

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

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## Introduction

A good deal 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 (Poit et al. 2004). Throughout the 1990s and into the 21st century, sub-Saharan Africa and South Africa specifically have experienced elevating adult mortality, which has been linked to escalating HIV prevalence over this period ((Ngom and Clark 2003, Hosegood et al. 2004). In this paper, we utilize a law of mortality, the Heligman-Pollard law, estimated via a Bayesian Melding procedure, to assess rapidly changing mortality schedules for men and women in a rural population of South Africa. Despite having been used in a variety of other-mostly economically developed - contexts, (Heligman and Pollard 1980, Rogers and Gard 1991, others) the Heligman-Pollard model has been under utilized in assessing this growing mortality. The ability of parametric models like the Heligman-Pollard to easily summarize changes in mortality over time via some elegant demographic parameter interpretations, make these methods especially valuable for researchers concerned with dynamic mortality profiles in the presence HIV/AIDS. The paper also reports on the relationship between growing HIV prevalence in this region and the change in the Heligman-Pollard parameters over this time period.

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 with a section on the Heligman-Pollard model and interpretation of its parameters. We follow with a description of the methods, results and discussion of our findings as related to the age pattern of mortality resulting from HIV.

## The sex-age pattern of adult mortality in the presence of HIV/AIDS

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

Despite limitations stemming from data which is often not of the highest quality, researchers have managed to identify several age and sex-specific effects of HIV.<sup>1</sup> Prior to the introduction of HIV/AIDS, life expectancy was gaining and probabilities of adult male deaths were in a reasonable range with fewer than half of all survivors from age 15 dying before age 60. In southern Africa, where the epidemic is advanced, male adult mortality is now considerably higher and over half of all those who make it to age 15 can expect to die before age 60 (Ngom and Clark 2003, Poit et al. 2001). 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 2004). This implausibility notwithstanding, INDEPTH data (2002) uncovered seven patterns of mortality from 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 and 55 for males and 20-45 for females (Ngom and Clark 2003). The age pattern of 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).

Female adult mortality levels tend to follow similar patterns to those for males but with some notable differences. Using data from 35 African countries affected by HIV, Ngom and Clark (2003, p. 6) find that overall mortality between ages 15 and 49 increases in similar increments for men and women as HIV prevalence increases but that the male and female profiles of increases in HIV/AIDS-related age-specific probabilities of death are dissimilar. This differential impact is largely driven by divergent ages of increased prevalence. As a consequence of the fact that sexual intercourse patterns are dominated by younger women

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<sup>1</sup>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 200). In addition to the lack of available vital registration data, 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 of data quality and underreporting extend to South Africa as well (Botha et al. 1985 , Tollman et al. 2001).

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).

It should come as no surprise that research concerning the relationship between adult HIV prevalence and age-specific mortality points to a strong association between these two phenomenon. Hosegood et al. using data from a rural South African province (2003, p. 669) reported that AIDS mortality reflects the HIV seroprevalence rate from approximately five years prior. Likewise, Porter and Zaba (2004, p. S23) and Ngom and Clark (2003) report a strong association between increases in adult HIV prevalence and increasing probabilities of adult death, although Ngom and Clark report little impact on mortality at other ages. Evidenced by the fact that countries with low prevalence levels experience probabilities of death between ages 15 and 60 of less than 40 percent while high prevalence areas experience probabilities of death of over half, it is likely that differentials in adult mortality levels are in large part explained by HIV/AIDS (Ngom and Clark 2003, p. 3; Porter and Zaba 2004; Hosegood et al. 2004 p. 670, Tollman et al. 2001).

In sub-Saharan Africa, HIV infection is a growing source of childhood mortality as well. 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 effected 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 (Debis and Ekpini 2002, De Cock et al. 2000 cited in Newell et al. 2004, Foster and Wiliamson 2000). 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, p. S32).

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, South Africa Department of Health 2001) and while there was virtually no HIV mortality at the beginning of the 1990s, by 2000 there was significant HIV-related mortality (Ngom and Clark 2003, Dorrington et al. 200). 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 (Hosegood et al. 2004, Tollman et al. 1999). These increases in mortality for South Africa mirror the reversals in life expectancy found in other parts of southern Africa (Hosegood et al. 2004, Tollman et al 1999).

## 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 (1980), presented in equation one, 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). Although, unlike many other mortality laws, like that put forth early on by Gompertz (1825), which covers the steep increase in mortality in late life, the Heligman-Pollard is a three-part model that covers the entire age range for all  $x > 0$ .<sup>2</sup>

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

Parametric methods, like the Heligman-Pollard, have several advantages for describing mortality, including smoothness, interpolation, analytic manipulation, parsimony, comparison and trends (Dellaportas 2001). These last two advantages, along with the ability of parametric methods to easily and succinctly summarize large amounts of data, 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 (Debn 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, Debn et al. 2005, Congdon 1993, Forfar and Smith 1987, Hartmann 1983, Rogers 1986 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 changes in less developed nations and has not no been used to assess mortality in a high HIV prevalence setting. 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.

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, Rogers and McKnown 1989, Rogers and Gard 1991). Figure one 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.<sup>3</sup>

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<sup>2</sup>For age 0 one can use a very small number like 0.00001 or the formula described in Rogers and Gard 1991, pg. 80

<sup>3</sup>Figures 1 and 2 are plotted using the following set of parameters, which can represent the nearly 100 probabilities of Brass' standard (Rogers and McKnown 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

