Using mathematical modelling to help explain the differential increase in HIV incidence in New South Wales, Victoria and Queensland: importance of other sexually transmissible infections


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Abstract. Background: Since 1999 there has been an increase in the number of HIV diagnoses in Australia, predominantly among men who have sex with men (MSM), but the magnitude of increase differs between states: ~7% rise in New South Wales, ~96% rise in Victoria, and ~68% rise in Queensland. Methods: Epidemiological, clinical, behavioural and biological data were collated into a mechanistic mathematical model to explore possible reasons for this increase in HIV notifications in MSM. The model was then used to make projections to 2015 under various scenarios. Results: The model suggests that trends in clinical and behavioural parameters, including increases in unprotected anal intercourse, cannot explain the magnitude of the observed rise in HIV notifications, without a substantial increase in a ‘transmission-increasing’ factor. We suggest that a highly plausible biological factor is an increase in the prevalence of other sexually transmissible infections (STI). It was found that New South Wales required an ~2-fold increase in other STI to match the data, Victoria needed an ~11-fold increase, and Queensland required an ~9-fold increase. This is consistent with observed trends in Australia for some STI in recent years. Future projections also indicate that the best way to control the current rise in HIV notifications is to reduce the prevalence of other STI and to promote condom use, testing for HIV, and initiation of early treatment in MSM diagnosed during primary infection. Conclusions: Our model can explain the recent rise in HIV notifications with an increase in the prevalence of other STI. This analysis highlights that further investigation into the causes and impact of other STI is warranted in Australia, particularly in Victoria.

Additional keywords: Australia, men who have sex with men.

Introduction

After a steady decline in HIV notifications during the 1990s in Australia, this current decade has seen an increase in the number of notifications, with the majority of cases involving men who have sex with men (MSM). Since 1999 there has been an ~44% increase in notifications among MSM. However, this trend is not uniform across all Australian states. For example, in New South Wales (NSW) there has been an ~7% rise, in Victoria (Vic.) there has been an ~96% increase, and an ~68% increase has been observed in Queensland (Qld).1 We seek to provide possible explanations for the differences in notifications between these three states, and to predict the future course of the HIV epidemic in these locations. To investigate the differences, we compiled available data from a variety of sources into a single mechanistic framework to understand how the various factors interact, and then estimated their relative importance in yielding an increase in new HIV infections. We developed a mathematical model to evaluate the HIV epidemics in MSM populations in NSW, Vic. and Qld, and incorporated data on time trends in various factors. These factors included: condom use, proportion of individuals diagnosed in primary HIV infection who received treatment upon diagnosis, proportion of MSM who test for HIV each year, proportion of treated patients with undetectable viral load, average number of sexual partners, and the proportion of MSM who disclose their serostatus. The model also considered other factors such as post-diagnosis behaviour change (possible increase or reduction in the number of casual partnerships), the frequency of sexual acts, and the increase in transmission due to the presence of other sexually transmissible infections (STI). We calibrated the model to match the observed number of HIV notifications in each state in 1999 and conducted rigorous uncertainty and sensitivity analyses.

Methods

Model structure

We constructed a deterministic compartmental model,2 formulated as a system of ordinary differential equations, to simulate the HIV epidemic in the MSM population in each state. The modelled population was divided into four main groups: uninfected with HIV (susceptible), HIV-infected but undiagnosed, diagnosed with HIV infection, and HIV-infected people on antiretroviral therapy. The HIV-infected
population in our model progresses through three stages of disease: from primary infection to chronic infection and eventually to AIDS (see Fig. 1). In our model each stage of infection was associated with a different viral load, which differentially determined the probability of transmission to a susceptible person during an act of penile–anal intercourse. We assumed that most Australian MSM will engage in both insertive and receptive acts. Thus, we assumed an average transmission probability per act that reflects both insertive and receptive penile–anal intercourse. The model thus comprised 10 groups in total (see Fig. 1 for a schematic illustration of the model showing the ‘flow’ of the population between the various disease states).

Parameter estimation and assumptions
We modelled the population of MSM in Australia, assuming a population size of 150 000–175 000 MSM nationally (~1–2% of the male population; see Appendix 1 for the proportion distributed among each state). Data were gathered on treatment in primary infection, HIV testing rates, number of casual partners, condom use, prevalence of other STI, and the rates of disclosure of serostatus among partners as reported in behavioural surveys conducted in each state for the years 1999 to 2006 (see Table 1). We also included other important factors that were not time dependent and remained constant over the period being modelled. Behaviour change post-diagnosis was one such factor; in this case, a multiplying factor acting on the number of sexual partners (range 0.4–1.1) was applied. This range specifies that there may be an increase or decrease in choosing new partners after HIV diagnosis, but on average there is a decrease in sexual partner acquisition. The frequency of sexual acts between regular partners was also important and ranged between 80 and 120 acts per year. A complete listing of parameters is given in Table A1 in the Appendix. Available

![Fig. 1. Schematic diagram of our compartmental model structure. Uninfected men who have sex with men (MSM) enter the model as susceptible (S). Upon infection, they move from the susceptible group into undiagnosed primary infection (I_P). From here a certain proportion will get tested and will then move into the diagnosed arm (to T_P, T_C or T_A, depending on disease stage). Those who are not diagnosed will continue to progress through the different disease stages (I_C or I_A). Once they eventually become diagnosed, a proportion will begin treatment (T_P, T_C or T_A; the proportion varies depending on the stage of infection). People can leave each group by ‘ageing’ out of the population or AIDS-related death once in AIDS stage (or at lower rates in chronic stage).]
Table 1. List of time-dependent parameters used in our mathematical model including graphs of changes in these parameters for New South Wales (squares), Victoria (triangles) and Queensland (diamonds)

