The effect of changes in condom usage and antiretroviral treatment coverage on human immunodeficiency virus incidence in South Africa: a model-based analysis

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This study aims to assess trends in human immunodeficiency virus (HIV) incidence in South Africa, and to assess the extent to which prevention and treatment programmes have reduced HIV incidence. Two models of the South African HIV epidemic, the STI (sexually transmitted infection)–HIV Interaction model and the ASSA2003 AIDS and Demographic model, were adapted. Both models were fitted to age-specific HIV prevalence data from antenatal clinic surveys and household surveys, using a Bayesian approach. Both models suggest that HIV incidence in 15–49 year olds declined significantly between the start of 2000 and the start of 2008: by 27 per cent (95% CI: 21–32%) in the STI–HIV model and by 31 per cent (95% CI: 23–39%) in the ASSA2003 model, when expressed as a percentage of incidence rates in 2000. By 2008, the percentage reduction in incidence owing to increased condom use was 37 per cent (95% CI: 34–41%) in the STI–HIV model and 23 per cent (95% CI: 14–34%) in the ASSA2003 model. Both models also estimated a small reduction in incidence owing to antiretroviral treatment by 2008. Increased condom use therefore appears to be the most significant factor explaining the recent South African HIV incidence decline.

Keywords: HIV incidence; mathematical model; sexual behaviour; South Africa

1. INTRODUCTION

The estimation of human immunodeficiency virus (HIV) incidence is critical to the evaluation of HIV control programmes, and to the monitoring of changes in HIV transmission patterns. However, the estimation of HIV incidence at a national level is challenging. Longitudinal studies, usually considered the gold standard in HIV incidence estimation, are typically conducted in surveillance sites that cannot be considered nationally representative, and the high cost of these surveys makes it impractical to conduct them at a national level. Laboratory tests that estimate HIV incidence on the basis of recent HIV infection have produced promising results in some settings, but a major concern has been the high proportion of HIV-positive specimens that yield ‘false recent’ results and the variability of this proportion between settings [1,2]. A variety of mathematical formulae have been proposed to estimate HIV incidence from cross-sectional HIV prevalence data [3–5], but confidence intervals around these estimates have generally been wide, particularly when considering HIV incidence patterns by age and sex [4–7]. This makes it difficult to achieve precision in estimating changes in HIV incidence over time, or in identifying the age groups in which HIV incidence is highest.

A fourth possible approach to estimate HIV incidence is through the use of dynamic mathematical models that are fitted to HIV prevalence data [8–10]. An advantage of this approach is that it allows the inclusion of HIV prevalence data from antenatal surveys as well as household surveys, and can potentially be extended to include mortality data, morbidity data [11] and sexual behaviour data [12]. This means that HIV incidence can potentially be estimated with greater accuracy and precision. A second major advantage is that dynamic mathematical models can be used to make inferences about how much of the change in HIV incidence is attributable to the impact of interventions [9]. This is important, as HIV incidence can decline in the absence of any...
intervention once the epidemic has reached a saturation threshold [13], and a simple analysis of whether HIV incidence is rising or falling is therefore of limited value when assessing the impact of HIV prevention programmes. However, mathematical modelling approaches also have drawbacks. Comparisons of different models of chlamydial infection [14] and influenza [15] demonstrate that different model structures can lead to substantially different conclusions regarding intervention impact. One model alone may therefore provide a misleading sense of precision if it is considered in isolation of other models.

Recent analysis suggests that adult HIV incidence has declined substantially in many African countries over the last decade [16], but it remains unclear how much of this decline is due to the impact of interventions. In South Africa, a recent analysis of household prevalence data has suggested a substantial decline in HIV incidence between the 2002–2005 and 2005–2008 periods [7]. However, there has been limited evaluation of the factors accounting for the decline in HIV incidence. Data from sexual behaviour surveys suggest that condom usage has increased substantially since 1998, but there is little evidence of changes in other sexual behaviours [17]. Although it is probable that increases in condom usage have contributed to the decline in HIV incidence, there is scepticism regarding the accuracy of self-reported condom usage data, with a number of studies showing significantly higher levels of consistent condom usage reported in traditional face-to-face interviews than in more anonymous interview formats, which are considered to be subject to less social desirability bias [18–20]. There is also speculation that some of the decline in HIV incidence may be attributable to the impact of antiretroviral treatment (ART), which has been rapidly rolled out in the South African public health sector since 2004 [21].

