A Parametric Investigation of Mortality at All Ages in a Rural, South African Population

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1 Introduction

A multitude of research has reported on the global mortality impact of HIV/AIDS with a considerable portion focused on documenting and analyzing HIV/AIDS related mortality in southern Africa as a consequence of the tremendous impact the epidemic has had in that region. Throughout the 1990s and into the 21st century, southern Africa experienced increasing adult mortality, which researchers have linked to escalating HIV prevalence over this period (UNAIDS, 2002; Ngom and Clark, 2003; Blacker, 2004; Porter and Zaba, 2004; Hosegood et al., 2004; Groenewald et al., 2005). In this paper, we utilize a parametric model, the Heligman-Pollard law of mortality, estimated via a Bayesian Melding with Incremental Mixture Importance Sampling (Poole and Raftery, 2000; Raftery and Bao, 2009), which allows us to estimate uncertainty around parameter estimates, to assess rapidly changing mortality schedules for males and females in a rural population of South Africa. Despite having been used in a variety of other, mostly economically developed contexts, the Heligman-Pollard model has been under utilized in the analysis of mortality in high HIV-prevalence settings. The ability of parametric models like the Heligman-Pollard to easily summarize changes in mortality over time via elegant demographic parameter interpretations, make these methods especially valuable for researchers concerned with dynamic mortality profiles in the presence HIV/AIDS. Moreover, after obtaining parameter estimates, we analyze the relationship between lagged HIV prevalence and those parameter values indicating the level and shape of mortality as well as employ a novel approach to estimating uncertainty intervals around life table columns.

We begin with a brief discussion of the trends and patterns in age-sex-specific HIV/AIDS-related mortality in sub-Saharan Africa since the beginning of the 1990s followed by an explanation of the Heligman-Pollard model and interpretation of its parameters. We continue with a description of the methods, results and discussion of our findings as related to the age pattern of mortality resulting from HIV.

1.1 Adult mortality in sub-Saharan Africa in the presence of HIV/AIDS

The impact of HIV/AIDS on mortality in southern Africa cannot be over stated. In sub-Saharan Africa, the epidemic has advanced to a generalized stage reaching rural communities and stretching beyond high-risk groups (Poit et al., 2001). Steadily escalating adult mortality over the past 15 to 20 years as a result of the rapid advancement of the epidemic has managed to wipe out previous gains in life expectancy garnered prior to the 1980s (Tollman et al., 1999; Ngom and Clark, 2003; Hosegood et al., 2004).

Our understanding of the role of HIV/AIDS in shaping adult mortality in sub-Saharan Africa is really a patchwork of information from a variety of data sources at varying levels of data collection. Indirect evidence on adult mortality gleaned from national and regional statistics including censuses and sample surveys furnish an understanding of trends in mortality at a national or regional level (Blacker, 2004) while community-based and cohort studies, which often collect data on the serostatus of study participants, including various Demographic Surveillance Sites throughout the region afford more direct evidence on the role of HIV/AIDS by comparing the mortality experience of infected and non-infected individuals (Zaba et al., 2004, 2007). In the following paragraphs, we will first briefly review the evidence from national censuses and regional statistics in order to document trends in adult mortality over the last 20 years. We will then turn to the direct evidence furnished by community-based studies to understand more nuanced impacts of HIV/AIDS including survival time since infection and the sex-age-pattern of mortality.

Absent high-quality vital registration systems[1], population censuses as well as sample surveys have become the main source of information for following demographic trends in most countries in sub-Saharan Africa and via techniques like intercensal survival and orphanhood methods, these sources point to escalating ${}_{45}q_{15}$ since the early 1990s for many countries in sub-Saharan Africa (Blacker, 2004). Based on the analysis of sibling histories asked about in Demographic and Health Surveys, Timæus and Jasseh (2004) report that by the turn of

[1]Data on mortality in developing regions can often be of meager quality due to poor vital registration systems (Porter and Zaba, 2004; Ngom and Clark, 2003). In addition, people with HIV/AIDS often die of some other more immediate cause, which is then recorded as the primary cause of death resulting in some underreporting of HIV/AIDS-related mortality. These issues extend to South Africa as well (Botha and Bradshaw, 1985; Tollman et al., 1999).

the century the probability of death before age sixty for someone surviving to age 15 was in the range of 30-60 percent up from 10-30 percent in the 1980s.

Although lacking in cause of death data, these data can indirectly inform on the impact of AIDS on adult mortality by linking high HIV prevalence with increasing mortality. Sibling histories from DHS data suggest a sharp increase in adult mortality after countries develop a generalized epidemic (Timæus and Jasseh, 2004). Likewise, UNAIDS (2002) reported that in countries where adult mortality was either declining or stabilizing prevalence was 5 percent or less while countries with increasing mortality had prevalence ranges of between 7 and 33 percent. In addition to the link to high HIV prevalence, national level data also suggest a strong age specific impact of the epidemic. Blacker (2004) notes that an examination of the age pattern of mortality increase can be quite useful in assessing the impact of HIV on adult mortality, specifically rapidly rising adult mortality that peaks earlier for women than for men. Ngom and Clark (2003) report elevated mortality at slightly younger ages for women than for men while Timæus and Jasseh (2004) note that the excess mortality cited above is concentrated at ages 25-39 for women and 30-44 for men.

While the strength of censuses and survey data lies in tracking the scale of change in adult mortality over certain time periods and in some cases linking these trends to HIV prevalence, community-based studies that collect data on the serostatus of participants make it possible to compare the mortality experiences of infected and uninfected individuals. These studies afford a much better understanding of the role of HIV in shaping adult mortality. First, community studies often furnish direct information on the survival times of infected individuals and this type of data can be particularly useful for those concerned with the age pattern of mortality. Depending on the start time of the epidemic and the age profile of incidence, the time between infection and death can have a large role in increasing mortality at specific ages. After following cohorts of infected individuals, research suggests survival times are roughly 9-11 years for individuals infected in their twenties with this interval shortening as age at infection increases (Porter and Zaba, 2004; Todd et al., 2007).

Direct evidence from community-based studies also gives us the best understanding of the impact of HIV on the level and age pattern of adult mortality. While background mortality (i.e. deaths from other causes like communicable diseases such as TB and malaria as well as variables affecting morbidity like poor nutrition) is higher in the developing world, contributing to excess mortality, mortality for infected individuals is much higher than for uninfected individuals and this increased mortality typically follows a sex-age-specific pattern (Porter and Zaba, 2004; Groenewald et al., 2005; Adjuik et al., 2006; Nyirenda et al., 2007; Smith et al., 2007; Zaba et al., 2007; Marston et al., 2007).

Owing to the fact that the sex-age specific HIV-related mortality schedule is influenced by a host of factors including age, sex, health, genetic endowment and environment and that the mortality schedule is a multifarious assemblage of many individuals disease experiences,

identification of a single universal HIV-related age pattern of mortality is implausible (Ngom and Clark, 2003). This implausibility notwithstanding, INDEPTH data uncovered seven patterns of mortality based on community data from all regions of Africa, two of which likely illustrate a substantial impact of HIV. These two patterns show significantly elevated mortality between ages 20 to 55 for males and 20 to 45 for females (INDEPTH, 2002). Similarly, in a review of serological studies, Porter and Zaba (2004) report that mortality rate ratios suggest high mortality in the middle age years as a result of high HIV prevalence while Smith et al. (2007) report the peak of male mortality was older (early 40s) than that of women (25-34). These sex differentials are largely driven by divergent ages of increased prevalence. As a consequence of the fact that sexual intercourse patterns are dominated by younger women with older men, women tend to experience increased prevalence at younger ages compared to men resulting in overall younger HIV/AIDS related mortality for women (Ngom and Clark, 2003; Porter and Zaba, 2004).

For South Africa, trends in HIV mortality largely follow those of southern Africa as a whole. South Africa is experiencing one of the most rapidly progressing HIV epidemics in the world (Hosegood et al., 2004). Antenatal prevalence amplified over the 1990s (Karim and Karim, 1999; Department of Health, Republic of South Africa, 2003) and while there was virtually no HIV mortality at the beginning of the 1990s, by 2000 research suggested significant HIV-related mortality (Ngom and Clark, 2003; Dorrington et al., 2002). Hosegood et al. (2004) reported that AIDS was the largest single cause of death in these rising mortality rates in South Africa. Similar to the sex-specific trends reported above for all of sub-Saharan Africa, women experience younger prevalence than men and the risk of dying from AIDS peaks earlier for women (25-39) than for men (30-44) in South Africa (Tollman et al., 1999; Hosegood et al., 2004; Groenewald et al., 2005). These increases in mortality for South Africa mirror the reversals in life expectancy found in other parts of southern Africa.

The age pattern of adult mortality is, of course, not stagnant and can be altered during the progression of the epidemic. Since this paper is concerned with assessing changes in age-specific mortality in the presence of HIV via a parametric time series approach, these trends are of significance to this analysis. As the epidemic matures and more people become infected, the age dependence of HIV mortality usually broadens (i.e. becomes less concentrated around a specific age) and becomes slightly older (Ngom and Clark, 2003).

In sub-Saharan Africa, HIV infection may be a growing source of childhood mortality as well. Similar to adult mortality in countries with generalized epidemics, data on child mortality is somewhat limited due to unreliable vital registration systems and meager cause of death information. An understanding of the impact of HIV on child mortality is restricted to data from small hospital-based studies or to population-based data that have been adjusted according to certain assumptions to arrive at estimated rates of child mortality caused by HIV/AIDS (Zaba et al., 2004). Although the effect of HIV on under-five mortality varies broadly by region, the impact appears to be substantial in southern Africa, where in the

worst affected countries, HIV may be causing up to half of all child deaths (Newell et al., 2004). Some 90 percent of pediatric infections occur in sub-Saharan Africa and because many HIV-infected children die before their fifth birthday, childhood mortality overall is intensified by HIV (Dabis and Ekpini, 2002; Foster and Williamson, 2000; De Cock et al., 2000) as cited in (Newell et al., 2004)). Given the high degree of vertical transmission - transmission from mother to child - we might also expect that increased seroprevalence in adult women may increase childhood mortality. The HIV/AIDS epidemic may also indirectly impact childhood mortality via maternal HIV status since children of HIV infected mothers are more likely to die than those of non-infected mothers (Newell et al., 2004; Zaba et al., 2004).

