

Assessing adult mortality in HIV-1-afflicted Zimbabwe (1998–2003)

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Objective To compare alternative methods to vital registration systems for estimating adult mortality, and describe patterns of mortality in Manicaland, Zimbabwe, which has been severely affected by HIV.

Methods We compared estimates of adult mortality from (1) a single question on household mortality, (2) repeated household censuses, and (3) an adult cohort study with linked HIV testing from Manicaland, with a mathematical model fitted to local age-specific HIV prevalence (1998–2000).

Findings The crude death rate from the single question (29 per 1000 person-years) was roughly consistent with that from the mathematical model (22–25 per 1000 person-years), but much higher than that from the household censuses (12 per 1000 person-years). Adult mortality in the household censuses (males 0.65; females 0.51) was lower than in the cohort study (males 0.77; females 0.57), while mathematical models gave a much higher estimate, especially for females (males 0.80–0.83; females 0.75–0.80). The population attributable fraction of adult deaths due to HIV was 0.61 for men and 0.70 for women, with life expectancy estimated to be 34.3 years for males and 38.2 years for females.

Conclusion Each method for estimating adult mortality had limitations in terms of loss to follow-up (cohort study), under-ascertainment (household censuses), transparency of underlying processes (single question), and sensitivity to parameterization (mathematical model). However, these analyses make clear the advantages of longitudinal cohort data, which provide more complete ascertainment than household censuses, highlight possible inaccuracies in model assumptions, and allow direct quantification of the impact of HIV.

Keywords Mortality; HIV infections/mortality; Censuses; Life tables; Cohort studies; Zimbabwe (source: MeSH, NLM).

Mots clés Mortalité; Infection à VIH/mortalité; Recensement; Tables survie; Etude cohorte; Zimbabwe (source: MeSH, INSERM).

Palabras clave Mortalidad; Infecciones por VIH/ mortalidad; Censos; Tablas de vida; Estudios de cohortes; Zimbabwe (fuente: DeCS, BIREME).

Arabic

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Voir page 195 le résumé en français. En la página 196 figura un resumen en español.

Introduction

Southern Africa is the region of the world most severely affected by the human immunodeficiency virus (HIV) epidemic. In Zimbabwe, disease prevalence in 15–49-year-olds is about 25%.¹ The epidemic has stabilized in some countries since the late 1990s, but this should not obscure the fact that the peak in prevalence will precede the peak in mortality by nearly a decade because of the long incubation of HIV/acquired immune deficiency syndrome (AIDS).² A stable epidemic is a result of equal numbers of

deaths and new infections — both of which were approximately 180 000 in Zimbabwe in 2003.³

In most Southern African countries, vital registration systems are unreliable because of substantial underreporting.⁴ Hospital records can be biased, especially in rural areas, since many people die at home and AIDS deaths could be underreported to protect surviving kin from stigma. Despite these problems in recordkeeping, empirical studies have been able to show the impact that the HIV epidemic is having on mortality in sub-Saharan Africa. Between 1990 and

1995, at a time when seroprevalence of HIV was 8% in Uganda's Masaka district, 40% of all adults deaths were due to HIV infection, which accounted for as much as 70% of all deaths in 25–44-year-olds.⁵ In the mid-1990s, half of all adult deaths in Mwanza, United Republic of Tanzania, and two-thirds of adult deaths in Rakai, Uganda, could be attributed to HIV.^{6,7} Mortality rates trebled from 1991 to 2003 in Namibia.⁸ In South Africa, AIDS is the leading cause of death.⁹

Various methods are used to assess the impact of HIV on rates and patterns

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of mortality. These include Demographic and Health Surveys, national census, sub-national household census, cohort studies, indirect estimation based on sibling and orphan survival, mathematical projection models, and even examination of parish registries and cemetery records.^{8–13} The population under observation in the ongoing Manicaland HIV/STD Prevention Project,^{14,15} provided a unique opportunity to analyse trends and compare different indicators of mortality. Using these data sources, we compared alternative methods for estimating adult mortality, and determined contemporary patterns of mortality in Manicaland, Zimbabwe.

Methods

Study population

The study population was resident in four subsistence-farming areas; two roadside trading centres; four forestry, tea, and coffee estates; and two small towns in the rural province of Manicaland in eastern Zimbabwe. All local residents were counted in an initial household census (July 1998–2000), which was repeated three years later in each site. Males aged 17–54 years and females aged 15–44 years were considered eligible for a concurrent cohort study of HIV transmission. The different age ranges were chosen to reflect the different age patterns of HIV infection in Zimbabwe.

Ethical approval for the study was granted by the Research Council of Zimbabwe (Number 02187) and the Applied and Qualitative Research Ethics Committee in Oxford, United Kingdom (N97.039).