The objective of this paper is to estimate HIV incidence trends in South Africa by fitting dynamic mathematical models to age-specific HIV prevalence data from national antenatal and household surveys, and to assess how much of the change in HIV incidence can be attributed to the impact of prevention and treatment programmes introduced prior to 2008. The potential for bias in the reporting of condom use is considered in the fitting of the model, and the levels of condom use most consistent with observed HIV prevalence trends are evaluated.

2. METHODS

2.1. Mathematical models

Two deterministic models of the South African HIV/AIDS epidemic are assessed: the STI (sexually transmitted infection)–HIV Interaction model [12] and the ASSA2003 AIDS and Demographic model [22,23]. Both models have been adapted for the purpose of the current analysis, and full details of the adapted models are included in electronic supplementary material, appendices A and B, respectively. Briefly, both models divide the South African population by age and sex, and the growth of the population over time is projected, starting from 1985, allowing for changing levels of mortality and fertility. The sexually active population is split into a number of risk groups, with rates of partnership formation depending on individuals’ age, sex and risk group. In both models, the rate of HIV acquisition is determined by assumed probabilities of HIV transmission per act of unprotected sex, which vary according to the individual’s age, sex and risk group, as well as the HIV stage of their infected partner. Both models allow for levels of condom usage to change over time, in response to increased condom availability and HIV awareness. A changing proportion of newly eligible adults are assumed to start ART each year [21], and individuals receiving ART are assumed to be roughly 90 per cent less infectious than individuals with untreated AIDS [24,25]. Key structural differences between the two models are summarized in electronic supplementary material, appendix C. The code for running the models is available from the first author on request.

2.2. Data sources

Both models are fitted to two HIV prevalence data sources: the 1997–2005 national antenatal clinic surveys [26] and the national household surveys conducted by the Human Sciences Research Council (HSRC) in 2005 and 2008 [27,28]. Although antenatal surveillance data are also available over the periods 1990–1996 and 2006–2009, these data have not been included in the model fitting, as the early antenatal surveys were biased towards urban areas, and the more recent antenatal surveys have followed a different sampling protocol [29]. HIV prevalence data from national household surveys in 2002 [30] and 2003 [31] have also not been included in the model fitting, as the HIV testing protocol in these surveys (saliva testing with no confirmatory testing of reactive specimens) differed from that in the 2005 and 2008 surveys, and response rates in the 2002 survey were relatively low. The ASSA2003 model is also fitted to reported death data over the 1997–2004 period [32], as it is a more demographically detailed model. Being more behaviourally detailed, the STI–HIV model is also fitted to data on numbers of sexual partners from the 2005 HSRC survey [12].

2.3. Model fitting

A Bayesian approach is adopted in fitting the models to the age-specific data. The first step in this analysis is the specification of a likelihood function to represent the ‘goodness-of-fit’ to South African HIV prevalence data, for a given set of model assumptions. This likelihood is calculated on the assumption that—if the model is a fair reflection of reality—differences between logit-transformed model estimates of HIV prevalence and logit-transformed survey estimates of HIV prevalence are normally distributed with zero mean, or with mean equal to a constant antenatal bias parameter in the case of the antenatal survey data. Full details of the method used to define the likelihood in respect of the HIV prevalence data are included in electronic supplementary material, appendix A. A separate likelihood function is defined with respect to the reported death data, for the ASSA2003
model. As detailed in electronic supplementary material, appendix B, this is calculated on the assumption that differences between the logarithm of the reported numbers of deaths and the logarithm of the modelled numbers of deaths are normally distributed, with mean equal to the logarithm of the proportion of adult deaths that are reported. Because this analysis of the ASSA2003 model allows for uncertainty in the extent of the under-reporting of deaths and in the extent of the antenatal bias, the calibration of the model to the death data and the antenatal data differs from that in the previously published ASSA2003 model [23]. Finally, the likelihood in respect of the reported number of partners makes allowance for social desirability bias, which differs according to individuals’ sex and marital status [12].

