

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 π 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 μ . 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 (λ) – 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, λS , 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 γ_P), (ii) remain undiagnosed and progress in disease to chronic infection stage (at a rate ω_P), or (iii) leave the sexually active population (at rate μ). 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 δ_A (we also include an HIV-related death rate, δ_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 ω), (ii) commencing antiretroviral therapy (at rate η), (iii) death (at rate δ), or (iv) leaving the sexually active population (at rate μ). Rates of movement into compartments of untreated HIV-infected and diagnosed men can be due to (i) newly diagnosed as HIV-infected (at rate γ) or (ii) previously treated men stopping antiretroviral therapy (at rate ν). 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

$$\frac{dI_P^N}{dt} = \gamma_P I_P(t) - I_P^N(t)(\mu + \omega_P + \eta_P),$$

$$\frac{dI_C^N}{dt} = \gamma_C I_C(t) + \omega_P I_P^N(t) + \nu_C T_C(t) + \nu_P T_P(t) - I_C^N(t)(\mu + \omega_C + \eta_C + \delta_C),$$

and

$$\frac{dI_A^N}{dt} = \gamma_A I_A(t) + \nu_A T_A(t) + \omega_C I_C^N(t) - I_A^N(t)(\mu + \eta_A + \delta_A),$$

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 η). 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 ν (individuals treated in primary infection could initiate an early treatment schedule and upon ceasing ART would move into chronic infection (at rate ν_P)). Treatment will delay the progression of disease, but HIV-infected patients on ART can still progress in their infection (at rates τ) 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

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

$$\frac{dT_C}{dt} = \eta_C I_C^N + \tau_P T_P(t) + (1 - p_A)\eta_A I_A^N(t) - T_C(t)(\mu + \nu_C + \tau_C + \delta_C^T),$$

and

$$\frac{dT_A}{dt} = p_A \eta_A I_A^N(t) + \tau_C T_C(t) - T_A(t)(\mu + \nu_A + \delta_T).$$

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, λ , 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 λ 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 regular partners. We use behavioural data¹⁻³ 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 $\theta_{\text{AIDS}} \cdot c$, where θ_{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

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

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
 ART, antiretroviral treatment; MSM, men who have sex with men; STI, sexually transmissible infections