1.2 The Heligman-Pollard law of mortality

We use the Heligman-Pollard law of mortality to assess mortality at all ages over a 14-year period. The Heligman-Pollard law (Heligman and Pollard, 1980), presented in equation 1, is one of many so-called laws of mortality. A law of mortality is simply a mathematical equation that produces age-specific mortality schedules usually as a function of age (Hartmann, 1987). Unlike many other mortality laws, like that put forth early on by Gompertz (1825), which describes the steep increase in mortality at older ages, the Heligman-Pollard is a three-part model that covers the entire age range for all x $>$ 0.[2]

$$f(x) = A^{(x+B)^C} + D \times e^{-E(ln(x)-ln(F))^2} + \frac{GH^x}{1+GH^x} \tag{1}$$

Parametric methods, like the Heligman-Pollard, have several advantages for describing mortality, including smoothness, interpolation, analytic manipulation, parsimony, comparison and trends (Dellaportas, 2001; Debón et al., 2005). These last two advantages, along with the ability of parametric methods to easily and succinctly summarize large amounts of data over many years, are especially valuable to the research at hand. The representation of mortality trends via parametric methods facilitates a time-series approach where by the shape and intensity of age-specific mortality curves can be elegantly summarized and compared over time (Debón et al., 2005; Congdon, 1993; Rogers and Gard, 1991; Hartmann, 1987).

Researchers have used the Heligman-Pollard model to document changes in mortality in a variety of contexts (Heligman and Pollard, 1980; Dellaportas, 2001; Debón et al., 2005; Congdon, 1993; Forfar and Smith, 1987; Hartmann, 1987; Roger, 1987) cited in (Dellaportas, 2001)). For instance, Heligman and Pollard's original paper (1980) tracks Australian mortality over the 20th century while Rogers and Gard (1991) document declining infant and young adult mortality over the 20th century in the U.S. Thus far, the time series approach using the Heligman-Pollard law has largely been under utilized in assessing mortality

[2]For age 0 one can use a very small number like 0.00001(the approach we use in this paper) or the formula described in Rogers and Gard (1991), pg. 80.

changes in less developed nations and has not been used to assess mortality in a high HIV prevalence setting probably because the model was not developed under these circumstances. Fortunately, we believe the model is flexible enough to capture the rise in adult mortality associated with HIV as it was intended originally to capture the"accident hump" hump associated with young adulthood in developed settings. The ability of the Heligman-Pollard to capture mortality at all ages and to characterize the level and shape of adult mortality make it well suited for the assessment of the increasing intensity of adult mortality over the 1990s in sub-Saharan Africa. In addition, because the model parameters have straight forward demographic interpretations (i.e. describing not only the level but the shape and location of adult mortality) we can easily link the parameter changes to lagged HIV prevalence fostering a better understanding of the impact of HIV in shaping adult mortality.

The eight parameters of the model control three age ranges of mortality - childhood mortality, young adult mortality and late life mortality - and have some convenient demographic interpretations (Heligman and Pollard, 1980; McNown and Rogers, 1989; Rogers and Gard, 1991). Figure 1 plots the resulting line from adding all three components together while also plotting the individual parts so as to give the reader an intuitive idea of the influence of each part of the model.[3]

Table one summarizes the parameter interpretations. The first three parameters describe early childhood mortality. Parameter A roughly approximates mortality at age one and can be taken as a measure of the intensity or level of childhood mortality (McNown and Rogers, 1989; Rogers and Gard, 1991; Hartmann, 1987). The second parameter is the age displacement variable (Rogers and Gard, 1991) and indicates the difference between mortality at age one and mortality at age 0. As the value of B increases, ${}_1q_0$ decreases below 0.5 and begins to approach ${}_1q_1$ (Rogers and Gard, 1991). Finally, C indicates how quickly mortality decreases during childhood and into the young adult years. Declines in A are consistent with decreasing child mortality (Hartmann, 1987). Parameters A, B and C all have domain (0, 1). Because of the potential direct impact of pediatric AIDS deaths and the indirect impact of adult AIDS mortality on childhood mortality, we expect the level of child mortality to increase from period to period. In other words, we should see increases in A for both sexes as time goes on and the epidemic grows.

The second part of the model was initially composed to model the accident hump in males and, to a somewhat lesser extent, maternal mortality in females (Heligman and Pollard, 1980; Hartmann, 1987; McNown and Rogers, 1989; Rogers and Gard, 1991). This hump would typically have a peak in the twenties but it may be higher in the case of HIV-driven mortality in sub-Saharan Africa. Parameter D is related to the level or intensity of young

[3]Figures 1 and 2 are plotted using the following set of parameters, which can represent the nearly 100 probabilities of Brass' standard (McNown and Rogers, 1989). A= 0.06008, B= 0.31087, C= 0.34431, D= 0.00698, E= 1.98569, F= 26.7107, G= 0.00022, H= 1.08800

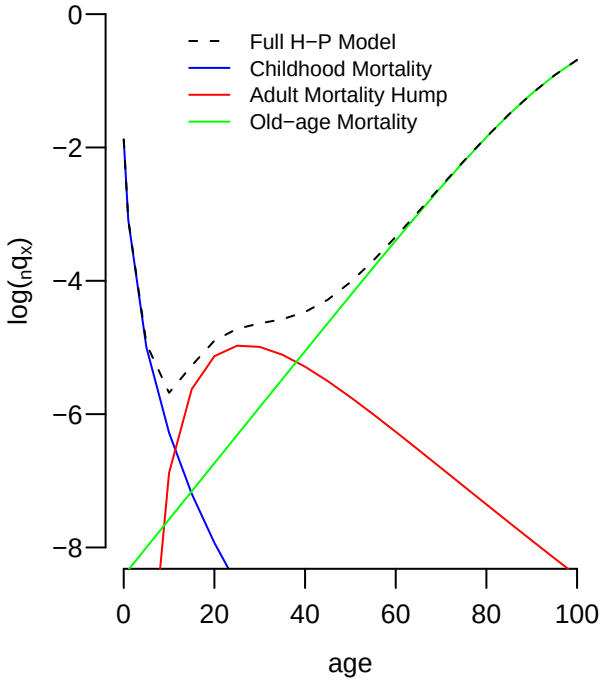

Figure 1: Decomposition of the Heligman-Pollard Model

adult mortality, E describes and is inversely related to the spread of the hump and F locates the position (Heligman and Pollard, 1980; McNown and Rogers, 1989; Rogers and Gard, 1991). Parameters D and E take domains (0, 1) and (0, ∞) respectively while the domain of F is less clear. This paper uses (15, 55) since we do not expect a large contribution of HIV deaths in ages older than 55. In light of the patterns and trends in HIV-related mortality, we expect increasing intensity of adult mortality for both sexes (increases in D). As the epidemic matures, we also expect a broadening of the adult mortality hump (decreasing E) and an increase in the location parameter, which should remain higher for males to reflect their older mortality.

It should be noted that if D is sufficiently low the other two parameters in this part of the curve do not influence the line very much. The eight panels of figure 2 illustrate the effect of each of the parameters individually. Each panel plots the resulting mortality schedule while changing the value of only a single parameter and holding all else constant. After an inspection of the equation and the panel for parameter D, one can see that the middle component of the Heligman-Pollard is composed of the product of D and the effects of E and F, so a small D essentially negates the effect of the spread and location parameters.

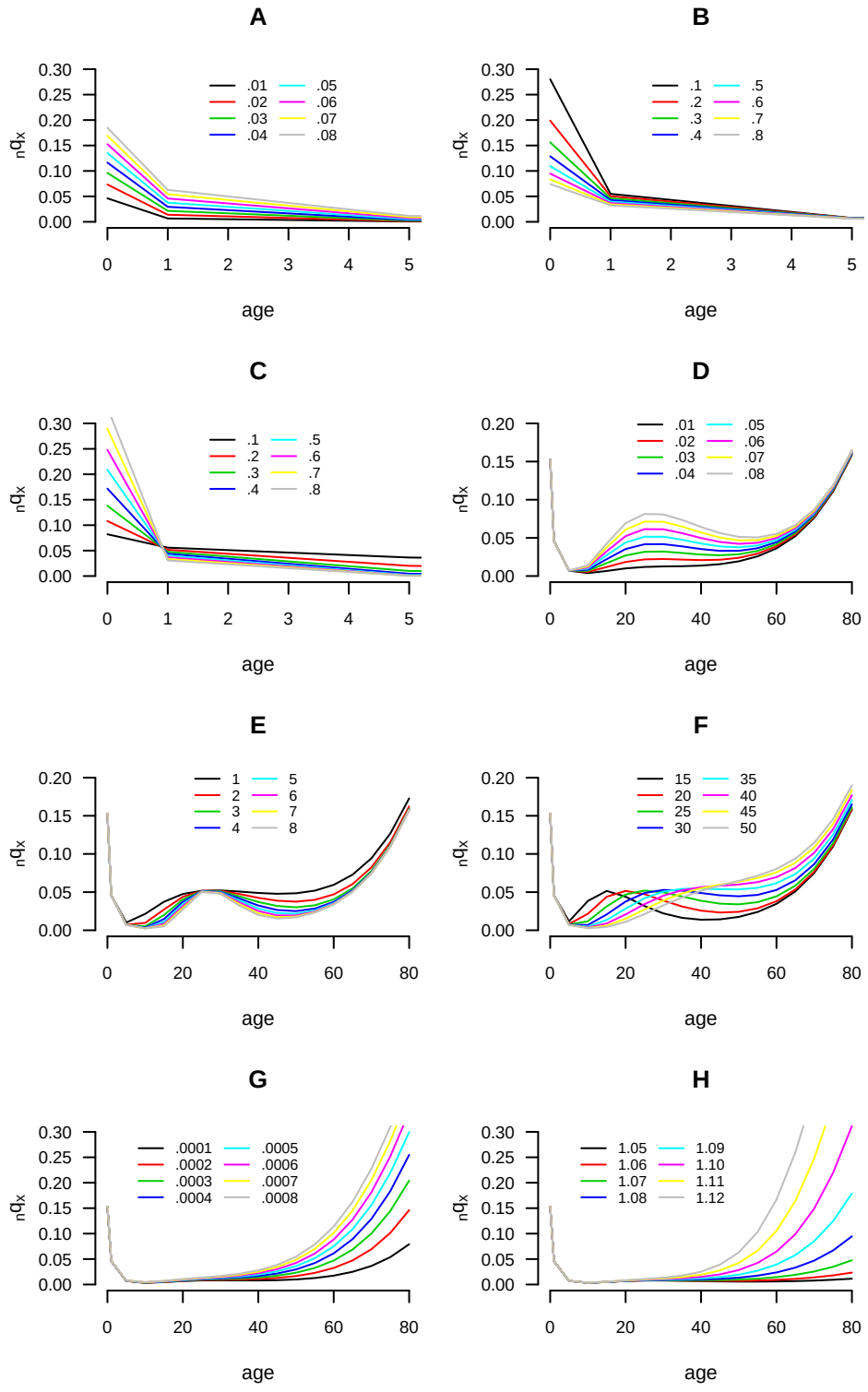

Figure 2: Resulting lines after changing the value of a single parameter while holding all others constant. For the plots of parameter E and F, D is set to .04 to give sufficient intensity to the hump.

