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Modelling HIV incidence and survival from age-specific seroprevalence after antiretroviral treatment scale-up in rural South Africa

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Objective: Our study uses sex-specific and age-specific HIV prevalence data from an ongoing population-based demographic and HIV survey to infer HIV incidence and survival in rural KwaZulu-Natal between 2003 and 2011, a period when antiretroviral treatment (ART) was rolled out on a large scale.

Design: Catalytic mathematical model for estimating HIV incidence and differential survival in HIV-infected persons on multiple rounds of HIV seroprevalence.

Methods: We evaluate trends of HIV incidence and survival by estimating parameters separately for women and men aged 15–49 years during three calendar periods (2003–2005, 2006–2008, 2009–2011) reflecting increasing ART coverage. We compare model-based estimates of HIV incidence with observed cohort-based estimates from the longitudinal HIV surveillance.

Results: Median survival after HIV infection increased significantly between 2003–2005 and 2009–2011 from 10.0 [95% confidence interval (CI) 8.8–11.2] to 14.2 (95% CI 12.6–15.8) years in women ($P < 0.001$) and from 10.0 (95% CI 9.2–10.8) to 14.0 (95% CI 10.6–17.4) years in men ($P = 0.02$). Our model suggests no statistically significant reduction of HIV incidence in the age-group 15–49 years in 2009–2011 compared with 2003–2005. Age-specific and sex-specific model-based HIV incidence estimates were in good agreement with observed cohort-based estimates from the ongoing HIV surveillance.

Conclusion: Our catalytic modelling approach using cross-sectional age-specific HIV prevalence data could be useful to monitor trends of HIV incidence and survival in other African settings with a high ART coverage.

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Introduction

KwaZulu-Natal in South Africa has one of the highest HIV prevalences in the world. Following the roll-out of HIV care and antiretroviral treatment (ART) in 2004, HIV-associated mortality decreased rapidly [1], leading to a substantial rise in life expectancy from 49.1 years in 2003 to 60.5 years in 2011 in our demographic surveillance site (DSS) in rural KwaZulu-Natal [2]. This rise in life expectancy is probably largely driven by the fact that HIV-positive adults in South Africa can have a near-normal life expectancy, provided that they start ART early enough and adhere to treatment [3]. Whereas in the initial phases of the ART programme, we observed little change in HIV prevalence and incidence locally [4], more recently, we observed both an increase of HIV

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prevalence in older age groups [5] and a decrease in risk of HIV acquisition with high ART coverage [6].

In the most recent National Strategic Plan, the South African National AIDS Council and the South African government confirm their strong commitment to reduce HIV incidence (the rate of new HIV infections) by 50% between 2012 and 2016 [7]. However, unlike HIV prevalence, which can be measured from nationally representative cross-sectional serological surveys, measurement of HIV incidence with any degree of accuracy and reliability is much more challenging [8]. Direct estimation of HIV incidence requires a cohort of individuals to be followed up prospectively and tested multiple times. Not only are such multiple measurements on the same individuals logistically difficult to achieve at a regional or national scale, but such data are also potentially subject to biases due to low consent rates for repeat testing [9] and loss to follow up due to migration. Although much effort has gone into trying to develop and validate novel biological assays that can reliably predict recent HIV infection [8,10–13], such assays were often found to require local calibration. An alternative approach to direct incidence estimation is to use a 'catalytic' modelling technique pioneered in the 1950s [14], which can potentially be applied to any age-stratified seroprevalence data sets, as long as sample sizes are large enough over wide age ranges.

Although a modelling approach has been previously applied to data from other sub-Saharan countries [15–23], we apply it here for the first time to multiple rounds of cross-sectional HIV surveys from rural KwaZulu-Natal, and for the first time in the context of a large-scale ART roll-out. Moreover, whereas previous work often relied on using external sources for HIV-associated mortality [15], we estimate differential HIV survival in parallel to HIV incidence using seroprevalence data only.

The aim of our study was thus to infer trends of HIV incidence and survival in rural KwaZulu-Natal from HIV prevalence data collected on a yearly basis between 2003 and 2011. This was achieved by applying a catalytic modelling approach on age-specific and sex-specific HIV seroprevalence patterns during three calendar periods (2003–2005, 2006–2008, 2009–2011), which coincide with increasing ART coverage. We used the model to simultaneously infer HIV incidence and survival during the three calendar periods and validate model-based incidence estimates with directly observed cohort incidence estimates obtained in the same setting between 2003 and 2011.