<table>
<thead>
<tr>
<th>Time-dependent parameter</th>
<th>Graph</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reported cases of unprotected acts with casual partners</td>
<td><img src="image1" alt="Graph" /></td>
<td>5–7,11</td>
</tr>
<tr>
<td>Change in number of sexual partners</td>
<td><img src="image2" alt="Graph" /></td>
<td>8–10</td>
</tr>
<tr>
<td>Disclosure of serostatus</td>
<td><img src="image3" alt="Graph" /></td>
<td>5–7,11,14,15</td>
</tr>
<tr>
<td>Testing rates</td>
<td><img src="image4" alt="Graph" /></td>
<td>5–7</td>
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</table>

(Continued next page)
data were insufficient to enable precise estimation of the prevalence of individual STI in each of the states, and to quantify the impact of these on HIV transmission. Therefore, we did not model STI individually but made the assumption that a certain proportion of MSM would have another STI, and that the presence of an STI would increase their susceptibility to HIV acquisition. There is strong evidence that both ulcerative and non-ulcerative STI can increase the probability of HIV transmission by augmenting HIV infectiousness and susceptibility; reciprocally, HIV infection can enhance the transmission of other STI. Several studies (in heterosexuals) estimate the relative risks of HIV infection due to infection with other STI in the range 2–24, but largely clustering between 2 and 5. We assume that this relative risk is equivalent in MSM and include a ‘transmission-increasing’ factor of 2–5 if another STI is present.

In order to model the impact of STI on HIV transmission, it was necessary to estimate the proportion of MSM with other STI as well as trends over time and by state. This was problematic for several reasons. First, although there have been indications that the prevalence of some STI, notably syphilis, has been increasing in MSM in Australia, most data are reported only as notifications, not as the proportion of tests that are positive. Furthermore, the National Centre for HIV Social Research reported significant increases in testing (10–20%) in the past few years. Second, much of the published data on STI in MSM in Australia is from the ‘Health in Men’ (HIM) study and the incidence of STI has decreased in this highly tested, Sydney-based, HIV-negative cohort over the past few years. Third, there is little data on trends in STI incidence and prevalence in MSM for the other states. Fourth, the most prevalent STI associated with HIV transmission is herpes simplex virus (HSV)-2 with prevalence in the HIM cohort estimated at ~23%, masking any trends that might be occurring with other STI. Of course, HSV-2 is latent for significant proportions of the time in infected people and virus is shed periodically; thus, the effective prevalence of HSV-2 in terms of increasing HIV transmissibility is likely to be lower. Given the uncertainty, we assumed that the average proportion of MSM with STI (ulcerative or non-ulcerative that contribute to increasing HIV transmissibility) is in the range 5–15% initially (that is, at 1999).

To investigate national HIV trends, we did not distinguish STI rates between states. There is strong evidence of a significant rise in the incidence (and prevalence) of STI in recent years.12,13,32

### Sampling and sensitivity analysis

Each parameter used in the model was assigned a uniform distribution between a minimum and maximum value, and Latin Hypercube Sampling33 was used to generate 10 000 samples from each distribution for each state. These parameter sets became input values for our model and Monte Carlo filtering was used to remove all parameter sets that could not generate the number of observed notifications in 1999 for

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**Table 1. (Continued)**

<table>
<thead>
<tr>
<th>Time-dependent parameter</th>
<th>Graph</th>
<th>References</th>
</tr>
</thead>
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<tr>
<td>Treatment during primary infection</td>
<td><img src="image1" alt="Graph" /></td>
<td>4</td>
</tr>
<tr>
<td>Proportion treated who achieve viral suppression</td>
<td><img src="image2" alt="Graph" /></td>
<td>23,42,43</td>
</tr>
</tbody>
</table>
each state. This left 4247 simulations for NSW, 3120 for Vic. and 3970 for Qld. These parameter sets then constituted the baseline for running simulations under several scenarios. Sensitivity analyses were carried out to determine important factors involved in the epidemic. The SaSAT software package was used to generate samples and to carry out the sensitivity analyses.

Further filtering was conducted for projecting the HIV epidemic over the next 10 years. A regression line was fitted to the notifications data, and only simulations that were within 10% of the regression line at 2006 were selected for projections. This left 1482 simulations for NSW, 443 for Vic. and 799 for Qld. A range of scenarios was simulated to predict the future dynamics of the epidemic including: (1) all parameters remaining constant at their 2006 values; (2) all parameters continuing on their current trend; and (3) all parameters returning to the 1999 values. We also investigated changes in testing rates, condom use, and rates of early treatment of MSM diagnosed in primary infection.

**Results**

Without a change in the prevalence of other STI, but using the available time-dependent data for all other parameters, our model indicated that the number of HIV diagnoses should have actually decreased in all three states since 1999 (Fig. 2). This is due to increases in the effectiveness of treatment, increases in testing rates, and a slight decrease in the average number of casual partners, but offset slightly by decreases in condom use.

During the period 1999–2006, NSW experienced an 7.25% increase in notifications (see Table 2). Without an increase in other STI, our model suggested that observed changes in condom use directly resulted in an increase in HIV notifications of 5.23% and changes in patterns of treatment during primary infection resulted in an 1.01% increase. That is, these two factors account for a large proportion of the HIV increase in NSW. However, the model indicated that these factors were offset by changes in testing rates, average number of casual partnerships, disclosure of serostatus, and most importantly the proportion of treated MSM that achieve viral suppression. Each of these factors reduces the number of new infections leading to an overall decline in HIV notifications as seen in Fig. 2a.

Similarly, in Vic. the model suggested that a decline in condom use and treatment in primary infection, and changes in testing rates contributed positively to the number of HIV notifications. In contrast to NSW, however, these three factors accounted for a very small proportion of the large observed increase in HIV notifications (Table 2); the impact of these factors was also found to be largely outweighed by the proportion of treated people achieving viral suppression and other key parameters. Overall, without an increase in other STI, our model yielded a decrease in the number of HIV notifications, in contrast with the observed trend (Fig. 2b).