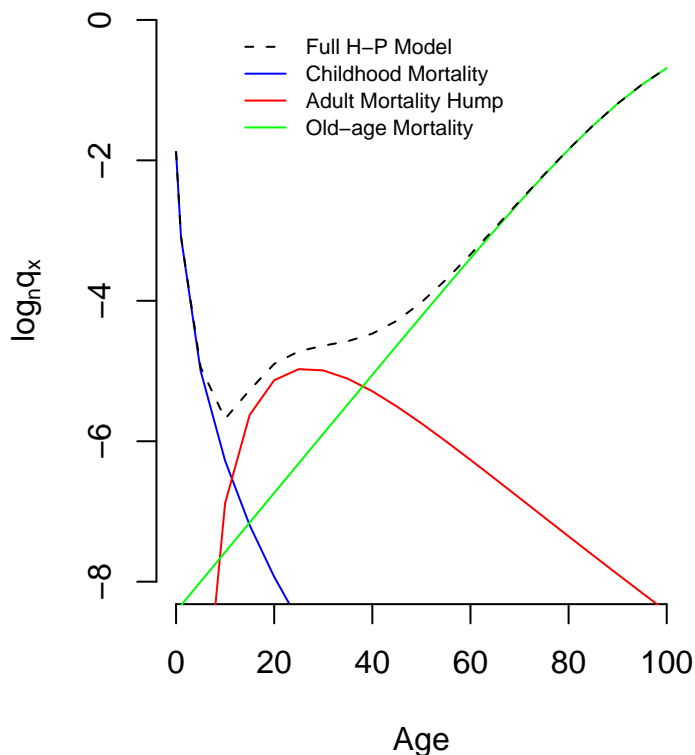


Figure 1: Decomposition of the Heligman-Pollard Model

Table 1 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 (Rogers and McKnown 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.

Table 1: Heligman-Pollard Parameters

Parameter	Description
A	Intensity of Childhood Mortality ( $\approx_1 q_1$ )
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 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, Rogers and McKnown 1989, Rogers and Gard 1991, Hartmann 1987). This hump would normally have a peak around age 20 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 adult mortality, E describes and is inversely related to the spread of the hump and F locates the position (Heligman and Pollard 1980, Rogers and McKnown 1989, Rogers and Gard 1991). Parameters D and E take domains  $(0, 1)$  and  $(0, \infty)$  respectively while the domain of F is less restrictive. This paper uses  $(15, 55)$  since we do not expect a large cotribution of HIV deaths in the oldest years. 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 spreads we might 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 matter much. The eight panels of figure two 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 other constant. After an inspection of the equation and the panel for parameter D, one can see that the effects of E and F are multiplied by D, so a small D essentially negates the effect of the spread parameter and location parameters.

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  $(0, \infty)$  respectively.

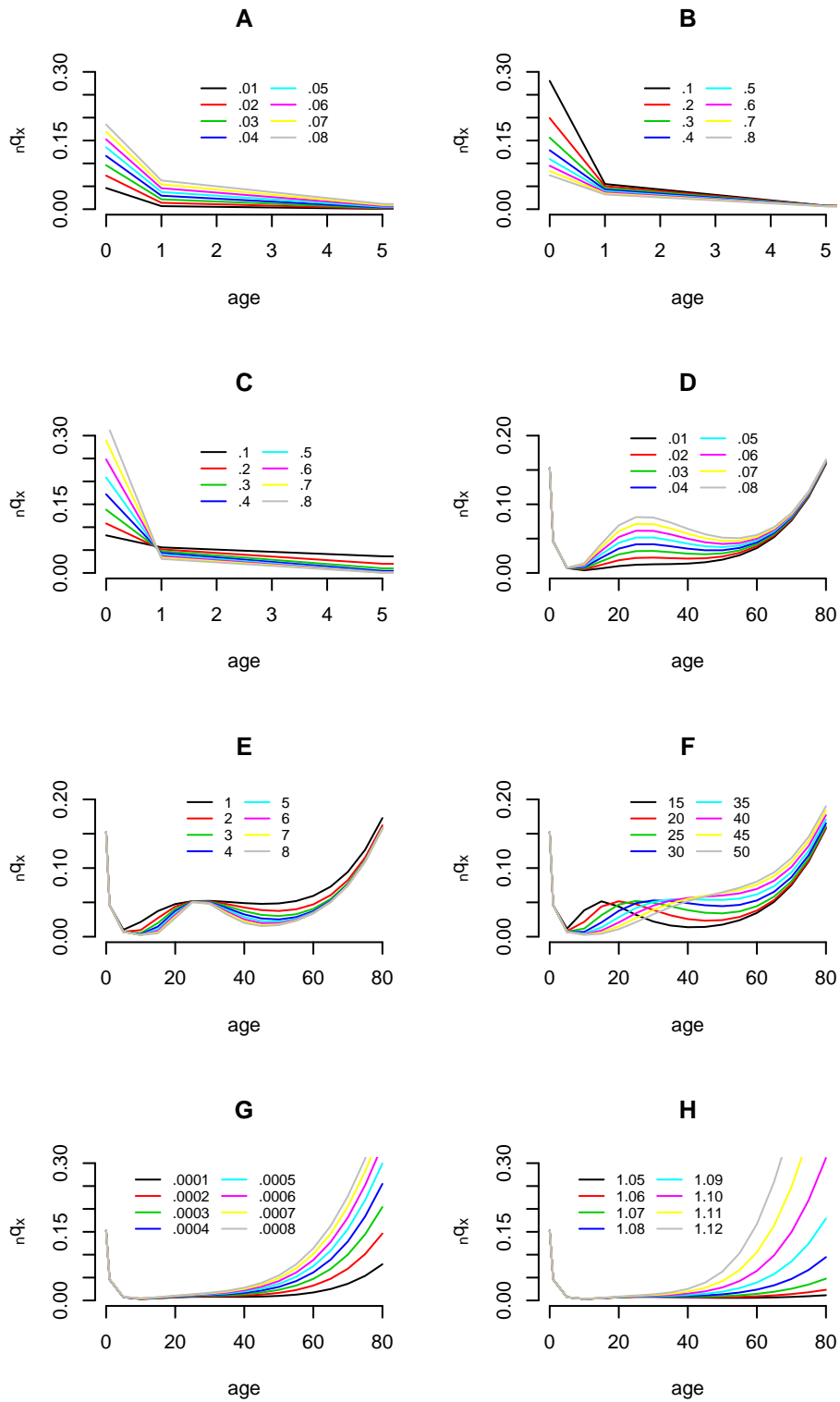


Figure 2: Resulting lines after changing the value of a single parameter while holding all others constant. For the plots of parameter E and F, D is set to .04 to give sufficient intensity to the hump.

As demonstrated with the discussion above concerning the effects of the middle three parameters, these 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 effect may be either diminished or intensified depending on the value of some other parameter.