Parameter	Description	Value	Ref.
c	Average number of sexual partnerships per year (undiagnosed MSM)	1–3 ^C	
θ_{AIDS}	Multiplying factor for the reduction in number of sexual partners for men in AIDS stage disease	0.1–0.4	
p_{anal}	Percentage of sexual partnerships in which penile–anal intercourse occurs	10–40%	6
f	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)	0.4–1.1	6–14
p_{disclose}	Proportion of partnerships in which serostatus is disclosed (in negotiating condom usage)	Regular Casual 0.8–0.9 ^C	1, 4, 15, 16
p_{condom}	Proportion of acts in which condoms are used		1, 4 ^C
ϵ	Efficacy of condom protection per act	0.85–0.9	17–21
W	Baseline viral load during chronic infection	10^4 – 10^5 copies/mL	22–26
V_{PI}	Average viral load at primary infection stage	$10^{6.5}$ – 10^8 copies/mL	22–24, 26, 27
V_A	Average viral load at AIDS	$10^{5.5}$ – $10^{6.5}$ copies/mL	24, 28, 29
V_T	Average viral load in effectively treated individual	10–100 copies/mL	30–32
P_s	Proportion of individuals on antiretroviral therapy in which viral load is suppressed		1, 7, 33, 34 ^C
β_C, β_C^N	Probability of HIV transmission per act from an individual in chronic stage of infection	0.0015–0.0025	35–40
$\beta_P, \beta_P^N, \beta_A, \beta_A^N$	Probability of HIV transmission per act from an individual in primary or AIDS stage of infection		5
$\beta_T^T, \beta_C^T, \beta_A^T$	Probability of HIV transmission per act from a treated individual		5, 41
P_{STI}	Proportion of HIV-negative MSM who have other STIs	0.05–0.15	42, 43
b_{STI}	The multiplicative increase in transmission probability due to the presence of other STIs	2–5	44–50
n_{reg}	Average number of anal intercourse acts per regular partner per week	1.6–2.4	51
n_{cas}	Average number of anal intercourse acts per casual partner (over duration of casual relationship)	1–2	16, 51
P_{Test}	Proportion of MSM who test for HIV infection each year		1 ^C
$1/\gamma_A$	Average time from the beginning of AIDS before individual is likely to be diagnosed with infection	2–4 months	
$1/\omega_P$	Average time for untreated individuals to progress from primary infection to chronic infection	3–9 months	23, 52, 53
$1/\omega_C$	Average time for individuals to progress from chronic infection to AIDS	8–12 years	22, 28, 54–57
P_p	Proportion of people diagnosed in primary infection who will commence treatment	A	
$1/V_p$	Average time to cease treatment for individuals with primary infection	6–12 months	A
P_C^C	Proportion of people who started ART in primary infection and continue ART after finishing dosing schedule	65–75%	A
P_C	Proportion of people in chronic infection who will commence treatment	65–75%	1, 4, 58
P_A	Proportion of people with AIDS who commence treatment that experience treatment failure	0–0.1	
$1/\eta_A$	Average time before individuals with AIDS commence therapy	1–3 months	
$1/\eta_C$	Average time before diagnosed individuals in chronic infection commence therapy	2–10 years	
$1/V_C$	Average time to cease treatment for individuals with chronic infection	6–12 years	1
$1/V_A$	Average time to cease treatment for individuals with AIDS	8–14 years	1
$1/\mu$	Average time for individuals to ‘retire’ out of sexually active population (no longer obtaining new partners)	30–35 years	56
δ_C	Proportion of untreated MSM in chronic infection who die each year	1–2%	59–63
δ_C^T	Proportion of treated MSM in chronic infection who die each year	1–2%	59–63
$1/\delta_A$	Average time until death from the onset of AIDS for untreated individuals	0.5–1.5 years	63–66
$1/\delta_T$	Average time until AIDS-related death for individuals in AIDS stage but on ART (with treatment failure)	0.5–5 years	56, 63, 65, 67–73

Table A1. Continued

Parameter	Description	Value	Ref.
$1/\tau_C$	Average time of disease progression for treated individual with chronic infection to progress to AIDS	$1/\omega_C < 1/\tau_C < 20$	
π	Number of new susceptible individuals entering the MSM population per year (this is ~3–3.5% of men)		
	Nationally	2000–2500 ^B	
	NSW	35–40%	
	VIC	22–27%	
	QLD	17–22%	

^AWe evaluated available data from primary infection cohorts on the percentage of HIV-infected MSM who commenced ART within 1 year of HIV diagnosis, including patients recruited to the Acute Infection and Early Disease Research Program (CORE 01) protocol established by the National Institutes of Health, and the Primary HIV and Early Disease Research: Australian Cohort (PHAEDRA) established by the National Centre in HIV Epidemiology and Clinical Research. This data has large uncertainty (summarised in reference 58), is limited in time and only includes NSW and VIC. Sample sizes are also not sufficient (as low as four in some years for VIC and six for NSW). Consequently, this has been used as a rough guide but we make assumptions in the trends in early treatment based on personal communication with clinicians (e.g. Prof. Tony Kelleher (NCHECR and Centre for Immunology at St Vincent's Hospital, t.kelleher@cfi.unsw.edu.au)). We estimate the basic anecdotal trends observed over the past few years, shown in Table 1 of the main text. However, since there are no firm data for the trends, we include greater uncertainty bounds on this time-dependent parameter than on the others (we use a multiplicative uncertainty range on these trends of 0.6–1.2). We also assume that the initial dosing schedule for these patients who commence treatment in primary infection is 6–12 months, after which time 60–70% of these patients will continue ART and the remaining patients will discontinue therapy until a later time.

^BThis leads to ~150 000–175 000 MSM nationally. The proportion of new MSM in NSW, VIC, QLD each year as a subset of the total National number are indicated.

^CFor each of these time-dependent parameters we include an uncertainty range of $\pm 5\%$.

