The potential impact of new generation molecular point-of-care tests on gonorrhoea and chlamydia in a setting of high endemic prevalence

Ben Hui, David P. Wilson, James S. Ward, Rebecca J. Guy, John M. Kaldor, Matthew G. Law, Jane S. Hocking and David G. Regan

The Kirby Institute, University of New South Wales, Sydney, NSW 2052, Australia.
Baker IDI Heart and Diabetes Institute, Alice Springs, NT 0871, Australia.
Centre for Women’s Health, Gender and Society, The University of Melbourne, Vic. 3053, Australia.

D Corresponding author. Email: bhui@kirby.unsw.edu.au

Appendix

Model dynamics

The modelled population is divided into 11 compartments (state), formulated as a system of ordinary differential equations as shown in (1). Each equation describes the change in the number of individuals in a particular compartment over time for a given set of parameter values. Individuals susceptible to infection ($S$) can become exposed ($E$) to infection through sexual contact with an infected partner. After an incubation period of $1/\mu_E$, an exposed individual will become infectious, either asymptotically ($A$ or $A_p$) or symptomatically ($Y$ or $Y_s$). A proportion of those with symptomatic infection ($Y$) are identified at the time of screening without undergoing a diagnostic test, and will enter the treatment state ($T$) directly. For simplicity we assumed constant proportions of 54% for male and 24% for female (1-2), regardless of screening coverage or diagnostic method. In addition, a proportion of those with asymptomatic infection ($A_p$) can enter the treatment state directly through presumptive treatment, although the impact of presumptive treatment is not investigated in the current study. An infectious individual will eventually recover, either naturally or through treatment, and will enter the recovered ($R$) state. Those in the recovered state are no longer infectious, and remain immune to new infections for a period of $1/\mu_R$.

If STI screening is in place, a proportion of those in the infectious state will be detected and treated accordingly. In this model, infectious individuals destined to be treated are represented by separate compartments. The $C_A$ compartment represents those with asymptomatic infectious identified through screening who will receive treatment after a delay of $\delta_{CT}$. Note that $C_A$ does not include those asymptomatic individuals who do not receive treatment through screening due to insufficient screening coverage, false-negative diagnoses, or who simply fail to return for treatment (they remain in compartment $A$). Similarly, the $C_{Y,CT}$ and $C_{Y,ST}$ compartments represent those with symptomatic infection identified through screening and who will proceed to treatment. Those in $C_{Y,CT}$ will receive treatment after a delay of $\delta_{CT}$, while those in $C_{Y,ST}$ will be treated immediately (or after a shorter delay of $\delta_{ST}$) due to presence of symptoms at the time of screening. See Figure 1 in the main document and the parameter tables for definition of parameters as well as the parameter values used in the model.

The model also includes various factors to adjust for possible changes in transmission probability, number of partners and number of sexual acts if a person is symptomatically infected or under treatment ($\psi$ and $\Psi$ respectively, with 0 being total elimination, to 1 being no changes). For the
current study, it was assumed that individuals who become symptomatically infected will reduce the number of acts in which they engage by 80%.

\[
\frac{dS}{dt} = -\lambda S + \mu R
\]
\[
\frac{dE}{dt} = \lambda S - \mu E
\]
\[
\frac{dA}{dt} = -\kappa E - \sigma_A \mu E - \Phi A + \Phi A
\]
\[
\frac{dY}{dt} = \sigma E - \Phi E + \Phi Y
\]
\[
\frac{dA_p}{dt} = -\kappa E \mu E - \frac{1}{\delta} A_p
\]
\[
\frac{dY_s}{dt} = \sigma E \mu E - \frac{1}{\delta} Y_s
\]
\[
\frac{dT}{dt} = \frac{1}{\delta} A_p + \frac{1}{\delta} Y_s + \frac{1}{\delta} C_A + \frac{1}{\delta} C_{Y,CT} + \frac{1}{\delta} C_{Y,ST} - \gamma T
\]
\[
\frac{dC_A}{dt} = \Phi A - \frac{1}{\delta} C_A
\]
\[
\frac{dC_{Y,CT}}{dt} = \Phi Y - \frac{1}{\delta} C_{Y,CT}
\]
\[
\frac{dC_{Y,ST}}{dt} = \Phi Y - \frac{1}{\delta} C_{Y,ST}
\]
\[
\frac{dR}{dt} = \gamma A + \gamma Y + \gamma T - \mu R
\]

Each model compartment is further stratified along gender (g, with 1 = male and 2 = female), age (a, with 1 = 15 to 19, 2 = 20 to 24, 3 = 25 to 29 and 4 = 30 to 34), level of sexual activity (k, with 1 = low and 2 = high), and frequency of continuous screening (n, with 1 = annual, 2 = every three months). For the remaining equations in this appendix, the subscripts \(g,a,k,n\) denote this stratification within a particular compartment.