## Methods and Data

### Data

We use two types of data in this analysis - mortality and prevalence data. We obtained mortality data come from Agincourt field site located in Mpumalanga province, South Africa, a rural area with HIV prevalence currently hovering around 30. Using age-specific (ages 0-100) death counts and person years for the period 1994-2007, we compute the age specific probabilities of death ( ${}_nq_x$ ). Then, 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.<sup>4</sup> The observed person years and deaths counts can be found in appendix A.

Because the individual year data is quite variable in terms of the  ${}_nq_x$  values, especially at older ages, and because sample sizes are somewhat small for individual years after the 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 ( $l_0\text{male} = 1,917$ ,  $l_0\text{female} = 1,873$ ), 1998-2001 ( $l_0\text{male} = 2,100$ ,  $l_0\text{female} = 2,049$ ), 2002-2004 ( $l_0\text{male} = 1,896$ ,  $l_0\text{female} = 1,822$ ) and 2005-2007 ( $l_0\text{male} = 1,974$ ,  $l_0\text{female} = 1,893$ ). The emerging mortality hump is easily recognizable in figure three, which presents the post-grouping mortality schedules for males and females.

### Methods

In the first part of this analysis we obtain parameter estimates via a Bayesian Melding procedure. The Heligman-Pollard model is usually estimated via least squares methods but over-parameterization has been revealed to be an issue using this technique. Dellaportas et al. (2001) used a Bayesian approach and reported that the over-parameterization issue is usually resolved. We, too, take a Bayesian approach using Bayesian Melding (Poole and Raftery 2000) with the posterior distributions estimated via an incremental mixture importance sampling procedure (Hesterberg 1995, Steele et al 2006, Bao and Raftery ND).<sup>5</sup> The Bayesian melding approach allows for the combination of expert opinion as well as past trends in inputs and outputs in the form of prior distributions.

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<sup>4</sup>We use the "solver" function in Excel as the optimizer for obtaining the persons at risk.

<sup>5</sup>See Bao and Raftery ND for a full accounting of Bayesian Melding using IMIS

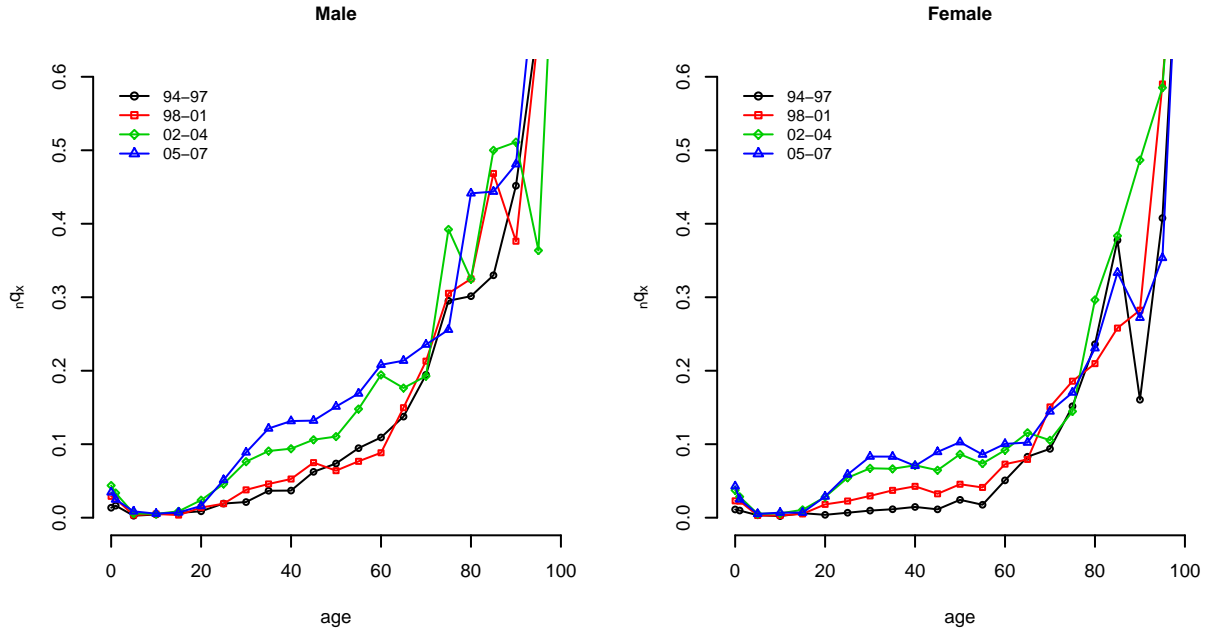


Figure 3: Age pattern of the probability of death, Agincourt 1994-2007

In a Bayesian framework, information on prior beliefs about the parameters can be incorporated in the form of probability densities  $p(\theta)$ , which is a vector of the parameter to be estimated. We define the prior distributions for the eight parameters by first estimating the model parameters and respective standard errors via maximum likelihood and then use a uniform distribution with bounds  $\pm 2 * se$  to create a uniform distribution four standard errors wide. Observed data,  $\mathbf{y}$ , are incorporated by way of specifying a likelihood,  $\mathcal{L}(\mathbf{y}|\theta)$ , which is the probability of observing the data for a given set parameters values. We can then update prior beliefs using Bayes' Theorem and the marginal density of the data,  $p(\mathbf{y})$ , to obtain the posterior distribution, which is used to make inference for  $\theta$ " (Clark, Thomas, Bao and Raftery ND). For each set of sampled parameter inputs, we derive the likelihood based on a binomial distribution.

Bayesian Melding is an appropriate technique when a deterministic model is used in the likelihood - the Heligman Pollard in this case. If  $M$  represents the model which transforms a set of parameter inputs,  $\theta$ , into a set of outputs, then  $\phi = M(\theta)$ . The bayesian approach combines information from the prior density for the model inputs and a likelihood for the outputs and data to produce a posterior distribution,

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

As stated above, a Bayesian approach, like the one used here, produces a posterior distribution of the estimated parameters. 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. Figures four and five present the plots of the posterior 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 400 separate lines resulting from the posterior distribution of parameters.