Our model requires estimates of the proportion of partnerships in which serostatus is disclosed in order to negotiate condom usage, p_{disclose} . We use data on the percentage of men who reported UAI and always disclosed serostatus,¹ and we included a $\pm 25\%$ 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.⁴ 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 $p_{\text{condom}}^{\text{reg}} < p_{\text{condom}}^{\text{cas}} < 10\%$.

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 β . However, if a condom is used as protection during intercourse then the probability of transmission is reduced. If ϵ 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 (η) and the proportion of these acts in which condoms are used (p_{condom}) to calculate the probability of transmission of infection per partnership over time. If β_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 $n \cdot p_{\text{condom}}$ and $n(1-p_{\text{condom}})$ discordant sex acts that involved protection or no protection is binomial: $(1-(1-\epsilon)\beta)^{n \cdot p_{\text{condom}}}$ and $(1-\beta)^{n(1-p_{\text{condom}})}$, respectively. Thus, together the probability of acquiring infection per discordant partnership per year is given by

$$\hat{\beta} = 1 - (1 - (1 - \epsilon)\beta)^{n \cdot p_{\text{condom}}} (1 - \beta)^{n(1-p_{\text{condom}})}$$

This expression is valid in the case of a standard transmission probability β . 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_{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

$$1 - (1 - (1 - \epsilon)\beta')^{n \cdot p_{\text{condom}}} (1 - \beta')^{n(1-p_{\text{condom}})},$$

where

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

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.*,⁵ namely,

$$\hat{\beta} = 2.45^{\log_{10} \left(\frac{\nu}{W}\right)} \beta_C,$$

where ν is the average viral load associated with a stage of infection, W is a baseline viral load taken at chronic infection, and β_C 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:

$$\begin{aligned} \lambda = & c_{\text{reg}} \left[p_{\text{disclose}}^{\text{reg}} \frac{\hat{\beta}_P^{\text{reg} | \text{low condom}} I_P + \hat{\beta}_C^{\text{reg} | \text{low condom}} I_C + \hat{\beta}_A^{\text{reg} | \text{low condom}} \theta_{\text{AIDS}} I_A}{S + I_P + I_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}} \frac{f(\hat{\beta}_P^{\text{reg} | \text{high condom}} I_P^N + \hat{\beta}_C^{\text{reg} | \text{high condom}} I_C^N + \hat{\beta}_A^{\text{reg} | \text{high condom}} \theta_{\text{AIDS}} I_A^N + \hat{\beta}_T^{\text{reg} | \text{high condom}} (T_P + T_C + \theta_{\text{AIDS}} T_A))}{S + I_P + I_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)} \\ & + (1 - p_{\text{disclose}}^{\text{reg}}) \frac{\hat{\beta}_P^{\text{reg} | \text{ave condom}} I_P + \hat{\beta}_C^{\text{reg} | \text{ave condom}} I_C + \hat{\beta}_A^{\text{reg} | \text{ave condom}} \theta_{\text{AIDS}} I_A}{S + I_P + I_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)} \\ & \left. + (1 - p_{\text{disclose}}^{\text{reg}}) \frac{f(\hat{\beta}_P^{\text{reg} | \text{ave condom}} I_P^N + \hat{\beta}_C^{\text{reg} | \text{ave condom}} I_C^N + \hat{\beta}_A^{\text{reg} | \text{ave condom}} \theta_{\text{AIDS}} I_A^N + \hat{\beta}_T^{\text{reg} | \text{ave condom}} (T_P + T_C + \theta_{\text{AIDS}} T_A))}{S + I_P + I_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}} \frac{\hat{\beta}_P^{\text{cas} | \text{low condom}} I_P + \hat{\beta}_C^{\text{cas} | \text{low condom}} I_C + \hat{\beta}_A^{\text{cas} | \text{low condom}} \theta_{\text{AIDS}} I_A}{S + I_P + I_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}} \frac{f(\hat{\beta}_P^{\text{cas} | \text{high condom}} I_P^N + \hat{\beta}_C^{\text{cas} | \text{high condom}} I_C^N + \hat{\beta}_A^{\text{cas} | \text{high condom}} \theta_{\text{AIDS}} I_A^N + \hat{\beta}_T^{\text{cas} | \text{high condom}} (T_P + T_C + \theta_{\text{AIDS}} T_A))}{S + I_P + I_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)} \\ & + (1 - p_{\text{disclose}}^{\text{cas}}) \frac{\hat{\beta}_P^{\text{cas} | \text{ave condom}} I_P + \hat{\beta}_C^{\text{cas} | \text{ave condom}} I_C + \hat{\beta}_A^{\text{cas} | \text{ave condom}} \theta_{\text{AIDS}} I_A}{S + I_P + I_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)} \\ & \left. + (1 - p_{\text{disclose}}^{\text{cas}}) \frac{f(\hat{\beta}_P^{\text{cas} | \text{ave condom}} I_P^N + \hat{\beta}_C^{\text{cas} | \text{ave condom}} I_C^N + \hat{\beta}_A^{\text{cas} | \text{ave condom}} \theta_{\text{AIDS}} I_A^N + \hat{\beta}_T^{\text{cas} | \text{ave condom}} (T_P + T_C + \theta_{\text{AIDS}} T_A))}{S + I_P + I_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] \end{aligned}$$