Written informed consent was obtained from all 9500 cohort participants, who were offered free HIV counselling and testing in addition to free treatment of other sexually transmitted diseases. Of each married couple, only one partner was selected. HIV serological testing was done on dried blood spots using a highly sensitive and specific antibody dipstick assay.¹⁶ Information on demographic, socioeconomic, and sexual behaviour data were collected from each individual through an interviewer-led questionnaire. Responses to sensitive questions about sexual behaviour were obtained through an informal system of voting. Respondents completed a “ballot” that they confidentially submitted to a locked box.¹⁷

Table 1. Mortality indices from single household question, household censuses, individual cohort study, and model predictions in Manicaland, Zimbabwe

Crude measures	Deaths	Time at risk (years)	Rate (per 1000 person-years)
Crude death rate			
Single question	817	28 302	29
Household censuses	1107	92 610	12
Cohort study	–	–	–
Model range	–	–	22–25
Crude adult mortality rate (15–59 years)			
Single question	–	–	–
Household censuses	669	52 513	13
Cohort study	404	17 949	23
Model	–	–	23–28
Standardized measures	Males	Females	
Adult mortality			
Household censuses	0.65	0.51	
Cohort study	0.77	0.57	
Model	0.80–0.83	0.75–0.80	
Adult mortality in the absence of HIV			
Household censuses	–	–	
Cohort study	0.34	0.27	
Model	0.40	0.33	
Life expectancy			
Best estimate ^a	34.3	38.2	
Model	36.6–37.8	35.83–37.8	
Life expectancy in the absence of HIV			
Best estimate ^a	48.8	52.5	
Model	56.1	58.5	

^a Combining estimated child survival based on HIV status, household censuses data corrected for under-ascertainment for ages not captured in the cohort study, and directly observed mortality rates from the cohort study.

Study design

HIV prevalence was 15% for males (17–44 years) and 21% for females (15–44 years) at baseline.¹⁸ In this analysis, we use three empirical data sources (repeated household censuses, a single question on household mortality, and an individual-based cohort with linked HIV testing) and a deterministic mathematical model.¹⁹

Household censuses

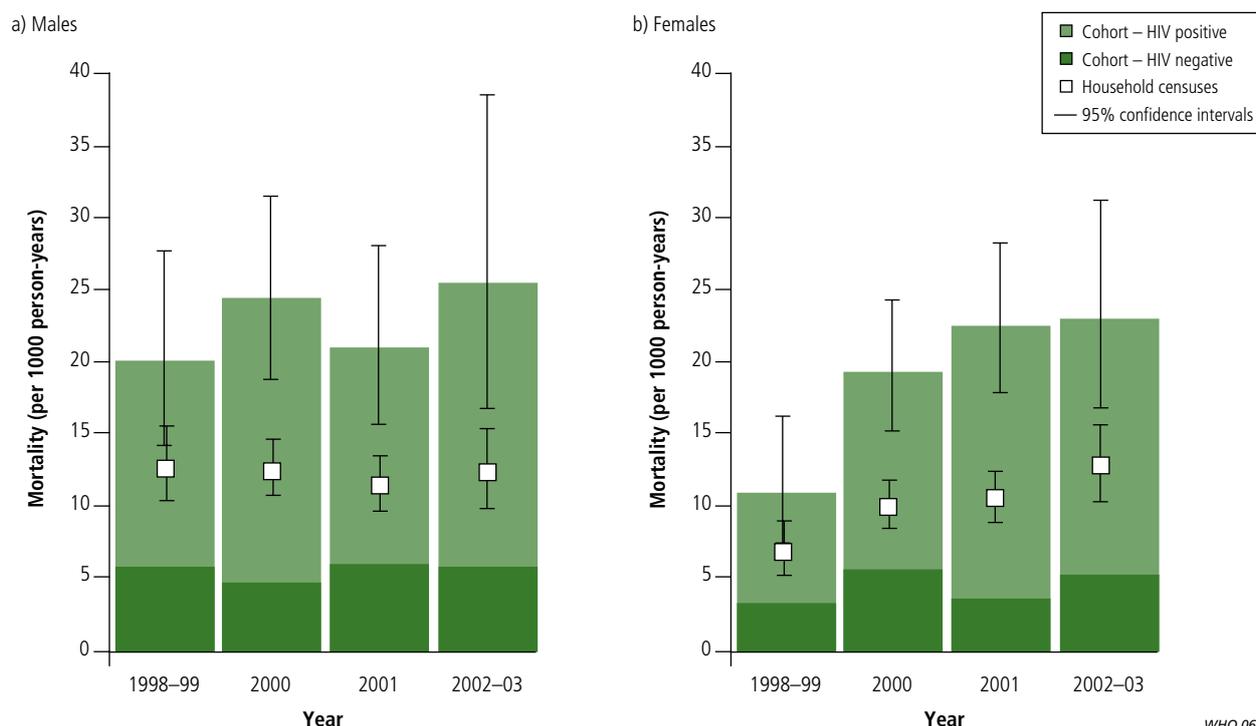
Longitudinal household censuses were done at 3-yearly intervals for each of the 12 study sites. A total of 28 986 household members were counted at round 1 and 5270 new household members or in-migrants were additionally counted at round 2. If a household member died in the course of follow-up, or in-migrated and died before follow-up, the month and year of death were recorded by interview with the

household head. The overall household follow-up rate was 82%. Households where all members out-migrated ($n = 736$, 8.6%) or were lost to follow-up for other reasons ($n = 832$, 9.7%) were not included in the analysis. The total number included in the analysis was 32 688.

Single household question

As part of the round 2 household census, respondents to the household questionnaire ($n = 6985$ households) were asked how many deaths there had been in the households in the past 12 months. The crude death rate was calculated by dividing the total number of deaths in the previous 12 months by the total number resident at the time of the census plus the total number of deaths. Recent in-migrants were included in both the numerator (deaths) and denominator (population).

Fig. 1. Annual mortality rates from household censuses and cohort study and proportion of HIV-positive deaths in Manicaland, Zimbabwe



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Individual cohort study

Selected participants ($n = 9842$) were given a structured questionnaire. HIV serological testing was done on dried blood spots using a highly sensitive and specific antibody dipstick assay.¹⁶ Follow-up was done at the same time as the household censuses. Out-migrants and those lost to follow up were not included. Out-migrants had similar HIV prevalence to non-migrants²⁰ so their exclusion may not have introduced major bias. Overall, follow-up in the individual closed cohort was 60.8% ($n = 5776$).