The second step in the Bayesian analysis is the specification of prior distributions to represent uncertainty regarding the key parameters in both models. Prior distributions are specified for 14 parameters in the STI–HIV model and 17 parameters in the ASSA2003 model (see electronic supplementary material, appendices A and B, respectively). Both models have been adapted to include a parameter to represent the extent of bias in female reporting of condom usage, \( u \). This parameter can take on any value between 0 and 1, with 0 implying no bias (i.e. national survey estimates of the proportion of sex acts that are protected), and 1 implying a very high level of bias (i.e. only women who report using condoms for contraception in Demographic and Health Surveys). Lines represent model estimates of trends in the proportion of women using condoms, for different values of the \( u \) parameter (black line, model \( u = 0 \); grey line, model \( u = 0.57 \); dashed line, model \( u = 1.00 \)). (a) STI–HIV model; (b) ASSA2003 model.

**2.4. Assessing the role of heterogeneity**

Since previously published models have suggested that the impact of interventions on HIV incidence is determined by the degree of heterogeneity in sexual behaviour [9,34,35], we hypothesized that the change in HIV incidence owing to ART and condoms would be related to the degree of heterogeneity in susceptibility. As a measure of heterogeneity in susceptibility, we calculate the coefficient of variation in HIV incidence rates between age, sex and risk groups.

The greater the variation in HIV incidence rates between age, sex and risk groups (within the 15–49 age range), the greater will be the coefficient of variation. Since the coefficient of variation in incidence may itself be affected by intervention programmes, the coefficient of variation is calculated only in the counterfactual scenario (iii) described previously. Associations between the coefficient of variation and key model outputs are evaluated using correlation coefficients and partial

![Figure 1. Trends in condom usage in women aged 15–24. Filled circles represent proportions of women reporting having used a condom the last time they had sex, in nationally representative household surveys. Unfilled circles represent proportions of sexually active women who report using condoms for contraception in Demographic and Health Surveys. Lines represent model estimates of trends in the proportion of women using condoms, for different values of the \( u \) parameter (black line, model \( u = 0 \); grey line, model \( u = 0.57 \); dashed line, model \( u = 1.00 \)). (a) STI–HIV model; (b) ASSA2003 model.](http://rsif.royalsocietypublishing.org/)
correlation coefficients that control for variation in the condom bias parameter [36].

To evaluate further the role of heterogeneity, the two models were compared with the results of a third model of the South African HIV epidemic, which does not divide the sexually active population into risk groups, but which does allow for age variation in sexual behaviour (i.e. assuming that there is no variation in sexual activity after controlling for age and sex). This model of Bacaeë©r et al. [37] was not modified from its originally published form and was not refitted to the HIV prevalence data (as with the other two models), but was included to illustrate the potential impact of assuming limited or no heterogeneity in behaviour, as this assumption is widely used in the HIV intervention modelling literature [38].

3. RESULTS

3.1. Model calibration

Both adapted models produce posterior estimates of HIV prevalence in pregnant women consistent with antenatal clinic survey estimates, in aggregated comparisons (figure 2) and in age-specific comparisons (electronic supplementary material, appendices A and B). Age-specific posterior estimates of HIV prevalence in the general population are also reasonably consistent with results from the 2005 and the 2008 national household surveys, although the ASSA2003 model tends to estimate a slightly higher HIV prevalence in young women, as well as a slightly higher prevalence in older men, when compared with the STI–HIV model (figure 3). ASSA2003 model estimates of mortality are also consistent with age- and sex-specific reported death data, after adjusting for incomplete reporting of deaths (electronic supplementary material, appendix B).