where the β 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

- 1 NSW, VIC and QLD Gay Periodic Surveys. 1998–2006.
- 2 Richters J. *HIV/AIDS, Hepatitis C & Related Diseases in Australia: Annual Report of Behaviour* 2006, National Centre in HIV Social Research, University of New South Wales: Sydney.
- 3 Crawford JM, et al. Number of risk acts by relationship status and partner serostatus: Findings from the HIM cohort of homosexually active men in Sydney, Australia. *AIDS Behav* 2006; 10: 325–31. doi: 10.1007/s10461-005-9057-3
- 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.
- 5 Quinn TC, et al. Viral load and heterosexual transmission of human immunodeficiency virus type 1. Rakai Project Study Group. *N Engl J Med* 2000; 342: 921–9. doi: 10.1056/NEJM200003303421303
- 6 *National Centre in HIV Social Research Annual Report of Trends in Behaviour*. 2006, University of New South Wales: Sydney.
- 7 Van de Ven P, et al. Undetectable viral load is associated with sexual risk taking in HIV serodiscordant gay couples in Sydney. *AIDS* 2005; 19: 179–84.
- 8 Marks G, et al. Meta-analysis of high-risk sexual behavior in persons aware and unaware they are infected with HIV in the United States: implications for HIV prevention programs. *J Acquir Immune Defic Syndr* 2005; 39: 446–53. doi: 10.1097/01.qai.0000151079.33935.79
- 9 Cleary PD, et al. Behavior changes after notification of HIV infection. *Am J Public Health* 1991; 81: 1586–90.
- 10 Colfax GN, et al. Sexual risk behaviors and implications for secondary HIV transmission during and after HIV seroconversion. *AIDS* 2002; 16: 1529–35. doi: 10.1097/00002030-200207260-00010
- 11 McCusker J, et al. Effects of HIV antibody test knowledge on subsequent sexual behaviors in a cohort of homosexually active men. *Am J Public Health* 1988; 78: 462–7.
- 12 Saah AJ, et al. Association of HLA profiles with early plasma viral load, CD4+ cell count and rate of progression to AIDS following acute HIV-1 infection. Multicenter AIDS Cohort Study. *AIDS* 1998; 12: 2107–13. doi: 10.1097/00002030-199816000-00005
- 13 Smith DK, et al. Design and baseline participant characteristics of the Human Immunodeficiency Virus Epidemiology Research (HER) Study: a prospective cohort study of human immunodeficiency virus infection in US women. *Am J Epidemiol* 1997; 146: 459–69.
- 14 Valleroy LA, et al. HIV prevalence and associated risks in young men who have sex with men. Young Men's Survey Study Group. *JAMA* 2000; 284: 198–204. doi: 10.1001/jama.284.2.198
- 15 Fogarty A, et al. The Health in Men and Positive Health cohorts: A comparison of trends in the health and sexual behaviour of HIV-negative and HIV-positive gay men, 2002–2005, National Centre in HIV Social Research Annual Report of Trends in Behaviour. 2006, University of New South Wales: Sydney.
- 16 Mao L, et al. “Serosorting” in casual anal sex of HIV-negative gay men is noteworthy and is increasing in Sydney, Australia. *AIDS* 2006; 20: 1204–6. doi: 10.1097/01.aids.0000226964.17966.75