Table 1: Heligman-Pollard Parameters

Parameter	Description
A	Intensity of Childhood Mortality ($\approx {}_1q_1$)
B	Measures the Difference between age 1 and 0 mortality probabilities
C	Captures decline in mortality during childhood
D	Intensity of young adult mortality
E	Varies inversely with the spread of young adult mortality hump
F	Location of the young adult mortality hump
G	Late life mortality (intercept of Gompertz curve at $x = 0$)
H	Late life mortality (slope of of Gompertz curve)

The last two parameters control the late life mortality section of the curve and describe the steep increase in mortality at these ages. Parameter G measures the base level of mortality at these ages (x=0) and H defines the rate of increase (Rogers and Gard, 1991). Parameters G and H are the intercept and slope of the Gompertz curve respectively and take domains (0, 1) and (0, ∞) respectively.

As demonstrated with the discussion above concerning the effects of the middle three parameters, each of the eight parameters should be interpreted in the presence of the others (Rogers and Gard, 1991). For instance, G and H influence the line as a result of their product as well as D with E and F; thus, their effects may be either diminished or intensified depending on the value of some other parameter.

2 Methods and Data

2.1 Data

We use two types of data in this analysis - mortality and prevalence data. We obtained mortality data from the Agincourt field site located in Mpumalanga province, South Africa, a rural area with HIV prevalence currently hovering around 30 percent. Using age-specific (ages 0-100) death counts and person years for the period 1994-2007, we computed the age specific probabilities of death (${}_nq_x$). In order to obtain the appropriate denominator values, we optimize the appropriate number of persons at risk of death in each age group (${}_nl_x$) based on the person years and deaths from that age range.[4] We report the observed person years and deaths counts in appendix A.

Because the individual year data is rather noisy in terms of the ${}_nq_x$ values, especially at older ages, and because sample sizes are somewhat small for individual years after the

[4]We use the "solver" function in Excel as the optimizer for obtaining the persons at risk with the target cell being l_0.

optimization (l_0(94-07) $\approx$ 400-550 persons/year), we grouped years based on similarity in their individual ${}_nq_x$ curves. Years are grouped as follows: 1994-1997 ($l_{0_{male}} = 1,917$, $l_{0_{female}} = 1,873$), 1998-2001 ($l_{0_{male}} = 2,100$, $l_{0_{female}} = 2,049$), 2002-2004 ($l_{0_{male}} = 1,896$, $l_{0_{female}} = 1,822$) and 2005-2007 ($l_{0_{male}} = 1,974$, $l_{0_{female}} = 1,893$). Figure 3 presents the post-grouping mortality schedules for males and females.

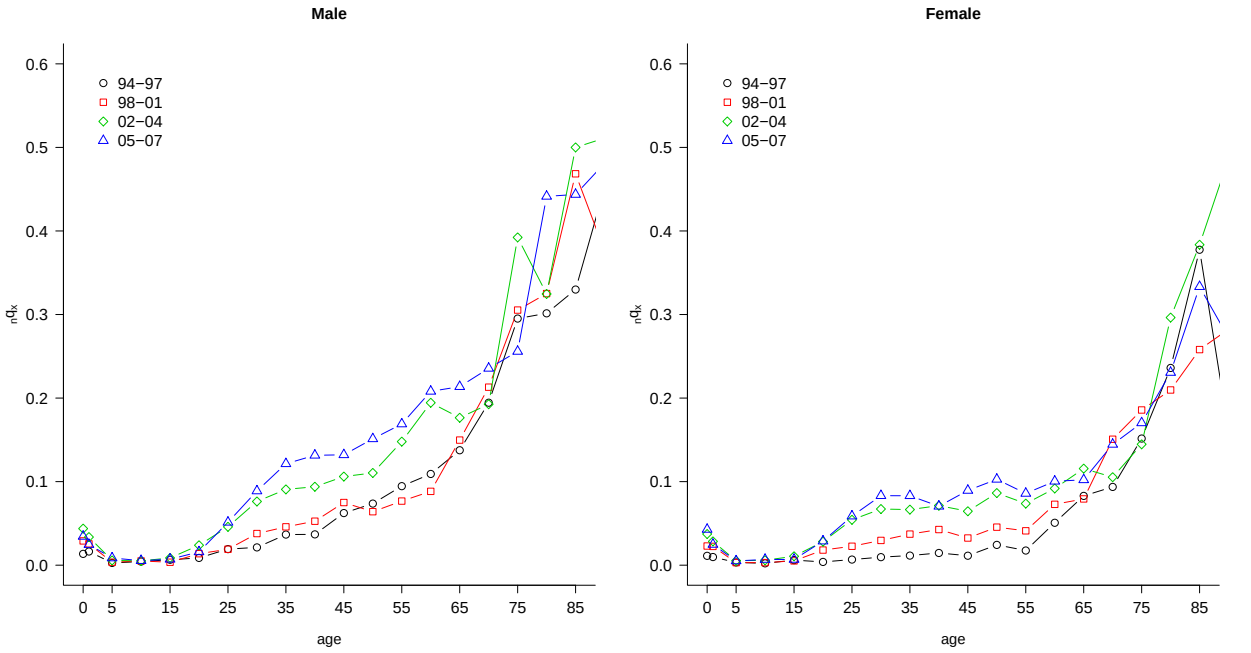

Figure 3: Age pattern of the probability of death, Agincourt 1994-2007

2.2 Methods

In the first part of this analysis, we obtain parameter estimates for the Heligman-Pollard model via Bayesian Melding with Incremental Mixture Importance Sampling with optimization (Poole and Raftery, 2000; Raftery and Bao, 2009). Not only does this approach address some estimation problems with the model (more on this below) but this procedure also allows us to estimate uncertainty around all parameter estimates and output (i.e. the model output, all age ${}_nq_x$). The Heligman-Pollard model is usually estimated via least squares methods but over-parameterization - a wide range of parameter estimates producing a similar fit - has been revealed to be an issue using this technique (Bebbington et al., 2007; Dellaportas, 2001; Congdon, 1993). Dellaportas (2001) used a Bayesian approach and reported that the over-paramterization issue is usually resolved via the use of informative priors (Congdon, 1993). One way to assess the over-parameterization problem is to examine the correlation between parameters. For the most part we do not see very high correlation among any pair of parameters save for G and H. The high correlation between G and H is to be expected

since they influence the line via their product. Another common estimation problem with the Heligman-Pollard occurs when the hump is flat. In this case, the hump parameters may take on values beyond a plausible range (Rogers and Gard, 1991; Hartmann, 1987; Heligman and Pollard, 1980). Parameter D may, for instance, be higher than expected, while the location parameter often places the hump in the high 90s or beyond age 100 essentially creating a flat curve through the adult years. Fixing one or more parameters and estimating the others tends to resolve this problem (Congdon, 1993; Rogers and Gard, 1991; Hartmann, 1987). Similarly, we restrict the range on parameter F to between 15 and 55, which keeps the hump parameters in plausible ranges.

2.2.1 Bayesian Melding

Bayesian Melding is an appropriate technique when a deterministic model is used in the likelihood (Raftery et al., 1995; Poole and Raftery, 2000) - the Heligman-Pollard in this case. The Heligman-Pollard model, denoted by M, transforms the set of eight parameter inputs, denoted θ, into age-specific probabilities of death, denoted ϕ, so that $\phi = M(\theta)$. Information on prior beliefs about the parameters can be incorporated in the form of probability densities, $p(\theta)$, where θ is a prior distribution for the parameter to be estimated. Observed data, $\mathbf{y}$, are incorporated by way of specifying a likelihood. We calculate the likelihood for the model outputs ($_nq_x$ values) from the binomial likelihood presented in equation 2.

$$\mathcal{L}(\mathbf{p}|\mathbf{x},\mathbf{n}) = \binom{n}{x} p^x (1-p)^{n-x} \tag{2}$$

The bayesian approach combines information from the prior density for the model inputs and the likelihood for the outputs and data to produce a posterior distribution of the inputs. The estimation of the posterior distribution is presented in equation 3 where the posterior parameter distribution is proportional to the likelihood times the prior.

$$p(\theta|\mathbf{y}) \propto \mathcal{L}(\mathbf{y}|M(\theta))p(\theta) \tag{3}$$

We approximate the posterior distribution using the Incremental Mixture Importance Sampling algorithm (Steele et al., 2006; Raftery and Bao, 2009). The algorithm builds on the SIR algorithm, which draws a large number of samples from the prior distribution of the model parameters, weights each sample by its likelihood and then resamples them with replacement and with the computed weights (Rubin, 1988). Occasionally the SIR algorithm does not perform well and few distinct values are present in the resample because a small number of large importance weights dominate the resample step. With IMIS, after calculating the likelihoods and weights as in the SIR algorithm, at each iteration a multivariate normal distribution centered at the highest importance weight is added to the current importance sampling distribution, thus forming a mixture and successfully representing parts of the parameter space that would be missed. If the initial sample from the prior misses a high probability region, IMIS may also not be able to cover that region. To address this problem, Raftery and Bao (2009) suggest inserting an optimization step after the initial

stage (i.e. drawing the samples from the prior distribution and calculating their weights) yielding a mixture of d multivariate normal distributions centered around a local maximum of the target distributions. New inputs drawn from the multivariate normal distributions from the optimization step are combined with the initial inputs and new likelihoods and weights are calculated. In the final resampling stage, IMIS resamples x number of inputs with replacement from the mixture distribution with the calculated weights forming the posterior parameter distribution.