3.2. Human immunodeficiency virus incidence trends

Although the STI–HIV and ASSA2003 models produce substantially different estimates of HIV incidence prior to 1996 (figure 4a), estimates of average HIV incidence in all adults aged 15–49 are similar after 2000 (table 1). Estimates of HIV incidence over the 2002–2005 period are consistent with the estimate of 2 per cent (95% CI: 1.2–3.0%) calculated using the synthetic cohort method [7], but estimates over the 2005–2008 period are slightly higher than the estimate of 1.3 per cent (95% CI: 0.6–2.5%) obtained using synthetic cohorts. The reduction in HIV incidence in adults aged 15–49, between the start of 2000 and the start of 2008, is estimated to be 27 per cent (95% CI: 21–32%) in the STI–HIV model and 31 per cent (95% CI: 23–39%) in the ASSA2003 model, when expressed as a percentage of HIV incidence in 2000. Despite these substantial reductions in HIV incidence, the lifetime risk of acquiring HIV remains high: if age-specific HIV incidence rates were to remain constant at their levels at the start of 2008 (figure 5), the chance of an uninfected 15 year old becoming infected before age 60 would be 55 per cent (95% CI: 51–58%) in females and 45 per cent (95% CI: 42–49%) in males, in the STI–HIV model. Corresponding lifetime risks in the ASSA2003 model are 46 per cent (95% CI: 42–49%) and 35 per cent (95% CI: 31–39%), respectively.

Although the two models estimate similar trends in HIV incidence over the 2000–2008 period, age-specific estimates of incidence differ substantially, with the ASSA2003 model suggesting a greater concentration of female HIV incidence in women aged 15–24 (figure 5b). The models also differ substantially in the extent to which they attribute the reduction in HIV incidence over the 2000–2008 period to the impact of increased condom use and ART. The STI-HIV model estimates that if there had been no ART or increase in condom use, HIV incidence would not have changed significantly between 2000 and 2008, while the ASSA2003 model estimates that in counterfactual scenario (iii) HIV incidence in 2008 would have been lower than the level in 2000 by 17 per cent (95% CI: 10–25%) (table 1 and figure 4b). Both models estimate that a significant part of the reduction in HIV incidence is due to increased condom usage, although the two models differ in the extent to which they attribute the change in incidence to the impact of condoms. By 2008, the percentage reduction in incidence owing to increased condom use was 37 per cent (95% CI: 34–41%) in the STI–HIV model and 23 per cent (95% CI: 14–34%) in the ASSA2003 model. Both models also estimate a small reduction in incidence owing to ART.

3.3. The role of heterogeneity

The variation in the estimated impact of condoms can be explained largely by differences in modelled heterogeneity in susceptibility to HIV. Both models estimate a significant positive association (r = 0.84) between the reduction in incidence that would have been expected in counterfactual scenario (iii), and the coefficient of variation in HIV incidence (figure 6a). The reduction in HIV incidence owing to increased condom use and ART in 2008, expressed as a percentage of the HIV incidence that would have been expected in counterfactual
scenario (iii) in 2000, is strongly negatively associated with the coefficient of variation in HIV incidence ($r = -0.82$ in the STI–HIV model and $r = -0.31$ in the ASSA2003 model). After controlling for the condom bias parameter, the association between the reduction in incidence owing to increased condom use/ART and the coefficient of variation becomes stronger in the ASSA2003 model ($r = -0.89$), but controlling for the condom bias parameter does not materially alter the other correlation coefficients (results not shown).

The ASSA2003 model estimates a greater coefficient of variation, on average, than the STI–HIV model (table 1), and the smaller estimated impact of increased condom use in the ASSA2003 model is therefore explained by the higher degree of heterogeneity in susceptibility estimated in ASSA2003.

The model of Bacáër et al. [37] produced a substantially lower coefficient of variation in HIV incidence than the other two models (table 1). Consistent with the associations noted previously, this model estimated that in the absence of increased condom use and ART, a dramatic increase in HIV incidence would have been expected between 2000 and 2008, and that increased condom use has led to much more dramatic declines in incidence than the other two models suggest. However, the model produced similar estimates of the overall reduction in HIV incidence between 2000 and 2008 (25%) and the lifetime risk of HIV infection.

Figure 3. HIV prevalence levels in the general population. Solid (ASSA2003 model) and dashed (STI–HIV model) lines represent posterior mean model estimates of HIV prevalence in the general population. Filled circles represent results from 2005 ((c) females; (d) males) and 2008 ((a) females; (b) males) national household surveys [27,28].