Materials and methods

Study setting

Since 2003, consecutive annual HIV sero-surveys have been conducted in the DSS in a rural subdistrict of uMkhanyakude in northern KwaZulu-Natal with a resident population of approximately 60 000 Zulu-speaking people. Although logistic details of the HIV sero-surveys have been described elsewhere [24,25], briefly, all adult persons eligible to participate on the basis of age and residency status are attempted to be contacted by a field team for participation in an HIV survey. During the interview, written informed consent is obtained before collecting information on health, sexual behaviour and biomedical measures including a finger prick sample for HIV assessment. In order to ensure comparability over time and across sexes, the data used in this analysis are restricted to persons aged 15–49 years who participated in the HIV surveillance in 2003–2011, as women aged 50 years or older and men aged 55 years or older were only included in HIV surveillance from 2007 onwards. Ethical approval for the HIV surveys and the Africa Centre Demographic Surveillance System was obtained from the Research Ethics Committee at the College of Health Sciences, University of KwaZulu-Natal.

Mathematical model-based estimates of incidence and survival

We apply a modified cumulative incidence and survival model [16,18] to estimate simultaneously age-dependent HIV incidence and differential mortality in HIV-infected persons. We evaluate trends by estimating model parameters (see below) separately for women and men during three calendar periods (2003–2005, 2006–2008, 2009–2011), which coincide with increasing local ART coverage by the local HIV treatment and care programme, which was a partnership between the Department of Health and the Africa Centre. The total number of patients initiating ART in the six clinics serving the DSS increased from 372 in 2003–2005 to 3528 in 2006–2008 and attained 5573 in 2009–2011 (C. Newell, personal communication). During 2011, it is estimated that 30.7% of all HIV-infected adults aged 15–49 years in the DSS were receiving ART [5].

We adapt the catalytic model of Gregson *et al.* [16], first, by using a lognormal functional form of incidence as proposed in Williams *et al.* [18] and, second, by fitting one survival parameter and three incidence parameters to yearly age-specific HIV-seroprevalence profiles of persons aged 15–49 years assuming homogeneity over time, grouped within three calendar periods.

The incidence function $f(a)$ (i.e. the age-dependent risk of acquiring HIV infection for a susceptible person of age a years) is assumed to follow a scaled offset lognormal distribution function [18] with parameters mean μ, standard deviation σ and scale θ and an offset corresponding to sexual debut fixed at 12 years:

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$$f(a) = \begin{cases} 0 & \text{if } a \leq 12 \text{ years} \\ \dfrac{\theta}{\sqrt{2\pi}\sigma(a-12)} \exp\left(-\dfrac{(\log(a-12)-\mu)^2}{2\sigma^2}\right) & \text{if } a > 12 \text{ years} \end{cases} \quad (1)$$

The probability of survival $s(x)$ after a duration of HIV infection of x years was assumed to follow the Weibull distribution,

$$s(x) = \exp\left(-\left(\frac{x}{\beta}\right)^{\alpha}\right), \quad (2)$$

where α and β are the shape and scale parameters, respectively. Unlike previous authors who fixed shape and scale parameters on the basis of external sources [26–28], we attempt to estimate the scale parameter β simultaneously from the seroprevalence data. We further conduct a model sensitivity analysis by varying the shape parameter α in a plausible range from 2 to 5 to see the effect of the shape parameter on incidence and survival summary statistics. Model comparisons were performed using Akaike information criterion (AIC). The results presented in the abstract and figures are based on models with the lowest AIC in the plausible range.

We obtain maximum likelihood estimates of the four parameters β, μ, σ, θ separately for men and women during three calendar periods C_1 (2003–2005), C_2 (2006–2008) and C_3 (2009–2011) by minimizing the negative binomial log likelihood [29]:

$$-\log(L) = -\sum_{t \in C_j} \sum_{a=15}^{49} [\log(F(a))R_a(t) + \log(1-F(a))(N_a(t)-R_a(t))] \quad (3)$$

where $R_a(t)$ and $N_a(t)$ are the observed number of HIV-positive individuals and total number of individuals who participated in HIV surveillance, respectively, having age a (years) during calendar year t, C_j is the calendar period [$j = 1,2,3$ and C_1=(2003,2004,2005), C_2=(2006,2007,2008), C_3=(2009,2010,2011)] and is the estimated HIV prevalence at age a given as in ref. [16],

$$F(a) = \frac{\int_0^a f(x)\exp\left(-\int_0^x f(y)dy\right)s(a-x)dx}{\int_0^a f(x)\exp\left(-\int_0^x f(y)dy\right)s(a-x)dx + \exp\left(-\int_0^a f(y)dy\right)} \quad (4)$$