Qld also experienced a large increase in notifications between 1999 and 2006. In the absence of an increase in other STI, the most important factors contributing to the rise in Qld were again found to be declines in condom use and trends in treatment during primary infection. The model also suggested that changes in testing rates and disclosure of serostatus contributed positively to the rise in HIV notifications. Condom use was the most influential of these factors, contributing to a 9.45% rise, followed by change in testing rates (1.73%), treatment during primary infection (0.69%) and disclosure of serostatus (0.01%) (Table 2). But when other factors such as treatment effectiveness were included, as for the other states, our model showed that overall Qld should have experienced a decline in HIV cases (Fig. 2c), if the prevalence of other STI had not risen.

Because our model simulations could not directly account for the number of HIV notifications in all three states without changes in other STI, we investigated the extent of the change required to match the notification data. Although the data on the prevalence of STI among MSM in each state are incomplete, there are clear indications of increases in the incidence of infectious syphilis, chlamydia and gonorrhoea in the past few years. Therefore, we used our model to estimate the magnitude of the increase in prevalence of other STI that would be required in order to match the observed data for each state. We implemented a linear increase in STI prevalence into the model. Figure 3 shows the adjusted simulations with an increasing STI factor. For NSW, we found that a 2-fold increase

![Fig. 2](image-url). Uncertainty analysis epidemic trajectories of the modelled number of HIV diagnoses if changes in the prevalence of other sexually transmissible infections are not considered. The black dots indicate the number of HIV diagnoses based on the surveillance data, and the red curves represent all simulated time courses for: (a) New South Wales, (b) Victoria and (c) Queensland. MSM, men who have sex with men.
in the prevalence of other STI from 1999 to 2006 was required to appropriately match the data. Victoria required an increase of ~9-fold for the model simulations to match the data. Queensland required an ~9-fold increase in the prevalence of other STI for the model simulations to match the data. These required increases are not inconsistent with the trends in notifications of other STI.12,13,32

We then used our model to predict the number of new HIV infections, HIV diagnoses and AIDS deaths until 2015 by simulating the epidemic over this period under a variety of scenarios. We found that if all parameters remained constant at the 2006 values, there would be an increase in the number of HIV diagnoses in Vic. and Qld, whereas there would be a decrease in NSW (Fig. 4). The same qualitative result was found if the parameters did not remain constant but continued in the direction of their current trends (Fig. 5); however, if the current trends continued there would be even greater increases in HIV notifications in Vic. and Qld (Fig. 5).

Table 2. Percentage change in HIV notifications (from 1999 to 2006) that are attributable to various factors

<table>
<thead>
<tr>
<th>Factor</th>
<th>NSW</th>
<th>Vic.</th>
<th>Qld</th>
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<tbody>
<tr>
<td>Number of HIV notifications among MSM in 1999</td>
<td>313</td>
<td>102</td>
<td>85</td>
</tr>
<tr>
<td>Increase in notifications data from 1999 to 2006 (linear regression)</td>
<td>7.25%</td>
<td>96.43%</td>
<td>67.69%</td>
</tr>
<tr>
<td>Increase in HIV notifications due to factor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in condom usage</td>
<td>5.23% (4.48, 6.03)</td>
<td>7.89% (6.78, 9.13)</td>
<td>9.45% (8.04, 10.97)</td>
</tr>
<tr>
<td>Change in casual partners</td>
<td>-0.45% (-2.20, -0.74)</td>
<td>-1.23% (-0.86, -1.62)</td>
<td>-0.57% (-0.39, -0.75)</td>
</tr>
<tr>
<td>Change in disclosure of serostatus</td>
<td>-0.11% (-0.33, -0.07)</td>
<td>-0.14% (-0.42, 0.12)</td>
<td>0.01% (0.01, 0.03)</td>
</tr>
<tr>
<td>Change in testing rates</td>
<td>-0.31% (-0.18, -0.46)</td>
<td>0.17% (0.03, 0.41)</td>
<td>1.73% (1.04, 2.52)</td>
</tr>
<tr>
<td>Change in treatment during primary infection</td>
<td>1.01% (0.43, 1.99)</td>
<td>0.52% (0.16, 1.15)</td>
<td>0.69% (0.45, 2.25)</td>
</tr>
<tr>
<td>Change in proportion treated that achieve viral suppression</td>
<td>-25.81% (-32.56, -20.26)</td>
<td>-56.13% (-62.2, -49.3)</td>
<td>-47.01% (-54.45, -39.97)</td>
</tr>
<tr>
<td>Change in condom usage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in other STI required to explain data</td>
<td>~2-fold increase</td>
<td>~11-fold increase</td>
<td>~9-fold increase</td>
</tr>
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</table>

Fig. 3. Uncertainty analysis epidemic trajectories of the modelled number of HIV diagnoses if changes in sexually transmissible infections (STI) are adjusted for each state. The black dots indicate the number of HIV diagnoses based on the surveillance data, and the red curves represent all simulated time courses for: (a) New South Wales – with a 2-fold increase in other STI, (b) Victoria – 11-fold increase in other STI, and (c) Queensland – 9-fold increase in other STI. MSM, men who have sex with men.

Fig. 4. Projected number of HIV notifications to 2015 in the case of parameters remaining constant at 2006 levels. The black dots indicate the surveillance data points, and the black line represents the best-fitting linear regression line through the data points for: (a) New South Wales, (b) Victoria and (c) Queensland. MSM, men who have sex with men.
We compared the expected impact of different interventions on the projected number of new HIV infections (as distinct from HIV notifications) in 2015 in all three states (Fig. 6). We found that the most effective way to reduce the number of new HIV infections was to reduce the prevalence of other STI. If the prevalence of other STI was changed immediately to 60%, then NSW would see a large increase in new infections, Vic. would see a decrease, and Qld would see a slight increase over the number expected if all parameters were maintained at their 2006 values. If reduced to 5%, then the epidemic is predicted to decline substantially in all states (Fig. 6). Not surprisingly, changes in condom use were also found to be highly influential in determining the future epidemic trajectories. Our projections indicated that if condoms were used in an average of 90% of acts (currently condom use is ~68% in NSW, ~69% in Vic. and ~63% in Qld), significant declines in HIV cases would be observed in NSW and Qld, but there would still be an increase in HIV cases in Vic. (Fig. 6).