Each age stratum spans 5 years, such that on average approximately one-fifth of each compartment will move to the next (older) age stratum each year. We assume that the total population size remains constant over time, i.e., the number of people leaving the oldest age group per unit time equals the number of people entering the youngest age group in the same time interval. We also ignore deaths as these are unlikely to occur as a consequence of infections with gonorrhoea or chlamydia and, as we are modelling a young population, the death rate from other causes can be assumed to be reasonably uniform across age-bands. We also assume all new entries to the youngest age group are susceptible. The effect of aging therefore will be modelled as follows:
\[
\frac{dK_{g,a,k,n}}{dt} = f_{\text{base}} \left( \frac{g_{a,k,n}}{5} - \frac{K_{g,a,k,n}}{5} \right) + \begin{cases} 
\frac{N_{g,4,k,n}}{5} & a = 1, K = S \\
0 & a = 1, K \neq S \\
\frac{K_{g,a-1,k,n}}{5} & a \neq 1 
\end{cases}
\]

\[
N_{g,a,k,n} = S_{g,a,k,n} + E_{g,a,k,n} + A_{g,a,k,n} + Y_{g,a,k,n} + A_{p,g,a,k,n} + Y_{g,ST,5} + T_{g,a,k,n} + C_{Y,CT,5} + C_{Y,CT,5} + R_{g,a,k,n}
\]

where \( f_{\text{base}}(K_{g,a,k,n}) \) denotes the ODE for compartment \( K \) as defined in (1).

**Transmission rate based on number of acts**

Assume for a population of size \( N \) (consisting of \( S \) susceptible and \( Y \) infectious individuals), the number of newly infected (\( \Delta Y \)) over a period of time \( \Delta T \) can be represented as a proportion of susceptible individuals who become infectious, and \( \Delta Y \) can then be represented as:

\[
\Delta Y = LS
\]

where \( L \) is a function of \( \Delta T \).

We define \( \eta \) as the number of new partners an individual acquires per unit time \( T \), such that \( \eta \Delta T \) new partners will be acquired, on average, over a period of \( \Delta T \). The probability of having \( n \) infectious partners over a period \( \Delta T \) can then be expressed by the following binomial term:

\[
\binom{\eta \Delta T}{n} \left( \frac{Y}{N} \right)^n \left( 1 - \frac{Y}{N} \right)^{\eta \Delta T - n}
\]

\[
(3)
\]

Assume \( L_\alpha \) is the probability of transmission to a susceptible individual from a single infectious partner (as defined in (9) below), then the probability of not becoming infected from \( n \) infectious partners \( L_\alpha \) is given by:

\[
L_\alpha = 1 - \sum_{n=0}^{\infty} \binom{\eta \Delta T}{n} \left( \frac{Y}{N} \right)^n \left( 1 - \frac{Y}{N} \right)^{\eta \Delta T - n}
\]

\[
(5)
\]

\( L \) is then equal to one minus the sum of \( L_\alpha \) over all \( n \), or

\[
L = 1 - \sum_{n=0}^{\infty} \binom{\eta \Delta T}{n} \left( \frac{Y}{N} \right)^n \left( 1 - \frac{Y}{N} \right)^{\eta \Delta T - n}
\]

\[
(6)
\]
Note that the sum of all $L_n$ forms a binominal series. Since the series converges for $L_a \leq 1$ and $Y < N$, (6) can be simplified as follows:

\[
L = 1 - \sum_{n=0}^{\infty} \left( \frac{\eta \Delta T}{n} \right) \left( 1 - L_a \frac{Y}{N} \right)^n \left( 1 - \frac{Y}{N} \right)^{\eta \Delta T - n} \\
= 1 - \left( 1 - L_a \frac{Y}{N} + 1 - \frac{Y}{N} \right)^{\eta \Delta T} \\
= 1 - \left( 1 - L_a \frac{Y}{N} \right)^{\eta \Delta T}.
\]  

