Appendix

Contents

1. Model of sexual behaviour

The modelling of partnership formation and dissolution, and the assumptions about coital frequencies and condom usage are the same in the network and frequency-dependent models. However, in the network model individuals are linked to specific partners when a new partnership is formed, based on a partner matching algorithm (which takes into account the same assumptions about mixing between age groups and risk groups as in the frequency-dependent model). The sections that follow describe the features of the sexual behaviour model in more detail; unless otherwise stated, these descriptions apply to both models. The final section, which describes the partner matching algorithm, applies only to the network model. Most of this material (with the exception of the partner matching algorithm) has been published previously [1], but is reproduced here for convenience.

1.1 Structure of sexual behaviour model: risk groups and relationship types

The population is divided into two broad risk groups: a high risk group (representing individuals with a propensity for concurrent sexual partners and commercial sex) and a low risk group (representing individuals who do not engage in concurrent partnerships or commercial sex). Within each of these risk groups a number of sub-groups (or states) are defined, based on the individual's current relationship status; movements between these states occur as individuals form new partnerships and end previously-formed partnerships. Figure S1 illustrates the state space that is defined for women in the high risk group. The model distinguishes between short-term (non-cohabiting) and long-term (cohabiting or marital) relationships; in addition the model allows for once-off sex acts between sex workers and clients. All long-term relationships are assumed to start as non-cohabiting relationships. For the sake of simplicity, it is assumed that individuals in the high risk group do not have more than two partners at any point in time (although high risk men can have contact with sex workers if they have two current partners). It is also assumed in the interests of simplicity that individuals do not have more than one long-term partner at any point in time, as rates of polygamy in South Africa are relatively low [2]. By definition, individuals in the low risk group cannot have more than one partner at any point in time, and many of the states that are defined for the high risk group (shaded in grey in Figure S1) therefore do not apply to the low risk group. Women engaging in sex work are assumed not to form short-term or long-term relationships during the periods in which they are active as sex workers. Only heterosexual partnerships are considered.

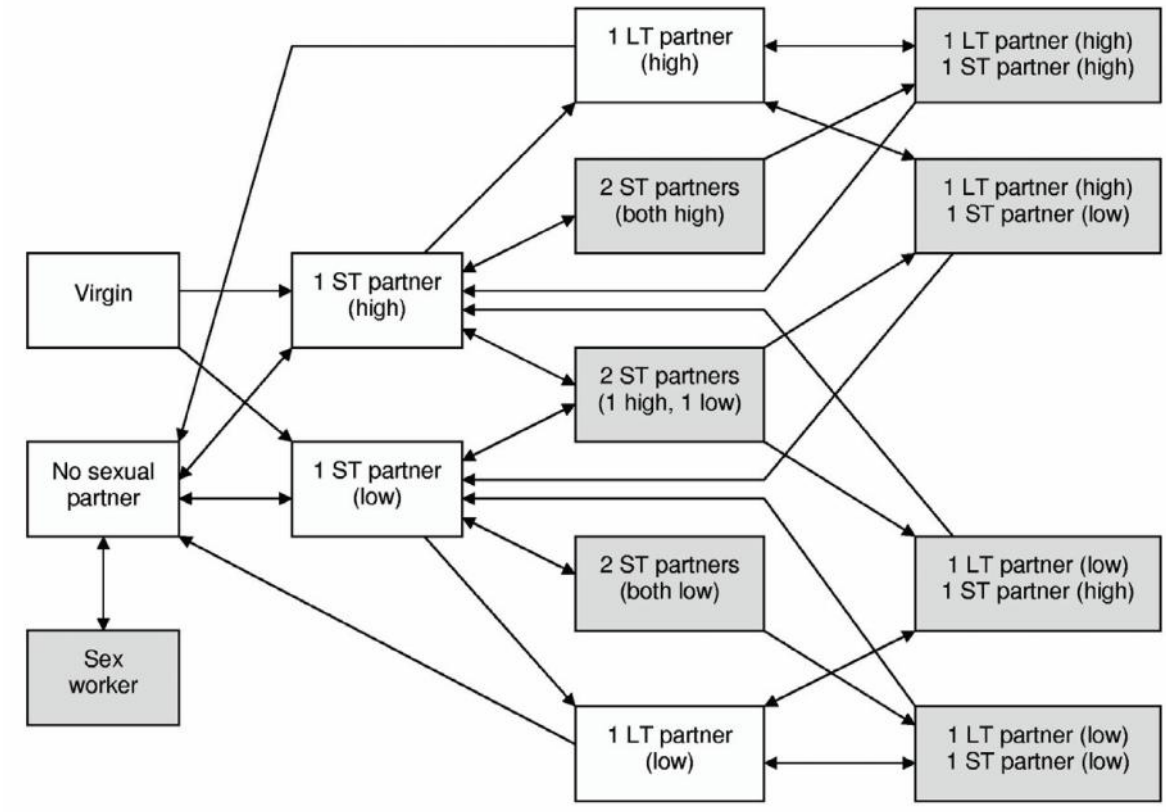

Figure S1: Multi-state model of sexual behaviour of 'high risk' females

LT = long-term (spousal). ST = short-term (non-spousal). 'High' and 'low' refer to the risk group of the sexual partner. The multi-state model for low risk females is the same as that shown here, except that the shaded states are omitted. The multi-state model for high risk men is also the same as that shown here, except that the 'sex worker' state is omitted.

The rates of transition between the different states differ according to the individual's age (rates are specified separately for each five-year age group). Some of the transition rates also depend on the individual's HIV status and stage of HIV disease. Although the frequency-dependent model does not stratify individuals according to their partner's STI status, it does stratify individuals according to their partner's risk group. The variables used in classifying the behavioural states are summarized in Table S1. Throughout section 1, $N^{s}_{g,i,j,l}(x,t)$ represents the number of individuals in the population of age x (a 5-year interval), sex g, and risk group i, in relationship type l with a partner in risk group j, and in HIV disease stage s at time t.

Table S1: Index variables

Symbol	Definition	State space
i	Individual risk group	01 = virgin with propensity for concurrency 02 = virgin with no propensity for concurrency 1 = Sexually experienced, high risk 2 = Sexually experienced, low risk 3 = Commercial sex worker (relevant to females only)
j	Risk group(s) of partner(s)	0 = no partner; 1 = 1 high risk partner; 2 = 1 low risk partner; 11 = 2 high risk partners; 12 = primary high risk & secondary low risk; 21 = primary low risk & secondary high risk; 22 = 2 low risk partners*
l	Relationship type	1 = short-term (non-marital) 2 = long-term (marital)†
x	Individual age group	10, 15, 20, …, 85
y	Partner age	10, 15, 20, …, 85
g	Sex	1 = male; 2 = female
s	HIV disease state	0 = uninfected; 1 = acute HIV; 2 = asymptomatic HIV; 3 = WHO clinical stage 3; 4 = AIDS; 5 = on ART
t	Time	0 to 40 (in years from mid-1985)

ART = antiretroviral treatment.

* Where the individual is in a marital relationship with one partner and a non-marital relationship with another, the first index refers to the risk group of the spouse and the second refers to the risk group of the other partner.

† Where the individual has two partners, this index refers to the nature of the primary partnership (the secondary relationship is always short-term). Where the individual has no partners, the index is omitted.

The fraction of the population in the high risk group has been set at 35% for men and 25% for women, based on South African studies evaluating the fraction of individuals reporting concurrent partnerships [3, 4].

1.2 Rates of short-term partnership formation

The parameter $c^{s}_{g,i,j,l}(x)$ is defined as the annual rate at which a sexually-experienced individual of sex *g* wishes to form new short-term partnerships if they are in risk group *i*, aged *x*, in HIV disease state *s*, and in relationship type *l* with a partner in group *j* (if the individual is currently single, $j = 0$ and the *l* subscript is omitted). A gamma probability density function is used to represent age differences in rates of partnership formation; for the purpose of calculating a constant rate over a five-year age interval, *x* is taken as the mid-point of the age interval (e.g. 17.5 in the 15-19 year age group). The rate at which individuals wish to form new partnerships is calculated as

$$c^{s}_{g,i,j,l}(x) = c_g \left(x - 17.5\right)^{\lambda_g - 1} \exp\left(-\alpha_g \left(x - 17.5\right)\right) \Omega_{g,i,j,l} \Phi(s)$$

where c_g is the desired rate in the baseline group (single, HIV-negative individuals in the high risk group who are aged 15-19), λ_g and α_g are the parameters of the gamma probability density function, $\Omega_{g,i,j,l}$ is an adjustment factor taking into account the individual's risk group and current relationship status, and $\Phi(s)$ is an adjustment factor that takes into account the individual's HIV status. The values assumed in the model are summarized in Table S2. These parameter values were previously estimated by fitting the frequency-dependent model to data

on numbers of current sexual partners, by age and sex, in a nationally-representative 2005 survey [5]. (The calibration to sexual behaviour data made allowance for misreporting of partner numbers, as evidenced by inconsistencies in the numbers of current partners reported by men and women.) The sexual behaviour parameters were also partially determined based on the age and sex patterns of HIV prevalence in nationally representative household surveys and antenatal surveys [5, 6]. Full details regarding the model fitting procedure are provided elsewhere [1].

Table S2: Parameters determining rates of short-term partnership formation, in sexually-experienced adults

Parameter	Assumed value		Source/explanation
	Males	Females	
c_g	7.3	14.6	[7] for women, male rate assumed to be half of female rate
α_g	3.98	4.14	Calibrated
λ_g	0.1486	0.2272	Calibrated
$\Omega_{g,i,j,l}$ for i=1, if j=0	1	1	-
$\Omega_{g,i,j,l}$ for i=1, if l=1 and j=1 or 2	0.64	0.54	Calibrated [1]
$\Omega_{g,i,j,l}$ for i=1, if l=2 and j=1 or 2	0.41	0.17	Calibrated [1]
$\Omega_{g,i,j,l}$ for i=1, if j=11, 12, 21 or 22	0	0	Maximum of 2 current partners
$\Omega_{g,i,j,l}$ for i=2, if $j\neq0$	0	0	Definition of low risk
$\Omega_{g,i,j,l}$ for i=2, if j=0	0.19	0.60	Calibrated [1]
$\Omega_{g,i,j,l}$ for i=3	0	0	No regular partners assumed for sex workers
$\Phi(0)$	1	1	-
$\Phi(1)$	1	1	No change in behaviour assumed during early disease
$\Phi(2)$	1	1	
$\Phi(3)$	0.65	0.65	[8-11]
$\Phi(4)$	0.25	0.25	[8-11]
$\Phi(5)$	0.80	0.80	[12, 13]

Because male and female demand for new partners may be inconsistent, it is necessary to balance the demand for new partnerships between the sexes. In the frequency-dependent model, this balancing is performed by setting the number of new partnerships formed to be the average of the number of new partnerships desired by women and the number of new partnerships desired by men (this averaging is performed separately for each possible combination of male and female risk group; mathematical details have been presented previously [1]). In the network model, the balancing is achieved by randomly changing the order in which individuals choose their sexual partners, from one time step to the next, with individuals being more or less likely to achieve their desired number of partners depending on how close they are to the front of the 'queue'. This ensures that the actual number of new partnerships formed will be (on average) halfway between the number of new partnerships desired by men and the number of new partnerships desired by women. Figure S2 shows that the network and frequency-dependent models produce similar estimates of the average numbers of short-term partners, although in the youngest age groups (15-24) the network model estimates a slightly higher average number of short-term partners.

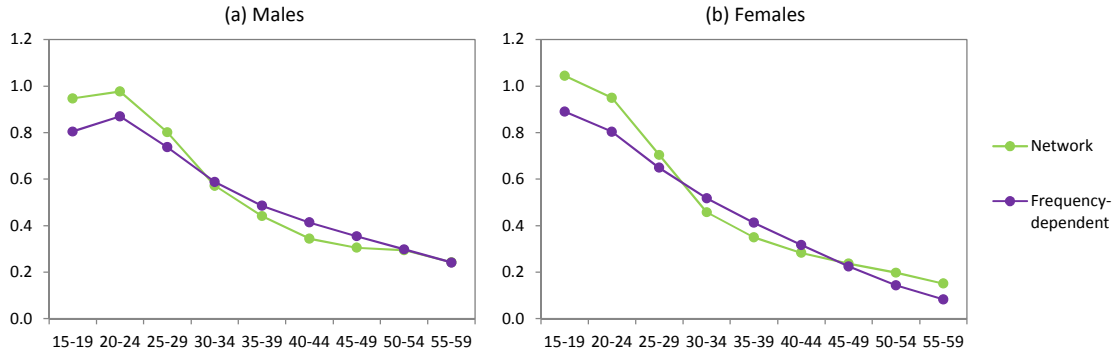

Figure S2: Average number of short-term partners at a point in time, per sexually-experienced individual

Results are calculated after simulating demographic and behavioural changes over a 20-year period. No HIV was introduced into either model. Results from the network model are from a single simulation.

1.3 Rates of sexual debut

Sexual debut is assumed to occur between the ages of 10 and 30. Sexual debut is assumed to occur upon entry into a short-term relationship, and the modelling of sexual debut is therefore similar to the modelling of the rates of short-term partnership formation. Rates of sexual debut are specified for each five-year age group, and correspond to the desired number of new sexual partners per period referred to in the previous section. The rates for the high risk group are specified in Table S3. Rates for the low risk group are assumed to be 50% of those in the high risk group, based on studies showing strong associations between early sexual debut and high risk behaviour later in life [3, 14, 15]. The assumed rates of sexual debut have been set in such a way that the overall fraction of youth who are sexually experienced, by age and sex, are roughly consistent with those reported in a 2005 national survey [5], except in the case of girls, where a degree of under-reporting is assumed to occur.

Table S3: Annual rates of sexual debut in high risk youth, by age and sex

Age group	10-14	15-19	20-24	25-29
Males	0.01	0.27	0.82	1.00
Females	0.05	0.52	0.91	1.00

1.4 Rates of marriage

The rates at which short-term partnerships become cohabiting or marital have been set in such a way that the model matches the observed proportions of the population in marital/cohabiting relationships, by age and sex, as reported in national censuses in 1996 and 2001, and in a 2007 national community survey (more detail on the parameters and calibration is provided elsewhere [1]). The fraction married is assumed to be the same for the high risk group and low risk group, which means that the rate at which short-term relationships become marital must be higher in the low risk group in order to compensate for the lower numbers of short-term partnerships in the low risk group. The rates at which short-

term relationships become marital are also assumed to depend on age and sex. Figure S3 shows that the modelled fraction of individuals who are in marital/cohabiting relationships is similar in the two models, by age and sex, although in men aged 25-39 the married fraction is lower in the network model than in the frequency-dependent model.

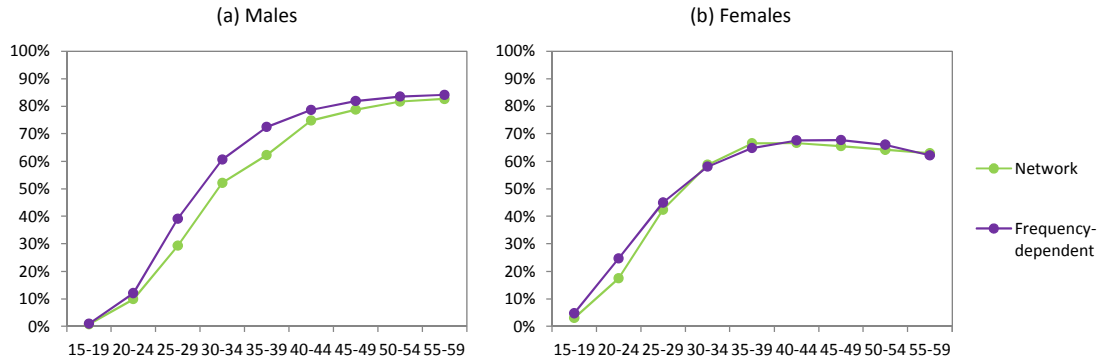

Figure S3: Fraction of individuals in marital/cohabiting relationships
Results are calculated after simulating demographic and behavioural changes over a 20-year period. No HIV was introduced into either model. Results from the network model are from a single simulation.