We also investigated the effect of patterns in the treatment of people diagnosed during the primary stage of HIV infection. Although the majority of seroconverters will not be detected with infection during primary infection, if early detection does

![Fig. 5. Projected number of HIV notifications to 2015 in the case of all parameters continuing their current trends. The black dots indicate the surveillance data points, and the black line represents the best-fitting linear regression line through the data points for: (a) New South Wales, (b) Victoria and (c) Queensland. MSM, men who have sex with men.](image)

![Fig. 6. Histograms showing the median number of projected HIV infections (as distinct from notifications) in 2015 for each scenario. The dashed red line corresponds to the number of infections simulated in 2006, and the solid orange line corresponds to the projected number of infections in 2015 if all parameters remain constant at their 2006 values.](image)
Notifications in 2006
Modelled infections 2006
Factors stay at 2006 levels
Current trends continue
STIs 60% prevalence
STIs 5% prevalence
50% condom use
90% condom use
40% testing rates
90% testing rates
Treat 30% in PHI
Treat 90% in PHI
Factors stay at 2006 levels

Number of HIV infections in Victoria
Fig. 6. (Continued)
occur and early treatment is provided then it has the effect of significantly reducing the high viral loads associated with primary infection, and this in turn reduces secondary transmission to others. Our findings indicate that increasing the proportion of people diagnosed in primary infection who receive treatment will lead to reductions in the number of new HIV cases. We investigated several coverage levels of treatment of people diagnosed in primary infection. Currently, $\sim$30–50% of people diagnosed in primary infection receive treatment. If this was increased to 90%, then decreases would be seen in both NSW and Qld. Victoria would still see an increase over the number of infections in 2006, but the numbers would be considerably lower than if all parameters were to remain constant. Treating 90% of people diagnosed in primary infection may be feasible; high treatment in primary infection was achieved in Australia during the late 1990s. The qualitative implications of this result are that any effort to increase treatment during primary HIV infection is likely to be beneficial at the population level. Of course, the degree of success in treating early infection is dependent on the number of people that are diagnosed early in their infection. Therefore, the rate of testing for HIV is highly important.

Increasing testing for HIV is beneficial in reducing further HIV transmissions because sexual behaviour generally changes upon diagnosis (to decrease transmission to partners). We found that increasing testing rates decreases the incidence of HIV. Currently $\sim$63% of MSM in NSW test for HIV each year, $\sim$56% in Vic. and $\sim$61% in Qld; testing rates have increased in all states since the levels in 1999 (of 60, 50 and 56%, respectively). This suggests that it is feasible to continue to increase testing rates. However, the maximum increase attainable is unknown. We investigated testing rates of 40, 50, 60, 70, 80 and 90%. Although testing 90% of MSM each year is likely to be unfeasible, we found that increasing testing has the potential to be highly beneficial in reducing the overall incidence of HIV in Australia (see Fig. 6).

Discussion

It was found that changes in condom use contributed to increases in HIV notifications in all three states. According to the data gathered from the Gay Periodic Surveys,\textsuperscript{3-7} in all three states there has been a slight increase in the percentage of unprotected acts during the period 1999–2006. However, this decline in condom use cannot completely explain the rise in notifications. Treatment during primary infection has decreased in each state during the studied time period. Because viral loads are very high in primary infection,\textsuperscript{35-40} the trends away from treatment during primary infection account for some of the rise in HIV notifications but are also unable to account for the magnitude of the observed rise. Indeed, the combination of changes in all variables could not account for the large rise in HIV notifications.

Testing rates in each state have recently increased modestly in all states. Our simulations suggested that the increased testing rates in NSW actually had a negative effect (-0.31%) on the number of HIV notifications (Table 2). In contrast, in Vic. and Qld the increases in testing had a small positive effect on HIV notifications, of 0.17 and 1.73%, respectively. Our model suggested that increasing testing can be beneficial in reducing the incidence of HIV. MSM diagnosed with HIV are likely to take behavioural measures to reduce their risk of transmission to other people, and if they receive treatment then their infectiousness will decrease substantially (especially if their infection is detected early). We have modelled a variety of testing levels and presented a ‘maximum’ achievable impact of increasing testing rates (to 90% of MSM each year). Although on its own such a high level of testing cannot turn the trends in HIV notifications in all states, our results suggest that testing is highly important and should be promoted further.

One factor that we have not attempted to model separately by state is serosorting, whereby MSM engage in unprotected anal intercourse only with men of the same serostatus as themselves. If successful, serosorting would reduce the risk of HIV transmission despite apparent increases in rates of unprotected anal intercourse. There are some data that suggest that serosorting may have been more successful in NSW than in Vic. or Qld.\textsuperscript{41} It is possible that this has contributed to differences in trends in HIV notifications by state, and currently this has not been captured in the models. If serosorting was more successful in NSW than the other states, then it is likely that less-dramatic differences in trends in rates of STI would be required to reproduce the observed trends in HIV notifications by state. However, we have considered differences between states in the trends in disclosure of serostatus in established sexual partnerships. It is much more likely that negotiated protection, based on serostatus, within partnerships will occur than the establishment of partnerships on the basis of serostatus (G. Prestage, National Centre in HIV Epidemiology and Clinical Research, pers. comm). It is also known that serosorting is more common among HIV-infected MSM than HIV-negative MSM. Thus, including serosorting is not likely to alter the incidence of new HIV infections. Of course, the impact of any serosorting among HIV-negative men is only as reliable as the frequency of HIV testing and degree of monogamy.