(7)

We define $\theta$ as the total number of sexual acts a person will engage in per unit time such that $\theta \Delta T$ acts will occur, on average, over the period $\Delta T$. Assuming these acts are distributed evenly between partners, then the number of acts per partner ($\alpha$) is given by:

\[
\alpha = \frac{\theta \Delta T}{\eta \Delta T} = \frac{\theta}{\eta}.
\]  

(8)

If $\beta$ is the probability of transmission per act, then the probability of transmission from an infectious partner ($L_a$) after $\alpha$ acts is given by:

\[
L_a = 1 - (1 - \beta)^\alpha.
\]  

(9)

Combining this with (7) above, we obtain

\[
\Delta Y = LS \\
= \left( 1 - \left( 1 - \beta \frac{Y}{N} \right)^{\eta \Delta T} \right) S.
\]  

(10)

Assuming all terms remain constant for small $\Delta T$, then by Taylor expansion of (10) across $\Delta T$ we obtain:

\[
\Delta Y = -\eta \ln \left( 1 - L_a \frac{Y}{N} \right) S \Delta T + O(\xi T^2) \\
= -\eta \ln \left( 1 - \left( 1 - \beta \frac{Y}{N} \right)^{\eta \Delta T} \right) S \Delta T + O(\xi T^2)
\]  

(11)

where $O(\xi T^2)$ is some function of order $\Delta T^2$.

As $\Delta T \to 0$, non-linear terms $O(\xi T^2)$ can be ignored, therefore we can state:
\[
\frac{dY}{dT} = \lim_{\Delta T \to 0} \frac{\Delta Y}{\Delta T} \\
\approx -\eta \ln \left( 1 - L_a \frac{Y}{N} \right) S \\
= -\eta \ln \left( 1 - \left( 1 - \beta \frac{Y}{N} \right) S \right).
\] (12)

Note that \(- \ln(1-x) \approx x\) for small \(x\), therefore if \(\beta \) or \(\frac{Y}{N}\) is small, then equation (12) can be reduced to a definition of the force of infection similar to those defined in many traditional epidemiological models, e.g., Garnett et al. (3-5):

\[
\frac{dY}{dT} \approx \eta L_a \left( \frac{Y}{N} \right) S = \eta \left( 1 - \beta \frac{Y}{N} \right) S.
\] (13)

However, for chlamydia and gonorrhoea, the transmission probability per act (\(\beta\)) is relatively large (in the range 0.12 to 0.4 in our model), therefore the simplification above is only valid if the prevalence of infection \(\frac{Y}{N}\) is small. For example, if \(\beta = 0.4\), \(a = 20\) and \(\frac{Y}{N} = 0.1\), then applying the \(- \ln(1-x) \approx x\) simplification will overestimate \(\frac{dY}{dT}\) by ~5%.

If there are \(m\) levels of infectivity in the model (with the number of individuals in each level being \(Y_1, Y_2, \ldots, Y_m\), each with their own \(\beta\) and \(\alpha\) values), then from equation (12) the total number of new infectious added to the population can be defined as the sum of all strata, such that:

\[
\frac{dY}{dt} \approx -\sum_{i=1}^{m} \eta_i \ln \left( 1 - \left( 1 - \beta_i \frac{Y_i}{N} \right) S \right).
\] (14)

**Force of infection**

The force of infection, denoted as \(\lambda\) in (1), is the rate at which susceptible individuals become infected in the population. Note that in this study we assume the natural histories of gonorrhoea and chlamydia infection are independent and independent of one another, i.e., transmissibility and susceptibility of either pathogen are not affected by co-infection with the other. We make this assumption because there is very little published data on the impact of co-infection on transmission. The force of infection term is a function of the prevalence of infection and the level of sexual contact in the population.