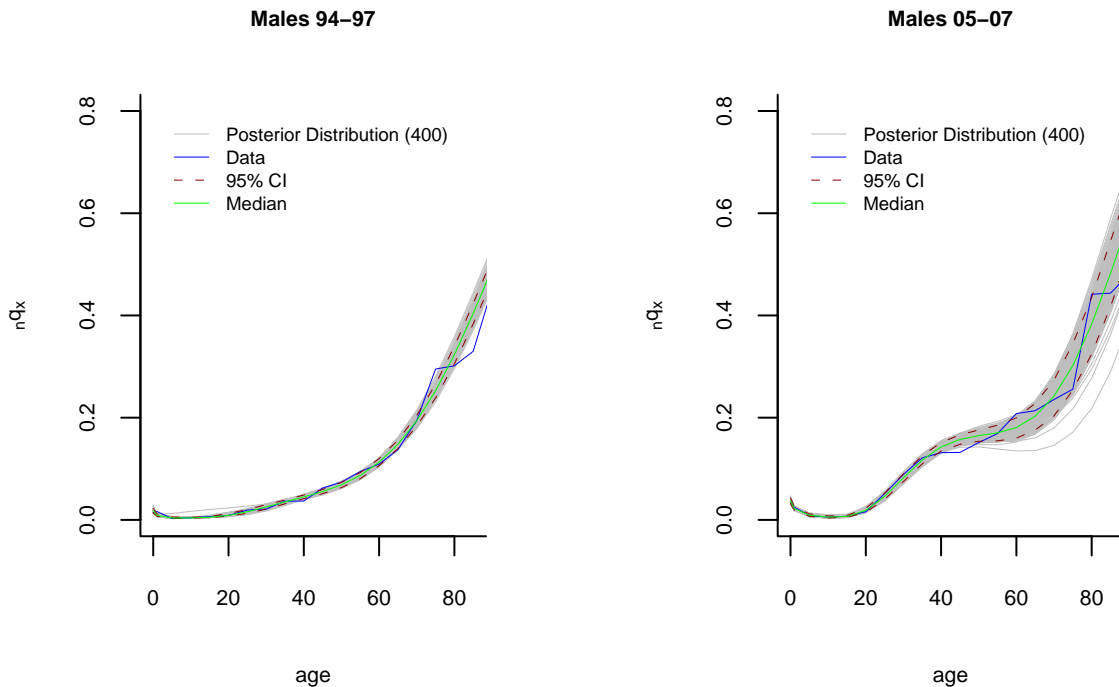


Figure 4: Plotted posterior distributions from a flat hump (Males 94-97) and an intense hump (Males 05-07)

### Relationship between parameters and HIV prevalence

Because of the established 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 relationship between the median value for each of the

parameter distributions and the mean HIV prevalence in the province with a five year lag. We expect that the correlations will show increased HIV prevalence to be positively related to both child and adult mortality intensity and positively related to the location parameter. Table three presents the mean HIV prevalence levels for five-year lagged periods of interest.<sup>6</sup>

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

## Results

Table three presents the median parameter estimates for each sex-specific group of years. We will first review trends in child mortality followed by an interpretation of the hump parameters.

**Childhood Mortality** Recall that we expected that childhood mortality should see 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 in each of the year groups. Figure six plots the value of parameters A, D, E and F over this period for both sexes. The upper left panel of figure six, which plots the time series of parameter A, bares this expectation out. The estimates of parameter A suggest increasing childhood mortality levels for both males and female although we only see a significant increase for males between periods one and two where the 95% confidence intervals do not overlap.

For child mortality neither sex has a consistent advantage over this period. In the 1990s, females enjoyed a childhood mortality intensity advantage, which reverses only in the final period. Although we do see a generally increasing trend in child mortality for both sexes, the level of male and female childhood mortality does not appear to be significantly different in any period.

**Adult Mortality** Trends in the level or intensity of adult mortality largely follow those of childhood mortality. The upper right panel of figure six plots parameter D for males and females over this period. Similar, to the trajectory of parameter A, parameter D also increases consistently from each period to the next and is significantly higher from each

<sup>6</sup>source: Department of Health, Republic of South Africa (1996, 1997, 1998, 2003, 2006, 2008)

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

	94-97	98-01	02-04	05-07
Males				
A	0.0153 ( 0.0117 - 0.0186 )	0.0290 ( 0.0223 - 0.0349 )	0.0331 ( 0.0210 - 0.0454 )	0.0339 ( 0.0285 - 0.0389 )
B	0.9278 ( 0.7756 - 0.9940 )	0.9455 ( 0.8226 - 0.9968 )	0.6600 ( 0.4623 - 0.8602 )	0.8779 ( 0.6970 - 0.9889 )
C	0.2470 ( 0.1704 - 0.3266 )	0.2397 ( 0.1832 - 0.2940 )	0.2853 ( 0.1976 - 0.3789 )	0.1919 ( 0.1573 - 0.2247 )
D	0.0186 ( 0.0154 - 0.0218 )	0.0369 ( 0.0320 - 0.0416 )	0.0659 ( 0.0578 - 0.0747 )	0.1383 ( 0.1292 - 0.1475 )
E	4.2099 ( 2.1433 - 6.2789 )	5.0254 ( 4.1491 - 5.8259 )	2.8357 ( 1.7898 - 3.7911 )	3.2903 ( 2.7552 - 3.8817 )
F	41.7839 ( 36.3298 - 47.0432 )	37.6341 ( 35.8483 - 39.3958 )	42.1647 ( 38.7163 - 45.3689 )	45.8965 ( 43.5435 - 48.2138 )
G	0.0016 ( 0.0014 - 0.0019 )	0.0009 ( 0.0008 - 0.0010 )	0.0027 ( 0.0026 - 0.0028 )	0.0003 ( 0.0002 - 0.0004 )
H	1.0736 ( 1.0712 - 1.0758 )	1.0833 ( 1.0820 - 1.0848 )	1.0674 ( 1.0654 - 1.0692 )	1.0983 ( 1.0921 - 1.1041 )
Females				
A	0.0128 ( 0.0078 - 0.0164 )	0.0224 ( 0.0156 - 0.0291 )	0.0243 ( 0.0170 - 0.0322 )	0.0407 ( 0.0304 - 0.0502 )
B	0.8954 ( 0.7033 - 0.9937 )	0.5930 ( 0.3330 - 0.8603 )	0.3923 ( 0.1340 - 0.6595 )	0.8812 ( 0.6196 - 0.9940 )
C	0.0921 ( 0.0493 - 0.1343 )	0.1909 ( 0.1392 - 0.2459 )	0.1523 ( 0.0943 - 0.2050 )	0.2092 ( 0.1525 - 0.2705 )
D	0.0081 ( 0.0054 - 0.0112 )	0.0263 ( 0.0236 - 0.0293 )	0.0657 ( 0.0610 - 0.0699 )	0.0967 ( 0.0896 - 0.1022 )
E	5.8950 ( 3.6253 - 8.6599 )	2.3411 ( 1.8499 - 2.8457 )	2.2812 ( 1.8346 - 2.7116 )	2.5920 ( 2.3104 - 2.8975 )
F	34.5173 ( 30.6406 - 38.2713 )	37.0586 ( 34.6584 - 39.5098 )	38.0472 ( 36.5055 - 39.6683 )	41.0491 ( 39.2749 - 43.3159 )
G	0.0003 ( 0.0002 - 0.0003 )	0.0003 ( 0.0002 - 0.0003 )	0.0001 ( 0.0001 - 0.0001 )	0.0001 ( 0.0000 - 0.0001 )
H	1.0962 ( 1.0929 - 1.1000 )	1.0918 ( 1.0882 - 1.0958 )	1.1081 ( 1.1007 - 1.1153 )	1.1121 ( 1.1087 - 1.1157 )