1.5 Mixing between risk groups

The parameter $°_{g,i,j}(t)$ is defined as the desired proportion of new short-term partners who are in risk group *j*, for an individual of sex *g* and in risk group *i* (*j* = 1 or 2 only) at time *t*. Mathematically, it is calculated according to the following formula:

$$°_{g,i,j}(t) = (1-£)¢_{ij} + £\frac{\sum_{u=0}^{2}\sum_{l}\sum_{y}\sum_{s=0}^{5} N^{s}_{g^*,j,u,l}(y,t)c^{s}_{g^*,j,u,l}(y)}{\sum_{v=1}^{2}\sum_{u=0}^{2}\sum_{l}\sum_{y}\sum_{s=0}^{5} N^{s}_{g^*,v,u,l}(y,t)c^{s}_{g^*,v,u,l}(y)},$$

where $¢_{ij}$ = 1 if *i* = *j* and 0 otherwise, g^* is the sex opposite to *g*, and £ is the degree of assortative mixing. The degree of assortative mixing can be any value from 0 to 1, with lower values of the parameter indicating greater tendency to form partnerships with individuals in the same sexual activity class. To our knowledge, there are no South African data sources that directly inform the choice of the £ parameter. However, in a previous Bayesian analysis, which involved fitting the frequency-dependent model to South African HIV prevalence data and sexual behaviour data, the posterior mean of the £ parameter was 0.56 [1]. This parameter value has been used in the present analysis.

1.6 Mixing between age groups

For both sexes, an age mixing matrix is specified, which determines the fraction of partners in each five-year age group, for individuals in each age group. Tables S4 and S5 show the age mixing matrices for women and men respectively. The female age mixing matrix is estimated based on the ages of spousal partners reported by women in the 1998 Demographic and Health Survey [16] and the age differences reported by women in non-spousal partnerships in smaller studies [5, 17-19]. The male age mixing matrix has been calculated to be consistent with the female age mixing matrix. In the network model, the age mixing matrices are taken into consideration when determining the relative probabilities of selecting different individuals as sexual partners (see section 1.11).

Table S4: Percentage of women's partners in each age group

	Age of male partner															
Female age	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
10-14	46.8	43.1	8.1	1.5	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
15-19	0.0	47.5	40.9	8.8	2.1	0.5	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
20-24	0.0	0.0	48.9	35.2	11.1	3.6	0.8	0.3	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
25-29	0.0	0.0	1.3	43.8	35.7	12.7	4.4	1.0	0.3	0.3	0.1	0.2	0.1	0.0	0.0	0.0
30-34	0.0	0.0	0.2	3.3	37.5	38.4	13.3	4.1	1.4	0.9	0.2	0.4	0.1	0.0	0.0	0.0
35-39	0.0	0.0	0.0	0.3	3.9	37.2	36.3	14.0	4.4	1.4	0.9	1.2	0.3	0.0	0.0	0.0
40-44	0.0	0.0	0.0	0.1	1.4	5.3	35.4	35.3	15.6	3.7	1.2	1.6	0.4	0.1	0.0	0.0
45-49	0.0	0.0	0.0	0.0	0.0	1.2	9.2	35.3	31.9	14.2	3.8	3.4	0.9	0.1	0.0	0.0
50-54	0.0	0.0	0.0	0.0	0.0	0.0	1.6	14.4	32.1	29.8	15.3	5.2	1.3	0.2	0.0	0.0
55-59	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.0	15.8	33.6	29.7	13.9	4.0	0.8	0.1	0.0
60-64	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.8	15.9	36.5	30.9	11.9	2.5	0.3	0.0
65-69	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.4	16.2	41.2	31.4	8.7	1.0	0.1
70-74	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.0	17.4	47.6	29.1	4.6	0.2
75-79	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	20.3	56.5	21.3	1.2
80-84	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.7	26.7	57.9	14.8
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.4	35.0	63.6

Table S5: Percentage of men's partners in each age group

	Age of female partner															
Male age	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
10-14	100.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
15-19	10.8	89.2	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
20-24	0.8	30.7	66.5	1.8	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
25-29	0.1	5.6	40.9	49.8	3.3	0.2	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
30-34	0.0	1.4	13.5	42.5	38.7	3.0	0.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
35-39	0.0	0.4	4.7	16.4	43.0	31.2	3.6	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
40-44	0.0	0.0	1.2	6.9	18.0	36.6	29.6	6.7	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
45-49	0.0	0.1	0.6	1.8	6.4	16.3	34.0	29.6	10.0	1.2	0.0	0.0	0.0	0.0	0.0	0.0
50-54	0.0	0.0	0.1	0.7	2.6	6.3	18.4	32.9	27.3	11.0	0.7	0.0	0.0	0.0	0.0	0.0
55-59	0.0	0.0	0.2	0.9	2.2	2.5	5.4	18.4	32.0	29.7	7.9	0.5	0.0	0.0	0.0	0.0
60-64	0.0	0.0	0.0	0.4	0.8	2.2	2.4	6.4	21.4	34.2	23.8	8.2	0.3	0.0	0.0	0.0
65-69	0.0	0.0	0.0	0.9	1.6	3.4	3.7	7.0	8.7	19.2	24.3	25.3	5.8	0.0	0.0	0.0
70-74	0.0	0.0	0.0	0.4	0.7	1.5	1.7	3.1	3.7	9.6	16.1	33.1	27.3	2.7	0.0	0.0
75-79	0.0	0.0	0.0	0.2	0.3	0.6	0.6	1.1	1.8	4.7	8.3	22.5	40.9	18.3	0.8	0.0
80-84	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.4	3.6	5.4	13.6	32.4	34.7	8.7	0.2
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.7	5.9	6.2	10.6	19.6	24.5	26.8	3.6

Figure S4 shows that the partner age distributions are roughly consistent in the network and frequency-dependent models. However, there are some differences, with the network model tending to estimate a higher fraction of women having partners in the same age group or younger. It is important to note that the frequency-dependent model does not directly

simulate the age distribution of sexual partners (unlike the network model); instead it assumes that the distribution of partner ages remains constant over time (as specified in Tables S4 and S5). The network model is expected to produce slightly different results because it reflects the effect of demographic changes to the population age profile and changes in the relative availability of partners at different ages.

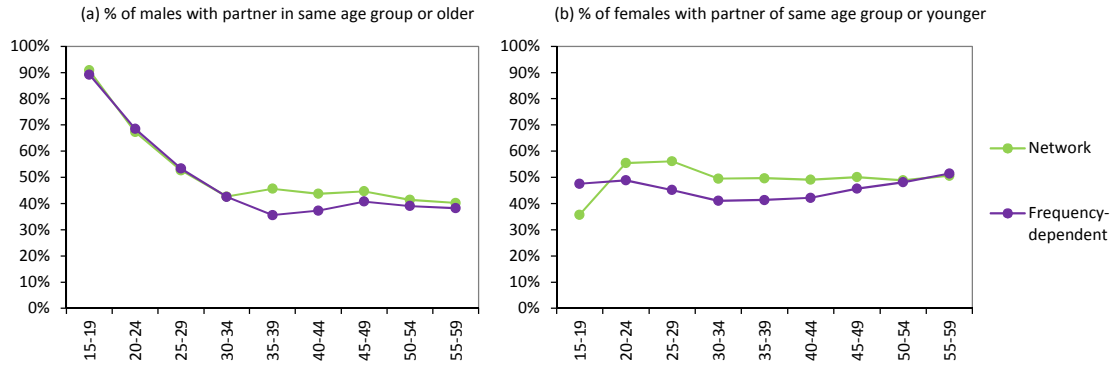

Figure S4: Partner age distributions

Results from the network model are calculated after simulating demographic and behavioural changes over a 20-year period. Results from the network model are from a single simulation, in which no HIV was introduced. Results from the frequency-dependent model are calculated from Tables S4 and S5, since the frequency-dependent model does not directly simulate the partner age distribution.

1.7 Rates of partnership dissolution and divorce

Partnerships can be terminated through death of either partner, through divorce (in the case of marital relationships) or through 'break up' (in the case of short-term relationships). In order to calculate rates of termination, it is necessary to define the following variables:

$D_{g,l}(x)$ = annual rate at which partnerships of type l dissolve, among individuals aged x, of sex g (ignoring mortality)

$f_g(y \mid x)$ = proportion of partners in age band y, if individual is of sex g and in age band x

$\alpha_g^s(x,t)$ = force of mortality at time t, in individuals aged x, of sex g, who are in HIV disease state s

For short-term relationships (l = 1), the annual rate of dissolution has been set at 2, which implies an average duration of short-term relationships equal to 6 months, roughly consistent with average durations of 3-12 months observed in African studies of non-spousal relationships [7, 20, 21]. For long-term relationships (l = 2), the rates of relationship dissolution have been estimated by multiplying estimated rates of divorce in 2004 (by age and sex) by a factor of 2 [22]. (This upward adjustment makes allowance for the fact that rates of dissolution are higher in cohabiting non-marital relationships than in marital relationships, and many married individuals are separated although not formally divorced.) The age- and sex-specific rates of divorce are tabulated in a previous publication [1]. The $f_g(y \mid x)$ values are contained in the age mixing matrices specified in the previous section (Tables S4 and S5), and the mortality rates depend on the model assumptions about non-AIDS mortality rates (by age and sex) and AIDS mortality.

For a man who is of age *x*, in group *i*, in relationship type *l* with a partner in group *j* at time *t*, the probability of the relationship being terminated over the time period $[t, t+d)$ is calculated in the frequency-dependent model as

$$1-\exp\left(-d\left(D_{1,l}(x)+\sum_{y} f_1(y \mid x)\frac{\sum_{s=0}^{5} N^{s}_{2,j,\bullet}(y,t)\ll^{s}_{2}(y,t)}{\sum_{s=0}^{5} N^{s}_{2,j,\bullet}(y,t)}\right)\right),$$

where $N^{s}_{g,j,\bullet}(y,t)$ represents the sum across all behavioural states involving a relationship type *l* with a partner in group *i*. A similar formula is used to determine the probability that a woman's partnership is terminated.

In the network model, a similar logic is applied, but because the individual is linked to a specific partner, and the survival of that partner is simulated separately, it is not necessary to include the mortality term in the above equation. (Whenever a death occurs, any relationships that the individual was in are automatically terminated.)

1.8 Commercial sex

Sexually experienced men are assumed to have contact with sex workers at an annual rate $w_{i,j,l}(x)$, which depends on their age (*x*), risk group (*i*) and current relationship status (represented by *j* and *l*). As in section 1.2, a gamma probability density function is used to represent the age differences in rates of male contact with sex workers; for the purpose of calculating a constant rate over each five-year age band, *x* is taken to be the midpoint of the age group (e.g. 17.5 for men aged 15 to 19). The following formula is used to calculate $w_{i,j,l}(x)$:

$$w_{i,j,l}(x) = w\,\mathtt{a}_{c}^{\tilde{}_{c}}\left(x-10\right)^{\tilde{}_{c}-1}\exp\left(-\mathtt{a}_{c}\left(x-10\right)\right)Y_{i,j,l}$$

where λ_c and α_c are the parameters of the gamma probability density function, and $Y_{i,j,l}$ is an adjustment factor to represent the man's risk group and relationship status. The assumed values of the parameters are summarized in Table S6. The base rate of sex worker contact (*w*, which applies to high risk men who currently have no partner and are aged 20-24) has been set in such a way that the demand for commercial sex is sufficient to match the estimated size of the South African sex worker population in a recent study [23], when it is assumed that sex workers have 750 clients per annum on average [24-31]. (The resulting estimate of the fraction of women who are sex workers is substantially higher than that estimated previously in the frequency-dependent model [1], although lower than that estimated empirically [23] because the empirical estimate is based on a broader definition of sex worker than is used in the model.) The *w* parameter has been calculated so that the average number of sex worker contacts per year, averaged across all men aged 15 to 49 at the start of the simulation, is 5, i.e.

$$w \equiv \frac{5\sum_{x=1}^{7}\sum_{i}\sum_{j}\sum_{l}\sum_{s} N^{s}_{1,i,j,l}(x\times 5,0)}{\sum_{x=1}^{7}\sum_{j}\sum_{l}\sum_{s} N^{s}_{1,1,j,l}(x\times 5,0)\,\alpha_c\,\gamma_c\left(x\times 5+2.5\right)^{\gamma_c-1}\exp\left(-\alpha_c\left(x\times 5+2.5\right)\right)Y_{i,j,l}}$$

Table S6: Assumed male rates of sex worker contact

Parameter	Value	Source/explanation
λ_c	0.165	Based on age differences in rates of
α_c	3.31	male contact with sex workers [32]
$Y_{i,j,l}$ for i=1 and j=0	1	-
$Y_{i,j,l}$ for i=1, l=1 and j=1 or 2	0.5	Assumption
$Y_{i,j,l}$ for i=1, l=2 and j=1 or 2	0.3	Assumption
$Y_{i,j,l}$ for i=1, l=1 and j=11, 12, 21 or 22	0.2	Assumption
$Y_{i,j,l}$ for i=1, l=2 and j=11, 12, 21 or 22	0.1	Assumption
$Y_{i,j,l}$ for i=2	0	Definition of low risk group

Since only men in the high risk group are assumed to have contact with sex workers, and since all sex workers are assumed to be recruited from the high risk group, no assumptions about mixing between risk groups are required for the purpose of modelling commercial sex. It is assumed in the interests of simplicity that clients have no preference regarding the age of their commercial sex contacts, although age preferences are accounted for implicitly by assuming a sex worker age distribution (based on data from Johannesburg [33]) and setting the age-specific rates of entry into sex work in such a way that the simulated age distribution remains roughly stable over time and matches that assumed. Women are assumed to remain active as sex workers for two years on average, before returning to the 'No sexual partner' state (Figure S1).

The number of new sex workers required over the period $[t,t+d)$ in order to satisfy male demand, $\Delta_c(t,t+d)$, is calculated as

$$\Delta_c(t,t+d) = \frac{1}{C}\sum_{s=0}^{5}\sum_{j}\sum_{l}\sum_{x} N^{s}_{1,1,j,l}(x,t)w_{1,j,l}(x) - \sum_{s=0}^{5}\sum_{x} N^{s}_{2,3,0}(x,t),$$

where C is the assumed annual number of clients per sex worker (750), and the second term in the equation is the total number of sex workers at the start of the time step. The probability that a woman in the high risk group, who has no partners and is aged x and in HIV disease state s at time t, becomes a sex worker over the period $[t,t+d)$ is calculated as

$$\frac{\Delta_c(t,t+d)W(x)\Phi(s)}{\sum_{s=0}^{5}\sum_{u} N^{s}_{2,1,0}(u,t)W(u)\Phi(s)},$$

where $W(x)$ is the factor by which the rate of recruitment into the 'sex worker' group is multiplied when the woman is of age x in order to match the target sex worker age profile, and $\Phi(s)$ is the factor by which the rate of recruitment into commercial sex is adjusted in HIV

disease stage *s* (these are the same as the factors assumed in modelling formation of short-term partnerships – see Table S2).

1.9 Frequency of sex

In short-term relationships, sex is assumed to occur at a rate of 3 times per month on average, based on South African studies reporting on coital frequencies among youth [19, 34, 35]. In long-term relationships, coital frequencies are assumed to depend on the age and sex of the individual. In married women, coital frequencies are assumed to reduce exponentially from a rate of 5 per month in the 20-24 age group, declining by a factor of 50% for each 20-year increase in age (i.e. reducing to 2.5 times per month in 40-44 year olds, to 1.25 times per month in 60-64 year olds, etc.). Coital frequencies in married men are calculated to be consistent with the assumed female frequencies, taking into account the age mixing matrices. These assumed frequencies of sex in spousal and non-spousal partnerships result in numbers of sex acts that are roughly consistent with the aggregate reported coital frequencies in the 15-24 and 25-49 age bands in a 2005 national household survey [5].