We have shown that the prevalence of other STI is a more important factor underlying the recent increase in HIV notifications than perhaps previously thought. Other STI may have had the greatest direct impact on the recent increase in HIV trajectories in Australia. It should be noted that our model did not link STI prevalence with condom use. In our model these factors have been decoupled, such that any changes in condom use and the prevalence of STI are independent. This is a limitation in our model. Future work will extend this analysis to model interacting infections and allow investigation of the indirect effects of condoms on reducing HIV by reducing the incidence (and thus prevalence) of other STI. However, our model indicates that the decrease in condom use alone (i.e. its direct effect in reducing HIV transmission) is not enough to account for the increases in HIV notifications. Our projections show that targeting STI in the community will be the most effective way to alter the epidemic trajectories. Condom use is also very important: our projections show that if condom use increased even moderately, then declines in new HIV infections would be observed. Of course, increasing condom use will also reduce the transmission of many other STI. We also suggest that increasing testing rates and subsequent early treatment of individuals diagnosed in primary infection will
have secondary benefits beyond the newly infected individuals in averting significant numbers of onward transmissions.

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Modelling HIV differences between Australian states

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Appendix:

Transmission model equations

The dynamic transmission model is represented by 10 ordinary differential equations. The mathematical description of our model is described here. In our model, we track the number of individuals to enter the susceptible men who have sex with men (MSM) population (S) at a rate of \( \pi \) per year. These individuals enter into the ‘pool’ of MSM, choosing sexual partners from the population. On average they leave the population of those choosing new sexual partners after an average of \( 1/\mu \) years. Thus, out of each compartment we include an outflow at rate \( \mu \). The other means by which susceptible individuals can leave this compartment is by becoming HIV-infected. The rate of flow in the number of people who become infected – that is, the force of infection (\( \lambda \)) – is defined below. Then, the rate of change in the total number of susceptible men at time \( t \) is given by

\[
\frac{dS}{dt} = \pi - (\mu + \lambda(t))S(t).
\]

Once an individual has become infected with HIV, he will initially have the status of undiagnosed with primary HIV infection (\( I_P \)). Thus, the number of MSM who leave the susceptible population per year, \( \lambda S \), becomes the source for the \( I_P \) compartment. There are three ways in which men can leave the undiagnosed primary HIV infection compartment: (i) become diagnosed as HIV-positive (at a rate \( \gamma_P \)), (ii) remain undiagnosed and progress in disease to chronic infection stage (at a rate \( \omega_P \)), or (iii) leave the sexually active population (at rate \( \mu \)). Accordingly, the rate of change in the total number of undiagnosed HIV-positive men in primary infection at time \( t \) is given by

\[
\frac{dI_P}{dt} = \lambda(t)S(t) - I_P(t)(\mu + \gamma_P + \omega_P).
\]

Similarly, the rate of change in the total number of undiagnosed HIV-positive men in chronic and AIDS stage infection at time \( t \) is given by

\[
\frac{dI_C}{dt} = \omega_P I_P(t) - I_C(t)(\mu + \gamma_C + \omega_C + \delta_C)
\]

and

\[
\frac{dI_A}{dt} = \omega_C I_C(t) - I_A(t)(\mu + \gamma_A + \delta_A),
\]

respectively, where the subscripts refer to the different disease stages and people in AIDS stage die of AIDS-related illnesses at a rate \( \delta_A \) (we also include an HIV-related death rate, \( \delta_C \) for people in the chronic stage of HIV infection).

Rates of movement out of compartments of untreated HIV-infected and diagnosed men can be due to (i) disease progression (at rate \( \omega \)), (ii) commencing antiretroviral therapy (at rate \( \eta \)), (iii) death (at rate \( \delta \)), or (iv) leaving the sexually active population (at rate \( \mu \)). Rates of movement into compartments of untreated HIV-infected and diagnosed men can be due to (i) newly diagnosed as HIV-infected (at rate \( \gamma \)) or (ii) previously treated men stopping antiretroviral therapy (at rate \( \nu \)). Then, the rate of change in the total numbers of diagnosed but untreated HIV-positive men in primary, chronic, and AIDS stages of infection at time \( t \) are given by

\[
\frac{dI_N^P}{dt} = \gamma_P I_P(t) - I_N^P(t)(\mu + \omega_P + \eta_P),
\]

\[
\frac{dI_N^C}{dt} = \gamma_C I_C(t) + \omega_P I_N^P(t) + \nu_C T_C(t) + \nu_P T_P(t) - I_N^C(t)(\mu + \omega_C + \eta_C + \delta_C),
\]

and

\[
\frac{dI_N^A}{dt} = \gamma_A I_A(t) + \nu_A T_A(t) + \omega_C I_N^C(t) - I_N^A(t)(\mu + \eta_A + \delta_A),
\]

where the subscripts refer to the respective disease stages.

Individuals diagnosed with HIV have the option of initiating antiretroviral therapy (ART). Based on the proportion of HIV-infected MSM who are on ART or initiate ART each year we determine the rate of movement from untreated diagnosed compartments to treatment compartments (denoted by \( \eta \)). The rates of initiating therapy are different for each stage of disease. Individuals on therapy can cease therapy until a later time (due to toxicities etc.), and we define the rate of ceasing treatment as \( \nu \) (individuals treated in primary infection could initiate an early treatment schedule and upon ceasing ART would move into chronic infection (at rate \( \nu_P \))). Treatment will delay the progression of disease, but HIV-infected patients on ART can still progress in their infection (at rates \( \tau \) and if
in AIDS-stage can still die of AIDS-related illnesses at a slower rate to untreated people (due to ineffective treatment for various possible reasons including drug resistance). Then, the rate of change in the total numbers of treated HIV-positive men in primary, chronic, and AIDS stages of infection at time $t$ are given by

$$\frac{dT_P}{dt} = \eta_P I_P(t) - T_P(t)(\mu + \nu_P + \tau_P).$$

$$\frac{dT_C}{dt} = \eta_C I_C + \tau_P T_P(t) + (1 - p_d)\eta_d I_d(t) - T_C(t)(\mu + \nu_C + \tau_C + \delta_C),$$

and

$$\frac{dT_A}{dt} = p_d\eta_d I_d(t) + \tau_C T_C(t) - T_A(t)(\mu + \nu_A + \delta_T).$$

Table A1 gives a full description of all of the parameters mentioned above, along with values that were used in the model.