- 17 Davis KR, Weller SC. The effectiveness of condoms in reducing heterosexual transmission of HIV. *Fam Plann Perspect* 1999; 31: 272–9. doi: 10.2307/2991537
- 18 Weller SC, Davis KR. Condom effectiveness in reducing heterosexual HIV transmission. *Cochrane Database Syst Rev* 2002; CD003255.
- 19 Pinkerton SD, Abramson PR. Effectiveness of condoms in preventing HIV transmission. *Soc Sci Med* 1997; 44: 1303–12. doi: 10.1016/S0277-9536(96)00258-4
- 20 Weller SC. A meta-analysis of condom effectiveness in reducing sexually transmitted HIV. *Soc Sci Med* 1993; 36: 1635–44. doi: 10.1016/0277-9536(93)90352-5
- 21 Fitch TJ, et al. Condom Effectiveness: Factors that influence risk reduction. *Sex Transm Dis* 2002; 29: 811–7. doi: 10.1097/00007435-200212000-00013
- 22 Rangsri R, et al. The natural history of HIV-1 infection in young Thai men after seroconversion. *J Acquir Immune Defic Syndr* 2004; 36: 622–9. doi: 10.1097/00126334-200405010-00011
- 23 Richardson BA, et al. Comparison of Human Immunodeficiency Virus Type 1 Viral Loads in Kenyan Women, Men, and Infants during Primary and Early Infection. *J Virol* 2003; 77: 7120–3. doi: 10.1128/JVI.77.12.7120-7123.2003
- 24 Simon V, Ho DD, Abdool Karim Q. HIV/AIDS epidemiology, pathogenesis, prevention, and treatment. *Lancet* 2006; 368: 489–504. doi: 10.1016/S0140-6736(06)69157-5
- 25 Sarr AD, et al. Viral dynamics of primary HIV-1 infection in Senegal, West Africa. *J Infect Dis* 2005; 191: 1460–7. doi: 10.1086/429409
- 26 Rodriguez RJ, et al. Comparison of serum and plasma viral RNA measurements in primary and chronic human immunodeficiency virus type 1 infection. *J Acquir Immune Defic Syndr Hum Retrovirol* 1997; 15: 49–53.
- 27 Lavreys L, et al. *Viral load during primary HIV-1 infection in a cohort of female commercial sex workers in Mombasa, Kenya*. Int Conf AIDS, 2000. 13: p. MoPeB2247.
- 28 Sabin CA, et al. Course of viral load throughout HIV-1 infection. *J Acquir Immune Defic Syndr* 2000; 23: 172–7.
- 29 Swindells S, et al. Predictive value of HIV-1 viral load on risk for opportunistic infection. *J Acquir Immune Defic Syndr* 2002; 30: 154–8.
- 30 Anekthananon T, et al. Safety and efficacy of a simplified fixed-dose combination of stavudine, lamivudine and nevirapine (GPO-VIR) for the treatment of advanced HIV-infected patients: a 24-week study. *J Med Assoc Thai* 2004; 87: 760–7.
- 31 Bonjoch A, et al. Long-term safety and efficacy of nevirapine-based approaches in HIV type 1-infected patients. *AIDS Res Hum Retroviruses* 2006; 22: 321–9. doi: 10.1089/aid.2006.22.321
- 32 Yozviak JL, Doerfler RE, Woodward WC. Effectiveness and tolerability of nevirapine, stavudine, and lamivudine in clinical practice. *HIV Clin Trials* 2001; 2: 474–6. doi: 10.1310/T0RR-TGY0-8QWB-8YT2
- 33 Blower S, et al. The antiretroviral rollout and drug-resistant HIV in Africa: insights from empirical data and theoretical models. *AIDS* 2005; 19: 1–14. doi: 10.1097/00002030-200501030-00001