In the network model, inconsistencies can emerge if coital frequencies are specified for men and women separately. The coital frequencies are therefore simulated for men only.

1.10 Condom usage

The probability of condom use is assumed to depend on the individual's age and sex, as well as the type of relationship that they are in. In addition, the model allows for changes in condom usage over time, as evidence suggests that there have been substantial increases in condom usage since the launch of various HIV communication programmes in the 1990s and early 2000s [36, 37]. However, it has been noted that the actual trends in HIV prevalence in South Africa appear inconsistent with the reported increases in condom usage, and this suggests that there may be some degree of social desirability bias in the reporting of condom use [38]. In the calibration of the models to the HIV prevalence data, we therefore make allowance for uncertainty regarding the extent of the bias in the self-reported data, allowing certain parameters to be interpolated between minimum and maximum values (the maxima corresponding to values that would be assumed if there were no reporting bias). A detailed explanation of the model of condom use has been published previously [38], but is repeated here for convenience.

The parameter $¥_{2,l}(x,t)$ represents the probability that a woman aged *x* uses a condom in an act of sex with a partner of type *l* at time *t*. This parameter is calculated in relation to a 'baseline' rate of condom usage, $¥^{*}$, which is the probability of condom use for a woman aged 15-19 in a short-term relationship in 1998 (1998 has been chosen as the baseline because it is the year for which the most condom usage data are available, and because there is little reliable data on condom usage prior to 1998). The following formula is used to calculate $¥_{2,l}(x,t)$:

$$\ln\left(\frac{¥_{2,l}(x,t)}{1-¥_{2,l}(x,t)}\right)=\ln\left(\frac{¥^{*}}{1-¥^{*}}\right)+ ¡_{l} +¬_{l}(x-15)+$$
$$\left[©_{l}^{i}+\left(©_{l}^{u}-©_{l}^{i}\right)\left(1-0.5^{(t/M_{l})^{Q_l}}\right)\right]$$

where

$\exp(¡_{l})$ = the odds of using a condom in relationship type *l*, relative to that in short-term relationhips (*l* = 1), in 1998;

$\exp(¬_{l})$ = the factor by which the odds of condom use reduces, per year of age;

$\exp(©_{l}^{i})$ = the initial odds of using a condom in relationship type *l*, in 1985 (before the onset of behaviour change), relative to the odds in 1998;

$\exp(©_{l}^{u})$ = the ultimate odds of using a condom in relationship type *l*, once behaviour change is at its maximum, relative to the odds in 1998;

M_{l} = the median time to behaviour change in relationships of type *l*, i.e. the time at which the log odds of condom use is half-way between its initial and ultimate levels (in years since 1985);

Q_{l} = the Weibull shape parameter controlling the speed of behaviour change in relationships of type *l*.

The term in square brackets represents the difference in condom usage (on a logit scale) between year *t* and 1998. A Weibull distribution is used to model the transition from the initial low levels of condom usage to the 'ultimate' levels of condom use. The logistic transformation prevents rates of condom use greater than 100%, and facilitates a 'logistic regression' interpretation of the condom parameters. Based on logistic regression models fitted to data on condom usage in the 1998 and 2003 South African DHSs [16, 39], it is assumed that the parameter $¬_{l}$ is -0.025 for both spousal and non-spousal relationships, and that the odds of condom usage in spousal relationships relative to that in non-spousal relationships ($\exp(¡_{2})$) is 0.46 in 1998. The proportion of African women reporting condom usage for contraceptive purposes was found to be 0.13% in the 1987-89 DHS [40], compared to 1.8% in the 1998 DHS, and on the basis of this information, the ratio of the initial odds of condom use to that in 1998 ($\exp(©_{l}^{i})$) is assumed to be 0.07 for both spousal and non-spousal relationships.

In interactions between sex workers and their clients, levels of condom usage were around 60% in 1998 [17, 33], compared with levels of around 20% in women aged 15-19 in the 1998 DHS. Based on this evidence, it is assumed that in 1998 the ratio of the odds of condom use in sex worker-client interactions to that in non-spousal relationships ($\exp(¡_{3})$) was 6.0. In the absence of information regarding age differences in condom use by sex workers, no age effect is assumed ($¬_{3}$ = 0). A study conducted in 1988 found that condom usage was reported by only about 20% of sex workers and their clients [41], and the odds ratio for condom use in 1985, relative to that in 1998 ($\exp(©_{3}^{i})$), is therefore set at 0.17. More recent studies suggest that levels of condom usage close to 90% may be possible [29, 42, 43], and the ratio of the ultimate odds of condom use to that in 1998 ($\exp(©_{3}^{u})$) is therefore set at 6.0. The parameter

Q_3 has been set at 5.22, to produce a trend in condom use consistent with these survey estimates.

The remaining parameters - ¥*, ©$_1^u$, ©$_2^u$, Q_1 and Q_2 - have been set separately for two scenarios: a scenario in which women are assumed to report accurately on their levels of condom use, and a scenario in which women are assumed to overstate their levels of condom use substantially. A condom reporting bias parameter, θ, is used to interpolate linearly between the parameter values in these two scenarios, with $\theta = 0$ corresponding to the scenario in which there is no bias and $\theta = 1$ corresponding to the scenario in which there is substantial over-reporting of condom use. The assumed parameter values for the two scenarios are summarized in Table S7. Parameters in the 'no bias' scenario were chosen so that the modelled proportions of young women using condoms were reasonably consistent with data on the proportion of young women reporting having used a condom the last time they had sex [5, 16, 44-46]. Parameters in the 'high bias' scenario were chosen so that the modelled proportions of young women reporting condoms were consistent with proportions of sexually active women who reported using condoms for contraceptive purposes in the Demographic and Health Surveys (on the assumption that these would be less affected by social desirability bias and would represent a minimum on the true rate of condom use). Although the assumptions about the relative levels of condom usage in the early stages of the epidemic and the levels of condom usage in sex workers are the same in all scenarios, these parameters were found to have little influence on HIV incidence, and potential bias in the estimation of these parameters is therefore of little consequence.

Table S7: Differences in condom usage parameters between scenarios

Parameter	Symbol	No bias scenario ($\theta = 0$)	High bias scenario ($\theta = 1$)
Probability of condom use in women aged 15-19, in short-term relationships, in 1998	¥*	0.20	0.08
Ultimate odds of condom use in short-term relationships, relative to the odds of condom use in 1998	$\exp(©_1^u)$	15	3
Ultimate odds of condom use in long-term relationships, relative to the odds of condom use in 1998	$\exp(©_2^u)$	7	1.5
Shape parameter controlling the speed of behaviour change in short-term relationships	Q_1	2.8	3.8
Shape parameter controlling the speed of behaviour change in long-term relationships	Q_2	1.8	3.6

For all relationship types, the median parameter M_l is calculated by noting that the 'baseline' parameters relate to 1998, and hence when $t = 13$ (i.e. in 1998)

$$©_l^i + (©_l^u - ©_l^i)\left(1 - 0.5^{(t/M_l)^{Q_l}}\right) = 0 .$$

The parameter M_l is therefore calculated as a function of $©_l^i$, $©_l^u$ and Q_l.

To ensure that male and female assumptions are consistent, the probability that a man uses a condom in a short-term or long-term relationship is calculated as

$$¥_{1,l}(x,t) = \sum_{y} f_1(y \mid x)¥_{2,l}(y,t),$$

where $f_1(y \mid x)$ is the probability that a female partner is aged *y*, if the male partner is aged *x*. The rate of condom use among clients of sex workers is the same as that estimated for sex workers, with no age dependency.

In the first analysis presented in the main text, it is assumed that there are no increases in condom usage over time (i.e. the term in square brackets is replaced with $©_l^i$ in all years). In this analysis, the θ parameter is set to 0.8, corresponding to the posterior mean estimated when the frequency-dependent model was previously fitted to South African HIV prevalence data [38]. In the second analysis presented in the main text, condom usage is allowed to change over time, as specified previously. When performing the uncertainty analysis for the HIV scenarios, θ values are randomly sampled from the uniform (0, 1) distribution. When performing the uncertainty analyses for the other STIs (conditional upon the best-fitting HIV parameters), the θ value is fixed at 0.63, the average of the parameters in the 100 best-fitting parameter combinations obtained from fitting the network model to the HIV data.

1.11 Partner matching algorithm in the network model

The most important difference between the network and frequency-dependent models is that the former models pair formation and links an individual to a specific partner whenever a new partnership is formed. The procedure for pair formation is as follows:

1. At the start of each week, we calculate for each individual the rate at which they wish to form new partnerships (this calculation is exactly the same as in the deterministic model). Suppose that for the i^{th} individual, c_i is the desired annual rate at which new partnerships are formed.
2. We then randomly generate a 'queue' of all individuals in the population (a new queue is randomly generated at the start of each time step, as a fixed queue would mean that some individuals are permanently advantaged/disadvantaged by their position in the queue, in contrast to the frequency-dependent model, which treats all individuals equally).
3. For the first person in the queue, we randomly assign a new relationship status at the end of the week. Depending on the individual's relationship status at the start of the week, one of seven possible events can occur: they can acquire a new high risk partner, acquire a new low risk partner, marry an existing high risk partner, marry an existing low risk partner, end a short-term relationship with a high risk partner, end a short-term relationship with a low risk partner, or get divorced. If none of these events occur, the 'new' relationship status of the individual at the end of the week is the same as that at the start. The method for calculating the probabilities of each of these events is exactly the same as in the frequency-dependent model.
4. If the new event is acquisition of a new high risk partner, a partner age group is randomly sampled from the specified partner age preference matrix (see Tables S4

and S5). A new partner is selected from the pool of potential high risk partners in the relevant 5-year age group as follows:

a) We calculate the sum of the rates at which individuals wish to form new partnerships, out of those people of the opposite sex in the high risk group who remain in the queue (i.e. individuals who have not yet been assigned a new relationship status). Mathematically, we are calculating for age group x,

$$N(x) = \sum_{j \in J(x)} c_j ,$$

where $J(x)$ is the set of high risk individuals aged x, of the opposite sex, who remain in the queue, and c_j is the annual rate at which individual j wishes to acquire new partners.

b) We assign sample weights to each of the individuals who is eligible to form a new relationship with the first individual. Mathematically, the weight assigned to individual j, if they are in the set $J(x)$, is $w_j = c_j / N(x)$.

c) A new partner is randomly selected from the set $J(x)$ using the sample weights. If set $J(x)$ is empty, a different age group is randomly selected, and a high risk partner from that risk group is randomly chosen, in the same way as before. However, if the second age group is also empty, the event assigned to individual i changes to acquisition of a new partner in the low risk group, and a partner is selected from the low risk group in the same way as for the high risk group (using the same sampled age groups). If there are no available partners in the low risk group, for either of the randomly sampled ages, the individual is assigned no change in relationship status.

The procedure is exactly the same if the new event is acquisition of a new low risk partner, except that the 'high' and 'low' risk labels in steps a-c are reversed.

5. The procedure outlined in steps 3 and 4 is repeated for the second person in the queue, and similarly for each subsequent individual. Note that each time an individual has a change in relationship status assigned to them, the relationship status of the associated partner automatically also gets updated, so that the partner gets removed from the queue of individuals waiting to be assigned a new relationship status.

The removal from the queue in the last step happens because we are only allowing a maximum of one new relationship event per individual, in each time step, to be consistent with the frequency-dependent model. For example, if individual A selects individual B as their new partner, then individual B has had an event assigned to them and cannot experience another event in the same time step. Similarly, if individual A is in a relationship with individual C at the start of the time step and does not end that relationship, then individual C cannot end the relationship when their turn in the queue comes (otherwise individual A would be both forming a new partnership and ending a partnership in the same time step). Because we are only allowing one event to occur in each time step, we avoid having to make assumptions about how partnership allocation is ordered when multiple partnerships can be assigned [47]. Although the assumption that only one partnership event can occur in each time step is not realistic, we are using weekly time steps to model changes in relationship status, and any loss of accuracy is therefore likely to be minimal. Contacts between sex workers and clients are not included in these relationship events.

Although sexual relationships are updated at weekly time steps, STI transmission and resolution can be updated more frequently. In the analyses that follow, HIV transmission and disease progression are updated at weekly time steps, but all other STIs are updated four times per week (to account for the rapid health seeking that may occur when STI symptoms develop).

2. Mathematical modelling of STI transmission and natural history

The sections that follow describe the modelling of the natural history of each STI and the assumptions about transmission probabilities per act of unprotected sex. Prior distributions are specified to represent the uncertainty around certain parameters. Most of the model assumptions and prior distributions are the same as described in previous publications [1, 48-51], although certain assumptions and prior distributions have been updated in light of recent evidence.

2.1 Mathematical model of gonorrhoea

Individuals who acquire gonorrhoea are assumed to either develop symptoms or remain asymptomatic, and eventually experience spontaneous resolution of infection if treatment is not sought. Symptomatic individuals are assumed to seek treatment at rate υ, which is effective in curing the infection with probability ψ. As there is some evidence of strain-specific immunity following recovery from gonorrhoea [52, 53], an additional state is defined to represent individuals who are temporarily immune following recovery. All individuals who experience spontaneous resolution of infection are assumed to enter this state. However, since successful early treatment of gonorrhoea does not appear to be followed by immunity [54], only a fraction (φ_2) of individuals are assumed to be immune if they have experienced resolution of infection following treatment. Immunity is assumed to wane at rate σ_3. This model of natural history and immunity is illustrated in Figure S5.

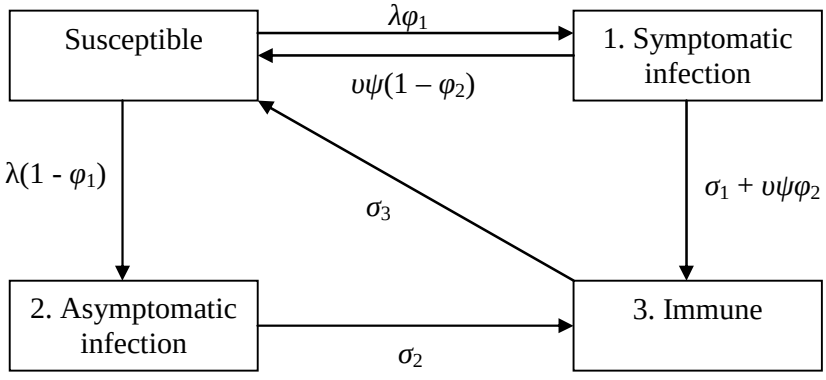

Figure S5: Multi-state model of the natural history of gonorrhoea

The prior distributions for the associated model parameters are specified in Table S8. For the most part these are the same as assumed previously [48, 50], but the prior distribution for the average duration of untreated gonorrhoea has a greater mean and standard deviation than that assumed previously, due to the recognized limitations of the available data [55]. Due to the lack of empirical data on transmission probabilities per act of sex, we have relied on estimates of transmission probabilities from other studies that have fitted mathematical models to gonorrhoea prevalence data.