The functions f and s in Eq. (4) correspond to the functions defined in Eqs. (1) and (2). The numerator of Eq. (4) represents individuals infected with HIV at age a, which is the integral of those initially infected at an earlier age who have survived to age a [16]. The denominator represents the sum of the expression in the numerator and the individuals who have not yet been infected by age a, so the denominator encompasses all individuals who have survived to age a.

The HIV prevalence data used for fitting the incidence and survival model thus consist of observed number of HIV-positive and number of participants in HIV surveillance stratified by year of age, by year of surveillance and by sex (see Supplemental Digital Content 1, http://links.lww.com/QAD/A372).

Optimization was performed using the default Nelder-Mead method of the *mle* function of the package *stats4* in R [30]. Differences in parameter estimates between calendar periods were assessed assuming that parameter estimates are (asymptotically) normally distributed [31] and that the standard error of the difference of two parameter estimates is the sum of the squares of the standard errors [32].

Observed cohort incidence

Observed cohort incidence was estimated from resident persons in the demographic surveillance area who participated at least twice in the annual HIV sero-surveys, whose earliest HIV result was negative and who were aged between 15 and 49 years at the time of the survey [6]. A uniformly distributed random seroconversion date was assigned between the last negative test and the first positive test. Incidence was calculated as the number of seroconversions in a given age and sex group divided by the total number of person-years of observations in that age and sex group between 2003 and 2011. Estimates and 95% confidence intervals (CIs) were obtained with Stata 11 (StataCorp, College Station, Texas, USA) using stptime.

Results

The age-specific observed and fitted model HIV prevalence estimates in women and men during the three calendar periods 2003–2005, 2006–2008 and 2009–2011 in Fig. 1 indicate a good visual model fit with observed data. The higher dispersion of the observed

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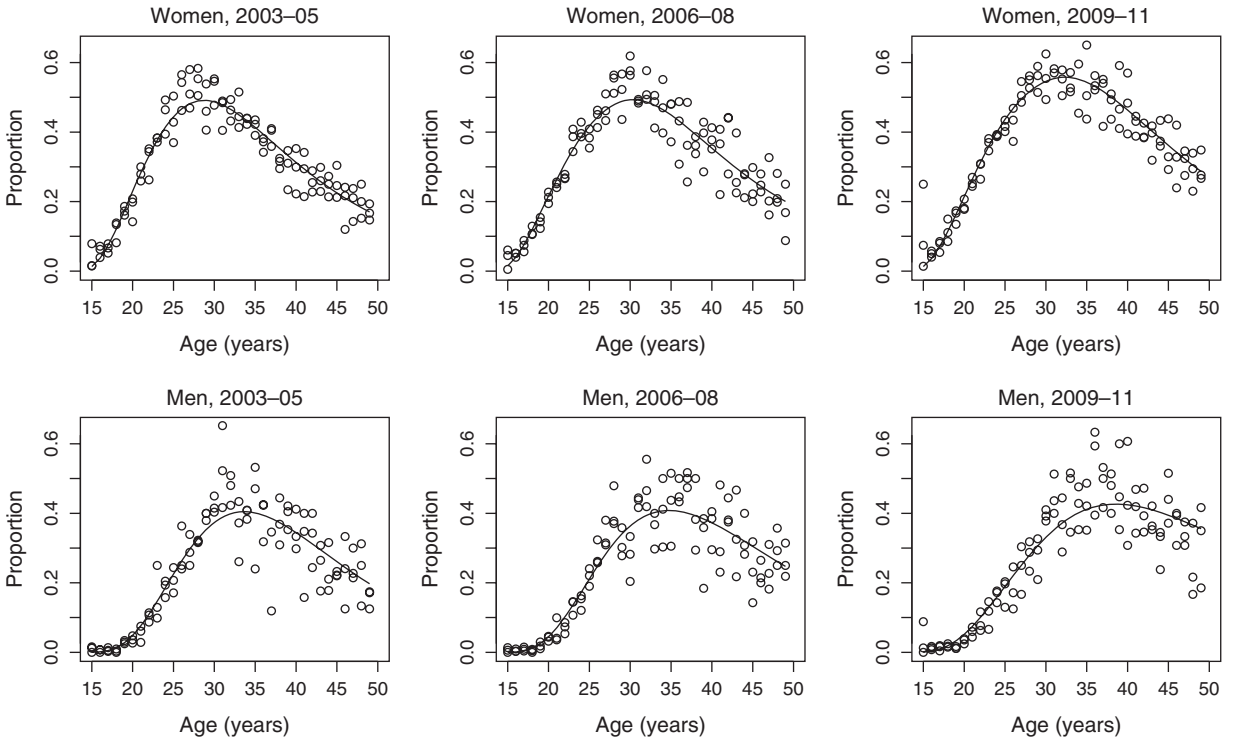