In this analysis we sample 400 sets of parameter values in the final resample (the result is a 400 X 8 matrix with each row corresponding to one draw of the final resample), which can then be used to calculate 400 separate ${}_nq_x$ lines for the entire age range. Figure 4 plots the posterior output distribution for the first period for males (a flat hump) and the last period for males (a more intense and concentrated hump). The gray cloud of lines is made up of the 400 separate lines resulting from the posterior distribution of parameters. The posterior distribution should be proportional to the likelihood multiplied by the density of the prior.

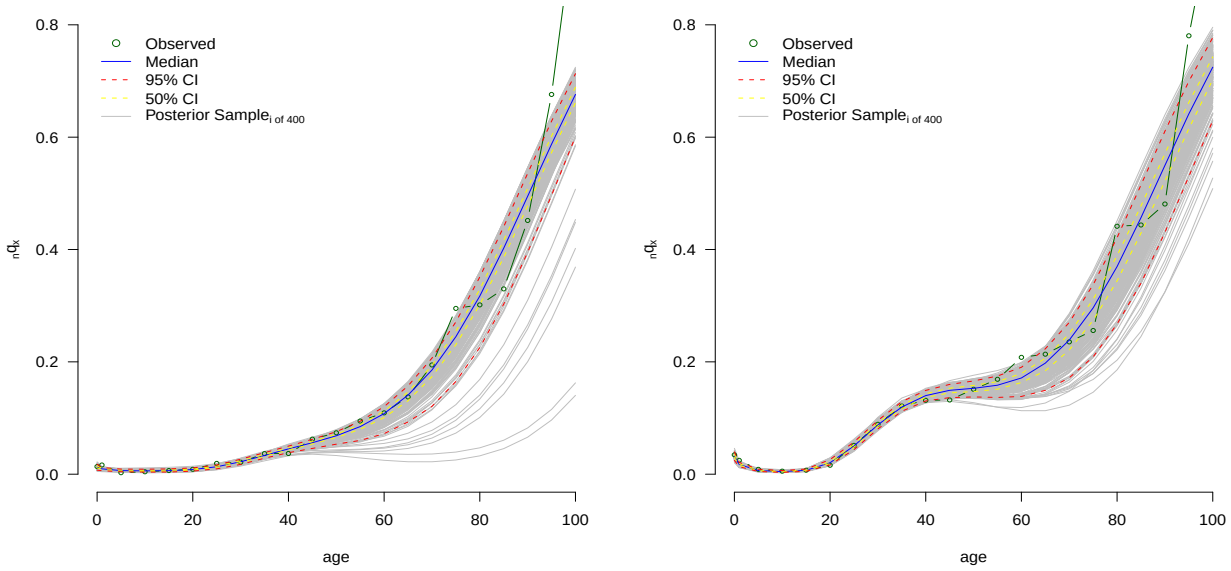

Figure 4: Plotted posterior output distributions from a flat hump (Males 94-97) and an intense hump (Males 05-07)

The prior distributions on the input parameters are defined as follows:

$$prior \begin{cases} A \sim Uniform[0, 0.25] \\ B \sim Uniform[0, 1] \\ C \sim Uniform[0, 1] \\ D \sim Uniform[0, 0.25] \\ E \sim Uniform[0, 20] \\ F \sim Uniform[15, 55] \\ G \sim Uniform[0, 0.01] \\ H \sim Uniform[1, 1.5] \end{cases} \tag{4}$$

2.3 Relationship between parameters and HIV prevalence

Because of the previously reviewed relationship between increased adult mortality and increased HIV-prevalence, in the second part of this analysis, we examine the relationship between the time series of parameter estimates, especially those related to the intensity and shape of the mortality hump, and HIV prevalence in the Mpumalanga province. Once we obtain the posterior distribution, we plot the median value for each of the parameter distributions by the mean HIV prevalence in the province with a five-year lag. We expect

that increased HIV prevalence will be positively related to both child and adult mortality intensity and positively related to the location parameter. Table 2 presents the mean HIV prevalence levels for five-year lagged periods of interest.[5]

Table 2: HIV Prevalence: Mpumalanga

Five-year lag period	HIV Prevalence
1990-1992	1.27
1993-1996	11.64
1997-1999	26.63
2000-2002	29.17

2.4 Calculating uncertainty around life table columns

An additional advantage of the Bayesian approach used here is the ability to estimate uncertainty bounds around both the model parameters and output. The posterior ${}_nq_x$ distribution means we have 400 individual schedules from which we can calculate 400 individual life tables for each sex-period specific posterior parameter distribution. This advantage affords a novel approach to life table column uncertainty estimation. In those situation where sample level data is used to construct a life table, the estimation of uncertainty around life table quantities, a problem which is not well addressed in the literature on life table construction (Lynch and Brown, 2005), is an important step in understanding the process of interest.

In light of past research which has sought to link declining life expectancy with increasing HIV prevalence and HIV related mortality, we calculate the posterior e_x output distribution from the 400 posterior life tables for each sex-specific period to assess whether there have been significant declines in life expectancy at birth and age 10 over the period of study here. Similar to the time series of parameter estimates, by calculating the posterior e_x schedule distribution at each period, we can assess the impact of increasing HIV prevalence on life expectancy.

3 Results

Table 3 presents the median parameter estimates for each sex-specific period.[6] We will first review trends in child mortality followed by an interpretation of the hump parameters.

[5]source: Department of Health, Republic of South Africa 1995; 1996; 1997; 1998; 2003; 2006

[6]A caution should be noted before reviewing the contents of table 3. We present the 95% CI intervals for each of the individual parameter distributions but each draw of the final resample to create the posterior distribution estimates the eight parameters simultaneously and evaluates their likelihood as a group. Thus, a high or low value of one parameter may be compensating for an extreme value of another parameter in order to better fit the observed mortality pattern.

Table 3: BM Median Results (95% CI in parentheses)

	94-97	98-01	02-04	05-07
Males				
A	0.0106	0.0297	0.0460	0.0262
	(0.0059 - 0.0148)	(0.0228 - 0.0365)	(0.0390 - 0.0529)	(0.0194 - 0.0321)
B	0.7829	0.9724	0.9652	0.7754
	(0.6673 - 0.8979)	(0.9248 - 0.9980)	(0.8928 - 0.9981)	(0.6930 - 0.8609)
C	0.0855	0.1791	0.3526	0.2083
	(0.0320 - 0.1501)	(0.1149 - 0.2336)	(0.2801 - 0.4191)	(0.1573 - 0.2589)
D	0.0243	0.0388	0.0726	0.1199
	(0.0160 - 0.0327)	(0.0324 - 0.0445)	(0.0618 - 0.0836)	(0.1062 - 0.1338)
E	5.2779	4.5021	3.1653	3.7761
	(3.4764 - 7.1116)	(3.0783 - 5.6768)	(1.5618 - 4.6151)	(2.9309 - 4.6871)
F	45.2412	38.5225	39.5636	42.2610
	(41.4002 - 49.0394)	(36.3215 - 40.5921)	(35.6854 - 43.7599)	(39.0647 - 44.7113)
G	0.0010	0.0006	0.0021	0.0009
	(0.0003 - 0.0017)	(0.0003 - 0.0009)	(0.0010 - 0.0034)	(0.0004 - 0.0015)
H	1.0798	1.0877	1.0717	1.0827
	(1.0725 - 1.0872)	(1.0822 - 1.0939)	(1.0630 - 1.0805)	(1.0772 - 1.0887)
Females				
A	0.0148	0.0275	0.0422	0.0356
	(0.0118 - 0.0181)	(0.0227 - 0.0334)	(0.0382 - 0.0473)	(0.0305 - 0.0417)
B	0.7852	0.9305	0.9792	0.8563
	(0.7047 - 0.8732)	(0.8589 - 0.9942)	(0.9520 - 0.9985)	(0.8085 - 0.9027)
C	0.1048	0.2120	0.2245	0.2373
	(0.0697 - 0.1409)	(0.1621 - 0.2568)	(0.2065 - 0.2447)	(0.2064 - 0.2669)
D	0.0126	0.0303	0.0688	0.0867
	(0.0083 - 0.0167)	(0.0266 - 0.0348)	(0.0642 - 0.0733)	(0.0808 - 0.0930)
E	14.5269	2.6527	2.5174	2.5266
	(13.0611 - 15.8447)	(1.9731 - 3.3408)	(2.1914 - 2.8271)	(2.0314 - 3.1009)
F	31.1959	36.5381	38.1891	40.6617
	(28.1650 - 34.0361)	(34.0823 - 39.2770)	(37.0759 - 39.2378)	(38.9361 - 42.2265)
G	0.0003	0.0003	0.0001	0.0001
	(0.0002 - 0.0004)	(0.0002 - 0.0004)	(0.0000 - 0.0001)	(0.0000 - 0.0002)
H	1.0958	1.0900	1.1110	1.1092
	(1.0904 - 1.1019)	(1.0824 - 1.0972)	(1.1049 - 1.1170)	(1.0976 - 1.1174)

Childhood Mortality Recall that we expected childhood mortality to experience increasing intensity over this period. In other words, we expect that parameter A, which roughly approximates ${}_1q_1$ and is an indicator of the level of childhood mortality, will increase from period to period. Figure 5 plots the value of parameters A, D, E and F over this period for both sexes. The upper left panel of figure 6, which plots the time series of parameter A, bears this expectation out except for the final period. The estimates of parameter A suggest increasing childhood mortality levels for both males and female through the 2002-2004 period until a we see a decrease from the third to the final period for both sexes.

Although, there appears to be a male mortality advantage during the first period, the model seems to underestimate the level of mortality during these years. The first component of model expects the usual rapid decline in child mortality traditionally observed at these age ranges but male infant mortality remains relatively flat over the first year of life in the first period for males - a feature the model has a difficult time fitting well.