Table S8: Gonorrhoea parameters

Parameter	Symbol	Prior distribution			
		Type	Mean	SD	Ref.
% of cases that become symptomatic					
Male	φ_1	Beta	0.90	0.05	[54, 56-60]
Female		Beta	0.40	0.15	[61, 62]
Average duration if untreated (weeks)					
Male	$1/\sigma_2$*	Gamma	20.0	10.0	[55, 62, 63]
Female		Gamma	20.0	10.0	[55, 62]
Average duration of immunity (weeks)	$1/\sigma_3$	Gamma	52.0	26.0	-
Proportion immune after treatment cure	φ_2	Uniform	0.50	0.29	-†
Transmission probability per act of sex					
Male-to-female	-	Beta	0.40	0.10	[61, 64-68]
Female-to-male	-	Beta	0.20	0.05	[65-70]
Fraction of symptoms correctly treated prior to introduction of SM	A	Beta	0.70	0.10	[71-74]

* Same parameter is used for symptomatic duration ($1/\sigma_1$) and asymptomatic duration ($1/\sigma_2$). † Due to the lack of evidence, a vague prior (uniform on the interval [0, 1]) is assumed.
SD = standard deviation, SM = syndromic management.

Suppose that we wish to estimate $R(t)$, the average number of partners an infected individual can be expected to transmit their infection to, within time t after acquiring infection, if all contacts are with susceptible partners and if infected individuals transmit their infection to susceptible partners at a constant rate β. (This is similar to the concept of "transmission potential" introduced by Fraser *et al* [75], except that it is defined as a function of time since infection.) In reality, the rate β differs according to the individual's characteristics and those of their partner(s), but for the purpose of deriving a simple summary measure, we will suppose it is constant. For the purpose of estimating $R(t)$, we also need to suppose that there is a constant rate of retirement from the sexually active population, ρ (this could be due to death or age-related reductions in sexual activity). Again, the parameter ρ differs between individuals in reality, but for the purpose of deriving a simple summary measure we will suppose it is constant. It can then be shown that

$$R(t) = \int_0^t \left\{ \varphi_1 \exp\left(-\left(\sigma_1 + \beta \cdot \rho\right)s\right) + \left(1-\varphi_1\right)\exp\left(-\left(\sigma_2 + \rho\right)s\right)\right\} ds$$

$$= \left[\frac{\varphi_1\left(1-\exp\left(-\left(\sigma_1 + \beta \cdot \rho\right)t\right)\right)}{\sigma_1 + \beta \cdot \rho} + \frac{\left(1-\varphi_1\right)\left(1-\exp\left(-\left(\sigma_2 + \rho\right)t\right)\right)}{\sigma_2 + \rho} \right]$$

In a frequency-dependent model, we are assuming that there is a constant rate of contact with susceptible partners, which does not change with respect to the duration of infection. In a pair or network model, however, the rate of contact with susceptible partners is not constant. For example, if individual A is in a monogamous relationship with individual B, and B transmits

gonorrhoea to A, A will not be at risk of transmitting gonorrhoea until either (1) individual A acquires a new partner, or (2) individual B's infection resolves and they become susceptible again. If individual A is 'low risk' (serially monogamous) there would be a further delay between when infection is acquired and the relationship with B ends before condition (1) can be met. Even if individual A has multiple partners at the time of acquiring infection from B, they will not initially be at risk of transmitting infection to one of those partners (B). In a frequency-dependent model, there is thus a period following initial infection when transmission risk is exaggerated, relative to what might be expected in a more realistic pair-formation or network model. The bias that exists in a frequency-dependent model is therefore likely to be proportional to the fraction of transmission that is expected to occur within some defined early phase of infection. We calculate this early transmission fraction $E(t)$ as

$$E(t) = \frac{R(t)}{R(\infty)} = \frac{\dfrac{\ddot{}_1\left(1-\exp\left(-\left(\pm_1 + {}^3\cdot + {}^\circ\right)t\right)\right)}{\pm_1 + {}^3\cdot + {}^\circ} + \dfrac{\left(1-\ddot{}_1\right)\left(1-\exp\left(-\left(\pm_2 + {}^\circ\right)t\right)\right)}{\pm_2 + {}^\circ}}{\dfrac{\ddot{}_1}{\pm_1 + {}^3\cdot + {}^\circ} + \dfrac{1-\ddot{}_1}{\pm_2 + {}^\circ}}.$$

This can be interpreted as the fraction of all transmission that would occur within time t after acquisition of infection if the infected individual had a high rate of turnover and all sex acts were with susceptible partners. Conveniently, this expression is independent of β, and variation between individuals is therefore not important. The choice of t is arbitrary, but has been set at 26 weeks to correspond to the assumed average duration of short-term relationships, since it is the time during which the infected individual remains with the partner who infected them that determines the extent of the bias. The ρ parameter has been set to 0.00086 per week in males and 0.00101 per week in females, based on average reductions in numbers of extramarital partners per year of age [1]. Neither the network model nor the frequency-dependent model assumes that there is an exponential decay in rates of extramarital sexual activity, and neither model assumes that STI transmission is limited to extramarital relationships. The above expression should therefore be regarded as an analytic approximation to the early transmission fraction, which could be more accurately calculated if one were to account for individual-specific sexual behaviour.

2.2 Mathematical model of chlamydial infection

The model of chlamydial infection is identical in structure to that used for gonorrhoea (see Figure S5), but parameters differ. As there is substantial evidence of partial immunity to chlamydial infection following recovery [76-80], a longer average duration of immunity is assumed. Since immunity is thought to be more significant when treatment is initiated in late disease than in early disease [81-83], it is assumed that only a proportion φ_2 of those who are successfully treated acquire immunity (since treated symptomatic individuals would tend to have a shorter duration of infection than individuals who experience spontaneous resolution). The prior distributions assigned to the various parameters are shown in Table S9; these are the same as the prior distributions assumed previously [48].

Table S9: Parameters for chlamydial infection

Parameter	Symbol	Prior distribution			
		Type	Mean	SD	Ref.
% of cases that become symptomatic					
Male	φ_1	Beta	0.30	0.15	[57, 59, 62, 84]
Female		Beta	0.15	0.08	[57, 62]
Average duration if untreated (weeks)					
Symptomatic	$1/\sigma_1$	Gamma	16.0	5.0	[62, 85]
Asymptomatic	$1/\sigma_2$	Gamma	90.0	15.0	[86-88]
Average duration of immunity (weeks)	$1/\sigma_3$	Gamma	520	200	-
Proportion immune after treatment cure	φ_2	Uniform	0.50	0.29	-*
Transmission probability per act of sex					
Male-to-female	-	Beta	0.12	0.06	[65-67]
Female-to-male	-	Beta	0.16	0.10	[65-67]
Fraction of symptoms correctly treated prior to introduction of SM	A	Beta	0.70	0.10	[71-74]

* Due to the lack of evidence, a vague prior (uniform on the interval [0, 1]) is assumed.
SD = standard deviation, SM = syndromic management.

The expression for the early transmission fraction, $E(t)$, is the same as that for gonorrhoea, since the model structure is identical.

2.3 Mathematical model of trichomoniasis

The model of trichomoniasis is identical in structure to that used for gonorrhoea (see Figure S5), but parameters differ. As with gonorrhoea, there is little evidence of immunity following successful treatment of the infection [89, 90], but it is possible that individuals may be temporarily immune following the spontaneous resolution of infection. Prior distributions for the various trichomoniasis parameters are shown in Table S10; these are the same prior distributions as assumed previously [48, 50].

Table S10: Trichomoniasis parameters

Parameter	Symbol	Prior distribution			
		Type	Mean	SD	Ref.
% of cases that become symptomatic					
Male	φ_1	Beta	0.40	0.10	[58-60, 84, 91]
Female		Beta	0.30	0.10	[92, 93]
Average duration of untreated infection					
Symptomatic males (weeks)	$1/\sigma_1$	Gamma	2.0	0.7	[94, 95]
Symptomatic females (weeks)		Gamma	20.0	7.0	-
Asymptomatic males (weeks)	$1/\sigma_2$	Gamma	15.0	5.0	[91, 96]
Asymptomatic females (weeks)		Gamma	150	50.0	[97, 98]
Average duration of immunity (weeks)		Gamma	52.0	26.0	-
Proportion immune after cure	φ_2	Uniform	0.50	0.29	-*
Transmission probability per act of sex					
Male-to-female	-	Beta	0.15	0.08	[99]
Female-to-male	-	Beta	0.04	0.02	[99]
Fraction of symptoms correctly treated prior to introduction of SM	A	Beta	0.40	0.15	[71, 72, 74]

* Due to the lack of evidence, a vague prior (uniform on the interval [0, 1]) is assumed.
SD = standard deviation

The expression for the early transmission fraction, $E(t)$, is the same as that for gonorrhoea, since the model structure is identical.

2.4 Mathematical model of syphilis

The course of syphilis is different from that of other STIs, and the structure of the model for this infection is shown in Figure S6. In the absence of treatment, individuals progress through four stages of infection: a short incubation period; primary syphilis, which is associated with genital ulcers; secondary syphilis, which is associated with more generalized symptoms; and latent syphilis. Individuals who recover from syphilis remain temporarily seropositive following the resolution of infection, and are assumed to be resistant to reinfection while seropositive. The extent to which they remain seropositive depends on the stage in which the infection resolves, with greater seropositivity in individuals who recover in the later stages of disease. The rate of health seeking is assumed to be lower in secondary syphilis than in primary syphilis, by a factor of χ, because the symptoms of secondary syphilis are more difficult to recognize. In addition, the probability that treatment is successful is assumed to be 30% lower for secondary syphilis than for primary syphilis, due to the greater difficulty of diagnosing secondary syphilis symptoms and the lower efficacy of penicillin in treating syphilis of longer duration [100]. Individuals who are asymptomatic, in the incubation and latent phases, are assumed not to seek treatment, but latent infection is assumed to resolve eventually at rate σ_4. Individuals are also assumed not to be infectious during the incubation and latent phases of infection.

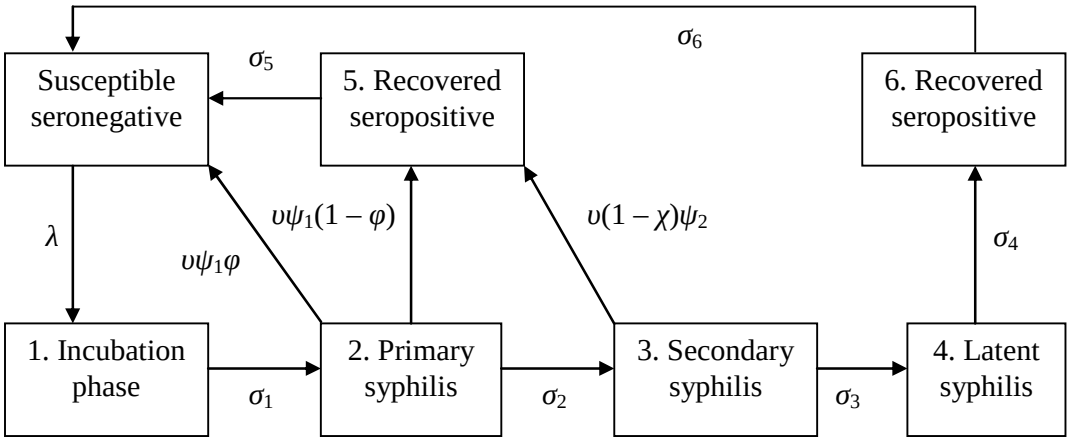

Figure S6: Model of syphilis

The prior distributions for the model parameters are summarized in Table S11. These distributions are the same as assumed previously [48], except that the mean and standard deviation of the prior on the male-to-female transmission probability have been increased to reflect high estimates from newly-identified sources [101].

Table S11: Syphilis parameters

Parameter	Symbol	Prior distribution Type	Mean	SD	Ref.
Average time (in weeks) from					
Infection to primary	$1/\sigma_1$	-	4.4*	-	[102]
Primary to secondary	$1/\sigma_2$	Gamma	6.6	2.0	[102]
Secondary to latent	$1/\sigma_3$	Gamma	15.6	4.0	[102]
Latent to spontaneous resolution	$1/\sigma_4$	Gamma	520	150	[67]
Recovery in early disease to seronegative	$1/\sigma_5$	Gamma	26.0	8.0	[103-105]
Recovery in late disease to seronegative	$1/\sigma_6$	Gamma	52.0	16.0	[103-105]
Proportion of primary cases seronegative immediately after successful treatment	φ	Beta	0.40	0.10	[106, 107]
Transmission probability per act of sex					
Male-to-female	-	Beta	0.25	0.10	[66, 101, 108, 109]
Female-to-male	-	Beta	0.15	0.05	[66, 101, 108, 109]
Fraction of symptoms correctly treated prior to introduction of syndromic management	A	Beta	0.70	0.10	[71-73, 110]
Reduction in health seeking: secondary syphilis	χ	Uniform	0.50	0.29	-

* Fixed parameter, not included in Bayesian analysis.
SD = standard deviation.

As before, we wish to estimate $R(t)$, the average number of partners an infected individual can be expected to transmit their infection to, within time t after acquiring infection, if all contacts are with susceptible partners and infected individuals transmit their infection to susceptible partners at a constant rate β. Individuals are assumed to be infectious only during the primary and secondary phases of disease. For the purpose of deriving an analytic approximation to $R(t)$, the time spent in the incubation phase is fixed at 4.4 weeks (Table S11), so that $R(t)$ is approximately equal to the average number of partners an infected individual transmits their infection to within $(t - 4.4)$ weeks after developing primary syphilis (ignoring the small probability of ceasing sexual activity during the incubation phase). Hence

$$R(t) \approx \int_{4.4}^{t} \exp\left(-\left(\pm_2 + \cdot_1 + \circ\right)(s-4.4)\right) ds + \int_{4.4}^{t}\int_{4.4}^{s} \exp\left(-\left(\pm_2 + \cdot_1 + \circ\right)(u-4.4)\right) \pm_2 \exp\left(-\left(\pm_3 + \left(1-\imath\right)\cdot_2 + \circ\right)(s-u)\right) du\, ds$$

for $t > 4.4$ weeks, where the first and second terms on the right-hand side of the equation correspond to transmission from primary and secondary syphilis respectively. From this we obtain

$$R(t) \approx \frac{1-\exp\left(-\left(\pm_2 + \cdot_1 + \circ\right)(t-4.4)\right)}{\pm_2 + \cdot_1 + \circ} + \frac{\pm_2}{\pm_2 + \cdot_1 - \pm_3 - \left(1-\imath\right)\cdot_2} \times \left[\frac{1-\exp\left(-\left(\pm_3 + \left(1-\imath\right)\cdot_2 + \circ\right)(t-4.4)\right)}{\pm_3 + \left(1-\imath\right)\cdot_2 + \circ} - \frac{1-\exp\left(-\left(\pm_2 + \cdot_1 + \circ\right)(t-4.4)\right)}{\pm_2 + \cdot_1 + \circ}\right]$$

The early transmission fraction is defined in the same way as before.

2.5 Mathematical model of genital herpes

Unlike the previously described infections, genital herpes is a chronic infection which is incurable. Although symptoms can be treated with antiviral treatment, access to antiviral treatment in South Africa has historically been very limited, and we therefore ignore treatment in this analysis. Figure S7 illustrates the model of genital herpes: the majority of newly-infected individuals are permanently asymptomatic, but a proportion φ develop a primary ulcer. This resolves at rate σ_1, but ulcers recur intermittently, at rate ξ (and resolve more rapidly, at rate σ_3). Over time these recurrences become less frequent, and we model this dynamic by transferring individuals to the 'permanently asymptomatic' state at rate σ_2.