Mathematical model

A mathematical model was constructed to compare observed patterns of mortality with projected mortality, based on HIV prevalence. Using a previously described model as our basis,¹⁹ we simulated the heterosexual transmission of HIV in a population stratified for age and sexual activity. Age-specific mortality in the absence of HIV was set at level 17 of the West model life table,²¹⁻²³ since this reflected most closely observed mortality in the HIV-negative cohort. Coale & Demeny's model life table was used as it is the most widely applied when little is known about the age pattern of mortality.

To give a range of plausible estimates of mortality rates, we created two model scenarios to give an upper and lower bound. The upper bound (higher mortality) assumed a declining epidemic (higher HIV prevalence before baseline) and used estimates for survival with HIV from an observational cohort study in rural Tanzania, since such data do not exist for Zimbabwean populations.²⁴ The lower bound assumed that the epidemic stabilized before baseline, and extracted estimates for survival with HIV from a meta-analysis of cohorts in industrialized countries²⁵ and applied these estimates to the demographic context of Zimbabwe.

In each scenario, the model was fitted to the observed baseline prevalence from the cohort study (year 20 in the model). Mortality estimates were examined for years 23-25.

To compare the crude death rate from model predictions to the cohort and household censuses, age-specific mortality rates were standardised to the appropriate population and summarized using the life table method (Table 1).

Life table methods for adult mortality indicators

A period life table for all age groups was constructed, first using the household

census data. Infant mortality estimates were derived from maternal birth history data obtained in the cohort study for the inter-survey period, and were decremented based on mother's HIV serostatus at baseline. The same technique could not be applied for child mortality because linked data were not collected from mothers on survival of children born before the first survey round. Therefore, child mortality was estimated indirectly using the Brass method — a procedure based on child survival as reported by women in different age groups.²⁶

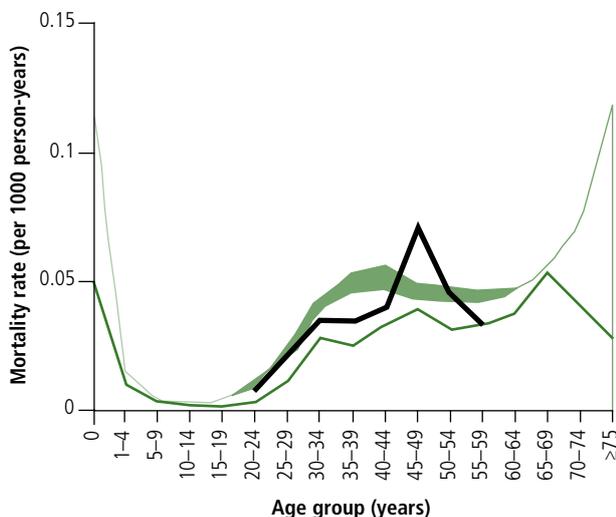
Using the cohort data and mathematical model projection of mortality rates, we constructed a period life table stratified by HIV status. Since data from the cohort were only available for ages 17-54 years for males and 15-44 years for females, the rest of the cohort life table was completed with household census data. This, in effect, assumed the same mortality for HIV-positive and HIV-negative groups in ages not captured by the cohort study.

Life expectancy

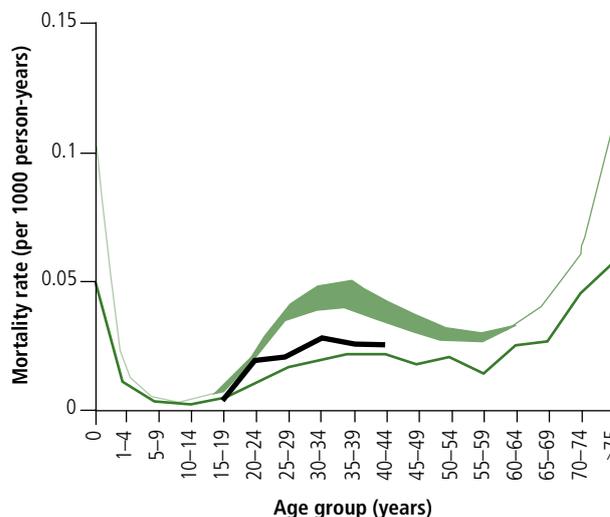
Life expectancy was calculated using life table methods.²³ The baseline rate of death was subtracted from HIV-positive

Fig. 2. Age-specific and sex-specific mortality rates (per 1000 person-years) comparing estimates from household censuses, the cohort study, and model predictions^a