- 34 Zhang H, et al. Human immunodeficiency virus type 1 in the semen of men receiving highly active antiretroviral therapy. *N Engl J Med* 1998; 339: 1803–9. doi: 10.1056/NEJM199812173392502
- 35 Vittinghoff E, et al. Per-contact risk of human immunodeficiency virus transmission between male sexual partners. *Am J Epidemiol* 1999; 150: 306–11.
- 36 DeGruttola V, et al. Infectiousness of HIV between male homosexual partners. *J Clin Epidemiol* 1989; 42: 849–56. doi: 10.1016/0895-4356(89)90098-X
- 37 Varghese B, et al. Reducing the risk of sexual HIV transmission: quantifying the per-act risk for HIV on the basis of choice of partner, sex act, and condom use. *Sex Transm Dis* 2002; 29: 38–43. doi: 10.1097/00007435-200201000-00007
- 38 Chesson HW, et al. HIV infections and associated costs attributable to syphilis coinfection among African Americans. *Am J Public Health* 2003; 93: 943–8.
- 39 Royce RA, et al. Sexual transmission of HIV. *N Engl J Med* 1997; 336: 1072–8. doi: 10.1056/NEJM199704103361507
- 40 Johnson AM, et al. Transmission of HIV to heterosexual partners of infected men and women. *AIDS* 1989; 3: 367–72. doi: 10.1097/00002030-198906000-00005
- 41 McCormick AW, et al. The effect of antiretroviral therapy on secondary transmission of HIV among men who have sex with men. *Clin Infect Dis* 2007; 44: 1115–22. doi: 10.1086/512816
- 42 Grulich AE, et al. Sexual behaviour and human herpesvirus 8 infection in homosexual men in Australia. *Sex Health* 2005; 2: 13–8. doi: 10.1071/SH04029
- 43 Jin F, et al. Epidemic syphilis among homosexually active men in Sydney. *Med J Aust* 2005; 183: 179–83.
- 44 Bautista CT, et al. Seroprevalence of and risk factors for HIV-1 infection among South American men who have sex with men. *Sex Transm Infect* 2004; 80: 498–504. doi: 10.1136/sti.2004.013094
- 45 Fleming DT, Wasserheit JN. From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection. *Sex Transm Infect* 1999; 75: 3–17.
- 46 Galvin SR, Cohen MS. The role of sexually transmitted diseases in HIV transmission. *Nat Rev Microbiol* 2004; 2: 33–42. doi: 10.1038/nrmicro794
- 47 Piot P, Laga M. Genital ulcers, other sexually transmitted diseases, and the sexual transmission of HIV. *BMJ* 1989; 298: 623–4.
- 48 Rottingen JA, Cameron DW, Garnett GP. A systematic review of the epidemiologic interactions between classic sexually transmitted diseases and HIV: how much really is known? *Sex Transm Dis* 2001; 28: 579–97. doi: 10.1097/00007435-200110000-00005
- 49 Simonsen JN, et al. Human immunodeficiency virus infection among men with sexually transmitted diseases. Experience from a center in Africa. *N Engl J Med* 1988; 319: 274–8.
- 50 Read TRH, et al. Risk factors for incident HIV infection in men having sex with men: a case-control study. *Sex Health* 2007; 4: 35–9. doi: 10.1071/SH06043
- 51 Crawford JM, et al. Number of risk acts by relationship status and partner serostatus: Findings from the HIM cohort of homosexually active men in Sydney, Australia. *AIDS Behav* 2006; 10: 325–31. doi: 10.1007/s10461-005-9057-3
- 52 Kaufmann GR, et al. Patterns of viral dynamics during primary human immunodeficiency virus type 1 infection. The Sydney Primary HIV Infection Study Group. *J Infect Dis* 1998; 178: 1812–5. doi: 10.1086/314480
- 53 Schacker TW, et al. Biological and Virologic Characteristics of Primary HIV Infection. *Ann Intern Med* 1998; 128: 613–20.
- 54 MAP Workshop. Extending public health surveillance of HIV infection: information from a five cohort workshop. (Multi-cohort Analysis Project). *Stat Med* 1993; 12: 2065–85. doi: 10.1002/sim.4780122203
