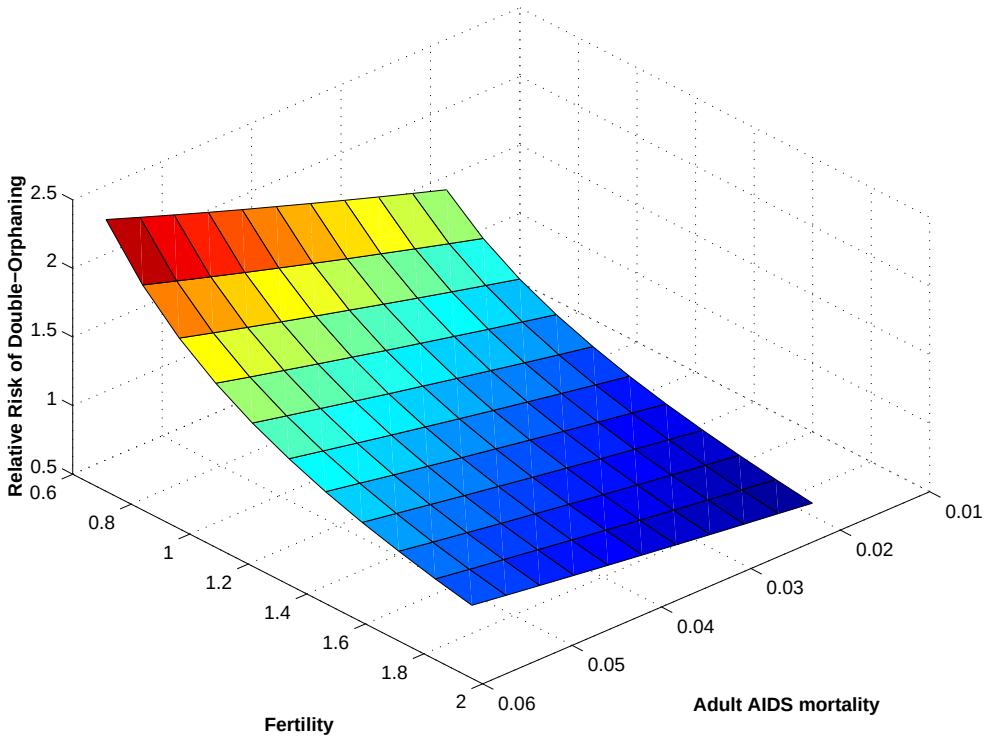

Figure 5: Surface plot showing the sensitivity of the fraction of double-orphans on parental AIDS mortality and fertility.