Adult Mortality Trends in the level or intensity of adult mortality largely follow those of childhood mortality but each consecutive period consistently outpaces the previous period for both sexes. The upper right panel of figure 5 plots parameter D for males and females over this period. Parameter D increases consistently from each period to the next and between both the second and third and third and fourth periods for both sexes the uncertainty intervals do not overlap. Figure 6 plots the median and 95% CI from the posterior ${}_nq_x$ distribution for each period. The significant increase in adult mortality over this period is apparent from the non-overlapping intervals depicted in figure 6. Similar effects can be seen by examining the hump component independently of the rest of the model, plotted in figure 7. From each period to the next, the adult mortality hump grows in intensity for both males and females.

Figure 8 plots just the hump component again but within sex for each period. Similar to figure7, the plot demonstrates growing intensity from period to period for both sexes and that by the final period, male intensity significantly outpaces female intensity (i.e. the peak of the male adult mortality hump is much higher than the peak for females). The upper right panel of figure5 suggests that adult mortality increased at a similar pace for both men and women until the final period when male mortality begins to intensify more quickly. For females, the largest increase occurs between the second and third periods. Figure 8 also confirms the typically "older" adult mortality for males associated with HIV related mortality with the male humps usually peaking at slightly older ages regardless of the intensity or spread of the hump.

Along with increasing intensity, increasing values of parameter F, plotted in the lower right panel of figure 5, suggest an aging mortality hump for both sexes. Recall that females tend to contract the disease at younger ages compared to men and thus experience elevated mortality at younger ages. The location parameter accurately reflects this previously observed pattern

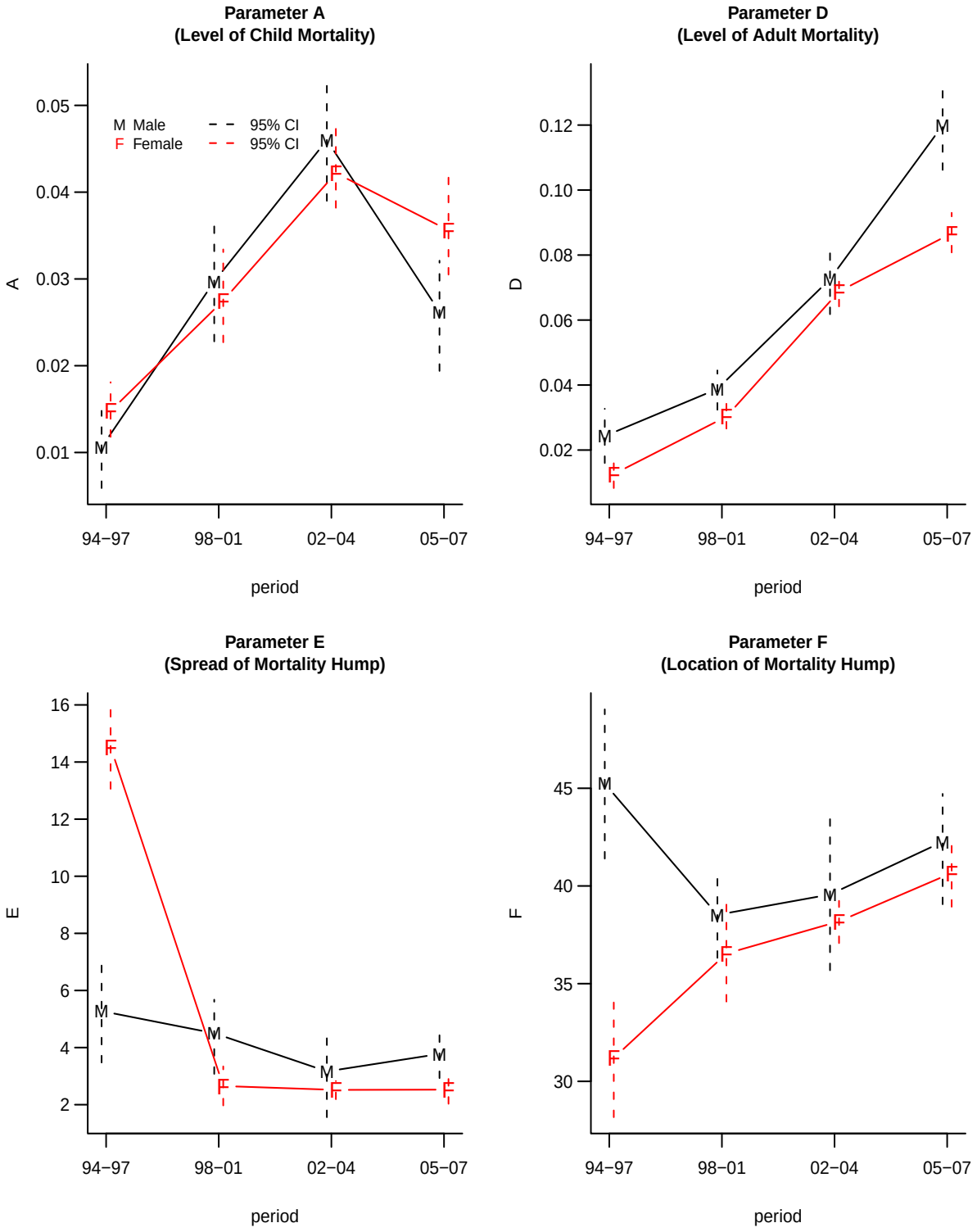

Figure 5: Times Series of A, D, E and F

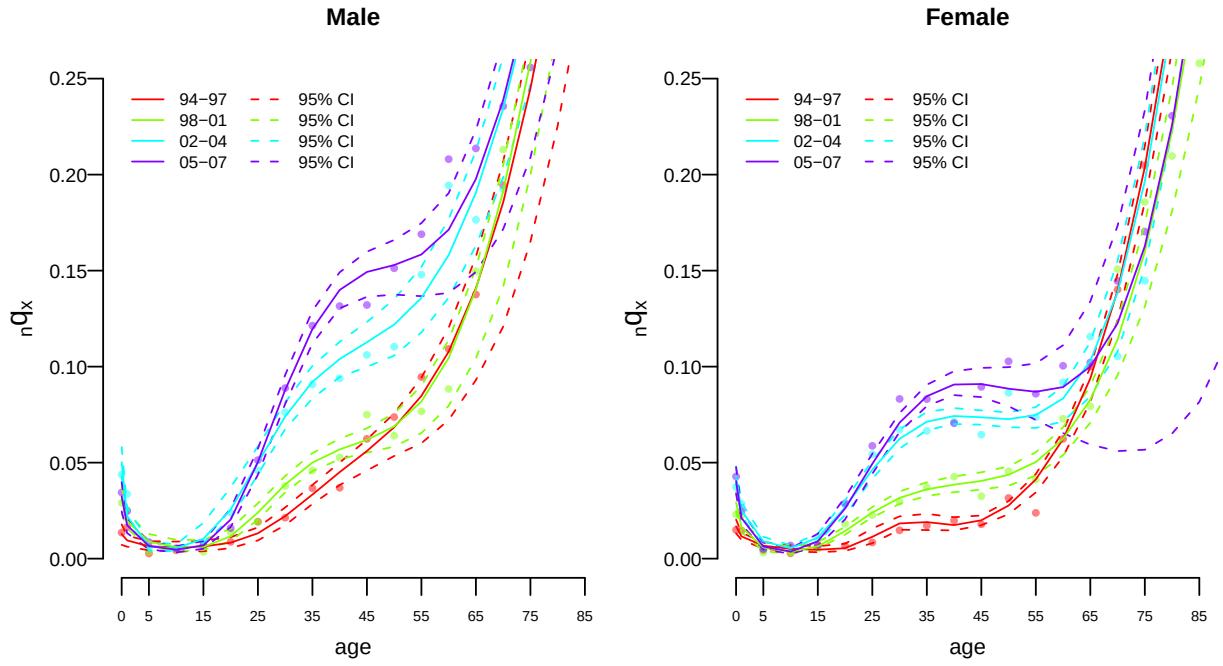

Figure 6: Median fitted curves with 95% CI plotted. Observed ${}_nq_x$ values are presented as lightly colored dots.

with the male median estimate of F consistently outpacing the female estimate, although, except for the first period, the male and female credible intervals overlap at each period. Females experience growing mortality from the early to the late 30s (probably reflecting the incidence profile for females with the roughly 9-11 years of survival after infection) while the male hump matures from the late 30s to the mid 40s after an initial decline between the first and second periods. The unusually high location parameter value in the first period for males probably results from a small increase in mortality around the late 40's to early 50s. Because this is the first period, it is unlikely that this small hump is resulting from HIV related deaths. Rather, HIV probably begins to impact the mortality schedule between the first and second period for males at which point the location and intensity parameters begin to pick up increasing male mortality in the late 30s consistent with previously observed HIV mortality patterns. Additionally, the location parameter is this case probably does not have a large impact given that the intensity is rather small. The change in the hump for males is best represented in figure 7 where one can see that between the first and second periods, the male hump increases slightly in intensity and shifts back to the expected age range.