Based on the relationship given in (14), the force of infection can be expressed as:

\[
\lambda_{g,a,k} = -\sum_{b=1}^{4} \sum_{j=1}^{2} \eta_{(3-g),b,j,a,k} \left( \sum_{m=1}^{8} \ln \left( 1 - \left( -B_{m}Bp_{m} \right) \right) \right).
\] (15)

where \(\eta_{(3-g),b,j,a,k}\) is the number of new partners an individual of gender \((3-g)\), age group \(b\) and activity group \(j\) may have with individuals of the opposite gender \(g\), age group \(a\) and activity group \(k\) per unit time. The value of parameter \(\eta_{(3-g),b,j,a,k}\) is determined by balancing the total number of new partners
across each population group, and the calculation steps required to achieve this are shown in equation (23).

In (15) above, $I_m$ is the prevalence of infection in the $m$-th population group and is given by:

\[
I_1 = \frac{A_{(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_2 = \frac{A_{p(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_3 = \frac{Y_{(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_4 = \frac{Y_{(3-g),b,j,a}}{N_{(3-g),b,j,a}}
\]

\[
I_5 = \frac{T_{(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_6 = \frac{C_{(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_7 = \frac{C_{Y,CT(3-g),b,j,a}}{N_{(3-g),b,j,a}}, I_8 = \frac{C_{Y,ST(3-g),b,j,a}}{N_{(3-g),b,j,a}}
\]

\[
N_{(3-g),b,j,a} = S_{(3-g),b,j,a} + E_{(3-g),b,j,a} + A_{(3-g),b,j,a} + Y_{(3-g),b,j,a}
\]

\[
+ A_{p(3-g),b,j,a} + Y_{(3-g),b,j,a} + T_{(3-g),b,j,a}
\]

\[
+ C_{(3-g),b,j,a} + C_{Y,CT(3-g),b,j,a} + C_{Y,ST(3-g),b,j,a} + R_{(3-g),b,j,a}.
\]

$B_m$ is the probability of no transmission from unprotected sex acts. It is a function of the transmission probability per act ($\beta_{(3-g)}$), the average number of acts per partner between the two population groups in question ($\alpha_{(3-g),b,j,a,k}$), and the proportion of acts in which protection is not used ($1-\nu_j$):

\[
B_m = \begin{cases} 
\left(\frac{1}{\beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{1,2,6}} 
& \text{m} \in \mathbb{Z}_{1,2,6} \\
\left(\frac{1}{\beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{4,7,8}} 
& \text{m} \in \mathbb{Z}_{4,7,8} \\
\left(\frac{1}{\beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{3}} 
& \text{m} \in \mathbb{Z}_{3}
\end{cases}
\]

The second and third lines of (17) include additional factors to reflect changes in the value of the transmission probability that are made when symptoms are present ($\Psi_\beta$) or treatment received ($\psi_\beta$).

Similarly, $B_{p,m}$ is the probability of no transmission from protected sex acts. The calculation is similar to that for unprotected acts given in (17), with additional parameter ($\xi$) to denote the efficiency of condoms in preventing transmission.

\[
B_{p,m} = \begin{cases} 
\left(\frac{1}{\xi \beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{1,2,6}} 
& \text{m} \in \mathbb{Z}_{1,2,6} \\
\left(\frac{1}{\xi \beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{4,7,8}} 
& \text{m} \in \mathbb{Z}_{4,7,8} \\
\left(\frac{1}{\xi \beta_{(3-g)}}\right)^{m \in \mathbb{Z}_{3}} 
& \text{m} \in \mathbb{Z}_{3}
\end{cases}
\]

The parameter $\alpha_{(3-g),b,j,a,k}$ denotes the average number sexual acts an individual of gender (3-g), age group $b$, and activity group $j$ has with individuals of the opposite gender $g$, age group $a$ and activity group $k$. It is assumed that the number of acts is divided equally among partners of same age and gender group, therefore $\alpha_{(3-g),b,j,a,k}$ is simply:

\[
\alpha_{(3-g),b,j,a,k} = \frac{\theta_{(3-g),b,j,a,k}}{\eta_{(3-g),b,j,a,k}},
\]