Figure 4. Posterior model estimates of HIV incidence trends in 15–49 year olds. Dark grey bars (STI–HIV model) and light grey bars (ASSA2003 model) represent posterior average estimates of HIV incidence rates, with error bars representing 95% CI. (a) Base scenario; (b) counterfactual scenario (iii), representing the HIV incidence levels that would have been expected if there had been no ART or change in condom use.
Bias in reported condom use

Both models suggest that self-reported condom usage is exaggerated, with posterior estimates of the condom bias parameter of 0.80 (95% CI: 0.66–0.91) and 0.66 (95% CI: 0.34–0.91) in the STI–HIV and ASSA2003 models, respectively. Posterior model estimates of trends in condom usage in women aged 15–24 and 25–49 are compared with proportions reported in national surveys in electronic supplementary material, appendices A and B.

Sensitivity analyses were conducted to assess the effect of assuming zero bias in the reporting of condom use (θ = 0), and the Metropolis algorithm was rerun for each model. In the STI–HIV model, this led to modelled HIV prevalence levels in young pregnant women following a steeper downward trend than those observed in recent surveys (results shown in electronic supplementary material, appendix A), which suggests that the assumption of zero bias in reporting of condom use is not plausible. Setting the condom bias parameter to zero in the ASSA2003 model led to little visible difference in the model fit to the HIV prevalence data (electronic supplementary material, appendix B), but led to a substantially greater

Table 1. Comparison of model estimates of HIV incidence in individuals aged 15–49.

<table>
<thead>
<tr>
<th></th>
<th>STI–HIV model estimate (95% CI)</th>
<th>ASSA2003 model estimate (95% CI)</th>
<th>Bacăër et al. estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV incidence at start of 2000 (%)</td>
<td>2.45 (2.26–2.66)</td>
<td>2.25 (2.13–2.38)</td>
<td>2.51</td>
</tr>
<tr>
<td>HIV incidence at start of 2008 (%)</td>
<td>1.79 (1.66–1.93)</td>
<td>1.55 (1.37–1.73)</td>
<td>1.87</td>
</tr>
<tr>
<td>average HIV incidence 2002–2005 (%)</td>
<td>2.11 (1.97–2.26)</td>
<td>1.90 (1.77–2.03)</td>
<td>2.86</td>
</tr>
<tr>
<td>average HIV incidence 2005–2008 (%)</td>
<td>1.86 (1.73–2.00)</td>
<td>1.62 (1.45–1.79)</td>
<td>2.19</td>
</tr>
<tr>
<td>percentage change in incidence that would have been expected in counterfactual scenario (iii), 2000–2008</td>
<td>3 (–5 to 14)</td>
<td>−17 (−25 to −10)</td>
<td>203</td>
</tr>
<tr>
<td>percentage reduction in incidence in 2008 owing to condomsb</td>
<td>37 (34–41)</td>
<td>23 (14–34)</td>
<td>85</td>
</tr>
<tr>
<td>percentage reduction in incidence in 2008 owing to ARTc</td>
<td>8.1 (6.0–9.4)</td>
<td>1.4 (0.7–2.6)</td>
<td>5.7</td>
</tr>
<tr>
<td>risk of acquiring HIV by age 60 if HIV-negative at age 15 at start of 2008: malesd (%)</td>
<td>45 (42–49)</td>
<td>35 (31–39)</td>
<td>51</td>
</tr>
<tr>
<td>risk of acquiring HIV by age 60 if HIV-negative at age 15 at start of 2008: femalesd (%)</td>
<td>55 (51–58)</td>
<td>46 (42–49)</td>
<td>55</td>
</tr>
<tr>
<td>coefficient of variation in HIV incidence rates in 2008, in counterfactual scenario (iii)</td>
<td>2.7 (2.4–3.1)</td>
<td>3.2 (2.9–3.6)</td>
<td>0.6</td>
</tr>
<tr>
<td>condom bias parameter</td>
<td>0.80 (0.66–0.91)</td>
<td>0.66 (0.34–0.91)</td>
<td>—</td>
</tr>
</tbody>
</table>

aCalculated as 1 – HIV incidence at start of 2008 divided by HIV incidence at start of 2000 (in counterfactual scenario (iii) the incidence rates are those that would have been expected in the absence of increased condom use and ART).
bCalculated as 1 – model estimate of HIV incidence in 2008 divided by estimate of incidence that would have been expected in 2008 in the absence of increased condom use.
cCalculated as 1 – model estimate of HIV incidence in 2008 divided by estimate of incidence that would have been expected in 2008 in the absence of ART.
dCalculated on the assumption that age-specific incidence rates remain constant after 2008.