Trends in the spread of the hump suggest a slight broadening of the mortality hump for both sexes although adult mortality remains similarly spread beyond the second period. The exceptionally high value for parameter E for the female first period is picking up a slight but

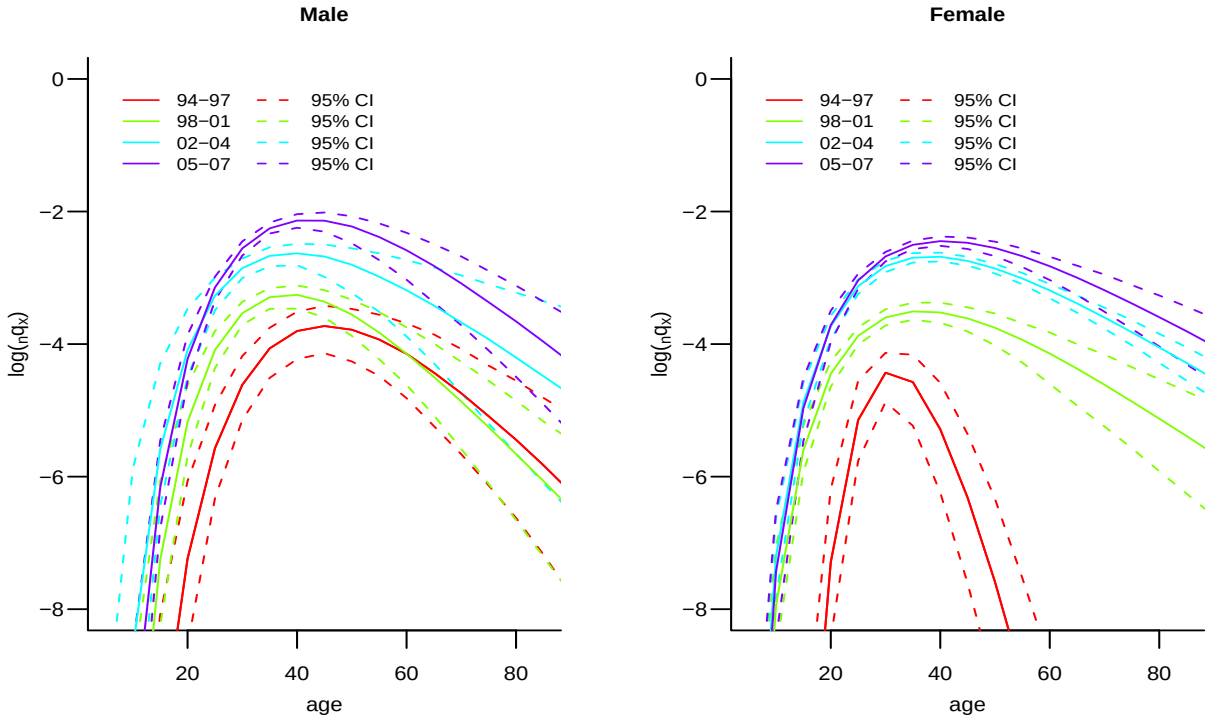

Figure 7: Hump Component for all periods and both sexes

concentrated bit of mortality in the late 20s to early 30s perhaps resulting from maternal mortality. Although the credible intervals overlap for all periods, after the initial period, females experience slightly broadening mortality. Similarly for males, after an initial decline in the parameter values, suggesting a broadening or adult mortality, the parameter values remain low. Figure 8 demonstrates the effect of the declining parameter value for both sexes. Comparing the latter period humps with those from the first two periods, one can see that not only does the hump become taller reflecting the increases in parameter D but the hump also broadens as the right half of the hump (ages 35+) becomes more horizontal. These changes reflect the spreading of increased adult mortality into the older middle adult year (35-45). Because survival time from infection to death is about 10 years and the epidemic in South Africa began later and at younger ages, the elevated mortality at older ages broadening the hump may reflect a broadening age profile of incidence and older ages at death. Likewise, because females tend to contract the virus earlier and die earlier we should see less of an increase in mortality at these older ages.

Using the mean prevalence from a period of equal length and five years prior to those under study here, we can assess the relationship between HIV prevalence rates and the Heligman Pollard parameters. Figure 9 plots the median parameter values for A, D, E and F along with the corresponding 5-year lagged HIV prevalence with the grey dashed line representing

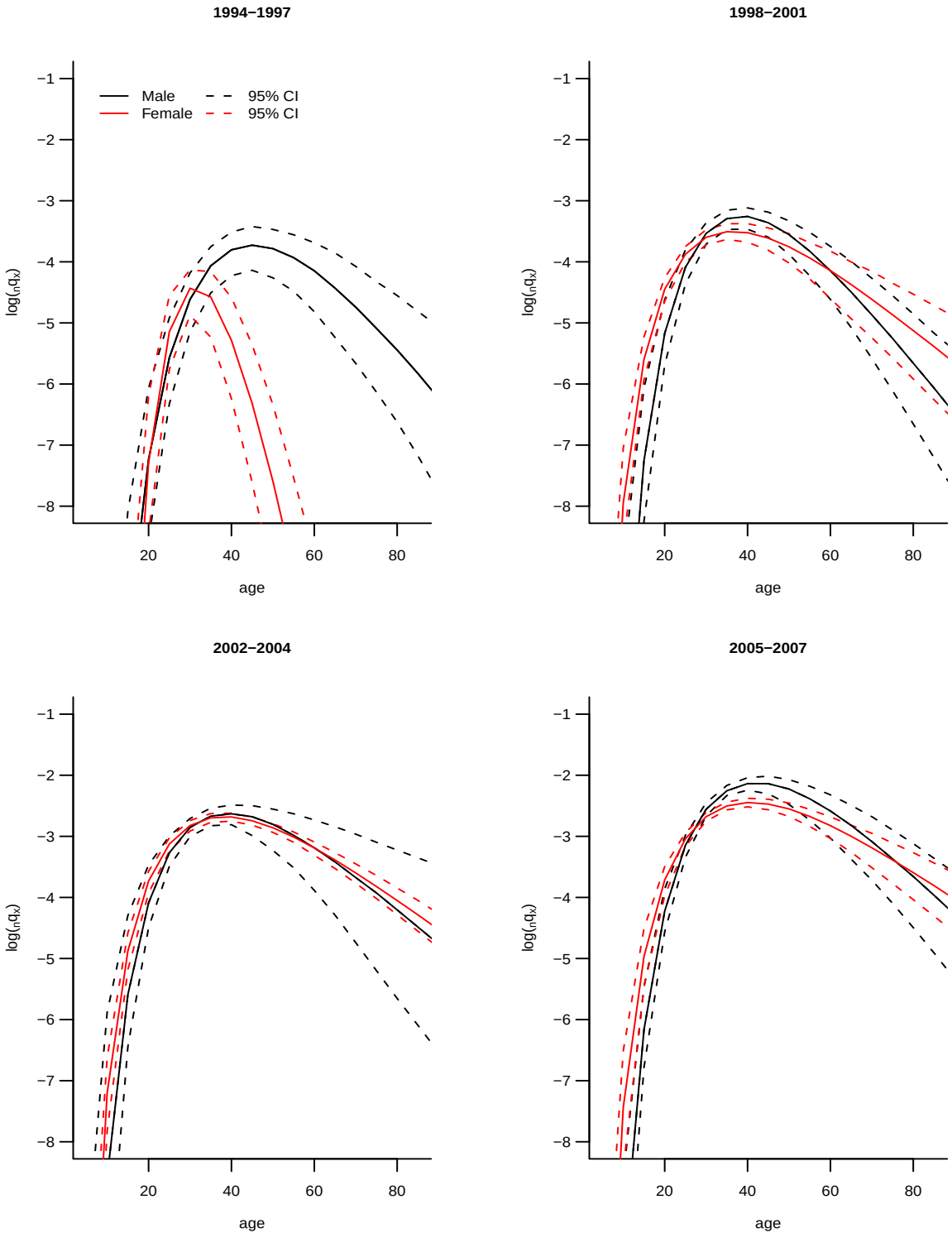

Figure 8: Hump Component for each sex for each period

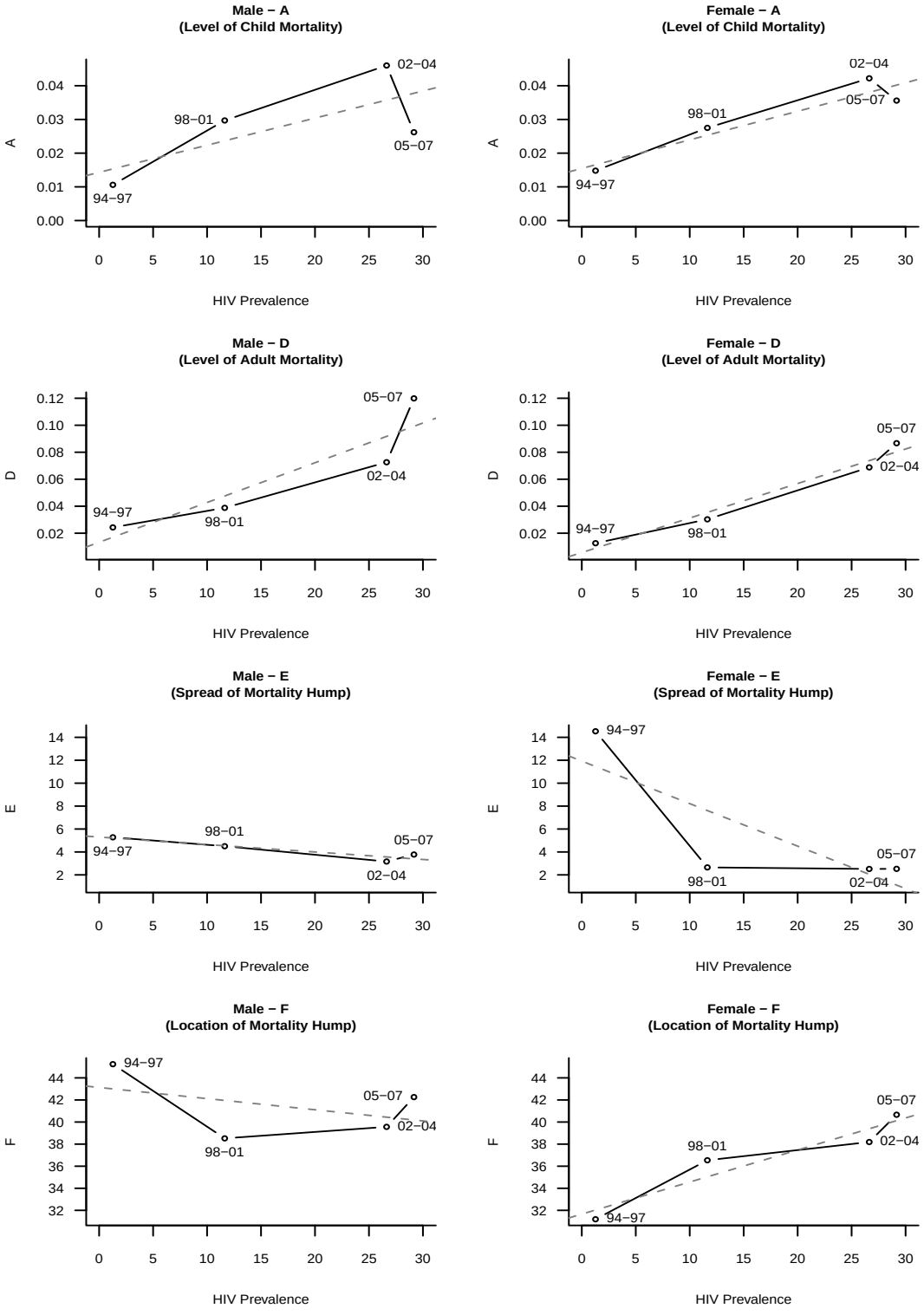

Figure 9: Selected Parameter Values by 5-year lagged HIV prevalence (OLS regression line in dashed grey)

a fitted OLS regression line. For all periods and both sexes, the intensity parameters, A and D increase with increasing HIV prevalence except for A during the final period for both sexes. As HIV prevalence increased the level of adult mortality increased consistently over these four periods for both sexes. The largest increase in intensity occurs between the second and third periods for females following the largest increase in prevalence while the largest male increase in intensity occurs between the third and final period. The later increase for males may result from longer survival periods from time of infection. Although small for both sexes, these results do suggest more widely spread mortality over the adult years as HIV prevalence increases. Save for the first period for males in which the hump is located late in the adult years due to increasing mortality around the late 40s, these results also indicate a trend of aging mortality for both males and females as HIV prevalence increases.