**Force of infection**

The force of infection, $\lambda$, is the dynamic rate at which susceptible individuals become infected with HIV. This function contains many of the factors that contribute to HIV transmission. Typically $\lambda$ is calculated as the average number of sexual partners each susceptible person has per year, multiplied by the probability that each new partner is HIV-positive, multiplied by the probability of HIV transmission occurring per partnership per year. Various factors contribute to each of these components.

**Number of sexual partners**

We distinguish between the numbers of casual sexual partners and the numbers of regular partners MSM are likely to have, on average, each year. We let $c_{cas}$ represent the number of casual partners and $c_{reg}$ represent the number of casual partners. We use behavioural data\(^1\)\(^-\)\(^3\) on the proportion of men who have 0, 1, 2-10, 11-50, >50 partners to calculate a weighted average at each available time point, to obtain the following trends. We also make the assumption that one partner is regular, on average, and the remaining partners are casual partners.

**Probability that new sexual partner is HIV-positive**

If there was homogeneous non-differential mixing and no change in sexual behaviour between any categories of MSM in our model, then the probability that a new partner is HIV-positive is simply the ratio of the number of HIV-infected men to the total number of men in the population. There is evidence of change in behaviour upon diagnosis and men in AIDS stage disease are likely to have reduced numbers of partners due to their sickness. If healthy undiagnosed and susceptible men have partners per year, then we model the number of partners per year that men with AIDS have as $\theta_{AIDS} \cdot c$, where $\theta_{AIDS}$ is a multiplying factor for the reduction in sexual activity due to the effect of illness. We model the number of partners that diagnosed men have per year as $f \cdot c$. Here, $f$ refers to the multiplicative increase or decrease in sexual activity; we consider both the possibility of increase or decrease since HIV-positive men may reduce risky sex to avoid infecting others or they may increase risky sex as they are no longer at risk of seroconverting. Thus, the probability of a new partner being HIV-positive is

$$\frac{I_P + I_C + \theta_{AIDS} I_A + f(I_P^N + I_C^N + \theta_{AIDS} I_A^N) + T_P + T_C + \theta_{AIDS} T_A)}{S + I_P + I_C + \theta_{AIDS} I_A + I_P^N + f(I_C^N + \theta_{AIDS} I_A^N) + T_P + T_C + \theta_{AIDS} T_A)\cdot}$$

Sexual partnerships are likely to be formed irrespective of HIV serology status. A proportion of men will disclose their HIV serostatus to their partner (which is generally reciprocated). We denote the proportion of men who disclose their serostatus to their partner as $p_{disclose}$. If serostatus is disclosed and a partnership is serodiscordant then we assume that condoms are used in the majority of acts, but if the partnership is thought to be seroconcordant then we assume that condom use will be low.\(^4\) The risk of transmission in the relationships thought to be seroconcordant is due to partners that are undiagnosed but HIV-infected. If serostatus is not disclosed, then we assume that there is average condom use (at the average level reported in survey studies) and that partners of any status compartment can be chosen.

Serosorting for the formation of partnerships is rare; particularly among HIV-negative MSM (it is more common among HIV-positive MSM) (G. Prestage, pers. comm., National Centre in HIV Epidemiology and Clinical Research). Therefore, we simplify our model by not including serosorting for the establishment of partners. Negotiating condom use based on disclosure of serostatus is relatively common and is an important aspect retained in our model.
### Table A1. Definitions, ranges and references for input parameters used in our mathematical model