where $\theta_{(3-g),b,j,a,k}$ is the total number of acts an individual of gender (3-g), age group $b$ and activity group $j$ has with all individuals of gender $g$, age group $a$ and activity group $k$ per unit time. The calculation of $\theta_{(3-g),b,j,a,k}$ is shown in equation (27).
Let $H_{3,g,b,j}$ and $\Theta_{3,g,b,j}$ be the number of partnerships and sexual acts, respectively, an individual of gender $(3-g)$, age group $b$, and activity group $j$ has per unit time. We also denote $\rho_{(3-g),b,j,a,k}$ as the probability of such an individual forming a partnership with an individual of gender $g$, age group $a$, and activity group $k$, and $P_{(3-g),b,j,a,k}$ as the proportion of sexual acts that they engage in with individuals of gender $g$, age group $a$, and activity group $k$. According to the formulation of Garnett et al. (3-5), $\rho_{(3-g),b,j,a,k}$ can be determined from the number of partnerships available and the degree of assortative mixing across age and activity group, given by $m_{age}$ and $m_{risk}$ in (20) below. A modified version of these equations is used in this model and is shown in (20). Adjustment factors to account for changes to the number of partnerships formed due to symptoms or treatment, denoted as $\Psi_\eta$ and $\psi_\eta$, respectively, can also be included in the model as follows:

$$
\rho_{(3-g),b,j,a,k} = M_{age} M_{risk} \\
M_{age} = m_{age} \delta_{g,a} \\
\delta_{g,a} = \left( \sum_{l=1}^{n} \left( \sum_{n=1}^{N_{g,a,k,n}} \frac{\sum_{m=1}^{2} WP_{g,a,l,n}}{\sum_{l=1}^{n} \sum_{n=1}^{N_{g,a,k,n}} WP_{g,a,l,n}} \right) \right) ; \\
M_{risk} = m_{risk} \delta_{k,j} \\
\delta_{k,j} = \left( \sum_{l=1}^{n} \left( \sum_{n=1}^{N_{g,a,k,n}} \frac{WP_{g,a,k,n}}{WP_{g,a,l,n}} \right) \right) \\
\delta_{g,a} = \left( \frac{\sum_{l=1}^{n} \left( \sum_{n=1}^{N_{g,a,k,n}} \frac{WP_{g,a,l,n}}{WP_{g,a,k,n}} \right) }{\sum_{l=1}^{n} \left( \sum_{n=1}^{N_{g,a,k,n}} WP_{g,a,l,n} \right) } \right) \rightarrow 1:0
$$

$$
WP_{g,a,k,n} = H_{g,a,k} \times (N_{g,a,k,n} + \Phi_{\eta} - 1 \left( Y_{g,a,k,n} + \sum_{x \in \mathcal{G}_{V,a,k}} C_{x a,k,a,k,n} \right) + \Phi_{\eta} - 1 \left( \sum_{a=1}^{3} \sum_{n=1}^{N_{g,a,k,n}} WP_{g,a,l,n} \right) ).
$$

As the number of partners available may change due to changes in infection and treatment status, partner acquisition rate must be adjusted accordingly. In this model, we use a modified version of the balancing equations developed in Garnett and Anderson (3):

$$
BP_{a,k,b,j} = \frac{\rho_{2,b,j,a,k} \sum_{m=1}^{2} WP_{2,b,j,n}}{\rho_{1,a,k,b,j} \sum_{n=1}^{2} WP_{1,a,k,n}}.
$$

If the number of partnerships across genders, age and risk groups are balanced across all strata (i.e., the total number of female partners from age-group $b$ and risk-group $j$ for males of age-group $a$ and risk-group $k$ equals the total number of male partners from age-group $a$ and risk-group $k$ for females. 

of age-group $b$ and risk-group $j$), then the solution of equation (22) will be one. If there is an imbalance, then the number of partnerships will be adjusted as follows:

$$
\eta_{2,b,j,a,k} = H_{2,b,j} \rho_{2,b,j,a,k} \left( P_{a,k,b,j} \right)_{\text{partner}}^{b_{\text{partner}}},
$$

$$
\eta_{1,a,k,b,j} = H_{1,a,k} \rho_{1,a,k,b,j} \left( P_{a,k,b,j} \right)_{\text{partner}}^{m_{\text{partner}}},
$$

(23)

The parameter $b_{\text{partner}}$ denotes degree of compromise between genders, ranging from $b_{\text{partner}} = 0$, where number of female partners would not change if there is an imbalance, to $b_{\text{partner}} = 1$, where the number of male partners would not change.