Fig. 1. Observed and fitted HIV prevalence as a function of age, sex and calendar period. Each circle represents the observed HIV prevalence estimate for a given survey year, a given year of age and sex. As each period contains three calendar years, there are three prevalence estimates (circles) for each year of age. Solid lines refer to fitted model based estimates of HIV prevalence. The shape parameter α of the Weibull survival distribution of HIV-infected individuals was fixed at a value of 5. Other parameters correspond to those reported in Table 1.

prevalence in older age groups (most pronounced among men) is indicative of lower numbers of HIV surveillance participants in these age groups (see Supplemental Digital Content 1, http://links.lww.com/QAD/A372). This lower sample size is both a reflection of smaller numbers of older men living in the DSS (see Fig. 2 in Tanser *et al.* [25]) and lower participation rates of older men in HIV surveillance [4].

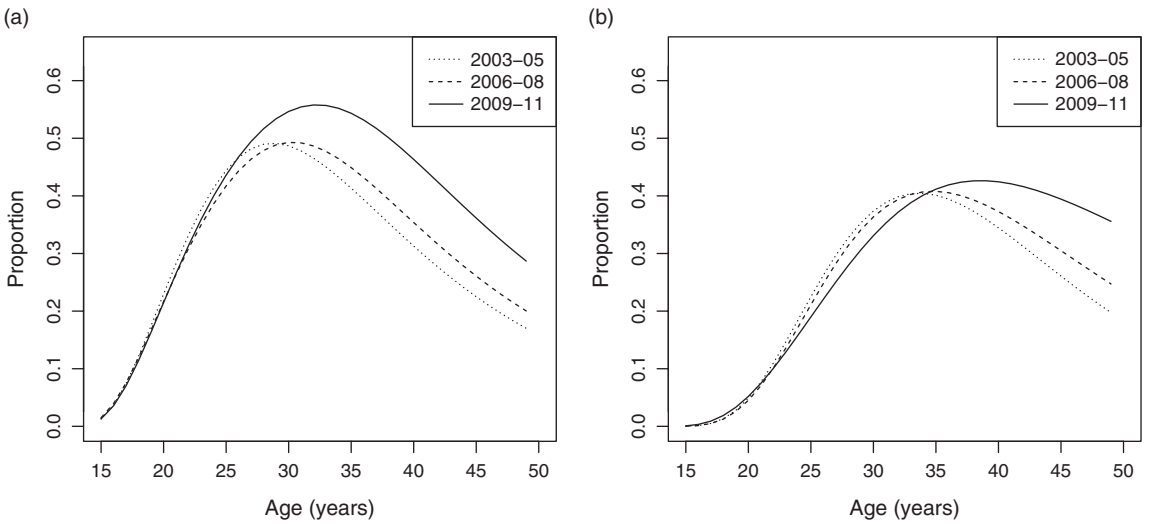

Fig. 2. Fitted HIV prevalence in women (a) and men (b) as a function of age. The shape parameter α of the Weibull survival distribution of HIV-infected individuals was fixed at a value of 5. Other parameter estimates correspond to those reported in Table 1.