eNegative percentage change should be interpreted as a percentage reduction.
ART, antiretroviral treatment.
reduction in HIV incidence in 2008 owing to condoms (41% (95% CI: 37–45%), closer to the 37% reduction estimated in the STI-HIV model (table 1)).

4. DISCUSSION

Despite significant structural differences, both of the models considered in this analysis suggest that adult HIV incidence in South Africa has declined significantly since the year 2000. In addition, both models suggest that most of this decline can be attributed to the effect of increased condom usage, and that some of the decline may be attributable to the impact of ART on the infectiousness of individuals with advanced HIV disease. The assumed increases in condom usage are consistent with the timing of increases in the distribution of male condoms in the South African public health sector [39] as well as the timing of behaviour change interventions (figure 7). School-based HIV/AIDS life-skills programmes were introduced in South Africa in 1998, and increases in condom use among youth have been found to be associated with exposure to these life-skills programmes [40], consistent with experience in other countries [41]. South African HIV communication programmes have been significantly strengthened in recent years, and exposure to these programmes appears to be associated with positive changes in knowledge and reductions in sexual risk behaviour and HIV risk [31,42–45]. Although behaviour changes other than increased condom usage may have played a role in the decline in HIV incidence, no other form of behaviour change has been consistently reported in South Africa [46], and other forms of behaviour change have therefore not been considered in this analysis.

Despite the encouraging evidence of the impact of increased condom usage, both models suggest that condom usage is significantly over-reported, with the
posterior mean of the condom bias parameter being substantially greater than the prior mean. This could be a reflection of social desirability bias, which may have been enhanced by HIV communication programmes that have heavily promoted condoms as an HIV prevention strategy. When the condom bias parameter was assumed to be zero in the STI–HIV model, the modelled HIV prevalence levels in young pregnant women followed a steeper downward trend than those observed in recent surveys, which suggests that the assumption of zero bias in reporting of condom use is not realistic. However, it is possible that the assumption of condom usage trends similar to those reported might produce results more consistent with recorded trends in HIV prevalence if condoms were assumed to have relatively low efficacy in preventing HIV transmission. We have assumed that condoms reduce the HIV transmission probability per act of sex by 90 per cent or more, but other modellers have noted that if the HIV transmission probability per partnership is nonlinearly related to the number of unprotected sex acts in the partnership, the impact of high levels of condom usage may be relatively modest [47].

This analysis suggests that ART may be playing a role in HIV prevention in South Africa, reducing the number of new HIV infections at the start of 2008 by as much as 10 per cent. At this time, adult ART coverage in South Africa was estimated to be only 40 per cent [21], and it is therefore likely that the impact of ART on HIV incidence would become more substantial as ART coverage is expanded, and as guidelines change to allow for initiation of ART in earlier stages of disease. However, this finding should be treated with caution, as there is much uncertainty regarding the behavioural changes associated with ART in the untreated population. A recent study in Kenya has suggested that incorrect beliefs about ART and ART-associated risk compensation might be causing increased levels of HIV risk [48], while studies in South Africa have suggested that ART is associated with increased uptake of HIV counselling and testing [49,50], which would be expected to reduce levels of risk behaviour. Neither of these factors has been taken into consideration in either model, and there is a need for further research to quantify the significance of these factors. Other models have also demonstrated that the preventive benefit of ART is highly dependent on the average duration of survival after ART initiation and the extent of behaviour change in treated individuals [51]—factors that remain uncertain. The smaller impact of ART in the ASSA2003 model is explained by the fact that this model assumes a greater reduction in the frequency of sex in the advanced stages of disease than the STI–HIV model, and consequently individuals in the late stages of disease (whether treated or untreated) contribute relatively little to total heterosexual transmission.