Similarly, the number of acts across different genders, age and risk groups must balance as well. Using a balancing approach similar to (23), we define:

$$
P_{\text{age},j,a,k} = M_{\text{age}} M_{\text{risk}}
$$

$$
M_{\text{age}} = m_{\text{age}} \delta_{b,a}
$$

$$
+ \left( -m_{\text{age}} \right) \left( \frac{\sum_{l=1}^{2} \sum_{n=1}^{2} WA_{g,a,l,n}}{\sum_{c=1}^{4} \sum_{l=1}^{2} \sum_{n=1}^{2} WA_{g,c,l,n}} \right)
$$

$$
M_{\text{risk}} = m_{\text{risk}} \delta_{b,j}
$$

$$
+ \left( -m_{\text{risk}} \right) \left( \frac{\sum_{a=1}^{2} WA_{g,a,k,n}}{\sum_{l=1}^{2} \sum_{n=1}^{2} WA_{g,a,l,n}} \right)
$$

$$
\delta_{x,y} = \left( y \right)_{x} \rightarrow 1:0
$$

$$
WA_{g,a,k,n} = \Theta_{g,a,k} \times
$$

$$
\left( N_{g,a,k,n} + \psi_{g} - \frac{1}{3} \sum_{a,k,n} C_{\text{d},g,a,k,n} + m_{\text{risk}} - \frac{1}{2} \gamma_{g,a,k,n} \right);
$$

(25)

$$
BA_{a,k,b,j} = \frac{P_{2,b,j,a,k} \sum_{n=1}^{2} WA_{2,b,j,n}}{P_{1,a,k,b,j} \sum_{n=1}^{2} WA_{1,a,k,n}};
$$

(26)

$$
\theta_{2,b,j,a,k} = \Theta_{2,b,j} P_{2,b,j,a,k} \left( A_{a,k,b,j} \right)_{\text{partner}}^{b_{\text{partner}}},
$$

$$
\theta_{1,a,k,b,j} = \Theta_{1,a,k} P_{1,a,k,b,j} \left( A_{a,k,b,j} \right)_{\text{partner}}^{m_{\text{partner}}},
$$

(27)

**Screening coverage and screened-treatment rate**
We define the screening coverage \( \omega \) as the proportion of the population to be screened over a period of time \( \tau_{scn} \). If the rate at which those identified by screening as positive (i.e., infectious) proceed to treatment, denoted as \( \Phi \) in the model, is constant over \( \tau_{scn} \), then it will be related to \( \omega \) as:

\[
\Phi = -\ln \left( 1 - \frac{\tau_{scn}}{\tau_{scn}} \right).
\]  

(28)

This is based on the screening rate equation defined in Bowden and Garnett (6). In this model, only a proportion of those screened will be identified as infected and will proceed to treatment. This proportion will be adjusted by the sensitivity of the screening test \( (\varepsilon) \) and the probability of a screened individual returning for treatment after a positive diagnostic \( (\tau) \).

The parameter \( \tau_{scn} \) denotes the length of time in which screening will be carried out. For example, for continuous annual screening, \( \tau_{scn} \) will be 1 year, whereas for a mass serial screening intervention implemented over a three weeks period, \( \tau_{scn} \) will take a value of approximately 0.06 years.

**Switches between screening group**

A proportion of individuals whose infections have resolved through treatment will enter the rescreening group, where they may be screened at different coverage or frequency (for example, once every three months rather than annually). Those in rescreening group will be screened and received treatment at different rate.

We denote \( \chi_i \) as the proportion of the population moving to rescreening group for a period of \( 1/\chi_o \), and the effect of rescreening is then described as follows:

\[
\begin{align*}
\frac{dK_{g,a,k,1}}{dt} &= f_{age} \left( \begin{array}{c}
\alpha_{g,a,k,1} \\
\varepsilon K_{g,a,k,2}
\end{array} \right) + \chi_o K_{g,a,k,1}, \\
\frac{dK_{g,a,k,2}}{dt} &= f_{age} \left( \begin{array}{c}
\alpha_{g,a,k,2} \\
\varepsilon K_{g,a,k,2}
\end{array} \right) + \chi_o K_{g,a,k,2}, \\
K &\in \begin{array}{c}
S^i, E, A, Y, A_p, Y_s, C_A, C_{Y,CT}, C_{Y,ST}, T
\end{array}
\end{align*}
\]

(29)

\[
\begin{align*}
\frac{dR_{g,a,k,1}}{dt} &= f_{age} \left( \begin{array}{c}
\alpha_{g,a,k,1} \\
\varepsilon Y_{g,a,k,1}
\end{array} \right) + \chi_o R_{g,a,k,1}, \\
\frac{dR_{g,a,k,2}}{dt} &= f_{age} \left( \begin{array}{c}
\alpha_{g,a,k,2} \\
\varepsilon Y_{g,a,k,2}
\end{array} \right) + \chi_o R_{g,a,k,2}
\end{align*}
\]

where \( f_{age}(K) \) is the ODE for state variable \( K \) in accordance with (2).