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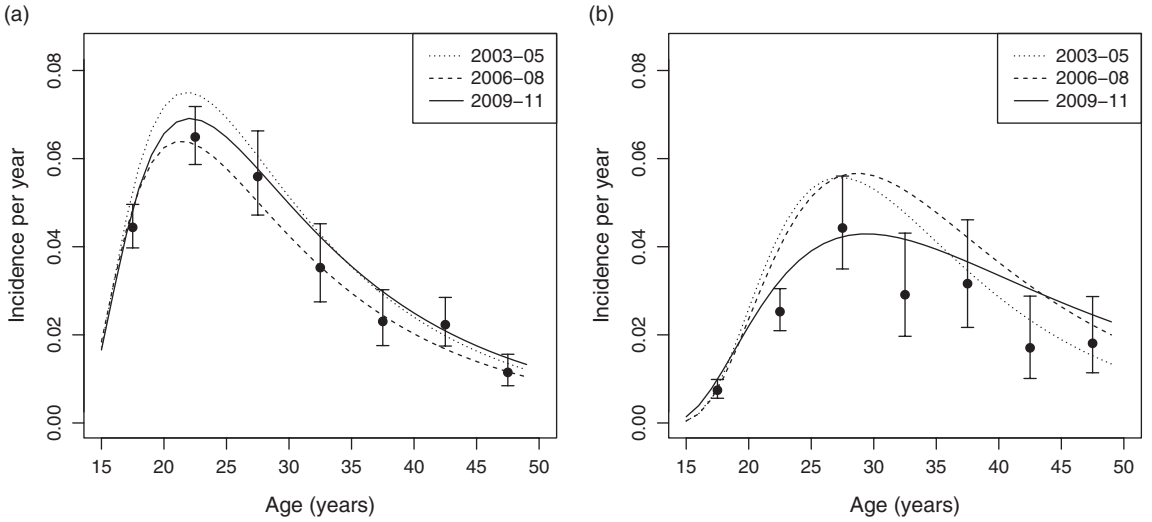

Fig. 3. Fitted HIV incidence in women (a) and men (b) as a function of age (each line represents different calendar periods) compared with observed cohort HIV incidence estimates (filled circles with 95% confidence bars) during the period 2003–2011. The shape parameter α of the Weibull survival distribution of HIV-infected individuals was fixed at a value of 5. Other parameter estimates correspond to those reported in Table 1.

When the HIV prevalence model fits of the three calendar periods are compared (Fig. 2), we observe virtually no change of HIV prevalence in younger women aged 15–24 years, but a substantial increase of HIV prevalence in women aged 30–49 years in 2009–2011 compared with the two earlier periods. In men, HIV prevalence in 2009–2011 is also higher than earlier periods, but the increase occurs at a later age than in women. During all three calendar periods, HIV prevalence in women is generally higher than in men throughout all age groups, with the exception of 45–49 year olds.

A comparison between fitted model-based and observed cohort-based incidence estimates for the full calendar period 2003–2011 is shown in Fig. 3. For women, the model incidence estimates are in broad agreement and overlap with the CIs of the observed cohort estimates, particularly for the two later calendar periods 2006–2008 and 2009–2011. In men, except for teenagers, the model-based incidence estimates tend to be considerably higher than the observed cohort estimates during the periods 2003–2005 and 2006–2008. For the latest period 2009–2011, the agreement between the two methods is somewhat better with an overlap of CIs occurring for four of the seven observed cohort age group estimates.

In women, incidence was marginally lower in the two later calendar periods over all age ranges, although this was not statistically different ($P = 0.932$ for parameter θ in Table 1). The age of peak incidence in women varied very little between calendar periods ranging between 21.4 and 22.1 years, with peak incidence ranging from 0.064 to 0.075 per year. Similarly in men, the age of peak incidence varied little ranging from 27.3 to 29.4 years, with peak incidence ranging from 0.043 to 0.057 per year (see Table 1). The age difference between men and women at peak incidence ranged between 5.5 and 7.3 years during the three calendar periods.

The cumulative incidence by age 50 years, which is equivalent to the probability of having acquired HIV by the age of 50 (assuming current age-specific incidence and in the absence of mortality), varied very little by calendar period, ranging from 0.713 to 0.768 in women and from 0.663 to 0.717 in men (Table 1 and Fig. 4). Although incidence in younger men was lower than in women, a higher incidence during middle ages translates into a similar cumulative incidence by the time men turn 50 years old.

For women, median survival after HIV infection was estimated at 10.0 (95% CI 8.8–11.2), 12.6 (95% CI 10.9–14.3) and 14.2 (95% CI 12.6–15.8) years during the calendar periods 2003–2005, 2006–2008 and 2009–11, respectively (Table 1). The difference of the survival scale parameter β between the first and last calendar period was highly significant ($P < 0.001$). For men, the respective median survival after HIV infection was estimated at 10.0 (95% CI 9.2–10.8), 9.8 (95% CI 6.4–13.2) and 14.0 (95% CI 10.6–17.4). The difference of the survival scale parameter β between the first and last calendar was borderline significant ($P = 0.03$).