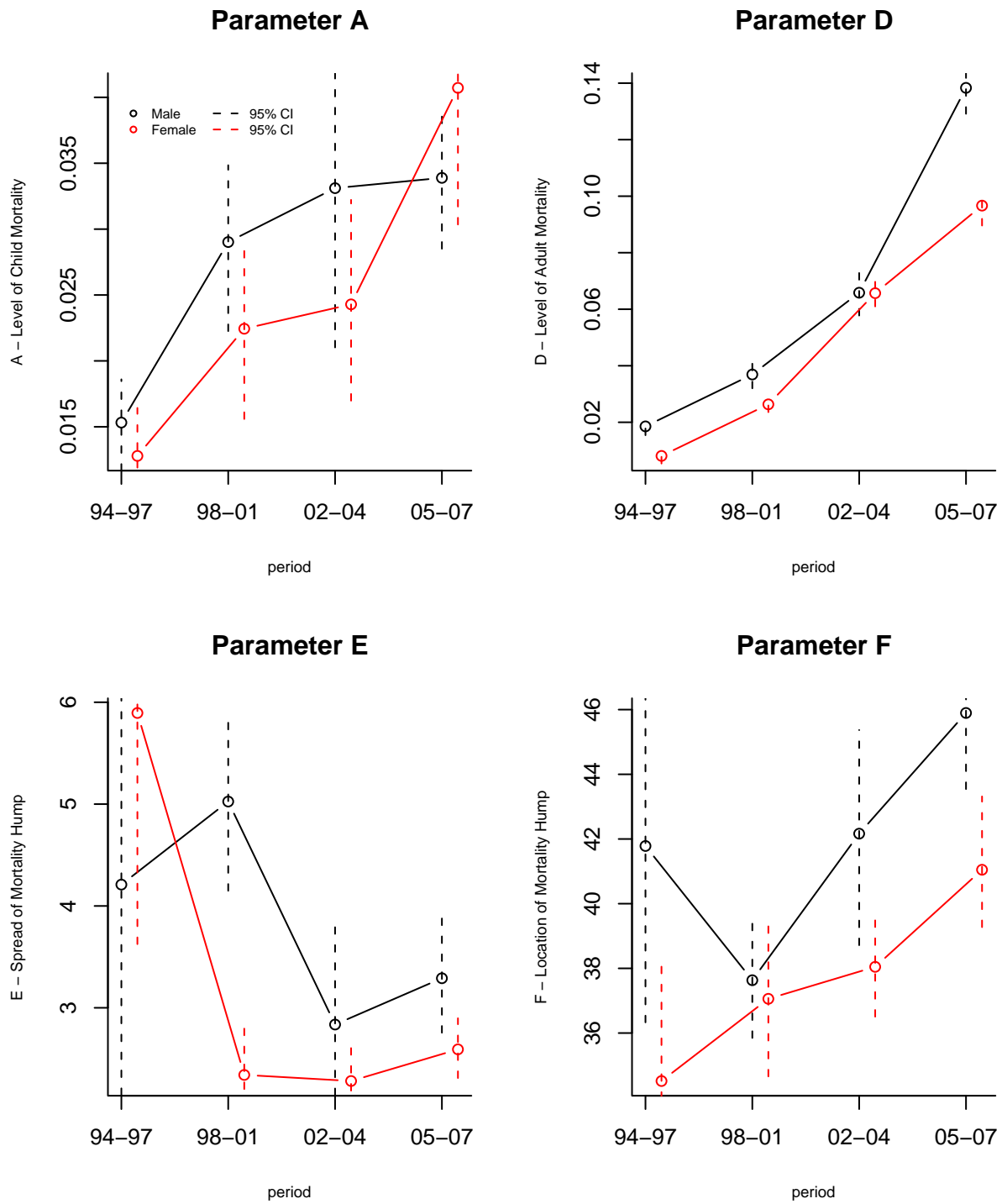


Figure 5: Times Series of A, D, E and F

period to the next for both males and females within sex. Figure seven plots the median lines for each group of years and for each sex over the adult years (15-60) while figure eight plots only the middle hump component of the HP model. It is clear from these plots that in all periods except for the third, the intensity of adult of mortality grows significantly from period to period. Likewise figure five suggests that the intensity of adult mortality is significantly higher for males in all periods except the third.