Both models found that heterogeneity in susceptibility to HIV had a significant influence on (i) the reduction in HIV incidence that would have been expected in the absence of interventions and (ii) the reduction in HIV incidence attributable to interventions. This was further demonstrated in a comparison with an independently developed and calibrated model of the South African model that allowed for only limited heterogeneity [37]. In the absence of interventions, high levels of heterogeneity imply that HIV saturates high-risk groups rapidly, precipitating a sharp decline in incidence as the number of susceptible individuals in the high-risk groups diminishes. High levels of heterogeneity also imply that when interventions are introduced, many individuals experience negligible reductions in HIV risk in absolute terms—either because they were at no risk of HIV to begin with, or because their HIV risk was so high that the reduction in risk per act of sex makes little difference to their cumulative risk of HIV acquisition over many sexual encounters. Higher heterogeneity in susceptibility is therefore associated with greater ‘natural’ declines in HIV incidence and smaller declines in incidence attributable to interventions.

The STI–HIV model allows for less heterogeneity in HIV risk than the ASSA2003 model, because it assumes that no individual has more than two concurrent partners, and because it does not restrict a high proportion of the population to a ‘not at risk’ group. The model of Bacaër and co-workers allows for only limited heterogeneity, because it assumes that susceptibility to HIV is determined only by an individual’s age and sex. The different model structures therefore lead to substantially different conclusions regarding the impact of interventions on HIV incidence. This is consistent with other HIV modelling studies that have compared different models of heterogeneity when evaluating the impact of male circumcision [34] and ‘test and treat’ interventions [35]. It is also consistent with comparisons of mathematical models of chlamydia [52] and gonorrhoea [53], which suggest that models that allow for greater heterogeneity typically estimate smaller benefits from prevention programmes. Caution is therefore warranted in the use of mathematical models to evaluate the impact of HIV prevention programmes, and comparison of multiple models may be advisable. To formulate more accurate models and to validate existing models will require empirical estimates of heterogeneity in HIV susceptibility, possibly from existing sentinel surveys [54,55].

Although the Bayesian approach to HIV incidence estimation has the advantage of drawing together data from multiple sources, and thus achieving greater accuracy and precision, model-derived HIV incidence estimates are determined to some extent by fixed model assumptions, which may introduce spurious precision. For example, it may be unrealistic to assume that household prevalence surveys provide unbiased estimates of HIV prevalence in the general population. Although non-response bias appears to be minimal if it is assumed that recorded demographic and behavioural characteristics fully account for all variation in HIV prevalence [56], recent studies have suggested that individuals who do not participate in HIV prevalence surveys are more likely to be HIV-positive, independent of their demographic and behavioural characteristics [57–59]. The assumption that the antenatal bias parameter is constant over time may also be unrealistic. To the extent that pregnant women are having relatively more unprotected sex than women in the general
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population, they may become relatively less representative of women in the general population as HIV communication programmes become more established [60]. If the antenatal bias parameter has in fact been increasing in recent years, the extent of the reduction in HIV incidence may have been underestimated. However, allowing for a linear change in antenatal bias over time did not result in significant increases in the percentage reduction in HIV incidence after 2000, either in the STI–HIV model or in the ASSA2003 model (see electronic supplementary material, appendices A and B).

Another limitation of this analysis is that it does not consider the role of other STIs in HIV transmission [61]. To the extent that increases in condom usage are likely to have reduced the prevalence of other STIs, the impact of increased condom usage on HIV incidence may have been underestimated. However, to the extent that STIs lead to increased heterogeneity in HIV susceptibility, the impact of condom usage on HIV incidence may also have been over-estimated (figure 6b). This analysis also does not consider the potential impact of improvements in STI treatment in South Africa in the late 1990s, although the effect of these changes on HIV incidence is thought to be relatively modest [62].

Although it is encouraging that HIV incidence in South Africa has reduced by close to 30 per cent between 2000 and 2008, HIV incidence rates in South Africa remain alarmingly high. If age-specific incidence rates were to remain constant at the levels estimated at the start of 2008, 40–50% of 15 year olds would become infected with HIV by age 60. This clearly leaves little room for complacency, and it is important that HIV prevention and treatment programmes be intensified.

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REFERENCES


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