Table 2 provides details of a sensitivity analysis of model estimates when varying the shape parameter α from 2 to 5. Higher values of α make the survival distribution more rectangular-shaped by lowering the variance. Although previous authors have tended to use values ranging from 2 to 2.5 on the basis of observed mortality in sero-converters prior to ART [15], our results suggest that a

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Table 1. Maximum likelihood parameter estimates (95% confidence intervals) for each calendar period and sex.

Parameter estimates	Women			Men			*p**	
	2003–2005	2006–2008	2009–2011	2003–2005	2006–2008	2009–2011	Women	Men
μ (mean of lognormal incidence function)	2.77 (2.71–2.83)	2.75 (2.65–2.85)	2.82 (2.72–2.92)	3 (2.93–3.07)	3.12 (2.96–3.28)	3.31 (3.06–3.56)	0.368	0.021
σ (standard deviation of lognormal incidence function)	0.69 (0.66–0.72)	0.72 (0.68–0.76)	0.72 (0.68–0.76)	0.52 (0.48–0.56)	0.56 (0.51–0.61)	0.67 (0.59–0.75)	0.377	0.001
θ (scaling factor of lognormal incidence function)	1.63 (1.42–1.84)	1.42 (1.18–1.66)	1.62 (1.38–1.87)	1.28 (1.13–1.43)	1.53 (0.88–2.18)	1.58 (0.91–2.25)	0.932	0.388
Peak incidence (per year)	0.075	0.064	0.069	0.056	0.057	0.043		
Age of peak incidence[a] (years)	21.8	21.4	22.1	27.3	28.6	29.4		
Cumulative incidence by age 50 years	0.768	0.713	0.756	0.680	0.717	0.663		
α (fixed shape parameter of survival after HIV infection)	5	5	5	5	5	5		
β (scale parameter of survival after HIV infection)	10.8 (9.5–12.1)	13.6 (11.8–15.4)	15.3 (13.6–17.0)	10.8 (9.9–11.7)	10.5 (6.8–14.2)	15.1 (11.5–18.7)	<0.001	0.03
Median survival after HIV infection[b] (years)	10.0 (8.8–11.2)	12.6 (10.9–14.3)	14.2 (12.6–15.8)	10.0 (9.2–10.8)	9.8 (6.4–13.2)	14.0 (10.6–17.4)	<0.001	0.03
Akaike information criterion	13256.1	14579.0	15701.3	5201.5	5034.7	5190.5		

*P value for t-test of difference between 2003–2005 and 2009–2011.
[a]The age of peak incidence corresponding to the mode of the offset lognormal distribution is given by $\exp(\mu - \sigma^2) + 12$.
[b]The median survival after HIV infection of the Weibull distribution with shape parameter α and scale parameter β is given by $\beta(\log(2))^{\frac{1}{\alpha}}$.
$\exp(\mu - \sigma^2) + 12$.

value of α equal to 5 provided the best fit for our data. Although model fit according to AIC could be improved by increasing the value of α beyond 5 (data not shown), higher values of α had a little impact on estimates of median survival, peak incidence and age at peak incidence. In particular, the increase of median survival after HIV infection between calendar periods 2003–2005 and 2009–2011 remains robust throughout a realistic parameter range.

Discussion

In this study, we use a catalytic model to provide useful insights on the evolution of the South African HIV epidemic following the large-scale roll-out of antiviral treatment. Our modelling approach is appealing because it is solely based on age-specific seroprevalence data. Such information is much more commonly available than HIV incidence derived from cohorts followed up over time. Although other authors have advocated synthetic cohort approaches on serial seroprevalence surveys [15,33], our substantially simpler approach provides reasonable HIV incidence estimates when validated against the 'gold standard' of the cohort methodology. However, even if the observed cohort and model-based incidence estimates are similar, one should be careful not to overinterpret results because there are potential selection biases in the data used for the two incidence estimation approaches and the biases could differ across the two approaches, for example due to lower consent rates for repeat testing [9]. However, it is important to note that recent work [5,34] has confirmed that survey nonparticipation does not lead to large biases in cross-sectional HIV prevalence estimation in this community. It would be interesting to apply our model to other age-specific HIV prevalence data sets in high prevalence settings to obtain incidence estimates that are impossible to obtain otherwise.