Life Expectancy Finally, we turn to changes in the life expectancy schedule. Table 4 reports life expectancy at birth and age 10 for the four period under investigation here for both sexes while figure 10 plots the median life expectancy schedule and 95% CI from the posterior e_x schedule distribution. These results indicate a precipitous decline in life expectancy over the entire period with the largest decreases occurring between the second and third periods for both sexes. Life expectancy at birth declines significantly by about four years for both men and women between the first and second periods while life expectancy at birth drops by roughly a decade from the late 1990s to the early 2000s.

Table 4: Life Expectancy at birth and age 10 for Agincourt 1994-2007 (95% CI in parentheses)

	Female e_0	Female e_{10}	Male e_0	Male e_{10}
1994-1997	73.44	66.00	67.26	59.12
	(72.44-74.66)	(65.01-67.16)	(65.55-72.18)	(57.58-64.06)
1998-2001	69.72	63.40	63.06	56.81
	(68.27-71.73)	(62.01-65.47)	(61.63-65.27)	(55.28-59.36)
2002-2004	59.72	54.42	53.50	47.89
	(58.63-61.43)	(53.41-56.11)	(51.99-55.07)	(46.51-49.56)
2005-2007	59.52	53.33	52.16	45.14
	(56.57-64.49)	(50.72-58.78)	(51.02-54.52)	(44.08-47.45)

Life expectancy at age 10 paints a similar picture. Although both sexes do experience decreases in e_{10} between the first and second periods, the uncertainty intervals overlap. The same is not true between the second and third periods where both sexes see significant declines. Again, the number of years a person can expect to live beyond age 10 plummets by nearly a decade between these two periods. In the 1998-2001 period, a female who survived to age 10 could expect to live close to 64 additional year while a male who survived to age 10 during that same period could expect an additional 57 years of life. Compare that to the

54 years for females and 47 years for males just four years later. Given the mean survival period from time of infection of 9-11 years, it is precisely at the turn of century we should begin to see the effects of escalating prevalence over the 1990s as reported in table 2.

Summary and Discussion

As the HIV epidemic matures, parametric methods can elegantly summarize changes in the mortality schedule over time. In this paper we have used one such method, the Heligman-Pollard law of mortality. Many of the changes in the Heligman Pollard parameters over this short period reflect an expanding and maturing HIV epidemic. Both the increasing childhood and adult mortality are consistent with the findings on age-specific HIV-related mortality. Although the increasing childhood mortality cannot be definitively attributed to direct pediatric AIDS deaths or to the indirect effect of adult AIDS mortality and prevalence on childhood survival, in light of the current research, certainly there is a sizable contribution of HIV to childhood survival.

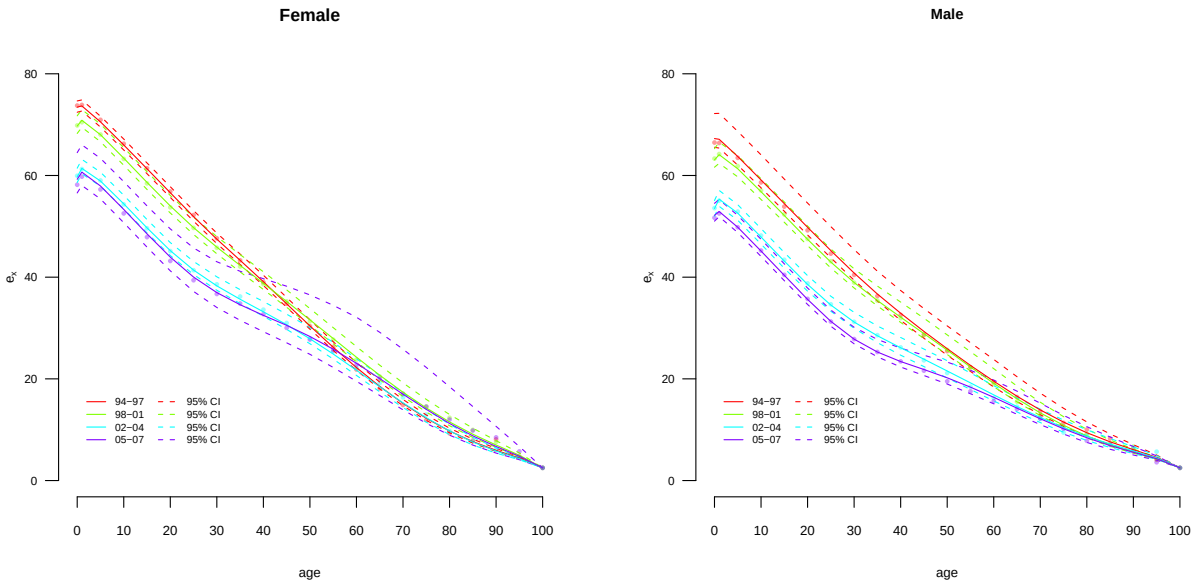

Figure 10: Posterior median e_x schedule with 95% CI. Observed data are represented as lightly colored dots.

Likewise, other features of a growing HIV epidemic are reflected in the hump parameters. The location parameter progresses to become slightly older for each group as some individuals live longer, while the older HIV-related mortality of men is quickly summarized

in the consistently higher location parameter. Likewise, as HIV prevalence increases and the age profile of incidence widens, the spread of adult mortality increases becoming less concentrated around a single age.

The advantages of modeling mortality with this type of model are numerous. We can not only parsimoniously describe mortality at all ages throughout the maturation of the epidemic, we can also link quantified descriptions of the level and shape of mortality to HIV prevalence. This approach certainly serves to elucidate our understanding of the impact of HIV/AIDS on both adult and child mortality. Likewise, because the model is flexible enough to model HIV-related mortality in the adult years it has other uses in the analysis of mortality in high HIV prevalence settings including smoothing empirical mortality schedules for use in constructing a model life table system for Africa as well making probabilistic mortality projections if the parameters are estimated in such a fashion.

Finally, the estimation procedure used here, Bayesian Melding with IMIS, results in a posterior parameter distribution which can be transformed via the model to a posterior distribution of ${}_nq_x$ schedules which can themselves be used to calculate posterior life tables. Because we have a distribution of life tables, calculating confidence intervals around life table columns is straight forward. Results from performing such a calculation here suggest a large impact of increasing HIV prevalence in declining life expectancy over this period. The posterior distributions of life expectancy at birth and age 10 suggest a significant role of HIV related mortality in the late 1990s and early 2000s for this population.

References

Adjuik, Martin, Tom Smith, Sam Clark, Jim Todd, Anu Garrib, Yohannes Kinfu, Kathy Kahn, Mitiki Mola, Ali Ashraf, Honorati Masanja, Kubaje Adazu, Jahit Sacarlal, Nurul Alam, Adama Marra, Adjima Gbangou, Eleuther Mwageni, and Fred Binka. 2006. "Cause-specifc mortality rates in sub-Saharan Africa and Bangladesh." *Bulletin of the World Health Organization* 84:181–188.

Bebbington, Mark, Chin-Diew Lai, and Ricardas Zitikis. 2007. "Modeling human mortality using mixtures of bathtub shaped failure distributions." *Journal of Theoretical Biology* 245:528–538.

Blacker, John. 2004. "The impact of AIDS on adult mortality: evidence from national and regional statistics." *AIDS* 18:S19–S26.

Botha, J.L. and D. Bradshaw. 1985. "African Vital Statistics - A Black Hole?" *South African Medical Journal* 67:977–981.

Congdon, Peter. 1993. "Statistical Graduation in Local Demographic Analysis and Projection." *Journal of the Royal Statistical Society: Series A* 156:237–270.

Dabis, François and Ehounou René Ekpini. 2002. "HIV-1/AIDS and maternal and child health in Africa." *Lancet* 359:2097–2104.

De Cock, Kevin M., Mary Glenn Fowler, Eric Mercier, Isabelle de Vincenzi, Joseph Saba, Elizabeth Hoff, David J. Alnwick, Martha Rogers, and Nathan Shaffer. 2000. "Prevention of mother to child HIV transmission in resource poor countries - translating research into policy and practice." *Journal of the American Medical Association* 283:1175–1182.

Debón, Ana, Francisco Montes, and Ramon Sala. 2005. "A comparison of parametric models for mortality graduation. Application to mortality data for the Valencia Region (Spain)." *SORT* 29:269–288.

Dellaportas, Petros. 2001. "Bayesian Analysis of Mortality Data." *Journal of the Royal Statistical Society: Series A* 164:275–291.

Department of Health, Republic of South Africa. 1995. "Fifth national HIV survey in women attending antenatal clinics of the public health services in South Africa, Oct/Nov." *Epidemiological Comments* 22.

Department of Health, Republic of South Africa. 1996. "Sixth national HIV survey of women attending antenatal clinics of the public health services in the Republic of South Africa, Oct/Nov 1995." *Epidemiological Comments* 23:3–15.

Department of Health, Republic of South Africa. 1997. "Seventh national HIV survey of women attending antenatal clinics of the public health services, October/November 1996." *Epidemiological Comments* 23:4–16.

Department of Health, Republic of South Africa. 1998. "Eighth annual national HIV sero-prevalence survey of women attending antenatal clinics in South Africa, 1997." .

Department of Health, Republic of South Africa. 2003. "National HIV and Syphilis Antenatal Sero-Prevalence Survey in South Africa: 2002." .