**Parameter fitting**

While the values of some model parameters are based on published data or values inferred by other transmission models, many are unavailable or unsuitable to replicate the level of prevalence observed in remote Indigenous communities in Australia. There are also parameters (e.g. the degree of assortative mixing) that are essentially model constructs, whose values must be simply assumed or set by trial and error. In this model, the values of these parameters are determined through a fitting process (genetic algorithm toolbox, Matlab (7)) that attempts to match the STI prevalences generated in the baseline model with those found in the baseline prevalence study (8). The parameters to be fitted in this model include:

- The assortative mixing index for age groups is assumed to be range 0.7 to 1.
• The assortative mixing index for activity group is assumed to be in the range 0.5 to 1.

• The number of sexual partners per year for each age and sexual activity group is assumed to be in the range 1 to 10 per year, based on sexual behaviour studies in Australia (9-10).

We did not include infection-specific biological parameters, such as the duration of infection, the probability of transmission and the proportion of infections that are symptomatic, in our parameter fitting process. This is because the values for these parameters have been extensively studied, and have considerably less uncertainty associated with them in comparison with the behavioural parameters or the model structure. The parameter values chosen for this model are comparable with those used in other STI transmission models of similar type.
### Parameter tables

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\beta$</td>
<td>Probability of transmission per act</td>
<td>Male: 0.20 Female: 0.40</td>
<td>(11-15)</td>
<td>Male: 0.16 Female: 0.12</td>
<td>(12, 16-18)</td>
</tr>
<tr>
<td>$1/\mu_E$</td>
<td>Average duration of incubation period</td>
<td>4 days</td>
<td>(19-20)</td>
<td>20 days</td>
<td>(17, 21-22)</td>
</tr>
<tr>
<td>$1/\mu_R$</td>
<td>Average duration of recovery period</td>
<td>7 days</td>
<td>Assumption</td>
<td>45 days</td>
<td>Assumption</td>
</tr>
<tr>
<td>$1/\gamma_A$</td>
<td>Average duration of untreated asymptomatic infection</td>
<td>178</td>
<td>(5, 23)</td>
<td>256</td>
<td>(12, 17, 24)</td>
</tr>
<tr>
<td>$1/\gamma_Y$</td>
<td>Average duration of untreated symptomatic infection</td>
<td>178</td>
<td>(5, 23)</td>
<td>112</td>
<td>(12, 25)</td>
</tr>
<tr>
<td>$1/\gamma_T$</td>
<td>Average duration of treated infection</td>
<td>1 day</td>
<td>Assumption</td>
<td>7 days</td>
<td>(26)</td>
</tr>
<tr>
<td>$\kappa_s$</td>
<td>Proportion of infections being symptomatic</td>
<td>Male: 0.45 Female: 0.14</td>
<td>(22, 27)</td>
<td>Male: 0.30 Female: 0.15</td>
<td>(12, 22)</td>
</tr>
<tr>
<td>$\sigma_Y$</td>
<td>Probability of a symptomatically infected individual being treated immediately at the time of screening</td>
<td>Male: 0.54 Female: 0.24</td>
<td>(1)</td>
<td>Male: 0.54 Female: 0.24</td>
<td>(1)</td>
</tr>
<tr>
<td>$\sigma_A$</td>
<td>Proportion of asymptotically infected treated through presumptive treatment</td>
<td>0</td>
<td>Not used in current analysis</td>
<td>0</td>
<td>Not used in current analysis</td>
</tr>
</tbody>
</table>