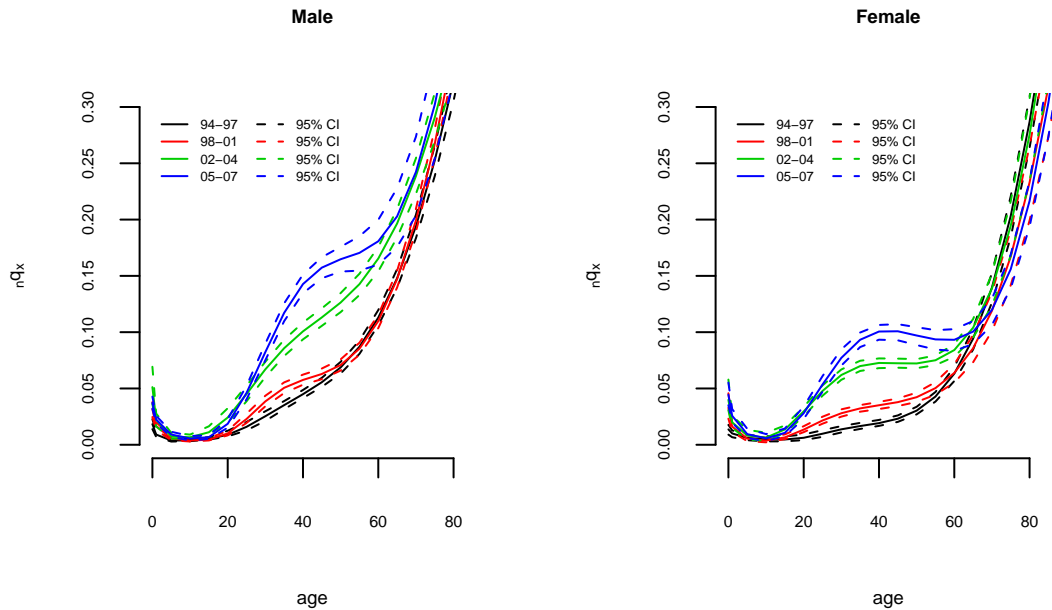


Figure 6: Median fitted curves with 95% CI plotted

Along with increasing intensity, increasing values of parameter  $F$ , plotted in the lower right panel of figure six, suggest an aging mortality hump for both sexes. Recall that females generally contract the disease at younger ages than men and thus die at younger ages. The location parameter accurately reflects this previously observed pattern with the median estimate of  $F$  being consistently higher for males. In the final period, the male mortality location parameter is significantly older than the female estimate of  $F$ . Females see growing mortality from the late 30s to around 40 while the male hump matures from about roughly 38 to 45. For males the value of parameter  $F$  actually decreases from the first period to the second, but because parameter  $D$  is not sufficiently large enough to create a hump, the age location parameter is somewhat irrelevant. As the intensity becomes sufficiently large to generate a hump (probably around the second period since the first is relatively flat), parameter  $F$  increases to reflect the ageing mortality hump.

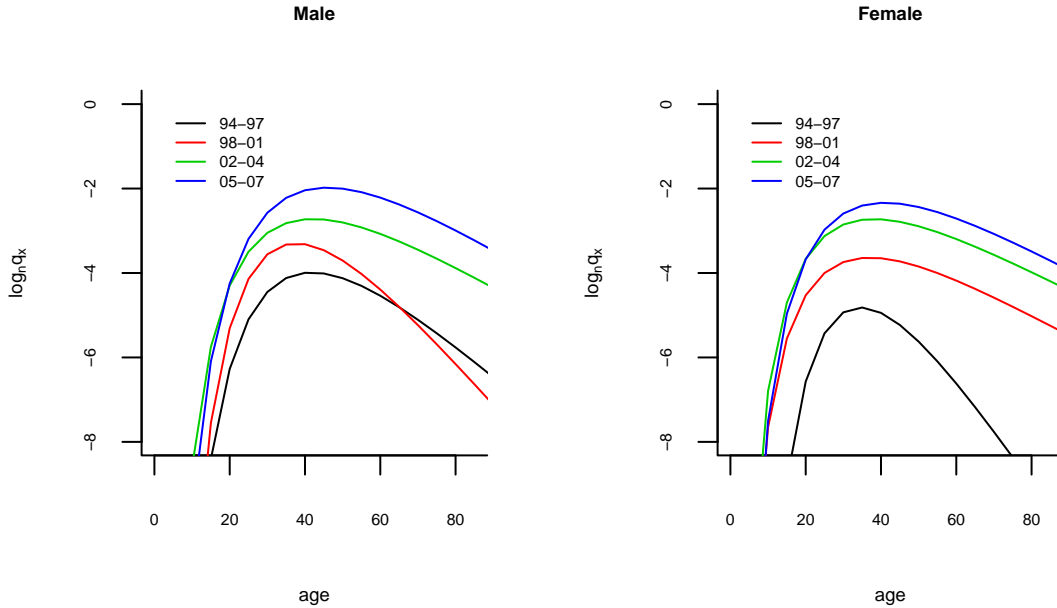


Figure 7: Hump Component for all periods and both sexes

Trends in the spread of the hump are somewhat less consistent than either the intensity or location of the hump but do show a general broadening of the mortality hump. Females see a significant decrease in  $E$  between the first two periods indicating a less concentrated adult mortality schedule while the spread parameter decreases significantly for men between the second and third periods. The large CIs around the estimates for parameter  $E$  in the first period reflect the wide range of values  $E$  can take on probably because parameter  $D$  is relatively small for both sexes in the first period. Although neither change is significant, both sexes do see a slight increase from period three to the final period suggesting a slight concentration.

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 eight plots the change in parameters  $A$ ,  $D$ ,  $E$  and  $F$  along with the corresponding 5-year lagged HIV prevalence. For all periods and both sexes, the intensity parameters,  $A$  and  $D$  increase with increasing HIV prevalence. As HIV prevalence increased so did the level of child and adult mortality over these four periods. Parameters  $E$  and  $F$  show less consistent patterns but there does appear to be a general increase in the location parameter for both men and women after the first period and a general decline in parameter  $E$  for both sexes although the majority of the decline appears to happen most rapidly for males between the second and third period whereas for females it occurs between the first and second period.