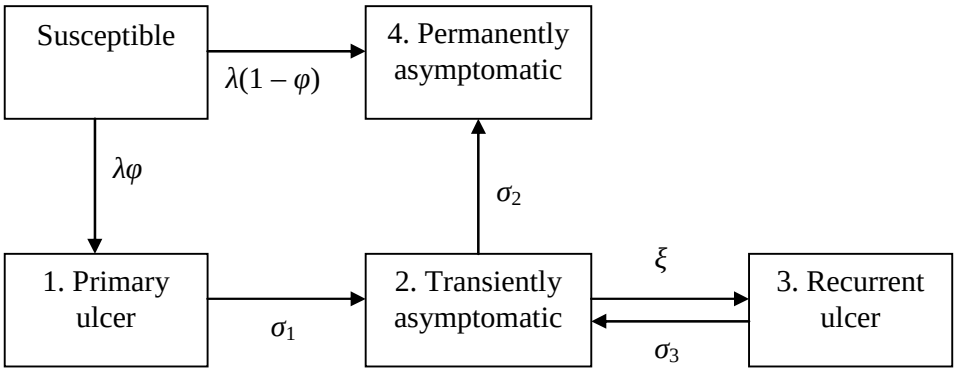

Figure S7: Model of genital herpes

Table S12 summarizes the prior distributions assumed for genital herpes parameters. It is assumed that individuals are more highly infectious when they are symptomatic than when they are asymptomatic (by a multiple Y), as the odds of detectable HSV-2 shedding in symptomatic infection is typically 10 to 20 times that in asymptomatic HSV-2 infection [111-114]. The assumed asymptomatic transmission probabilities specified in Table S12 are therefore lower than those observed in the studies cited, to make allowance for the increases in infectiousness during the symptom recurrences. It is also assumed that HSV-2 infectiousness is doubled if the infected partner is co-infected with HIV [115, 116], and that HIV co-infection increases the rate at which genital ulcers recur [11, 117]. As HIV co-infected individuals do not appear to experience reductions in ulcerative recurrences over time, it is further assumed that individuals co-infected with HIV do not progress from the transiently asymptomatic state to the permanently asymptomatic state.

Table S12: Genital herpes parameters

Parameter	Symbol	Prior distribution			
		Type	Mean	SD	Ref.
Proportion of cases becoming symptomatic	φ	Beta	0.15	0.05	[118-121]
Annual incidence of symptomatic recurrences in the transiently asymptomatic state					
Males	ξ	Gamma	6.0	1.0	[122-126]
Females		Gamma	3.0	0.5	
Average duration of primary ulcer (weeks)	$1/\sigma_1$	-	2.6*	-	[122, 123]
Average duration of recurrent ulcer (weeks)	$1/\sigma_3$	-	1.1*	-	[111, 123, 127-129]
Annual rate of transition from transiently asymptomatic to permanently asymptomatic	σ_2	Gamma	0.1	0.02	[124]
Transmission probability per act of sex with infected asymptomatic partner					
Client to sex worker	-	Beta	0.002	0.0005	[130]
Male-to-female, non-spousal relationship	-	Beta	0.0095	0.0038	[131, 132]
Female-to-male, non-spousal relationship	-	Beta	0.0065	0.0026	[131, 132]
Male-to-female, spousal relationship	-	Beta	0.0009	0.00036	[133]
Female-to-male, spousal relationship	-	Beta	0.00015	0.00006	[133]
Multiple by which transmission probability increases if infected partner is symptomatic	Y	Gamma	15	5	[111-114]

* Fixed parameter, not included in uncertainty analysis.
SD = standard deviation.

For the purpose of approximating the early transmission fraction, we will treat the 'transiently asymptomatic' and 'recurrent ulcer' states as one combined state, since the time spent symptomatic is very short relative to the time spent asymptomatic. If β is the average rate of transmission from asymptomatically-infected individuals, then the average rate of transmission from the combined state is

$$Y^* \approx Y\frac{\P}{\P+\pm_3} + \frac{\pm_3}{\P+\pm_3},$$

from which it follows that $Y^* \approx (Y\P+\pm_3)/(\P+\pm_3)$. Similarly, the average rate of transition out of the combined state (into the permanently asymptomatic state) is $\pm_2^* \approx \pm_2\pm_3/(\P+\pm_3)$. For the purpose of deriving an analytic approximation to the early transmission fraction, it is assumed that primary ulcers persist for a fixed duration of 2.6 weeks (Table S12). We thus approximate $R(t)$ as

$$R(t) \approx (1-¨)\int_0^t \exp(-°s)\,ds + ¨\left[2.6Y + \int_{2.6}^t Y^* \exp\left(-(\pm_2^*+°)(s-2.6)\right)ds + \int_{2.6}^t\int_{2.6}^s \exp\left(-(\pm_2^*+°)(u-2.6)\right)\pm_2^* \exp(-°(s-u))\,du\,ds\right]$$

for $t > 2.6$ weeks. From this it follows that

$$R(t) \approx \frac{(1-\ddot{})}{\circ}(1-\exp(-\circ t))+\ddot{} \left[2.6Y + \frac{Y^{*}-1}{\pm_2^{*}+\circ}(1-\exp(-(\pm_2^{*}+\circ)(t-2.6))) + \frac{1}{\circ}(1-\exp(-\circ(t-2.6)))\right]$$

and the early transmission fraction is defined in the same way as before.

2.6 Mathematical model of HIV

A four-stage model is used to describe the course of HIV disease in the absence of antiretroviral treatment (ART), with σ_i representing the weekly rate of transition out of stage i (in the absence of ART) and β_i representing the weekly rate of HIV transmission from infected individuals in stage i to susceptible partners. Individuals who develop AIDS-related symptoms are assumed to start ART with probability φ, but φ can increase above 1 if the number of individuals starting ART exceeds the number of individuals progressing to AIDS (Figure S8).

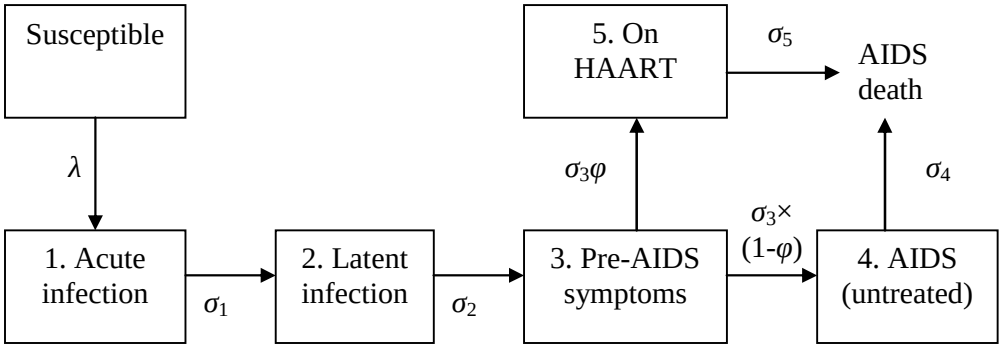

Figure S8: Model of HIV/AIDS
In cases where the ratio of ART initiates to new AIDS cases (φ) exceeds 1, the rate of transition from pre-AIDS to AIDS is set to 0.

Table S13 summarizes the prior distributions assigned to the parameters in the HIV model. The relative infectiousness during the pre-AIDS symptomatic stage is assumed to be intermediate between that in the asymptomatic stage and that in the untreated AIDS stage, with $_3/_2 = \sqrt{_4/_2}$. HIV transmission probabilities per act of unprotected sex are specified separately for short-term and long-term relationships. In addition to the priors on the biological parameters, we have specified a prior on the extent of the bias in self-reported levels of condom usage, since this has previously been shown to be an important parameter in the calibration of the deterministic model to HIV prevalence data [38].

Table S13: Prior distributions for parameters in HIV model

Parameter	Symbol	Prior distribution Type	Mean	SD	Ref.
Average time spent in absence of ART (years)					
Acute infection	$1/\sigma_1$	-	0.25†	-	[134]
Latent infection	$1/\sigma_2$	-	5.16†	-	[135, 136]
Pre-AIDS symptoms	$1/\sigma_3$	-	4.14†	-	[135, 136]
AIDS	$1/\sigma_4$	-	1.96†	-	[135, 136]
Average annual AIDS mortality after ART start	σ_5	-	0.033	-	[137]
Relative infectiousness during acute HIV	β_1/β_2	Gamma	16	6	[134, 138, 139]
Relative infectiousness during untreated AIDS	β_4/β_2	Gamma	7	2	[140]
Transmission probability: non-spousal partners*					
Client-to-sex worker	-	-	0.003†	-	[130, 141-143]
Male-to-female (ST relationships)	-	Beta	0.012	0.005	[144, 145]
Female-to-male	-	Beta	0.008	0.003	[146, 147]
Transmission probability: spousal partners*					
Male-to-female	-	Beta	0.002	0.00075	[148-150]
Female-to-male	-	Beta	0.002	0.00075	[148-150]
HIV prevalence in high risk females in 1990	V_0	Uniform	0.02	0.0087	[151, 152]

* Average across untreated disease stages, using average time spent in each stage as weights. † Fixed parameter, not included in uncertainty analysis.
SD = standard deviation.

A prior distribution is also assigned to represent the uncertainty around parameter V_0, the initial HIV prevalence in high risk females aged 15-49 in 1990 (although the simulation begins in 1985, it is convenient to start the simulation of HIV transmission in 1990 because stochastic variation in HIV trajectories is less substantial when the HIV epidemic is initialized using a higher initial HIV prevalence). Since the observed antenatal HIV prevalence in 1990 was 0.76% [151], and since HIV prevalence in the general population of women aged 15-49 tends to be lower than the antenatal prevalence [152], it is likely that the overall HIV prevalence in women aged 15-49 would not have been greater than 0.76%. Since high risk females are assumed to comprise 25% of the sexually experienced female population, and since the initial prevalence of HIV is assumed to be concentrated only in the high risk group, this implies that the prevalence of HIV in high risk females in 1990 could not have been greater than 0.76%/0.25 = 3.04%. We therefore set the prior on the V_0 parameter to be uniform on the range [0.01, 0.03). This initial HIV prevalence is adjusted by a set of scaling factors to determine the initial HIV prevalence by sex and by 5-year age group, based on the relative levels of HIV prevalence in males and females in different age groups in a 1991 survey in KwaZulu-Natal [153]. Suppose that $s_g(x)$ represents the scaling factor for high risk individuals of age x and sex g, in the high risk group, and that $v_{g,r}(x)$ represents the initial HIV prevalence (in 1990) in individuals of sex g and risk group r, who are aged x. We calculate $v_{g,r}(x)$ as

$$v_{g,r}(x) = \begin{cases} V_0 s_g(x) & \text{for } r = 1 \text{ (high risk)} \\ 0 & \text{for } r = 2 \text{ (low risk)} \end{cases}$$

The values assumed for the $s_g(x)$ scaling factors, based on the KwaZulu-Natal survey, are summarized in Table S14.

Table S14: Assumed ratios of initial HIV prevalence to average prevalence in females aged 15-49 (high risk group)

	15-19	20-24	25-29	30-34	35-39	40-44	45-49
Males (g = 0)	0.04	0.44	1.00	0.69	0.58	0.48	0.29
Females (g = 1)	1.11	1.24	1.24	0.95	0.68	0.54	0.38

As with syphilis and genital herpes, we calculate $R(t)$ by starting with the simplifying assumption that the time spent in initial infection phase is fixed at 13 weeks (Table S13) and we ignore retirement from the sexually active population during this initial phase. Then for $t > 13$ weeks

$$
\begin{aligned}
R(t) \approx\ & {}_1 \times 13 + {}_2 \int_{13}^{t} \exp\left(-\left(\circ + \pm_2\right)(s-13)\right) ds \\
& + {}_3 \int_{13}^{t} \int_{13}^{s} \exp\left(-\left(\circ + \pm_2\right)(u-13)\right) \pm_2 \exp\left(-\left(\circ + \pm_3\right)(s-u)\right) du\, ds \\
& + {}_4 \int_{13}^{t} \int_{13}^{s} \int_{13}^{u} \exp\left(-\left(\circ + \pm_2\right)(v-13)\right) \pm_2 \exp\left(-\left(\circ + \pm_3\right)(u-v)\right) \pm_3 \\
& \qquad \times \exp\left(-\left(\circ + \pm_4\right)(s-u)\right) dv\, du\, ds \\
=\ & {}_1 \times 13 + \frac{{}_2}{\circ + \pm_2}\left(1 - \exp\left(-\left(\circ + \pm_2\right)(t-13)\right)\right) \\
& + \frac{{}_3 \pm_2}{\pm_2 - \pm_3}\left[\frac{1 - \exp\left(-\left(\circ + \pm_3\right)(t-13)\right)}{\circ + \pm_3} - \frac{1 - \exp\left(-\left(\circ + \pm_2\right)(t-13)\right)}{\circ + \pm_2}\right] \\
& + \frac{{}_4 \pm_2 \pm_3}{\pm_2 - \pm_3}\left[\left(\frac{1}{\pm_3 - \pm_4} - \frac{1}{\pm_2 - \pm_4}\right)\frac{1 - \exp\left(-\left(\circ + \pm_4\right)(t-13)\right)}{\circ + \pm_4}\right. \\
& \qquad \left. - \frac{1 - \exp\left(-\left(\circ + \pm_3\right)(t-13)\right)}{\left(\pm_3 - \pm_4\right)\left(\circ + \pm_3\right)} + \frac{1 - \exp\left(-\left(\circ + \pm_2\right)(t-13)\right)}{\left(\pm_2 - \pm_4\right)\left(\circ + \pm_2\right)}\right]
\end{aligned}
$$

if ART initiation is ignored.

2.7 Bacterial vaginosis

Following the scoring method proposed by Nugent *et al* [154], women are categorized into one of three groups according the relative abnormality of their vaginal flora: normal (score of 3 or less), intermediate (score of 4 to 6) or bacterial vaginosis (score of 7 to 10). In addition, women with bacterial vaginosis are divided between symptomatic and asymptomatic states. These different states and the possible movements between states are illustrated in Figure S9. As bacterial vaginosis is difficult to treat effectively, the model allows for both complete cure (return to the "normal vaginal flora" state) and partial cure (return to the "intermediate vaginal flora" state), following either symptom treatment or screening interventions. Initial estimates of the rates of transition between states (unrelated to treatment) were obtained by fitting a Markov chain model to data on changes in vaginal flora patterns in untreated women [155].

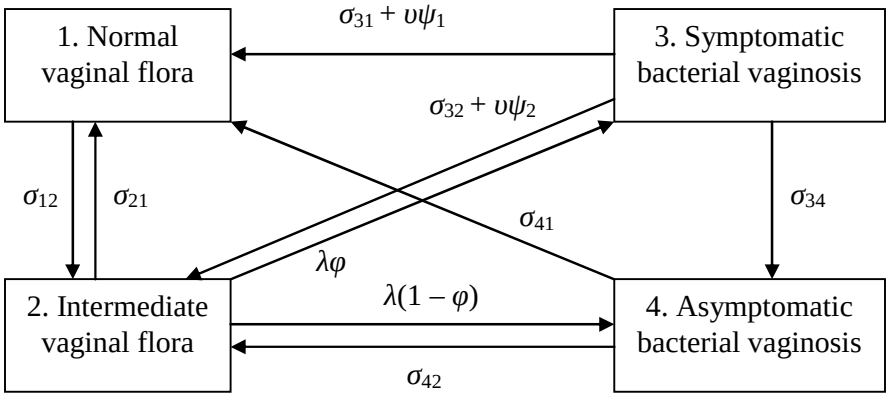

Figure S9: Model of bacterial vaginosis

Table S15 summarizes the bacterial vaginosis parameters, and the data sources on which these are based. Although bacterial vaginosis is generally not regarded as an STI, there is strong evidence to suggest that women who have multiple partners experience bacterial vaginosis more frequently than women who are monogamous [156-160], and that virgins are significantly less susceptible to bacterial vaginosis than women who are sexually experienced [160, 161]. It is therefore assumed that the rate at which women progress from the intermediate state to the bacterial vaginosis state is doubled if they currently have more than one partner or are engaging in commercial sex, and is halved if they currently have no sexual partner.