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>$c$</td>
<td>Average number of sexual partnerships per year (undiagnosed MSM)</td>
<td>1-3</td>
<td>$^c$</td>
</tr>
<tr>
<td>$\theta_{\text{AIDS}}$</td>
<td>Multiplying factor for the reduction in number of sexual partners for men in AIDS stage disease</td>
<td>0.1-0.4</td>
<td></td>
</tr>
<tr>
<td>$p_{\text{anal}}$</td>
<td>Percentage of sexual partnerships in which penile-anal intercourse occurs</td>
<td>10-40%</td>
<td>6</td>
</tr>
<tr>
<td>$f$</td>
<td>Multiplying factor for the average change in number of sexual partners post-diagnoses of HIV infection (this reflects a possible range from 50% decrease to 10% increase)</td>
<td>0.4-1.1</td>
<td>6-14</td>
</tr>
<tr>
<td>$p_{\text{disclose}}$</td>
<td>Proportion of partnerships in which serostatus is disclosed (in negotiating condom usage)</td>
<td>Regular: 0.8-0.9, Casual: 0.8-0.9</td>
<td>1, 4, 15, 16</td>
</tr>
<tr>
<td>$p_{\text{condom}}$</td>
<td>Proportion of acts in which condoms are used</td>
<td>0.85-0.9</td>
<td>17-21</td>
</tr>
<tr>
<td>$\psi$</td>
<td>Efficacy of condom protection per act</td>
<td>0.85-0.9</td>
<td>17-21</td>
</tr>
<tr>
<td>$W$</td>
<td>Baseline viral load during chronic infection</td>
<td>$10^4 - 10^5$ copies/mL</td>
<td>22-26</td>
</tr>
<tr>
<td>$V_{\text{pf}}$</td>
<td>Average viral load at primary infection stage</td>
<td>$10^6 - 10^7$ copies/mL</td>
<td>22-24, 26, 27</td>
</tr>
<tr>
<td>$V_d$</td>
<td>Average viral load at AIDS</td>
<td>$10^4 - 10^5$ copies/mL</td>
<td>24, 28, 29</td>
</tr>
<tr>
<td>$V_T$</td>
<td>Average viral load in effectively treated individual</td>
<td>10-100 copies/mL</td>
<td>30-32</td>
</tr>
<tr>
<td>$p_s$</td>
<td>Proportion of individuals on antiretroviral therapy in which viral load is suppressed</td>
<td>1, 7, 33, 34c</td>
<td></td>
</tr>
<tr>
<td>$\beta_C$, $\beta_N$</td>
<td>Probability of HIV transmission per act from an individual in chronic stage of infection</td>
<td>0.0015-0.0025</td>
<td>35-40</td>
</tr>
<tr>
<td>$\beta_{C}$, $\beta_{N}$, $\beta_C$, $\beta_N$</td>
<td>Probability of HIV transmission per act from an individual in primary or AIDS stage of infection</td>
<td>0.05-0.15</td>
<td>42, 43</td>
</tr>
<tr>
<td>$\beta_C$, $\beta_N$, $\beta_C$, $\beta_N$</td>
<td>Probability of HIV transmission per act from a treated individual</td>
<td>0.05-0.15</td>
<td>42, 43</td>
</tr>
<tr>
<td>$P_{\text{STI}}$</td>
<td>Proportion of HIV-negative MSM who have other STIs</td>
<td>2-5</td>
<td>44-50</td>
</tr>
<tr>
<td>$b_{\text{STI}}$</td>
<td>The multiplicative increase in transmission probability due to the presence of other STIs</td>
<td>2-5</td>
<td>44-50</td>
</tr>
<tr>
<td>$n_{\text{eg}}$</td>
<td>Average number of anal intercourse acts per regular partner per week</td>
<td>1.6-2.4</td>
<td>51</td>
</tr>
<tr>
<td>$n_{\text{cas}}$</td>
<td>Average number of anal intercourse acts per casual partner (over duration of casual relationship)</td>
<td>1-2</td>
<td>16, 51</td>
</tr>
<tr>
<td>$P_{\text{test}}$</td>
<td>Proportion of MSM who test for HIV infection each year</td>
<td>1c</td>
<td></td>
</tr>
<tr>
<td>$1/\gamma_A$</td>
<td>Average time from the beginning of AIDS before individual is likely to be diagnosed with infection</td>
<td>2-4 months</td>
<td></td>
</tr>
<tr>
<td>$1/\gamma_p$</td>
<td>Average time for untreated individuals to progress from primary infection to chronic infection</td>
<td>3-4 months</td>
<td>23, 52, 53</td>
</tr>
<tr>
<td>$1/\gamma_c$</td>
<td>Average time for individuals to progress from chronic infection to AIDS</td>
<td>8-12 years</td>
<td>22, 28, 54-57</td>
</tr>
<tr>
<td>$P_p$</td>
<td>Proportion of people diagnosed in primary infection who will commence treatment</td>
<td>▲</td>
<td></td>
</tr>
<tr>
<td>$1/\nu_p$</td>
<td>Average time to cease treatment for individuals with primary infection</td>
<td>6-12 months</td>
<td>▲</td>
</tr>
<tr>
<td>$P_{\text{pf}}$</td>
<td>Proportion of people who started ART in primary infection and continue ART after finishing dosing schedule</td>
<td>65-75%</td>
<td>▲</td>
</tr>
<tr>
<td>$P_C$</td>
<td>Proportion of people in chronic infection who will commence treatment</td>
<td>65-75%</td>
<td>1, 4, 58</td>
</tr>
<tr>
<td>$P_d$</td>
<td>Proportion of people with AIDS who commence treatment that experience treatment failure</td>
<td>0-0.1</td>
<td></td>
</tr>
<tr>
<td>$1/\nu_d$</td>
<td>Average time before individuals with AIDS commence therapy</td>
<td>1-3 months</td>
<td></td>
</tr>
<tr>
<td>$1/\nu_C$</td>
<td>Average time before diagnosed individuals in chronic infection commence therapy</td>
<td>2-10 years</td>
<td></td>
</tr>
<tr>
<td>$1/\nu_c$</td>
<td>Average time to cease treatment for individuals with chronic infection</td>
<td>6-12 years</td>
<td>1</td>
</tr>
<tr>
<td>$1/\nu_A$</td>
<td>Average time to cease treatment for individuals with AIDS</td>
<td>8-14 years</td>
<td>1</td>
</tr>
<tr>
<td>$1/\mu$</td>
<td>Average time for individuals to ‘retire’ out of sexually active population (no longer obtaining new partners)</td>
<td>30-35 years</td>
<td>56</td>
</tr>
<tr>
<td>$\delta_C$</td>
<td>Proportion of untreated MSM in chronic infection who die each year</td>
<td>1-2%</td>
<td>59-63</td>
</tr>
<tr>
<td>$\delta_{C}$</td>
<td>Proportion of treated MSM in chronic infection who die each year</td>
<td>1-2%</td>
<td>59-63</td>
</tr>
<tr>
<td>$1/\delta_d$</td>
<td>Average time until death from the onset of AIDS for untreated individuals</td>
<td>0.5-1.5 years</td>
<td>63-66</td>
</tr>
<tr>
<td>$1/\delta_T$</td>
<td>Average time until AIDS-related death for individuals in AIDS stage but on ART (with treatment failure)</td>
<td>0.5-5 years</td>
<td>56, 63, 65, 67-73</td>
</tr>
</tbody>
</table>
Our model requires estimates of the proportion of partnerships in which serostatus is disclosed in order to negotiate condom usage, \( p_{\text{disclose}} \). We use data on the percentage of men who reported UAI and always disclosed serostatus,\(^1\) and we included a ±25% uncertainty on the data.