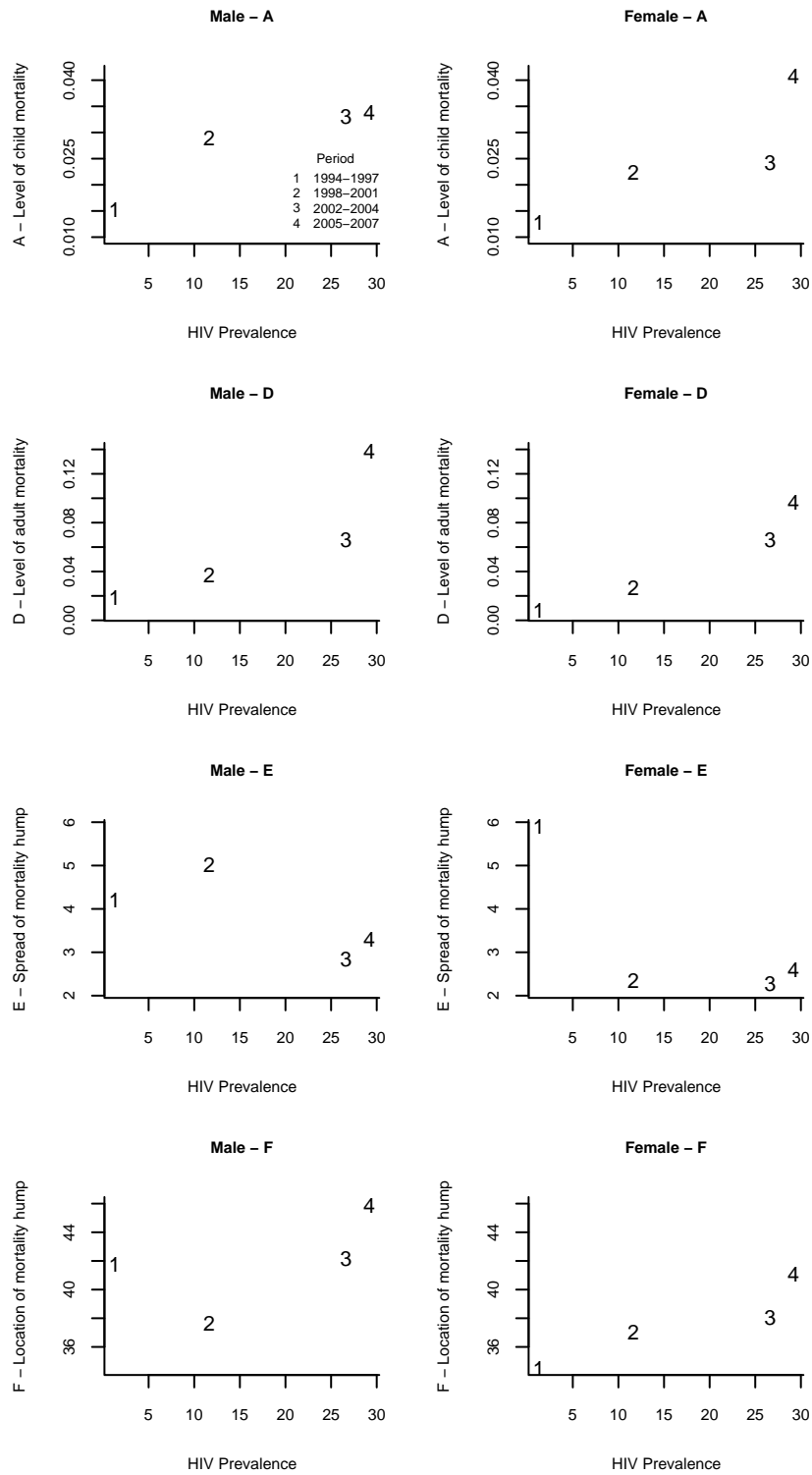


Figure 8: Selected Parameter Values by 5-year lagged HIV prevalence

## Summary

As the HIV epidemic matures, changes in the mortality schedule can be easily summarized via parametric methods. 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 mortality and adult mortality are consistent with the findings on age-specific HIV-related mortality. The losses of previous gains in life expectancy as a result of HIV are reflected in the growing intensity of mortality among both adults and children. Although the increasing childhood mortality cannot be clearly 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 large contribution of HIV to childhood survival.

Likewise, other features of a growing HIV epidemic are reflected in the hump parameters. The location parameter progresses to be 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 ( $F$ ) value. From the second to the third period, mortality broadens as expected but in the latter two periods, mortality narrows and becomes slightly more concentrated for both men and women. The decreasing spread may be reflecting stabilization in HIV-related mortality as prevalence reaches a plateau. Also consistent with prior research, increasing HIV prevalence contributes to both elevated child and adult mortality.