*Table 1: Parameters specific to gonorrhoea and chlamydia used in the model.*
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Low activity group:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age</td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15-19</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20-24</td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24-29</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30-34</td>
<td>0.8</td>
</tr>
<tr>
<td>$H_{g,a,k}$</td>
<td>Number of new partners acquired per year(^1)</td>
<td>High activity group:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age</td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15-19</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20-24</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24-29</td>
<td>2.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30-34</td>
<td>2.5</td>
</tr>
<tr>
<td>$\Theta_{g,a,k}/52$</td>
<td>Number of sexual acts per week</td>
<td>2</td>
<td>(28), assumed to be same for all modelled group</td>
</tr>
<tr>
<td>$\psi_\beta$</td>
<td>Proportional adjustment to transmission probability due to symptomatic infection</td>
<td>1</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\psi_\eta$</td>
<td>Proportional adjustment to number of new partners per year due to symptomatic infection</td>
<td>1</td>
<td>Assumption</td>
</tr>
</tbody>
</table>

\(^1\) $g = 1$ for male, $g = 2$ for female; $a = 1$ for age 15-19, $a = 2$ for age 20-24, $a = 3$ for age 24-29, $a = 4$ for age 30-34; $k = 1$ for low activity, $k = 2$ for high activity.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\psi_{\theta}$</td>
<td>Proportional adjustment to number of acts per year due to symptomatic infection</td>
<td>0.2</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\psi_{\beta}$</td>
<td>Proportional adjustment to transmission probability due to treatment</td>
<td>0</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\psi_{\eta}$</td>
<td>Proportional adjustment to number of new partners per year due to treatment</td>
<td>1</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\psi_{\vartheta}$</td>
<td>Proportional adjustment to number of acts per year due to treatment</td>
<td>0</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\xi$</td>
<td>Preventative efficacy of condoms</td>
<td>0.95</td>
<td>(14)</td>
</tr>
<tr>
<td>$\nu$</td>
<td>Condom usage for high activity group</td>
<td>age &lt; 30: 0.3, age 30+: 0.1</td>
<td>(29-30)</td>
</tr>
<tr>
<td>$m_{\text{risk}}$</td>
<td>Assortativity index for mixing between activity groups (range: 0-1, with 1 being fully assortative)</td>
<td>0.70</td>
<td>Fitted against prevalence data</td>
</tr>
<tr>
<td>$m_{\text{age}}$</td>
<td>Assortativity index or mixing between age groups (range: 0-1, with 1 being fully assortative)</td>
<td>0.50</td>
<td>Fitted against prevalence data</td>
</tr>
<tr>
<td>$b_{\text{parner}}$</td>
<td>Balancing factor for number of partnerships</td>
<td>0.5</td>
<td>Assumption</td>
</tr>
<tr>
<td>$b_{\text{acts}}$</td>
<td>Balancing factor for number of acts</td>
<td>0.5</td>
<td>Assumption</td>
</tr>
</tbody>
</table>

Table 2: Sexual behaviour parameters used in the model.
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Parameter</th>
<th>Value</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma$</td>
<td>Proportion of population screened (per year or per screening session)</td>
<td>Baseline: 0.44</td>
<td>Baseline: (2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention: Varies</td>
<td></td>
</tr>
<tr>
<td>$r$</td>
<td>Probability of a screened individual who is diagnosed positive returning for treatment</td>
<td>Baseline: 0.85</td>
<td>Baseline: (2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention: 0.6</td>
<td>Intervention: (14, 31-33)</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>Sensitivity of screening test</td>
<td>Gonorrhoea: 0.95</td>
<td>Gonorrhoea: (34-36)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chlamydia: 0.98</td>
<td>Chlamydia: (37-38)</td>
</tr>
<tr>
<td>$\delta_{CT}$</td>
<td>Average time delay between screening and the commencement of treatment (assuming treatment is not offered at time of screening).</td>
<td>26 days</td>
<td>(2)</td>
</tr>
<tr>
<td>$\delta_{ST}$</td>
<td>Average time delay between consultation and the commencement of treatment for a symptomatic patient</td>
<td>5 days</td>
<td>Assumption</td>
</tr>
<tr>
<td>$\delta_p$</td>
<td>Average time delay between consultation and the commencement of presumptive treatment</td>
<td>7 days</td>
<td>Not used in current analysis</td>
</tr>
</tbody>
</table>

Table 3: Parameter specific to screening.
Reference