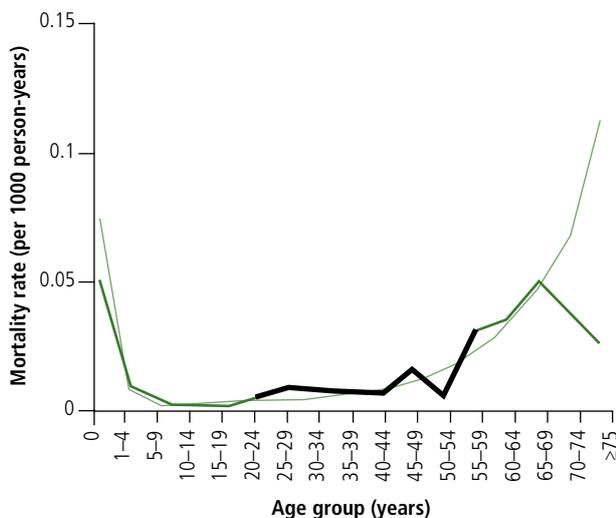
a) Males: HIV positive and HIV negative



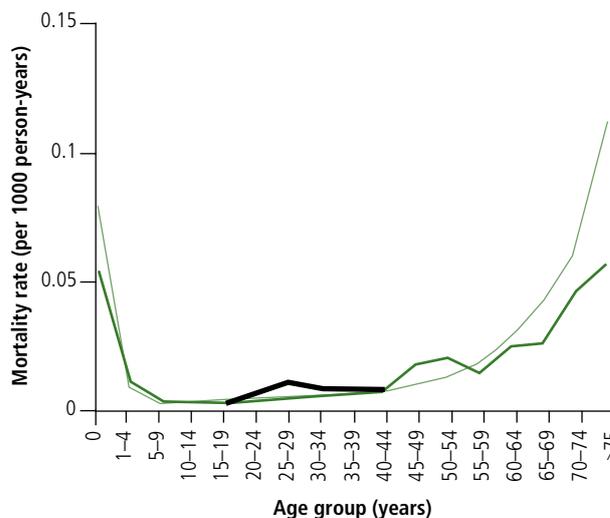
b) Females: HIV positive and HIV negative



c) Males: HIV positive excluded



d) Females: HIV positive excluded



— Household censuses — Cohort study — Model predictions

^aThe shaded area illustrates the range of the model predictions, based on two extreme assumptions about the duration of survival after HIV infection and the phase of the HIV epidemic in Zimbabwe.

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groups to identify the true mortality from HIV. We also calculated life expectancy from birth in the population and life expectancy without the effect of HIV. Inputs for child mortality (0–10 years) for HIV-negative children were based on the West model life table, corresponding to a life expectancy of 50 years. Mortality in HIV-positive children was based on models by Marston et al.,²⁷ which estimated the

survival of children born HIV-positive as 67% at year 1, 39% at year 5 and 13% at year 10. We assumed an HIV prevalence of 30.7% at birth amongst children born to HIV-positive women.²⁸ Furthermore, we assumed that mortality was underestimated in the household censuses in the age groups not measured in the cohort study (10–14 years, >45 years for females; and 10–16 years, >55

years for males) at the same level that it was underestimated in the observed age groups (12% for females, 19% for males). The difference between the life expectancies should be taken as a conservative estimate of the mortality impact of HIV, since we assume no difference in HIV-positive and HIV-negative mortality in the age ranges not captured in the cohort study. The cumulative probability

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of dying from HIV was calculated by dividing the number of people who died from HIV by the size of the model cohort alive at age 15 years.²³

Results

There were 817 deaths based on responses to the single question “how many deaths have there been in the household in the past 12 months?”. In the individual cohort study, there were 404 (males 184; females 220) deaths, with date of death missing in 37 (9%) of cases. In total, 1107 (males 592; females 515) deaths were reported in the three years between the first and second census, with date of death missing in 85 (8%) cases.

Crude mortality rate

The crude death rate was substantially lower when measured by the household censuses (12 per 1000 person-years) as compared to the single question on household mortality (29 per 1000 person-years), which was slightly higher than the range of model estimates (22–25 per 1000 person-years) (Table 1).

Adult mortality

Adult mortality was also much lower when measured by the household censuses (males 0.65; females 0.51) compared with that in the cohort study (males 0.77; females 0.57), which was slightly lower than the model estimates (males 0.80–0.83; females 0.75–0.80) (Table 1b). Despite there being a lower

mortality rate measured by the household census, its level of statistical precision was higher given the larger size of the population ($n = 28\,986$ compared with $n = 5776$ in the cohort (as shown by the width of the confidence intervals in Fig. 1).

Gender

Female mortality was lower than male mortality (20 compared with 26 per 1000 person-years) contrary to model predictions, when rates were standardized to the cohort age structure (Table 1).

Over the whole study period, mortality was higher in males than in females (Table 1). The age-adjusted mortality rate ratio for males to females was 1.25 (95% confidence interval (CI) = 1.1–1.4; $P < 0.001$) based on the household census data and 1.4 (95% CI = 1.1–1.7; $P < 0.001$) based on the individual cohort study (Table 1 and Fig. 1).

Temporal patterns

These figures mask different temporal patterns in men and women. No clear pattern was observed in male mortality, but female adult mortality increased over the study period (Fig. 1). Female mortality increased at an annual rate ratio of 1.19 (Poisson regression 95% CI = 1.06–1.35; $P = 0.003$; no evidence that a non-linear model provided better fit, likelihood ratio test $P = 0.45$). Levels of female mortality reached that of males by the last 2 years of the study. Furthermore, the proportion of female deaths (from the cohort study) who were HIV-

positive at baseline increased from 70% in the first half of the study (1998–2000: 69 HIV positive/98 deaths) to 82% in the second half (2001–03: 100 HIV positive/122 deaths).

Age-specific mortality rates

Fig. 2 shows the age-specific mortality rates for all individuals and with HIV-positive individuals excluded. The household census data, coupled with the indirect Brass method based on child survival, produced lower estimates for child mortality and old-age mortality compared with the model. The model reflects the observation of higher mortality for females compared with males from ages 15–29 years, but produces substantially higher overall female mortality than is observed in the data. Fig. 2 c), which combines the household census-derived estimates for child and old-age mortality with HIV-negative male adults from the cohort, fits closely to model predictions for adults. Fig. 2 d) suggests that the level of female mortality due to AIDS predicted by the model may be too high. The higher mortality in males aged 55–69 years and females aged 45–49 years suggests that there is considerable AIDS mortality in these groups that is not captured in the cohort study due to the age-inclusion criteria.