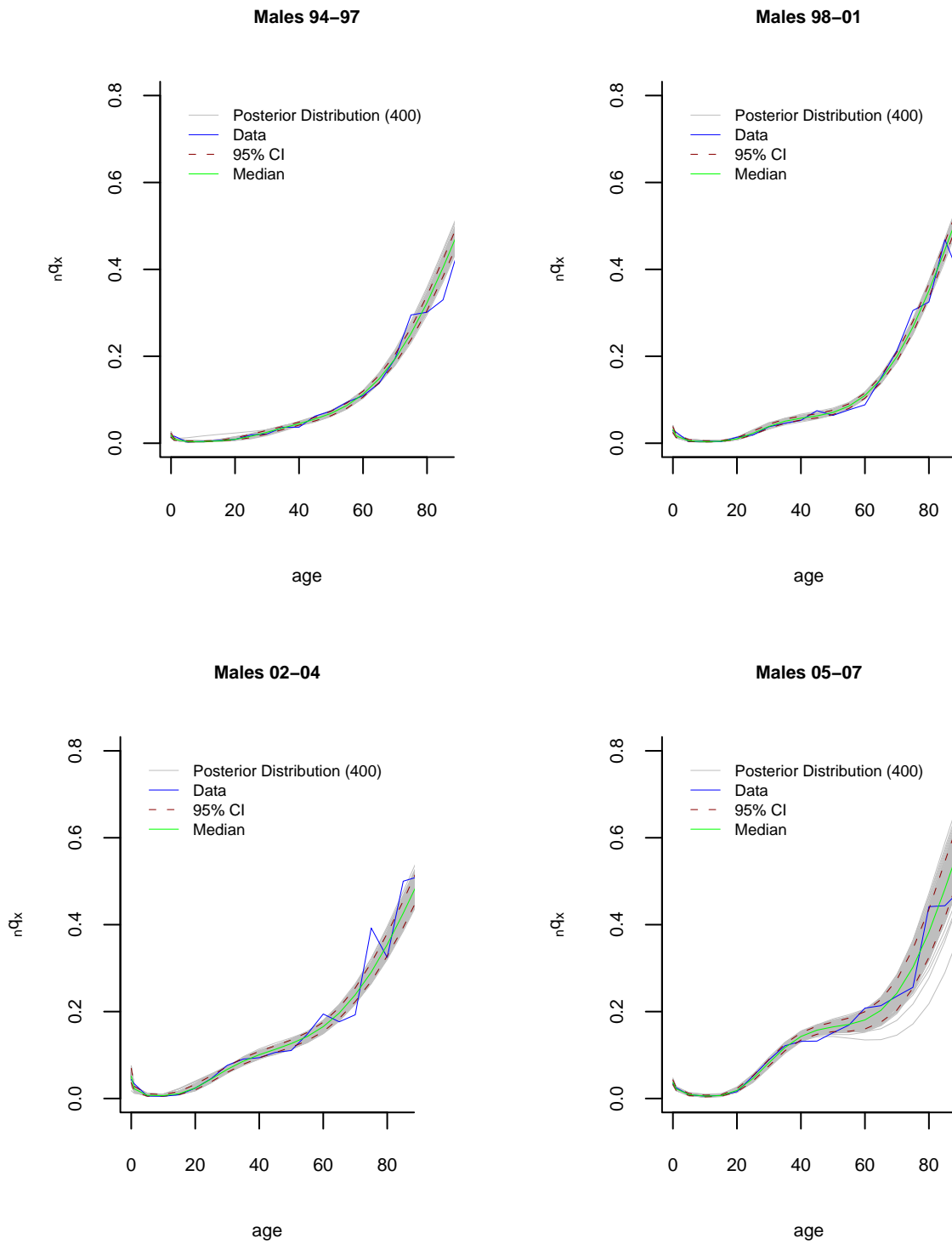
## Appendix A - Observed Person Years and Deaths, Females

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

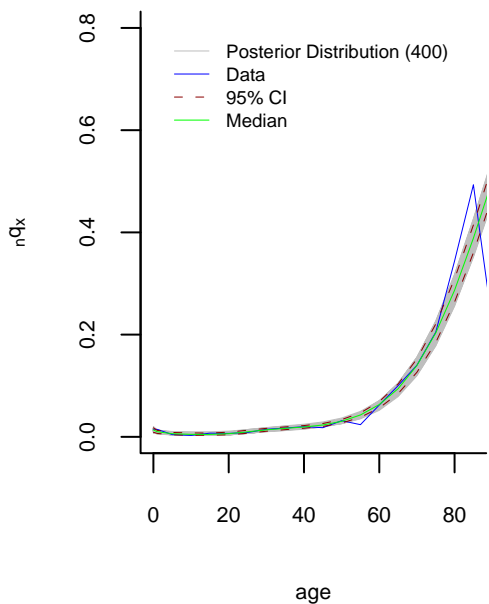
## Appendix A - Observed Person Years and Deaths, Males

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

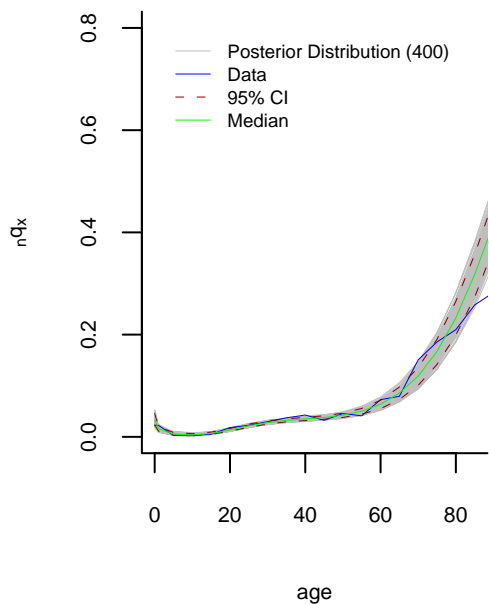
## Appendix B - Plotted posterior distributions



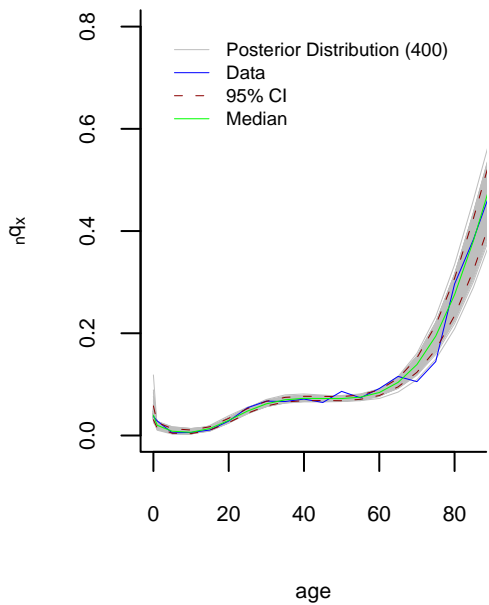
**Females 94-97**



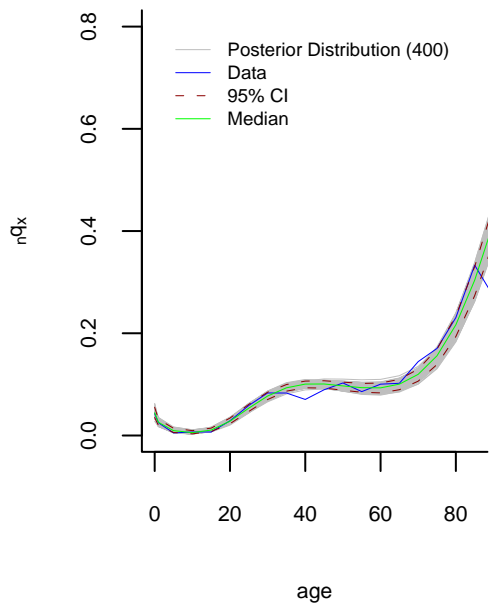
**Females 98-01**



**Females 02-04**



**Females 05-07**



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