Table S15: Bacterial vaginosis parameters

Parameter	Symbol	Value	Ref.
Proportion of cases that become symptomatic	φ_1	0.25	[162, 163]
Weekly rates of transition			
Bacterial vaginosis to normal flora	σ_{31}, σ_{41}	0.008	[155]
Bacterial vaginosis to intermediate flora	σ_{32}, σ_{42}	0.051	[155]
Normal flora to intermediate flora	σ_{12}	0.030	[155]
Intermediate flora to normal flora	σ_{21}	0.069	[155]
Intermediate flora to bacterial vaginosis*	λ	0.100	[155]

* Applies to women who currently have one sexual partner. The rate is doubled in women with multiple partners and halved in women who have no partner.

2.8 Vaginal candidiasis

Vaginal candidiasis is caused by various *Candida* species, which inhabit the vaginal mucosa either as commensals or as pathogens [164]. To reflect the dual nature of the infection, the model divides women infected with *Candida* into 'asymptomatic candidiasis' and 'symptomatic candidiasis' states (Figure S10). Some *Candida* colonies may persist for long periods of time, even after appropriate treatment, with colonies being too small to be detected by culture methods, and it has been suggested that this may account for the frequent 'relapse' of symptoms after treatment in women with recurrent vaginal candidiasis [165-167]. The

model therefore assumes that 'asymptomatic candidiasis' is an intermediate state between 'uninfected' and 'symptomatic candidiasis'. As with bacterial vaginosis, symptomatic vaginal candidiasis is difficult to cure effectively, and the model therefore allows for both complete cure (return to the 'uninfected' state) and partial cure (return to the 'asymptomatic candidiasis' state).

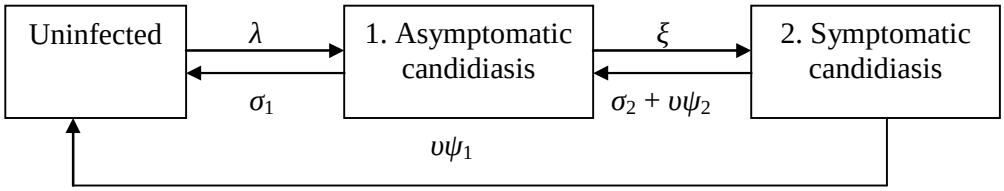

Figure S10: Model of vaginal candidiasis

Table S16 summarizes the vaginal candidiasis parameters, and the data sources on which these are based. Because vaginal candidiasis is rare in women who are not of reproductive age [167-169], and because the prevalence of candidiasis is particularly high during pregnancy [170, 171], the incidence of vaginal candidiasis is assumed to be proportional to women's age-specific fertility rates. The λ parameter in Table S16 relates to HIV-negative women aged 15 to 19, and this incidence rate is scaled up or down, in proportion to the age-specific fertility rates, in order to produce the asymptomatic candidiasis incidence rates at other ages. The λ parameter in Table S16 is also multiplied by a factor of 1.5 in HIV-positive women who have pre-AIDS symptoms, and by a factor of 2 in women who have untreated AIDS, to allow for observed increases in the incidence of vaginal candidiasis in the more advanced stages of HIV infection [11, 172-175].

Table S16: Vaginal candidiasis parameters

Parameter	Symbol	Value	Ref.
Average duration of symptoms (weeks)	$1/\sigma_2$	12.0	[176]
Average time to clearance of asymptomatic infection (weeks)	$1/\sigma_1$	26.0	[170]
Annual incidence of asymptomatic infection	λ	0.80	[172, 177, 178]
Annual incidence of symptoms	ξ	0.52	[170, 179-181]

2.9 STI treatment

The modelling of STI treatment has been described previously [49]. In the case of genital ulcer and discharge symptoms, it is assumed that the rate of health seeking (υ) depends on the individual's age and sex: for adults aged 20 and older, the weekly rate of seeking treatment is 0.23 in women and 0.57 in men [17, 182-184], and these rates are halved in adolescents [185, 186]. In sex workers the weekly rate of health seeking is assumed to be 0.9 [17, 186].

Rates of STI cure are determined by defining the following symbols:

$\cdot\ {}^{d}_{g}(t)$ = probability that an individual of sex *g*, experiencing symptoms of disease *d*, is cured if they seek treatment at time *t*

$r_{g,h}$ = % of individuals of sex *g* who seek STI treatment in health sector *h* (*h* can take on values of 0, 1 or 2, corresponding to the public health sector, formal private health sector and traditional healers respectively)

$\Omega_h(t)$ = % of health workers in sector *h* who correctly follow syndromic management protocols at time *t* (*h* = 0 or 1 only)

A_g^d = % of individuals of sex *g*, with symptoms of STI *d*, who receive effective treatment if the health worker is *not* following syndromic management protocols (ignoring the potential effect of drug shortages)

Z_g^d = % of individuals of sex *g*, with symptoms of STI *d*, who receive effective treatment if the health worker is following syndromic management protocols (ignoring the potential effect of drug shortages)

$V(t)$ = % reduction in the probability of cure in public STI clinics as a result of drug shortages, at time *t*

s_A^d = probability that STI *d* is cured if treated with effective drugs

s_T^d = probability that STI *d* is cured if treated by a traditional healer

The probability of cure is then calculated as:

$$\cdot\,{}_g^d(t) = \left[r_{g,0}\left(\left(1-\Omega_0(t)\right)A_g^d + \Omega_0(t)Z_g^d\right)\left(1-V(t)\right) + r_{g,1}\left(\left(1-\Omega_1(t)\right)A_g^d + \Omega_1(t)Z_g^d\right)\right]s_A^d + r_{g,2}s_T^d$$

This is the weighted average probability of cure in the different health sectors, where the weights are the proportions of individuals seeking treatment in each sector, $r_{g,h}$. In the case of bacterial vaginosis and vaginal candidiasis, the $\cdot\,{}_g^d(t)$, s_A^d and s_T^d symbols are modified slightly by adding a subscript after the first to indicate whether cure is complete (1) or only partial (2), as explained previously.

Of men who seek treatment, 45% are assumed to seek treatment in the public health sector, 40% seek treatment in the formal private sector and the remaining 15% seek treatment from traditional healers [17, 182, 187-189]. For women, the corresponding proportions are 60%, 30% and 10% respectively. For the formal health sectors, probabilities of effective treatment being provided prior to syndromic management (A_g^d) are difficult to determine precisely, and these parameters have therefore been assigned prior distributions to represent the associated uncertainty (see Tables S8-S11). Probabilities of cure, if effective treatment is provided, have been set to 90% for most STIs [49]. For the informal health sector, treatment is assumed not to be effective (ς_T =0).

The time-varying parameters are summarized in Table S17. Rates of ART initiation are the same as those assumed previously [190], and due to lack of recent data have been assumed to remain at 2010 levels after 2010. In the case of gonorrhea, probabilities of treatment success have been adjusted to take into account rising levels of ciprofloxacin resistance in recent years [191-193]: the treatment effectiveness parameter (ς_A) is reduced in proportion to the product of the fraction of gonorrhoea cases treated with ciprofloxacin and the fraction of gonorrhoea cases that are ciprofloxacin-resistant (Table S17). This adjustment ceases to apply

after 2008, when syndromic management guidelines were revised to recommend the use of ceftriaxone in place of ciprofloxacin [194].

Table S17: Time-varying treatment parameters

Year	% of providers correctly using syndromic management protocols		% of public clinics with STI drug shortages	Source	Ratio of new ART patients to new AIDS cases	% of gonorrhoea cases that are ciprofloxacin-resistant	% of gonorrhoea cases that are treated with ciprofloxacin
	Private	Public					
1993	0%	0%	20%		0.000	0%	50%
1994	3%	10%	20%		0.000	0%	50%
1995	7%	30%	18%		0.000	0%	50%
1996	11%	50%	16%	[195][a]	0.000	0%	50%
1997	15%	65%	13%	[196][b]	0.000	0%	50%
1998	18%	75%	10%	[197][a]	0.000	0%	50%
1999	21%	78%	8%	[74][b]	0.000	0%	50%
2000	23%	80%	6%		0.035	0%	50%
2001	25%	80%	5%	[198][b]	0.046	0%	50%
2002	27%	80%	4%	[199][a]	0.048	0%	50%
2003	29%	80%	4%	[200][a]	0.085	4%	50%
2004	31%	80%	4%		0.220	10%	50%
2005	33%	80%	4%		0.402	16%	50%
2006	35%	80%	4%		0.480	25%	50%
2007	37%	80%	4%		0.650	38%	50%
2008	39%	80%	4%		1.004	55%	50%
2009	41%	80%	4%		1.290	70%	0%
2010	43%	80%	4%		1.656	82%	0%
2011	45%	80%	4%		1.656	90%	0%
2012	47%	80%	4%		1.656	96%	0%

[a] Public health sector. [b] Private health sector.

In the first analysis presented in the main text, where endemic STI prevalence levels are estimated on the assumption of no change to the behavioural and treatment assumptions from the baseline values in 1985, it is assumed that no STIs are treated according to syndromic management guidelines ($\Omega_h(t) = 0$), and all other time-varying parameters are held constant at the values in the first row of Table S17.

3. Modelling fitting procedure

The models are fit separately for each STI, using the sexual behaviour assumptions described previously. A disadvantage of fitting the same model of sexual behaviour to multiple STI prevalence data sources is that the system is more heavily constrained when the same sexual behaviour assumptions have to be used for all STIs, and this makes calibration challenging. However, the advantage of fitting the same model of sexual behaviour to multiple STI prevalence data sources is that data specific to one STI can provide insights into sexual behaviour that might not be obvious when calibrating the model for other STIs, and this can lead to improved confidence in the sexual behaviour assumptions. For example, patterns of HIV prevalence by age and sex were important in determining relative levels of sexual risk behaviour at different ages in men and women, when calibrating an earlier version of the frequency-dependent model [1]. Calibration of the frequency-dependent model to HIV prevalence trends at young ages has also suggested that self-reported levels of condom usage

are likely to overstate actual levels of condom use [38]. In the current analysis, it was found that the network model could not match the high levels of gonorrhoea and syphilis prevalence observed in South Africa unless the assumed rate of commercial sex activity was increased substantially (in line with a recent study [23]). The sections that follow describe the methods and data sources employed in fitting the models for each STI.

3.1 HIV

The approach adopted in defining the likelihood for the HIV data is similar to that described previously [38], but updated to include more recent HIV prevalence data. We consider first the likelihood in respect of the antenatal survey HIV prevalence data [201], for the period 1997-2012 and for 5-year age groups 15-19, …, 35-39. Suppose that $H_{x,t}(\boldsymbol{\theta})$ is the model estimate of HIV prevalence in pregnant women aged x to $x + 4$, in year t, where the vector $\boldsymbol{\theta}$ represents the values of the model input parameters. The corresponding prevalence of HIV actually measured in the antenatal survey is represented by $y_{x,t}$. It is assumed that if $\boldsymbol{\theta}$ is the true set of parameter values, then the difference between the logit-transformed model estimate and the logit-transformed observed prevalence is normally distributed. The mean of this normal distribution represents the extent of antenatal bias, which could arise due to various sampling biases. The variance of the distribution is assumed to be composed of a 'model error' term (representing model error in the estimation of prevalence), and a 'survey error' term (representing the uncertainty around the survey estimate due to binomial variation and cluster variation in the survey). More formally, it is assumed that

$$\log\left(\frac{y_{x,t}}{1-y_{x,t}}\right) = \log\left(\frac{H_{x,t}(\boldsymbol{\theta})}{1-H_{x,t}(\boldsymbol{\theta})}\right) + b + m_{x,t} + £_{x,t},$$

where b is the antenatal bias parameter, $m_{x,t} \sim N(0, \pm^2_m)$ and $£_{x,t} \sim N(0, \pm^2_{x,t})$. The latter two terms represent the model error and the survey error respectively. The logit transformations ensure that the error terms are closer to normality and that the model error terms are roughly independent of the level of HIV prevalence. For a given parameter combination $\boldsymbol{\theta}$, the mean of the antenatal bias parameter in 1997 and subsequent years is estimated using the formula

$$\hat{b} = \frac{1}{80}\sum_{x}\sum_{t=1997}^{2012}\left(\log\left(\frac{y_{x,t}}{1-y_{x,t}}\right) - \log\left(\frac{H_{x,t}(\boldsymbol{\theta})}{1-H_{x,t}(\boldsymbol{\theta})}\right)\right).$$

Antenatal data collected prior to 1997 are not included in the likelihood definition, in part because the antenatal sampling protocol prior to 1997 was not designed to be nationally representative, and in part because 95% confidence intervals were incorrectly calculated in the early antenatal surveys (not accounting for clustering of observations by clinic). The $\pm^2_{x,t}$ values are estimated from the 95% confidence intervals that have been published for the post-1996 survey estimates. The $\pm^2_m$ parameter is estimated as

$$\pounds_m^2 = \frac{1}{80}\sum_x\sum_t\left(\log\left(\frac{y_{x,t}}{1-y_{x,t}}\right)-\log\left(\frac{H_{x,t}(\boldsymbol{\theta})}{1-H_{x,t}(\boldsymbol{\theta})}\right)-\hat{b}\right)^2-\pm_{x,t}^2,$$

subject to a minimum of zero. The likelihood in respect of the antenatal data is calculated based on the assumption that the error terms are normally distributed:

$$L(\mathbf{y}\mid\boldsymbol{\varphi})=\prod_x\prod_t\left(2\circledR\left(\pounds_m^2+\pm_{x,t}^2\right)\right)^{-0.5}\exp\left[-\frac{\left(\text{logit}\left(y_{x,t}\right)-\text{logit}\left(H_{x,t}(\boldsymbol{\theta})\right)-\hat{b}\right)^2}{2\left(\pounds_m^2+\pm_{x,t}^2\right)}\right],$$

where **y** represents the matrix of $y_{x,t}$ values, across age bands 15-19 to 35-39, and across calendar years 1997 to 2012.

We define a likelihood in respect of the HSRC household survey HIV prevalence data [5, 46, 202] using a similar approach to that adopted for the antenatal survey data. The most important difference is that the household survey bias term is excluded (on the assumption that household surveys provide unbiased estimates). The likelihood is calculated separately for 2005, 2008 and 2012, for males and females, and for each 5-year age band from 15-19 up to 55-59. Although a household survey was also conducted in 2002 and 2003, these data have been excluded as they were obtained using a single saliva test with no confirmatory testing, and survey response rates were relatively low [44].

For the purpose of the model calibration, we randomly generate a set of 100 000 parameter combinations by sampling from the prior distributions in Table S13. (This sample is larger than the sample of 20 000 chosen for other STIs because the larger amount of HIV data means that the likelihood function is much more sharply 'peaked', and hence a larger sample is needed to identify a set of parameters with similarly high likelihood values.) The likelihood function (the products of the likelihood values in respect of the antenatal and HSRC data) is calculated for each of the 100 000 parameter combinations, and the 100 parameter combinations with the highest likelihood values are analysed further. This analysis is performed separately for the network and frequency-dependent models.

3.2 Genital herpes, syphilis, gonorrhoea, chlamydia and trichomoniasis

The approach to defining the likelihood for STIs other than HIV is the same as that described previously [48], with some modifications to the data sources used. Briefly, a beta-binomial distribution is used to model the observed variation in the fraction of individuals testing positive for the STI of interest in different studies. The beta distribution parameters are set to take into account both (a) the expected variation in the true STI prevalence between different studies populations and (b) the expected variation in the performance (sensitivity and specificity) of the diagnostic method used in the study of interest. Although we previously specified a prior distribution to represent the variance in respect of (a), we have fixed these variance parameters at the posterior means estimated previously [48] for the purpose of the present analysis.

The data sources on which the likelihood values are based are summarized in Tables S18-S22. These are the same data sources as considered previously [48], except that (a) data from the period prior to 1990 have been excluded, and (b) the systematic review of STI prevalence data has been updated to identify more recent STI prevalence data. Data from the period prior to 1990 have been excluded because the simulation starts in 1985 and a 5-year 'burn-in' was deemed necessary in order to remove dependence on the assumed baseline STI prevalence levels.