Impact of HIV on adult mortality

Overall, HIV-positive men and women had mortality rates 8.6 and 10.2 times higher, respectively, than those who were

Table 2. Age-stratified adult mortality from the individual cohort, Manicaland, Zimbabwe

Age (years)	HIV negative		HIV positive		Rate ratio (95% CI ^a)	PAF ^b
	Deaths/person-years	Mortality per 1000 person-years	Deaths/person-years	Mortality per 1000 person-years		
Males						
17–24	19/2373	8.0	2/91	21.8	3.9 (0.9–16.6)	0.10
25–34	15/1534	9.8	42/651	64.4	7.0 (3.9–12.7)	0.64
35–44	7/896	7.8	45/496	90.6	10.5 (4.8–23.4)	0.77
45–54	11/786	14.0	42/242	173.5	12.0 (6.2–3.2)	0.72
All	52/5590	9.3	131/1482	88.4	8.6 (6.2–11.8)	0.61
Females						
15–24	20/2609	7.7	23/415	55.3	9.6 (5.3–17.5)	0.54
25–34	8/2030	3.9	68/1147	59.2	15.2 (7.3–31.7)	0.84
35–44	19/2314	8.2	61/868	70.2	7.7 (4.6–12.9)	0.65
45–54	2/958	2.1	11/176	62.2	32.4 (7.2–146)	0.83
All	49/7912	6.2	163/2608	62.5	10.2 (7.4–14.1)	0.70

^a CI = confidence interval.

^b PAF = population attributable fraction.

HIV negative (from cohort study; Table 2). Overall, 61% of mortality in males and 70% of mortality in adult females can be attributed to HIV in this population. HIV-associated mortality in males was highest in the 45–54 year age group (174 per 1000 person-years) whereas in females, the peak was in the 35–44 year age group (70 per 1000 person-years) (Table 2).

In the absence of HIV-associated mortality, adult mortality would be 0.34 for males and 0.27 for females (compared with 0.77 and 0.57, respectively, as observed with HIV-associated mortality).

The probability of a man or woman who survived to age 15 years dying of HIV by age 60 years was 0.51 and 0.35, respectively (Fig. 3).

Impact of HIV on life expectancy

The empirical estimate of life expectancy from birth was calculated to be 34.3 years for males and 38.2 for females. Based on the decrement life table, life expectancy at birth in the absence of HIV would be 48.8 for males (14.5 year increase) and 52.5 (14.3 year increase) for females. The model projections of life expectancy for men ranged from 36.6 to 37.8 and for women from 35.8 to 37.8, with HIV reducing life expectancy by a median of 19 years for men and 22 years for women (Table 1).

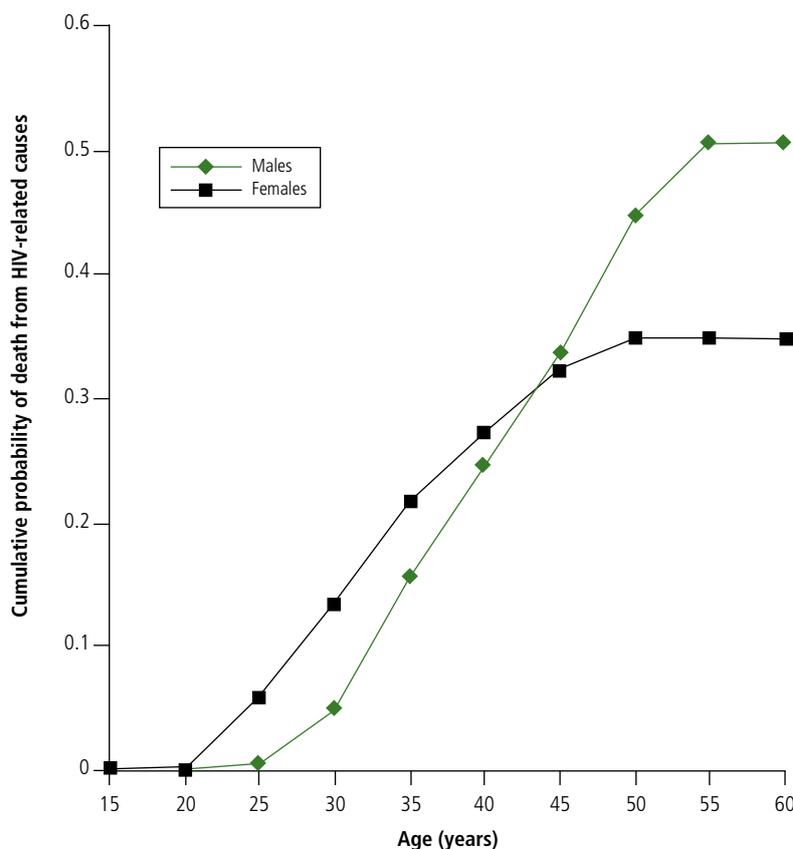
Discussion

By any measure, patterns of mortality in Manicaland, Zimbabwe are extremely troubling. HIV, the cause of 61% of adult male and 70% of adult female deaths, has reduced life expectancy to 34 years and 38 years, respectively. Given present HIV prevalence and mortality rates, males have a 51% chance and women have a 35% chance of dying from HIV between the ages of 15 and 60 years. Female mortality increased throughout the survey period and, in 2001, reached the level of male mortality.