Our model further provides additional evidence of a survival benefit of HIV-infected persons following ART roll-out between 2003 and 2011 in a high HIV prevalence setting in rural South Africa. Our findings, which are solely derived from cross-sectional seroprevalence data, are in agreement with previous epidemiological findings from longitudinal demographic surveillance in our setting [1,35,36] showing a significant reduction of HIV-associated mortality and increased life expectancy [2]. The increase of survival occurred at the same time as ART coverage in the area was scaled up by the South African government with the help of PEPFAR funding [3]. One of the side effects of this increased survival is a noticeable increase of HIV prevalence at the population level, particularly in women aged 30–49 years and in men aged 45–49 years. This in agreement with recent findings from our DSS showing that the increase in HIV prevalence was largely driven by the older age groups

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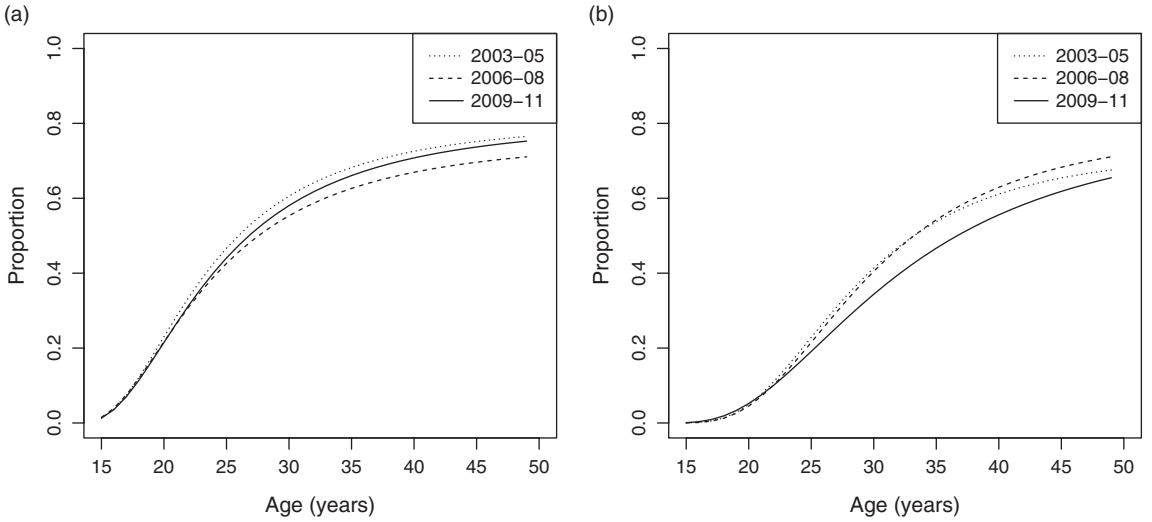

Fig. 4. Fitted cumulative incidence in women (a) and men (b) as a function of age. The shape parameter α of the Weibull survival distribution of HIV-infected individuals was fixed at a value of 5. Other parameter estimates correspond to those reported in Table 1.

in which the largest proportions of HIV-infected people received ART [5].

Our study confirms previous findings of age-mixing patterns in this [6,9,37] and other sub-Saharan settings [38], particularly in that the peak of HIV incidence occurs approximately 5–7 years earlier in women than in men. In this rural setting in KwaZulu-Natal, recent work has suggested that age-disparity within a sexual partnership (i.e. 'sugar-daddies') did not increase the risk of HIV acquisition among young women (Harling *et al.*, unpublished observation). Although our model allowed incidence functions to vary between sexes, for future work, it would be interesting to extend our modelling framework to directly estimate sexual mixing patterns ('who acquires infection from whom'), which was a popular approach for respiratory infections [39,40] prior to the availability of empirical social contact data [41].

From a public health perspective, our modelled incidence estimates were not suggestive of a major decrease of HIV incidence, which is one of the major goals set by the South African government in the years to come [7]. One possible reason for this is that our model could have been underpowered to detect a small reduction of incidence [42]. A review of several transmission models has predicted that an incidence decrease in South Africa is likely to fall in the range of 15–25% by 2010 following the roll-out of ART [43]. Moreover, a recent study [36] in our setting has shown that treatment levels have to reach a relatively high coverage (between 20 and 30% of all HIV-positive individuals on treatment) to have a significant effect on reducing the risk of acquiring HIV infection at the population level. Coverage levels of 30% in adults aged between 15 and 49 years were only obtained in 2011 [5]. It should be noted that the study by Tanser *et al.* [6], which also included individuals older than 50 years, found that the decline in the risk of acquisition of HIV infection with increasing ART coverage was more pronounced in the older age groups (>35 years of age). Taken together, this might suggest that incidence reductions are more likely to have occurred in older age groups due to higher access and uptake of ART.