**Condom use**

In regular relationships that are serodiscordant, we assume that average condom usage is high. Based on the Futures study,\(^4\) we assume condoms are used in 75–85% of anal intercourse acts between discordant MSM. However, in regular relationships that are thought to be seroconcordant we assume that average condom usage is relatively low; we assume condoms are used in 5–10% of acts.\(^2\) In casual relationships, serological disclosure is not as common as in regular relationships, but if the MSM in a casual relationship determines the relationship is serodiscordant then we assume condoms are used in 95–100% of acts. We assume that condoms are used more frequently in casual partnerships than in regular partnerships; thus, if it is thought that a casual relationship is seroconcordant then \( p_{\text{condom}} < p_{\text{cas}} < 10\% \).

### Probability of HIV transmission per discordant partnership per year

We denote the probability of HIV-transmission from an infected male to an uninfected male during a single unprotected act of anal intercourse by \( \beta \). However, if a condom is used as protection during intercourse then the probability of transmission is reduced. If \( \epsilon \) is the efficacy of condoms then the transmission probability per protected act is \( (1-\epsilon)\beta \). We consider the average number of coital acts per partner per unit time (\( \eta \)) and the proportion of these acts in which condoms are used (\( p_{\text{condom}} \)) to calculate the probability of transmission of infection per partnership over time. If \( \beta_i \) is the probability of HIV-transmission during a single coital act in a discordant partnership with protection type \( i \) (condom or no protection), then the probability of remaining uninfected after the single act is \( (1-\beta_i) \). Since each discordant coital act results in either transmission of infection or not (two possible outcomes), we have a Bernoulli trial, assuming each act is independent and has equal transmission for each protection option.

Accordingly, the probability of remaining uninfected after all \( n \) condom and \( n(1-p_{\text{condom}}) \) discordant sex acts that involved protection or no protection is binomial: \( (1-(1-\epsilon)\beta)^{n p_{\text{condom}}} (1-\beta)^n (1-p_{\text{condom}})^n \), respectively. Thus, together the probability of acquiring infection per discordant partnership per year is given by

\[
\hat{\beta} = 1 - (1-(1-\epsilon)\beta)^{n p_{\text{condom}}} (1-\beta)^n (1-p_{\text{condom}})^n.
\]

This expression is valid in the case of a standard transmission probability \( \beta \). But the presence of other sexually transmissible infections, both ulcerative and non-ulcerative, can increase the transmission of HIV. Therefore, we consider the proportion of men who have other sexually transmissible infections (\( p_{\text{STI}} \)) and the multiplicative increase in the transmission...
probability due to the presence of other infections ($b_{STI}$). Accordingly, the probability of acquiring infection per discordant partner per year is adjusted to become

$$1 - (1 - (1 - \epsilon)\beta')^{n_{\text{cond}}}(1 - \beta')^{n(1 - \beta_{\text{cond}})}$$

where

$$\beta' = (1 - p_{\text{STI}}) \beta + p_{\text{STI}} b_{\text{STI}} \beta.$$

### Combining factors for the resultant force of infection function

The force of infection is not as simple as multiplying each of the components together. This is because each compartment of HIV-infected person will have a different transmission probability. Average HIV viral load differs between disease stages and in individuals effectively treated with combination antiretroviral therapy. To calculate the transmission probabilities for each of these compartments we employ the relation described by Quinn et al.,

$$\beta = 2.45^{\log_{10} \nu} \beta_C,$$

where $\nu$ is the average viral load associated with a stage of infection, $W$ is a baseline viral load taken at chronic infection, and $\beta$ is the transmission probability for someone in chronic infection. That is, for each $\log_{10}$ increase in viral load there is a $2.45$ times increase in the transmission probability.

Taken together, our expression for the force of infection is given by:

$$\lambda = c_{\text{reg}} \left[ p_{\text{disclose}}^{\text{reg}} \left( \beta_{\text{reg}}^{\text{low condom}} l_p + \beta_{\text{reg}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right) 
+ p_{\text{disclose}}^{\text{reg}} \left( \beta_{\text{reg}}^{\text{high condom}} l_p + \beta_{\text{reg}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{reg}}) \left[ \beta_{\text{reg}}^{\text{low condom}} l_p + \beta_{\text{reg}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$

$$+ c_{\text{cas}} \left[ p_{\text{disclose}}^{\text{cas}} \left( \beta_{\text{cas}}^{\text{low condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right) 
+ p_{\text{disclose}}^{\text{cas}} \left( \beta_{\text{cas}}^{\text{high condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{cas}}) \left[ \beta_{\text{cas}}^{\text{low condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{cas}}) \left[ \beta_{\text{cas}}^{\text{high condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{cas}}) \left[ \beta_{\text{cas}}^{\text{high condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{cas}}) \left[ \beta_{\text{cas}}^{\text{low condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$

$$+ (1 - p_{\text{disclose}}^{\text{cas}}) \left[ \beta_{\text{cas}}^{\text{high condom}} l_p + \beta_{\text{cas}}^{\text{ave condom}} l_c + \theta_{\text{AIDS}} I_A + f(I_P^N + I_C^N + \theta_{\text{AIDS}} I_A^N + T_P + T_C + \theta_{\text{AIDS}} T_A) \right]$$
where the $\beta$ parameters are each specified by the transmission probability per partnership per year as defined above and based on the various behavioural and biological parameters (including number of acts for each type of relationship, condom usage, and viral loads affecting the transmission probabilities).

References

4. Grierson J, Thorpe R, Pitts M. HIV Futures 5: Life as we know it, monograph series number 60. 2006, The Australian Research Centre in Sex, Health and Society, Latrobe University, Melbourne, Australia.