Comparing the measures of mortality from different methods, certain consistencies as well as discrepancies emerge. Similar estimates of crude mortality derived from the single question and the model, and adult male mortality as measured by the cohort study and the model, suggested the accuracy of these measures.

First, in terms of inconsistencies, mortality rates derived from the house-

Fig. 3. Cumulative probability of death from HIV-related causes aged 15–60 years for males and females in Manicaland, Zimbabwe^a



^aAdult mortality is highly dependent on HIV-associated mortality, and is calculated as the probability that a person who lives until 15 years will die before the age of 60. The present value is 0.79 for men and 0.59 for women.

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hold censuses were substantially lower than the other estimates. Because the respondents in household interviews were required to report on the survival of all members of a household, there may be a tendency to underreport, especially with a long recall period of three years. Demographic surveillance systems typically rely on a 1-year recall period. Further exacerbating under-ascertainment, households can often dissolve or relocate after a death.²⁹ These biases partly explain the surprising finding that both the household census and the individual cohort give much lower estimates of crude mortality than the single question. The two household methods could be expected to give similar results, but the finding that the single household question was probably more accurate makes clear the importance of limiting the time frame to reduce recall period and the chance of the household dissolving.

Second, as a measure of crude mortality, the single question about deaths

in the household in the past 12 months was fairly accurate with respect to the range of model projections. This is encouraging, considering the widespread use of this tool in surveys and censuses, although in the Manicaland data, it remains unclear whether there are multiple biases that combine to produce an accurate result. For example, underreporting may occur for reasons stated above and overreporting may occur if respondents included deaths that actually occurred more than one year ago.⁴

Third, the cohort data produced estimates of adult mortality that were consistent with the model for men, but much lower than predicted for women. It is unlikely that female deaths would be ascertained more poorly than male deaths, which suggests inaccurate parameterization of the model. Fitting the model to age-specific HIV prevalence at baseline may not have fully captured the extent to which the HIV epidemic spread in males before becoming widespread in females or how behaviour or mixing

patterns may have changed through time,¹⁹ or, perhaps, differential losses to follow-up of deaths.

Did losses to follow-up contribute to the lower mortality estimates derived from the cohort study compared with the model? Overall, the HIV prevalence at baseline was lower in individuals who were not found at follow-up ($n = 3646$, 19%) than those who were found or were known to have died ($n = 6162$, 25%). However, this does not necessarily imply that individuals lost to follow-up had lower mortality. Losses to follow-up are a mixed group. Known out-migrants, who left the study sites for employment or other reasons, may be relatively healthy and truly have lower mortality rates but individuals who were not found at all may have migrated home or near a health centre to receive care for an eventually fatal illness. So it is possible this subgroup of out-migrants were at an advanced stage in their HIV illness and therefore had higher overall mortality despite their somewhat lower HIV prevalence.

Taking into consideration these limitations, our best estimates of life expectancy (males 34.3 years; females 38.2 years) were similar to model estimates (35.8 to 37.8 years) and 2001 WHO estimates (males 37.1 years; females 36.5),²¹ which were all higher than estimates from the National AIDS

Council of Zimbabwe (32 years).³ Our empirical measure of adult mortality (based mainly on the individual cohort study) is remarkably consistent with other estimates. Using sibling histories from Demographic and Health Surveys, Jasseh & Timæus³⁰ calculated adult mortality in Zimbabwe to be 0.57 for women and 0.73 for men, compared with our measures of 0.57 and 0.77, respectively. This represents a substantial rise from the early nineties when AIDS-related deaths became prevalent in the Manicaland area³¹ and in Zimbabwe as a whole.⁴

Our estimates that 61% of male and 70% of female adult mortality is attributable to HIV in this population (where prevalence is 15% for males and 21% for females) are higher than equivalent figures for Kisesa, United Republic of Tanzania (45% for men and 54% for women) and the Masaka region, Uganda (40% for both sexes) from the mid-1990s where HIV prevalence was less than 10%.^{5,6} The higher population attributable fraction estimates from Rakai, Uganda at 66% for men and 80% for women (when HIV prevalence was 16%) can be attributed to lower mortality levels in the HIV-negative baseline group.⁷

This collection of diverse data has allowed the direct comparison of different estimates of mortality in the same

population. Each of these methods has advantages and will continue to be used in various study designs but these analyses make clear the advantages of longitudinal cohort data. These provided more complete ascertainment than repeated household censuses and served to highlight possible inaccuracies in model predictions.

The startling model-based predictions made over 10 years ago, that HIV could be responsible for three-quarters of all deaths,³² have proven to be true in this rural Zimbabwean population. However, the current results do suggest that reductions in HIV-associated mortality achieved through the use of antiretroviral therapy, for example, would have a major impact on general mortality patterns. ■

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Competing interests: GPG has acted as a consultant for, and/or received grants from, GlaxoSmithKline, Aventis Pasteur, Merck, and Abbott Pharmaceuticals. GPG also chaired a meeting of WHO in 2003 to develop a consensus on the importance of unsafe injections in HIV epidemiology. SG owns shares in GlaxoSmithKline and Astra Zeneca.

Résumé

Evaluation de la mortalité adulte dans une région fortement touchée par le VIH-1 du Zimbabwe (1998-2003)

Objectif Comparer différentes méthodes utilisées en l'absence de données d'état civil pour estimer la mortalité adulte et décrire les schémas de mortalité au Manicaland, une région du Zimbabwe gravement touchée par le VIH.