One of the innovative aspects of our modelling approach is that we were able to estimate the increase of survival after HIV acquisition without having to rely on external mortality sources. Although previous authors have fixed both shape and scale parameters on the basis of observed survival in HIV-infected cohorts, we were able to estimate one of these, the scale parameter. Although it is possible in principle to simultaneously fit the shape and scale parameters, in practice, we found that the maximum likelihood estimates of the shape parameter tended to infinity whereas the likelihood converged to a finite value. This could be due to insufficient sample sizes at older ages, so that our data did not contain enough information to successfully estimate all parameters. The problem is likely to be amplified by correlation among parameters, which is why we decided to keep the shape parameter constant and estimate the remaining parameters as other researchers have done.

One limitation of our study is that our catalytic model does not take advantage of the longitudinal set-up of our HIV-surveillance, but rather treats yearly cross-sectional sero-surveys as independent data points. Obviously, this is a crude simplification and other authors have proposed more complicated models with a demographically correlated period/cohort or serial approaches [15,44]. However, we do take advantage of the longitudinal set-up in our model validation exercise. For future work, it would be of great benefit to refine model estimates within

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Table 2. Sensitivity analysis of shape parameter α on median survival, age at peak incidence and peak incidence estimates.

	Women			Men			Difference early – late[a]	
	2003–2005	2006–2008	2009–2011	2003–2005	2006–2008	2009–2011	Women	Men
$\alpha = 2$								
Median survival after HIV infection	6.7	8.9	9.7	6.8	6.9	9.6	3	2.8
Age at peak incidence	22.7	22.0	23.0	28.0	29.2	30.8	0.3	2.8
Peak incidence	0.098	0.079	0.087	0.073	0.072	0.055	−0.012	−0.018
Akaike information criterion	13 269.5	14 596.3	15 719.0	5208.6	5040.0	5201.4		
$\alpha = 3$								
Median survival after HIV infection	8.7	11.2	12.6	8.8	8.8	12.2	3.9	3.4
Age at peak incidence	22.2	21.6	22.4	27.6	28.8	30.0	0.2	2.4
Peak incidence	0.083	0.069	0.075	0.062	0.062	0.047	−0.012	−0.015
Akaike information criterion	13 262.5	14 587.1	15 709.4	5204.9	5037.1	5195.7		
$\alpha = 4$								
Median survival after HIV infection	9.6	12.1	13.7	9.7	9.5	13.4	4.1	3.7
Age at peak incidence	21.9	21.5	22.2	27.4	28.7	29.6	0.3	2.2
Peak incidence	0.077	0.066	0.071	0.058	0.058	0.044	−0.006	−0.012
Akaike information criterion	13 258.5	14 582.0	15 704.2	5202.8	5035.6	5192.4		
$\alpha = 5$								
Median survival after HIV infection	10.1	12.6	14.2	10.1	9.8	14.0	4.1	3.9
Age at peak incidence	21.8	21.4	22.1	27.3	28.6	29.4	0.3	2.1
Peak incidence	0.075	0.064	0.069	0.056	0.057	0.043	−.006	−0.013
Akaike information criterion	13 256.1	14 579.0	15 701.3	5201.5	5034.7	5190.5		

[a]Difference between 2003–2005 and 2009–2011.

a Bayesian framework by integrating other existing data sources such as age-specific HIV-associated mortality that can provide additional information on the underlying relevant age-dependent epidemiological processes.

Our modelling study thus provides further evidence of the substantial demographic impact of ART scale-up between 2004 and 2011 in our rural community in South Africa in terms of improving survival of HIV-infected persons. If current trends of local ART coverage are maintained or increased, we will expect survival of HIV-infected persons to increase further, as predicted by mathematical modelling [43]. Because our modelling approach is based on cross-sectional seroprevalence data rather than longitudinal cohort data, it could prove useful to monitor trends of HIV survival and incidence in other African settings with increasing ART coverage.

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J.M., F.T., T.B. and M.L. designed the study; J.M. and E.G. coded the catalytic model in R and conducted the analysis; E.G. and F.T. provided the cohort-based incidence estimates; all authors contributed to the drafting of the manuscript.

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Conflicts of interest

There are no conflicts of interest.

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