- 55 MAP Workshop. Marker paths. (Multi-cohort Analysis Project). *Stat Med* 1993; 12: 2099–126. doi: 10.1002/sim.4780122205
- 56 Law MG, et al. Modelling the effect of combination antiretroviral treatments on HIV incidence. *AIDS* 2001; 15: 1287–94. doi: 10.1097/00002030-200107060-00011
- 57 Kilmarx PH, et al. Disease progression and survival with human immunodeficiency virus type 1 subtype E infection among female sex workers in Thailand. *J Infect Dis* 2000; 181: 1598–606. doi: 10.1086/315469
- 58 Glenday K, et al. *HIV antiretroviral treatment differences by state in Australia*. In Preparation, 2007.
- 59 Bonnet F, et al. Causes of death among HIV-infected patients in the era of highly active antiretroviral therapy, Bordeaux, France, 1998–1999. *HIV Med* 2002; 3: 195–9. doi: 10.1046/j.1468-1293.2002.00117.x
- 60 Keiser O, et al. All cause mortality in the Swiss HIV Cohort Study from 1990 to 2001 in comparison with the Swiss population. *AIDS* 2004; 18: 1835–43. doi: 10.1097/00002030-200409030-00013
- 61 Lewden C, et al. Factors associated with mortality in human immunodeficiency virus type 1-infected adults initiating protease inhibitor-containing therapy: role of education level and of early transaminase level elevation (APROCO-ANRS EP11 study). The Antiproteases Cohorte Agence Nationale de Recherches sur le SIDA EP 11 study. *J Infect Dis* 2002; 186: 710–4. doi: 10.1086/342047
- 62 Petoumenos K, Law MG. Risk factors and causes of death in the Australian HIV Observational Database. *Sex Health* 2006; 3: 103–12. doi: 10.1071/SH05045
- 63 Krentz HB, Kliwer G, Gill MJ. Changing mortality rates and causes of death for HIV-infected individuals living in Southern Alberta, Canada from 1984 to 2003. *HIV Med* 2005; 6: 99–106. doi: 10.1111/j.1468-1293.2005.00271.x
- 64 Luo K, et al. The role of initial AIDS-defining illness in survival following AIDS. *AIDS* 1995; 9: 57–64. doi: 10.1097/00002030-199501000-00008
- 65 Costello C, et al. HIV-1 subtype E progression among northern Thai couples: traditional and non-traditional predictors of survival. *Int J Epidemiol* 2005; 34: 577–84. doi: 10.1093/ije/dyi023
- 66 Li Y, et al. Improving survival following AIDS in Australia, 1991–1996. National HIV Surveillance Committee. *AIDS* 2000; 14: 2349–54. doi: 10.1097/00002030-200010200-00016
- 67 Wilson DP, Kahn J, Blower SM. Predicting the epidemiological impact of antiretroviral allocation strategies in KwaZulu-Natal: the effect of the urban-rural divide. *Proc Natl Acad Sci USA* 2006; 103: 14228–33. doi: 10.1073/pnas.0509689103
- 68 Barbour JD, et al. Higher CD4+ T cell counts associated with low viral pol replication capacity among treatment-naive adults in early HIV-1 infection. *J Infect Dis* 2004; 190: 251–6. doi: 10.1086/422036
- 69 Egger M, et al. Impact of new antiretroviral combination therapies in HIV infected patients in Switzerland: prospective multicentre study. Swiss HIV Cohort Study. *BMJ* 1997; 315: 1194–9.
- 70 Egger M, et al. Prognosis of HIV-1-infected patients starting highly active antiretroviral therapy: a collaborative analysis of prospective studies. *Lancet* 2002; 360: 119–29. doi: 10.1016/S0140-6736(02)09411-4

- 71 Hogg RS, *et al.* Improved survival among HIV-infected individuals following initiation of antiretroviral therapy. *JAMA* 1998; 279: 450–4. doi: 10.1001/jama.279.6.450
- 72 Mocroft A, *et al.* Changing patterns of mortality across Europe in patients infected with HIV-1. EuroSIDA Study Group. *Lancet* 1998; 352: 1725–30. doi: 10.1016/S0140-6736(98)03201-2
- 73 Palella FJ Jr, *et al.* Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. *N Engl J Med* 1998; 338: 853–60. doi: 10.1056/NEJM199803263381301