Table S18: Syphilis prevalence estimates

Study	Year	Sample	Location	n	Prev.	Diagnostic
Coetzee [203]	1990-2	ANC	Cape Town	1973	5.2%	Non-trep. + trep.
Opai-Tetteh *et al* [204]	-	ANC	Durban	200	11.0%	Non-trep. + trep.
Bam *et al* [205]	1990	ANC	Bloemfontein	971	15.7%	Non-trep. + trep.
Hoosen *et al* [206]	-	ANC	Durban	32	12.0%	Non-trep. + trep.
Qolohle *et al* [207]	1993	ANC	Durban	363	9.4%	Non-trep. + trep.
Govender *et al* [208]	1994	ANC	Durban	168	12.0%	Non-trep. + trep.
Kharsany *et al* [209]	1994	ANC	Durban	52	26.9%	Non-trep.
Sturm *et al* [210]	1995	ANC	Hlabisa	327	12.0%	Non-trep. + trep.
Sturm *et al* [211]	1996	ANC	Hlabisa	327	8.4%	Non-trep. + trep.
Mashiane *et al* [212]	1997	ANC	Pretoria	3000	12.4%	Non-trep. + trep.
Dawadi *et al* [213]	1997-8	ANC	Hewu	271	8.5%	Non-trep.
Myer *et al* [214]	1998-2000	ANC	Hlabisa	7391	7.5%	Non-trep.
Sturm *et al* [210]	1999	ANC	Hlabisa	245	6.0%	Non-trep. + trep.
	2002	ANC	Hlabisa	449	2.0%	Non-trep. + trep.
Bronzan *et al* [215]	2001-2	ANC	Eastern Cape	1250	6.3%	Non-trep. + trep.
Mothupi *et al* [216]	2002	ANC	Johannesburg	16537	1.0%	Non-trep. + trep.
Frohlich *et al* [217]	2002	ANC	Vulindlela	39	2.6%	Non-trep. + trep.
Sebitloane *et al* [218]	2003-5	ANC	Durban	801	5.2%	Non-trep.
Dinh *et al* [219]	2005-6	ANC	Gauteng	982	4.0%	Non-trep.
			Northern Cape	1026	5.8%	Non-trep.
Devjee *et al* [220]	2005	ANC	Durban	1856	5.4%	Non-trep. + trep.
Perti *et al* [221]	-	ANC	Johannesburg	390	1.3%	Non-trep.
Ramjee *et al* [130]	1996-2000	CSW	KZN	395	31.4%	Non-trep. + trep.
Steen *et al* [222]	1996-7	CSW	Virginia	407	33.8%	Non-trep. + trep.
Dunkle *et al* [27]	1996-7	CSW	Johannesburg	295	25.6%	Non-trep. + trep.
Williams *et al* [17]	1998	CSW	Khutsong	121	23.3%	Non-trep. + trep.
Williams *et al* [223]	2000	CSW	Khutsong	93	34.4%	Non-trep. + trep.
Vickerman *et al* [224]	2000	CSW	Johannesburg	310	11%	Non-trep.
Ndhlovu *et al* [189]	2001	CSW	Khutsong	101	21.0%	Non-trep. + trep.
Schneider *et al* [225]	1994	FPC	Bushbuck-ridge	249	5.0%	Non-trep. + trep.
Wilkinson *et al* [163]	-	FPC	Hlabisa	189	8.0%	Non-trep. + trep.
Kharsany *et al* [209]	1994	FPC	Durban	55	21.8%	Non-trep.
Hoosen *et al* [226]	-	FPC	Durban	40	8.0%	Non-trep. + trep.
Fehler *et al* [227]	-	FPC	Johannesburg	210	8.6%	Non-trep. + trep.
Frohlich *et al* [217]	2002	FPC	Vulindlela	221	2.3%	Non-trep. + trep.
Cronje *et al* [228]	-	HH, F 20-49	Urban FS	403	15.5%	Non-trep. + trep.
		HH, F 20-49	Rural FS	465	12.3%	Non-trep. + trep.
Colvin *et al* [229]	1995	HH, F 15-49	Hlabisa	142	8.5%	Non-trep. + trep.
Williams *et al* [17]	1998	HH, F 15-59	Khutsong	712	9.7%	Non-trep. + trep.
Auvert *et al* [144]	1999	HH, F 15-24	Khutsong	622	4.5%	Non-trep. + trep.
Colvin *et al* [229]	1995	HH, M 15-49	Hlabisa	86	9.3%	Non-trep. + trep.
Williams *et al* [17]	1998	HH, M 15-59	Khutsong	475	6.1%	Non-trep. + trep.
Auvert *et al* [144]	1999	HH, M 15-24	Khutsong	560	1.8%	Non-trep. + trep.
Williams *et al* [223]	2000	HH, M 15-19	Khutsong	606	8.1%	Non-trep. + trep.
Ndhlovu *et al* [189]	2001	HH, M 15-59	Khutsong	532	5.0%	Non-trep. + trep.
Auvert *et al* [230]	2002	HH, M 15-49	Orange Farm	438	3.2%	Non-trep. + trep.

ANC = antenatal clinic attenders. CSW = commercial sex workers. F = females. FPC = family planning clinic attenders. FS = Free State. HH = households. KZN = KwaZulu-Natal. M = males. Non-trep. = non-treponemal assay. Prev. = prevalence. Trep. = treponemal assay.

Table S19: Gonorrhoea prevalence estimates

Study	Year	Sample	Location	n	Prev.	Diagnostic
Hoosen *et al* [206]	-	ANC	Durban	32	6.0%	Culture
Govender *et al* [208]	1994-5	ANC	Durban	168	3.0%	Culture
Kharsany *et al* [209]	1994	ANC	Durban	52	5.8%	Culture
Rours *et al* [231]	1996-7	ANC	Johannesburg	766	8.5%	LCR on urine
Sturm *et al* [211]	1996	ANC	Hlabisa	327	7.8%	Culture
Sturm *et al* [210]	1999	ANC	Hlabisa	245	7.0%	PCR on swabs
Sturm *et al* [232]	-	ANC	Hlabisa	185	7.6%	-*
Sturm *et al* [210]	2002	ANC	Hlabisa	449	4.0%	PCR on swabs
Odendaal *et al* [233]	2002	ANC	Cape Town	343	0.9%	Culture
Frohlich *et al* [217]	2002	ANC	Vulindlela	48	4.2%	SDA on swabs
Moodley *et al* [234]	2008-10	ANC	Durban	1459	6.4%	SDA on swabs
Ramjee *et al* [130]	1996-2000	CSW	KZN	387	10.3%	Culture
Steen *et al* [222]	1996-7	CSW	Virginia	407	17.3%	LCR on urine
Dunkle *et al* [27]	1996-7	CSW	Johannesburg	295	23.3%	LCR on urine
Williams *et al* [17]	1998	CSW	Khutsong	121	15.7%	LCR on urine
Williams *et al* [223]	2000	CSW	Khutsong	93	16.1%	LCR on urine
Vickerman *et al* [224]	2000	CSW	Johannesburg	310	25%	LCR on urine
Ndhlovu *et al* [189]	2001	CSW	Khutsong	101	10.0%	LCR on urine
Schneider *et al* [225]	1994	FPC	Bushbuckridge	249	3.0%	LCR on urine
Wilkinson *et al* [163]	-	FPC	Hlabisa	189	4.0%	Culture
Kharsany *et al* [209]	1994	FPC	Durban	55	5.5%	Culture
Hoosen *et al* [226]	-	FPC	Durban	40	5.0%	Culture
Fehler *et al* [227]	-	FPC	Johannesburg	210	8.6%	LCR on urine
Kleinschmidt *et al* [235]	1999-2001	FPC	Orange Farm	538	3.9%	LCR on urine
Frohlich *et al* [217]	2002	FPC	Vulindlela	226	6.6%	SDA on swabs
Colvin *et al* [229]	1995	HH, F 15-49	Hlabisa	137	5.8%	LCR on urine
Williams *et al* [17]	1998	HH, F 15-59	Khutsong	712	6.9%	LCR on urine
Auvert *et al* [144]	1999	HH, F 15-24	Khutsong	622	10.9%	LCR on urine
Williams *et al* [223]	2000	HH, F 15-49	Khutsong	893	8.6%	LCR on urine
Ndhlovu *et al* [189]	2001	HH, F 15-59	Khutsong	878	11.0%	LCR on urine
Pettifor *et al* [236]	2002-3	HH, F 15-19	Peri-urban	2624	3.5%	PCR on urine
		HH, F 20-24	townships	2002	3.5%	PCR on urine
Hurkchand *et al* [237]	2002	HH, F 20-49	Mbalenhle	399	4.7%	PCR on urine
O'Leary *et al* [238]	2010	HH, F 15-19	Eastern Cape	329	10.6%	Aptima on urine
Colvin *et al* [229]	1995	HH, M 15-49	Hlabisa	85	2.4%	LCR on urine
Williams *et al* [17]	1998	HH, M 15-59	Khutsong	475	3.4%	LCR on urine
Auvert *et al* [144]	1999	HH, M 15-24	Khutsong	560	2.9%	LCR on urine
Williams *et al* [223]	2000	HH, M 15-49	Khutsong	606	3.3%	LCR on urine
Ndhlovu *et al* [189]	2001	HH, M 15-59	Khutsong	532	4.0%	LCR on urine
Pettifor *et al* [236]	2002-3	HH, M 15-19	Peri-urban	2389	1.1%	PCR on urine
		HH, M 20-24	townships	1455	3.2%	PCR on urine
Hurkchand *et al* [237]	2002	HH, M 20-49	Mbalenhle	291	3.9%	PCR on urine
O'Leary *et al* [238]	2010	HH, M 15-19	Eastern Cape	330	1.8%	Aptima on urine

* Diagnosed by culture and a series of genetic tests, which in combination would have had very high sensitivity and specificity.

ANC = antenatal clinic attenders. CSW = commercial sex workers. F = females. FPC = family planning clinic attenders. HH = households. KZN = KwaZulu-Natal. LCR = ligase chain reaction test. M = males. PCR = polymerase chain reaction test. Prev. = prevalence. SDA = strand displacement amplification.

Table S20: Chlamydial infection prevalence estimates

Study	Year	Sample	Location	n	Prev.	Diagnostic
Hoosen *et al* [206]	-	ANC	Durban	32	41.0%	DIF
Kharsany *et al* [209]	1994	ANC	Durban	52	19.2%	DIF
Rours *et al* [231]	1996-7	ANC	Johannesburg	766	12%	LCR on urine
Sturm *et al* [211]	1996	ANC	Hlabisa	327	12.9%	DIF
Sturm *et al* [210]	1999	ANC	Hlabisa	245	11.0%	PCR on swabs
Sturm *et al* [232]	-	ANC	Hlabisa	185	13.5%	-*
Sturm *et al* [210]	2002	ANC	Hlabisa	449	11.0%	PCR on swabs
Odendaal *et al* [233]	2002	ANC	Cape Town	343	11.7%	PCR on swabs
Frohlich *et al* [217]	2002	ANC	Vulindlela	48	8.3%	SDA on swabs
Govender *et al* [239]	2005	ANC	Cape Town	219	18.7%	PCR on swabs
Moodley *et al* [234]	2008-10	ANC	Durban	1459	17.8%	SDA on swabs
Ramjee *et al* [26]	1996-7	CSW	KZN	145	16.4%	DIF
Steen *et al* [222]	1996-7	CSW	Virginia	407	14.3%	LCR on urine
Dunkle *et al* [27]	1996-7	CSW	Johannesburg	295	8.4%	LCR on urine
Williams *et al* [17]	1998	CSW	Khutsong	121	9.1%	LCR on urine
Williams *et al* [223]	2000	CSW	Khutsong	93	12.9%	LCR on urine
Vickerman *et al* [224]	2000	CSW	Johannesburg	310	17%	LCR on urine
Ndhlovu *et al* [189]	2001	CSW	Khutsong	101	8.0%	LCR on urine
Schneider *et al* [225]	1994	FPC	Bushbuckridge	249	12.0%	LCR on urine
Wilkinson *et al* [163]	-	FPC	Hlabisa	189	8.0%	DIF
Kharsany *et al* [209]	1994	FPC	Durban	55	12.7%	DIF
Hoosen *et al* [226]	-	FPC	Durban	40	15.0%	DIF
Fehler *et al* [227]	-	FPC	Johannesburg	210	18.1%	LCR on urine
Kleinschmidt *et al* [235]	1999-2001	FPC	Orange Farm	539	14.1%	LCR on urine
Frohlich *et al* [217]	2002	FPC	Vulindlela	226	8.8%	SDA on swabs
Colvin *et al* [229]	1995	HH, F 15-49	Hlabisa	140	6.4%	LCR on urine
Williams *et al* [17]	1998	HH, F 15-59	Khutsong	712	8.1%	LCR on urine
Auvert *et al* [144]	1999	HH, F 15-24	Khutsong	622	14.6%	LCR on urine
Williams *et al* [223]	2000	HH, F 15-49	Khutsong	893	13.8%	LCR on urine
Ndhlovu *et al* [189]	2001	HH, F 15-59	Khutsong	878	12.0%	LCR on urine
Auvert et al [230]	2002	HH, F 15-49	Orange Farm	492	6.9%	PCR on urine
Pettifor *et al* [236]	2002-3	HH, F 15-19	Semi-urban	2624	9.1%	PCR on urine
		HH, F 20-24	townships	2002	10.8%	PCR on urine
Hurkchand *et al* [237]	2002	HH, F 20-49	Mbalenhle	399	6.5%	PCR on urine
O'Leary *et al* [238]	2010	HH, F 15-19	Eastern Cape	329	23.1%	Aptima on urine
Colvin *et al* [229]	1995	HH, M 15-49	Hlabisa	90	5.6%	LCR on urine
Williams *et al* [17]	1998	HH, M 15-59	Khutsong	475	5.2%	LCR on urine
Auvert *et al* [144]	1999	HH, M 15-24	Khutsong	560	4.8%	LCR on urine
Williams *et al* [223]	2000	HH, M 15-49	Khutsong	606	12.4%	LCR on urine
Ndhlovu *et al* [189]	2001	HH, M 15-59	Khutsong	532	7.0%	LCR on urine
Auvert et al [230]	2002	HH, M 15-49	Orange Farm	438	6.2%	PCR on urine
Pettifor *et al* [236]	2002-3	HH, M 15-19	Semi-urban	2389	3.5%	PCR on urine
		HH, M 20-24	townships	1455	10.1%	PCR on urine
Hurkchand *et al* [237]	2002	HH, M 20-49	Mbalenhle	291	8.2%	PCR on urine
O'Leary *et al* [238]	2010	HH, M 15-19	Eastern Cape	330	8.2%	Aptima on urine

* Diagnosed by culture and a series of genetic tests, which in combination would have had very high sensitivity and specificity.

ANC = antenatal clinic attenders. CSW = commercial sex workers. DIF = direct immunofluorescence. F = females. FPC = family planning clinic attenders. HH = households. KZN = KwaZulu-Natal. LCR = ligase chain reaction test. M = males. PCR = polymerase chain reaction test. Prev. = prevalence. SDA = strand displacement amplification.