Department of Health, Republic of South Africa. 2006. "National HIV and Syphilis Antenatal Sero-Prevalence Survey in South Africa: 2005." .

Dorrington, Rob, David Bourne, Debbie Bradshaw, Ria Laubscher, and Ian M. Timæus. 2002. "The Impact of HIV/AIDS on Adult Mortality in South Africa." Technical report, Burden of Disease Research Unit, Medical Research Council.

Forfar, D.O. and D.M. Smith. 1987. "The Changing shape of English Life Tables." *Transactions of the Faculty of Actuaries* 40:98–133.

Foster, Geoff and John Williamson. 2000. "A review of current literature of the impact of HIV/AIDS on children in sub-Saharan Africa." *AIDS* 14:S275–S284.

Gompertz, Benjamin. 1825. "On the Nature of the Function Expressive of the Law of Human Mortality, and on a New Mode of Determining the Value of Life Contingencies." *Philosophical Transactions of the Royal Society of London* 115:513–583.

Groenewald, Pam, Nadine Nannana, David Bourne, Ria Laubscher, and Debbie Bradshaw. 2005. "Idetiying deaths from AIDS in South Africa." *AIDS* 19:193–201.

Hartmann, Michael. 1987. "Past and Recent Attempts to Model Mortality at All Ages." *Journal of Official Statistics* 3:19–36.

Heligman, Larry and John H. Pollard. 1980. "The Age Pattern of Mortality." *Journal of the Institute of Actuaries* 107:49–80.

Hosegood, Victoria, Anna-Maria Vaenneste, and Ian M. Timæus. 2004. "Levels and causes of adult mortality in rural South Africa: the impact of AIDS." *AIDS* 18:663–667.

INDEPTH. 2002. "INDEPTH Mortality Patterns for Africa." In *Population and Health in Developing Countries*. Ottawa: IDRC Press.

Karim, Q. Abdool and Salim S. Abdool Karim. 1999. "Epidemiology of HIV Infection in South Africa." *AIDS* 13:4–7.

Lynch, Scott M. and J. Scott Brown. 2005. "A New Approach to Estimating Life Tables with Covariates and Constructing Interval Estimates of Life Table Qunatities." *Sociological Methodology* 35:189–237.

Marston, Milly, Jim Todd, Judith R. Glynn, Kenrad E. Nelson, Ram Rangsin, Tom Lutalo, Mark Urassa, Sam Biraro, Lieve Van der Paal, Pam Sonnenberg, and Basia Żaba. 2007. "Estimating 'net' HIV-related mortality and the importance of background mortality rates." *AIDS* 21:S65–S71.

McNown, Robert and Andrei Rogers. 1989. "Forecasting Mortality: A Parameterized Time Series Approach." *Demography* 26:645–660.

Newell, Marie-Louise, Heena Brahmbhatt, and Peter D. Ghys. 2004. "Child mortality and HIV infection in Africa: a review." *AIDS* 18:S27–S34.

Ngom, Pierre and Samual Clark. 2003. "Adult Mortality in the Era of HIV/AIDS: Sub-Saharan Africa." New York. Population Division, United Nations.

Nyirenda, Makandwe, Victoria Hosegood, Till Bärnighausen, and Marie-Louise Newell. 2007. "Mortality levels and trends by HIV serostatus in rural South Africa." *AIDS* 21:S73–S79.

Poit, Peter, Michael Bartos, Peter D. Ghys, Neff Walker, and Bernhard Schwartländer. 2001. "The global impact of HIV/AIDS." *Nature* 410:968–973.

Poole, David and Adrian Raftery. 2000. "Inference for Deterministic Simulation Models: The Bayesian Melding Approach." *Journal of the American Statistical Association* 95:1244–1255.

Porter, Kholoud and Basia Zaba. 2004. "The empirical evidence for the impact of HIV on adult mortality in the developing world: data from serological studies." *AIDS* 18:S9–S17.

Raftery, Adrian and Le Bao. 2009. "Estimating and Projecting Trends in HIV/AIDS Generalized Epidemics Using Incremental Mixture Importance Sampling." Technical Report 560, Department of Statistics, University of Washington.

Raftery, Adrian, G.H. Givens, and J.E. Zeh. 1995. "Inference from a deterministic population dynamics mode for bowhead whales." *Journal of the American Statistical Association* 90:402–416.

Roger, Andrei. 1987. "Parameterized multistate population dynamics and projections." *Journal of the American Statisitcal Association* 81:48–61.

Rogers, Andrei and Kathy Gard. 1991. "Applications of the Heligman/Pollard Model Mortality Schedule." *Population Bulletin of the United Nations* 30:79–105.

Rubin, D. 1988. "Using the SIR algorithm to simulate posterior distributions." In *Bayesian Statistics 3: proceedings of the third valencia international meeting, June 1-5, 1987*, edited by J.M. Bernardo, M.H. DeGroot, D.V. Lindley, and A.F.M. Smith. Clarnedon Press.

Smith, Jennifer, Phyllis Mushati, and Felicia Kurwa. 2007. "Changing patterns of adult mortality as the HIV epidemic matures in Manicaland, eastern Zimbabwe." *AIDS* 21:S81–S86.

Steele, Russell J., Adrian Raftery, and Mary J. Edmond. 2006. "Computing Normalizing Constants for Finite Mixture Models via Incremental Mixture Importance Sampling (IMIS)." *Journal of Computational and Graphical Studies* 15:712–734.

Timæus, Ian M. and Momodou Jasseh. 2004. "Adult Mortality in Sub-Saharan Africa: Evidence from Demographic and Health Surveys." *Demography* 41:757–772.

Todd, Jim, Judith R. Glynn, Milly Marston, Tom Lutalo, Sam Biraro, Wambura Mwita, Vinai Suriyanon, Ram Rangsin, Kenrad E. Nelson, Pam Sonnenberg, Dan Fitzgerald, Etienne Karita, and Basia Żaba. 2007. "Time from HIV seroconversion to death: a collaborative analysis of eight studies in six low and middle-income countries before highly active antiretroviral therapy." *AIDS* 21:S55–S63.

Tollman, Steven M., Kathleen Kahn, Michel Garenne, and John S.S. Gear. 1999. "Reversal in mortality trends: evidence from the Agincourt field site, South Africa, 1992– 1995." *AIDS* 13:1091–1097.

UNAIDS. 2002. "Report on the global HIV/AIDS epidemic."

Zaba, Basia, Milly Marston, Amelia C. Crampin, Raphael Isingo, Sam Biraro, Till Bärnighausen, Ben Lopman, Tom Lutalo, Judith R. Glynn, and Jim Todd. 2007. "Age-specific mortality patterns in HIV-infected individuals: a comparative analysis of Africa community study data." *AIDS* 21:S87–S96.

Zaba, Basia, Alan Whiteside, and J. Ties Boerma. 2004. "Demographic and socioeconomic impact of AIDS: taking stock of the empirical evidence." *AIDS* 18:S1–S7.

Appendix A - Observed Person Years and Deaths, Females

1994-1997		1998-2001		2002-2004		2005-2007	
PY	Deaths	PY	Deaths	PY	Deaths	PY	Deaths
3771.5	59	3581.8	83	2382.8	91	2567.8	114
16100.3	60	14373.1	82	10216.0	75	9555.2	60
20370.5	17	19605.3	12	13282.0	13	12666.4	11
17551.3	9	18797.4	12	14319.4	17	13365.6	18
14853.5	21	15989.0	17	12842.0	27	13536.5	17
13002.8	17	13739.6	50	10774.3	62	11542.8	67
10749.3	18	11466.2	51	9056.7	101	9354.3	113
9163.4	27	9439.5	56	7358.1	103	7436.0	129
7026.6	25	8027.5	61	6220.5	86	6172.2	107
5831.2	23	6200.4	54	4953.4	73	5418.5	79
3973.7	14	5034.3	34	4130.0	56	4153.5	78
3248.9	21	3496.2	33	3174.8	57	3526.6	76
3140.5	15	3114.2	26	2298.6	35	2577.5	47
3042.2	38	2860.8	44	2139.9	41	2039.0	43
2834.0	60	2930.6	48	1903.7	47	1894.7	41
1742.7	53	2260.2	73	2091.9	46	1725.9	53
1200.9	55	1389.5	56	1091.5	33	1682.0	63
441.0	37	720.9	37	731.4	51	710.4	37
209.1	28	258.2	16	225.7	22	447.0	38
99.1	6	99.0	9	109.8	14	110.9	7
37.3	8	40.1	9	35.0	6	55.6	5
46.0	9	21.4	4	9.2	2	14.9	1

Appendix A - Observed Person Years and Deaths, Males

1994-1997		1998-2001		2002-2004		2005-2007	
PY	Deaths	PY	Deaths	PY	Deaths	PY	Deaths
3795.5	55	3485.6	103	2332.6	104	2587.7	91
16183.6	66	14306.2	91	10041.9	86	9321.9	58
20452.0	12	19640.2	12	13139.5	15	12326.2	21
17469.8	15	18880.2	17	14463.3	15	13113.1	13
15055.9	18	16318.0	13	12885.9	23	13803.1	18
11798.2	22	13571.6	36	11080.0	53	11514.0	37
9543.1	37	10626.8	41	8730.0	82	9418.5	100
7803.9	34	8508.8	65	6696.8	107	7005.0	131
5863.0	44	6727.7	63	5439.4	104	5505.2	142
4833.8	37	5074.3	56	4082.4	81	4488.1	127
3810.7	48	4317.5	67	3233.4	73	3244.9	92
2698.6	41	3138.9	41	2762.8	65	2759.4	91
2461.2	49	2414.3	38	1823.0	58	2102.4	78
1617.2	38	2090.5	38	1637.4	71	1389.4	65
1529.5	47	1410.4	45	1101.4	43	1350.3	65
1190.9	54	1202.1	57	898.0	39	722.2	39
848.9	58	888.6	66	556.7	55	674.8	40
328.6	25	503.9	39	455.1	35	356.1	44
126.1	12	194.3	25	175.9	23	265.2	31
45.5	7	51.6	4	60.0	10	69.9	9
13.5	2	16.8	4	12.4	3	22.2	8
32.7	2	11.0	1	0.1	0	3.0	0