Méthodes L'étude a comparé des estimations de la mortalité adulte obtenues à partir de 1) une simple question sur la mortalité dans les foyers, 2) des recensements successifs auprès des ménages et 3) une étude de cohorte portant sur des adultes dont les données sont reliées aux résultats de dépistage du VIH au Manicaland, avec celles fournies par un modèle mathématique adapté à la prévalence locale par âge du VIH (1998-2000).

Résultats Le taux de mortalité brut obtenu par le biais de la question unique sur la mortalité au sein du foyer (29 pour 1000 personnes-année) était assez proche de celui fourni par le modèle mathématique (22 à 25 pour 1000 personnes-année), mais beaucoup plus élevé que celui issu des recensements auprès des ménages (12 pour 1000 personnes-année). Le taux de mortalité adulte obtenu par recensement auprès des ménages (hommes

0,65 ; femmes 0,51) était plus faible que celui tiré de l'étude de cohorte (hommes 0,77 ; femmes 0,57), alors que le modèle mathématique donnait une estimation beaucoup plus élevée, surtout pour les femmes (hommes 0,80 à 0,83 ; femmes 0,75 à 0,80). La proportion de décès d'adultes imputables au VIH parmi la population considérée était de 0,61 pour les hommes et de 0,70 pour les femmes, l'espérance de vie étant estimée à 34,3 ans pour les hommes et à 38,2 ans pour les femmes.

Conclusion Chaque méthode d'estimation de la mortalité adulte se heurtait à des limites : sujets perdus de vue (étude de cohorte), sous-évaluation (recensement auprès des ménages), transparence des procédures sous-jacentes (question unique) et sensibilité à l'égard des paramètres (modèle mathématique). Toutefois, ces analyses font bien ressortir les avantages d'une étude de cohorte longitudinale, qui fournit une évolution plus complète que les recensements auprès des ménages, révèle les inexactitudes éventuelles des hypothèses du modèle et permet de quantifier directement l'impact du VIH.

Resumen

Evaluación de la mortalidad de adultos en un país afectado por el VIH-1, Zimbabwe (1998–2003)

Objetivo Comparar métodos alternativos a los sistemas de registro civil para calcular la mortalidad de adultos, y describir las pautas de mortalidad en Manicaland, Zimbabwe, país que se ha visto gravemente afectado por el VIH.

Métodos Comparamos las estimaciones de la mortalidad adulta obtenidas a partir de 1) una sola pregunta sobre la mortalidad familiar, 2) censos de hogares repetidos, y 3) un estudio de cohortes de adultos que incluía pruebas del VIH, realizado en Manicaland, con un modelo matemático ajustado a la prevalencia local por edad de la infección por VIH (1998–2000).

Resultados La tasa bruta de mortalidad obtenida a partir de la pregunta única (29 por 1000 años-persona) concordaba bastante con la arrojada por el modelo matemático (22-25 por 1000 años-persona), pero era muy superior a la deducida de los censos de hogares (12 por 1000 años-persona). La mortalidad de adultos según estos censos (hombres: 0,65; mujeres: 0,51) fue inferior a la estimada con el estudio de cohortes (hombres: 0,77; mujeres:

0,57), mientras que los modelos matemáticos arrojaron valores mucho mayores, especialmente para las mujeres (hombres: 0,80 - 0,83; mujeres: 0,75 - 0,80). La fracción atribuible poblacional de defunciones de adultos debidas al VIH fue de 0,61 para los hombres y 0,70 para las mujeres, con una esperanza de vida estimada en 34,3 años para los primeros y 38,2 años para las últimas.

Conclusión Todos los métodos de estimación de la mortalidad de adultos presentaron limitaciones, relacionadas con el seguimiento (estudio de cohortes), la subapreciación (censos de hogares), la transparencia de los procedimientos empleados (pregunta única) y la sensibilidad a la parametrización (modelo matemático). Sin embargo, estos análisis destacan las ventajas de los datos de cohortes longitudinales, que aportan información más completa que los censos de hogares, ponen de relieve las posibles inexactitudes de las hipótesis del modelo, y permiten cuantificar directamente el impacto del VIH.

Arabic

References

1. World Health Organization. *UNAIDS at a country level: Progress report*. Geneva: WHO; 2004.
2. Anderson RM, May RM, Boily MC, Garnett GP, Rowley JT. The spread of HIV-1 in Africa: sexual contact patterns and the predicted demographic impact of AIDS. *Nature* 1991;352:581-9.
3. *The HIV and AIDS Epidemic in Zimbabwe*. Harare: National Aids Council; 2004.
4. Feeney G. The impact of HIV/AIDS on adult mortality in Zimbabwe. *Pop Dev Rev* 2001;27:10.
5. Nunn AJ, Mulder DW, Kamali A, Ruberantwari A, KengeyaKayondo JF, Whitworth J. Mortality associated with HIV-1 infection over five years in a rural Ugandan population: cohort study. *BMJ* 1997;315:767-71.
6. Urassa M, Boerma JT, Isingo R, Ngalula J, Ng'weshemi J, Mwaluko G, et al. The impact of HIV/AIDS on mortality and household mobility in rural Tanzania. *AIDS* 2001;15:2017-23.
7. Sewankambo NK, Gray RH, Ahmad S, Serwadda D, Wabwire-Mangen F, Nalugoda F, et al. Mortality associated with HIV infection in rural Rakai District, Uganda. *AIDS* 2000;14:2391-400.
8. Notkola V, Timaeus IM, Siiskonen H. Impact on mortality of the AIDS epidemic in northern Namibia assessed using parish registers. *AIDS* 2004;18:1061-5.
9. Groenewald P, Nannan N, Bourne D, Laubscher R, Bradshaw D. Identifying deaths from AIDS in South Africa. *AIDS* 2005;19:193-201.
10. Timaeus, IM. Impact of the HIV epidemic on mortality in sub-Saharan Africa: evidence from national surveys and censuses. *AIDS* 1998;12 Suppl 1:S15-27.
11. Tollman SM, Kahn K, Garenne M, Gear JS. Reversal in mortality trends: evidence from the Agincourt field site, South Africa, 1992-1995. *AIDS* 1999;13:1091-7.
12. Sanders EJ, Araya T, Kebede D, Schaap AJ, Nagelkerke ND, Coutinho RA. Mortality impact of AIDS in Addis Ababa, Ethiopia. *Ethiop Med J* 2003;41 Suppl 1:35-42.
13. Todd J, Balira R, Grosskurth H, Mayaud P, Moshafiq G, et al. HIV-associated adult mortality in a rural Tanzanian population. *AIDS* 1997;11:801-7.
14. Lopman BA, Garnett GP, Mason PR, Gregson S. Individual level injection history: a lack of association with HIV incidence in rural Zimbabwe. *PLoS Med* 2005;2:142-6.

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15. Gregson S, Nyamukapa CA, Garnett GP, Mason PR, Zhuwau T, Carael M, et al. Sexual mixing patterns and sex-differentials in teenage exposure to HIV infection in rural Zimbabwe. *Lancet* 2002;359:1896-903.
16. Ray CS, Mason PR, Smith H, Rogers L, Tobaiwa O, Katzenstein DA. An evaluation of dipstick-dot immunoassay in the detection of antibodies to HIV-1 and 2 in Zimbabwe. *Trop Med Int Health* 1997;2:83-8.
17. Gregson S, Mushati P, White PJ, Mlilo M, Mundandi C, Nyamukapa C. Informal confidential voting interview methods and temporal changes in reported sexual risk behaviour for HIV transmission in sub-Saharan Africa. *Sex Transm Infect* 2004;80 Suppl 2:ii36-42.
18. Boerma JT, Gregson S, Nyamukapa C, Urassa M. Understanding the uneven spread of HIV within Africa: comparative study of biologic, behavioral, and contextual factors in rural populations in Tanzania and Zimbabwe. *Sex Transm Dis* 2003;30:779-87.
19. Gregson S, Garnett GP. Contrasting gender differentials in HIV-1 prevalence and associated mortality increase in eastern and southern Africa: artefact of data or natural course of epidemics? *AIDS* 2000;14 Suppl 3:S85-99.
20. Mundandi C, Vissers D, Voeten H, Habbema D, Gregson S. No difference in HIV incidence and sexual behaviour between out-migrants and residents in rural Manicaland, Zimbabwe. *Trop Med Int Health*. In press.
21. World Health Organization. *Life Tables for 191 Countries*. Geneva: WHO; 2001.
22. Coale AJ, Demeny P. *Region Model Life Tables and Stable Populations*. London: Academic Press; 1983.
23. Preston SH, Heuveline P, Guillot M. *Demography: Measuring and modelling population processes*. Oxford: Blackwell Publishers; 2001.
24. Porter K, Zaba B. The empirical evidence for the impact of HIV on adult mortality in the developing world: data from serological studies. *AIDS* 2004; 18 Suppl 2:S9-S17.
25. Time from HIV-1 seroconversion to AIDS and death before widespread use of highly-active antiretroviral therapy: a collaborative re-analysis. Collaborative Group on AIDS Incubation and HIV Survival including the CASCADE EU Concerted Action. Concerted Action on SeroConversion to AIDS and Death in Europe. *Lancet* 2000;355:1131-7.
26. Manual X: *Indirect techniques for demographic estimation*. New York: United Nations; 1983.
27. Marston M, Zaba B, Salomon JA, Brahmbhatt H, Bagenda D. Estimating the net effect of HIV on child mortality in African populations affected by generalized HIV epidemics. *J Acquir Immune Defic Syndr* 2005;38:219-27.
28. Zijenah LS, Moulton LH, Iliff P, Nathoo K, Munjoma MW, Mutasa K, et al. Timing of mother-to-child transmission of HIV-1 and infant mortality in the first 6 months of life in Harare, Zimbabwe. *AIDS* 2004;18:273-80.
29. Mushati P, Zvidzai C, Lewis JJ, Gregson S. *Adult mortality and the economic sustainability of households in towns, estates and villages in AIDS-afflicted eastern Zimbabwe*. In: XV International AIDS Conference, Bangkok, Thailand, 2004.
30. Timaeus IM, Jasseh M. Adult mortality in sub-Saharan Africa: evidence from Demographic and Health Surveys. *Demography* 2004;41:757-72.
31. Gregson S, Anderson RM, Ndlovu J, Zhuwau T, Chandiwana SK. Recent upturn in mortality in rural Zimbabwe: evidence for an early demographic impact of HIV-1 infection? *AIDS* 1997;11:1269-80.
32. Gregson S, Garnett GP, Anderson RM. Is HIV-1 likely to become a leading cause of adult mortality in sub-Saharan Africa? *J Acquir Immune Defic Syndr* 1994;7:839-52.