Table S21: Trichomoniasis prevalence estimates

Study	Year	Sample	Location	n	Prev.	Diagnostic
Hoosen *et al* [206]	-	ANC	Durban	32	19.0%	Wet mount
Govender *et al* [208]	1994-5	ANC	Durban	168	21.0%	Wet mount
Kharsany *et al* [209]	1994	ANC	Durban	52	51.9%	Culture
Funk *et al* [240]	1995	ANC	Pretoria	798	12.3%	Wet mount
Sturm *et al* [211]	1996	ANC	Hlabisa	327	41.4%	Culture
Sturm *et al* [210]	1999	ANC	Hlabisa	245	32.0%	PCR on swabs
Sturm *et al* [232]	2001	ANC	Hlabisa	185	36.8%	-*
Sturm *et al* [210]	2002	ANC	Hlabisa	449	27.0%	PCR on swabs
Frohlich *et al* [217]	2002	ANC	Vulindlela	48	20.8%	PCR on swabs
Sebitloane *et al* [218]	2003-5	ANC	Durban	801	10.7%	Culture
Moodley *et al* [234]	2008-10	ANC	Durban	1459	15.3%	PCR on swabs
Ramjee *et al* [130]	1996-2000	CSW	KZN	392	35.7%	Wet mount
Dunkle *et al* [27]	1996-7	CSW	Johannesburg	295	16.8%	Wet mount
Schneider *et al* [225]	1994	FPC	Bushbuckridge	249	18.0%	Wet mount
Wilkinson *et al* [163]	-	FPC	Hlabisa	189	14.0%	Culture
Kharsany *et al* [209]	1994	FPC	Durban	55	25.5%	Culture
Hoosen *et al* [226]	-	FPC	Durban	40	20.0%	Culture
Fehler *et al* [227]	-	FPC	Johannesburg	210	10.6%	Culture
Kleinschmidt *et al* [235]	1999-2001	FPC	Orange Farm	547	7.5%	Culture
Frohlich *et al* [217]	2002	FPC	Vulindlela	226	23.9%	PCR on swabs
Cronje *et al* [228]	-	HH, F 20-49	Urban FS	405	29.6%	Wet mount
		HH, F 20-49	Rural FS	470	27.4%	Wet mount
O'Leary *et al* [238]	2010	HH, F 15-19	Eastern Cape	329	5.8%	Aptima on urine
Charumilind *et al* [241]	2003	HH, M 20-54	Johannesburg	1458	5.6%	PCR on urine
O'Leary *et al* [238]	2010	HH, M 15-19	Eastern Cape	330	0.0%	Aptima on urine

* Diagnosed by culture and a series of genetic tests, which in combination would have had very high sensitivity and specificity.

ANC = antenatal clinic attenders. CSW = commercial sex workers. F = females. FPC = family planning clinic attenders. FS = Free State. HH = households. KZN = KwaZulu-Natal. M = males. PCR = polymerase chain reaction test. Prev. = prevalence.

Table S22: Genital herpes (HSV-2) prevalence estimates

Study	Year	Sample	Location	n	Prev.	Diagnostic
Sturm [242]	2002	ANC	Hlabisa	417	65.0%	Western blot
Perti *et al* [221]	-	ANC	Johannesburg	390	58.7%	Western blot
Ramjee *et al* [130]	1996-2000	CSW	KZN	416	84.0%	ELISA
Mlaba *et al* [243]	-	FPC	Johannesburg	210	73.0%	-*
Auvert *et al* [144]	1999	HH, F 15-24	Khutsong	771	53.3%	ELISA
Jewkes *et al* [244]	2003	HH, F 15-24	Eastern Cape	1416	29.3%	-*
Auvert (personal communication)	2002	HH, F 18-24	Orange Farm	132	45.5%	ELISA
		HH, F 25-49		285	87.7%	
	2007	HH, F 18-24		477	34.4%	
		HH, F 25-49		723	81.6%	
	2010	HH, F 18-24		434	32.3%	
		HH, F 25-49		783	78.0%	
	2012	HH, F 18-24		1050	32.2%	
		HH, F 25-49		1753	76.6%	
Auvert *et al* [144]	1999	HH, M 15-24	Khutsong	718	17.0%	ELISA
Jewkes *et al* [244]	2003	HH, M 15-24	Eastern Cape	1360	10.2%	-*
Jean *et al* [245]	2002	HH, M 18-29	Orange Farm	225	20.9%	ELISA
		HH, M 30-49		151	58.9%	
	2007	HH, M 18-29		735	11.7%	
		HH, M 30-49		235	57.9%	
	2010	HH, M 18-29		699	13.4%	
		HH, M 30-49		321	59.8%	
	2012	HH, M 18-29		1971	11.4%	
		HH, M 30-49		927	54.4%	

* Diagnosed by a number of tests, which in combination would have had very high sensitivity and specificity. ANC = antenatal clinic attenders. CSW = commercial sex workers. ELISA = enzyme linked immunosorbent assay. F = females. FPC = family planning clinic attenders. HH = households. KZN = KwaZulu-Natal. M = males. Prev. = prevalence.

Assumptions about the sensitivity and specificity of the different assays are summarized in Table S23. Again, these are the same assumptions as made previously [48], except that new assays have been added to the list (SDA on swabs and Gen-Probe/Aptima on urine).

Table S23: Assumed sensitivity and specificity of different diagnostics

STI	Diagnostic	Sex	Sensitivity		Specificity		Ref
			Mean	SD	Mean	SD	
Syphilis	Non-treponemal + treponemal assays	M, F	0.956	0.02	1	0	[246-249]
	Non-treponemal assay	M, F	0.956	0.02	0.98	0.02	[106, 107, 249-251]
Gonorrhoea	Culture	F	0.742	0.193	0.998	0.005	[252]
	LCR on urine	M	0.921	0.029	1	0	[57, 253]
		F	0.838	0.191	1	0	[252]
	PCR on urine	M	0.904	0.029	0.997	0.007	[254]
		F	0.556	0.202	0.987	0.018	[254]
	PCR on swabs	F	0.942	0.046	0.992	0.012	[254]
	SDA on swabs	F	0.955	0.998	0.051	0.003	[255-257]
	Aptima on urine	M, F	0.886	0.998	0.138	0.001	[258-260]
Chlamydial infection	Direct immunofluorescence	F	0.763	0.02	0.988	0.008	[261-263]
	LCR on urine	M	0.875	0.121	1	0	[252]
		F	0.866	0.121	1	0	[252]
	PCR on urine	F	0.833	0.139	0.995	0.007	[254]
	PCR on swabs	F	0.855	0.116	0.996	0.005	[254]
	SDA on swabs	F	0.868	0.993	0.105	0.009	[255-257, 264]
	Aptima on urine	M, F	0.939	0.995	0.045	0.001	[258-260]
Trichomoniasis	Wet mount	F	0.59	0.123	0.991	0.021	[265]
	Culture	F	0.689	0.131	1	0	[232, 266-268]
	PCR on swabs	F	0.95	0.05	0.98	0.024	[269]
	PCR on urine	M	0.95	0.05	0.98	0.024	-
	Aptima on urine	M, F	0.855	0.991	0.108	0.008	[270, 271]
Genital herpes	ELISA	M, F	0.95	0.951	0.03	0.037	[272-276]
	Western blot	M, F	0.914	1	0.03	0	[272, 273]

F = females. LCR = ligase chain reaction. M = males. PCR = polymerase chain reaction. SD = standard deviation. SDA = strand displacement amplification.

4. Model fits to STI prevalence data

Figure S11 shows the estimates of antenatal HIV prevalence, for the 100 best-fitting parameter combinations in both models. The two models give a similarly good fit to the data: although the network model provides a slightly better fit to the prevalence trend in the 15-19 age group, the network model also tends to slightly over-estimate HIV prevalence in women aged 20-29 in recent years. Both models are roughly consistent with the antenatal HIV prevalence data from the pre-1997 period, although these data have not been used in defining the likelihood function.

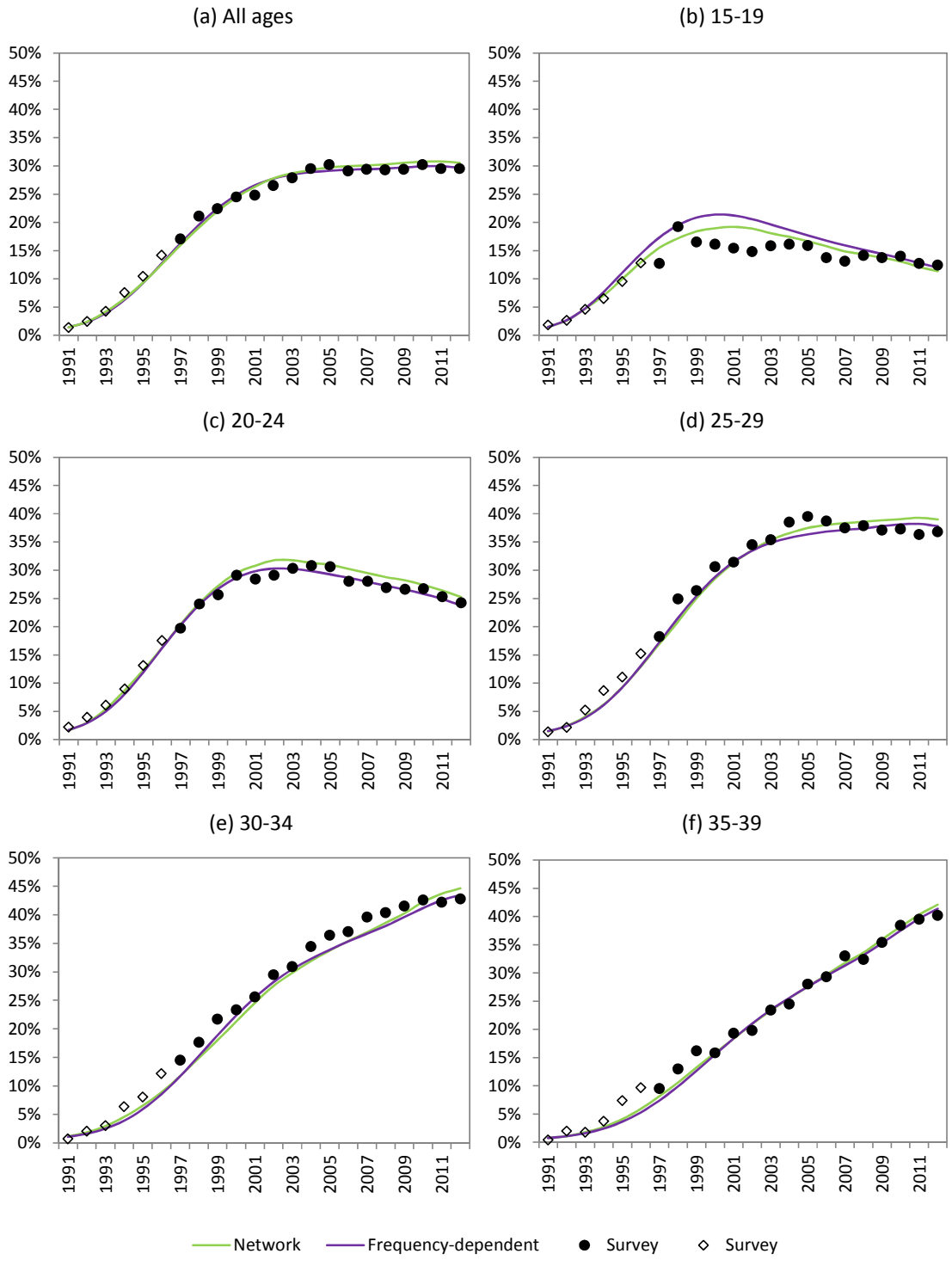

Figure S11: HIV prevalence in pregnant women

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Closed circles represent data used in the likelihood definition, while open diamonds represent data not used in the likelihood definition.

Figure S12 shows the HIV prevalence in the general population, stratified by age and sex. Again, both models provide a similarly good fit to the data, although both models appear to over-estimate HIV prevalence among older men in the most recent survey. This may be because both models assume that the rate of ART uptake is the same in men and women, when in reality men are be less likely to be treated, and hence less likely to survive with HIV to older ages [277].

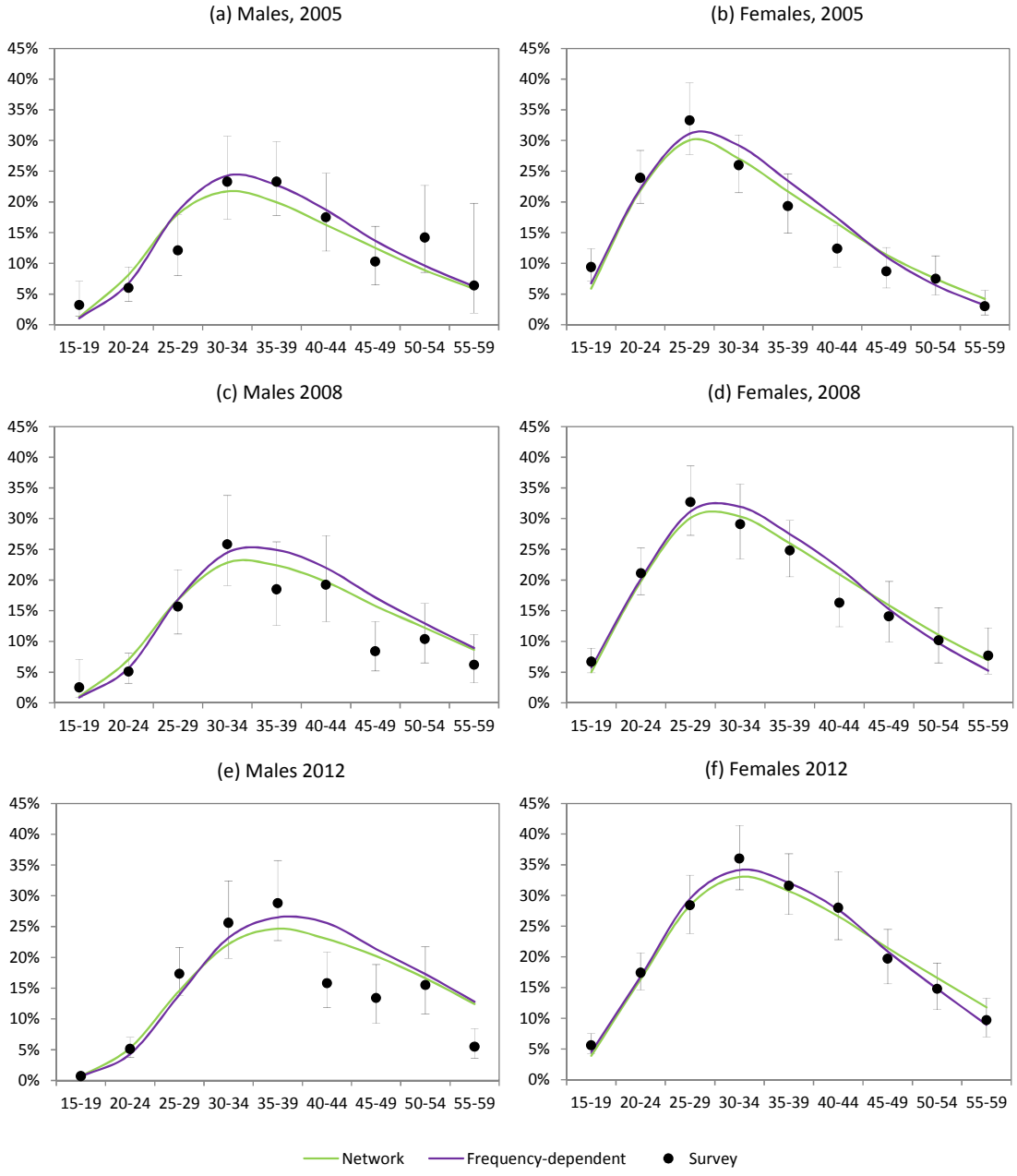

Figure S12: HIV prevalence by age and sex

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Survey data are from the national household surveys conducted by the HSRC in 2005, 2008 and 2012.

Figure S13 shows the estimates of the seroprevalence of syphilis. Both models are roughly consistent with the empirical data, and it is reassuring that both models predict a decline in antenatal syphilis prevalence consistent with that observed in the national antenatal surveys, even though the nationally-representative survey data have not been included in the likelihood definition. However, there is substantial variation in the empirical estimates of syphilis prevalence, which is a reflection of the non-standard way in which the data have been collected (studies have been conducted in different communities, using different diagnostics, and are mostly based on relatively small sample sizes).

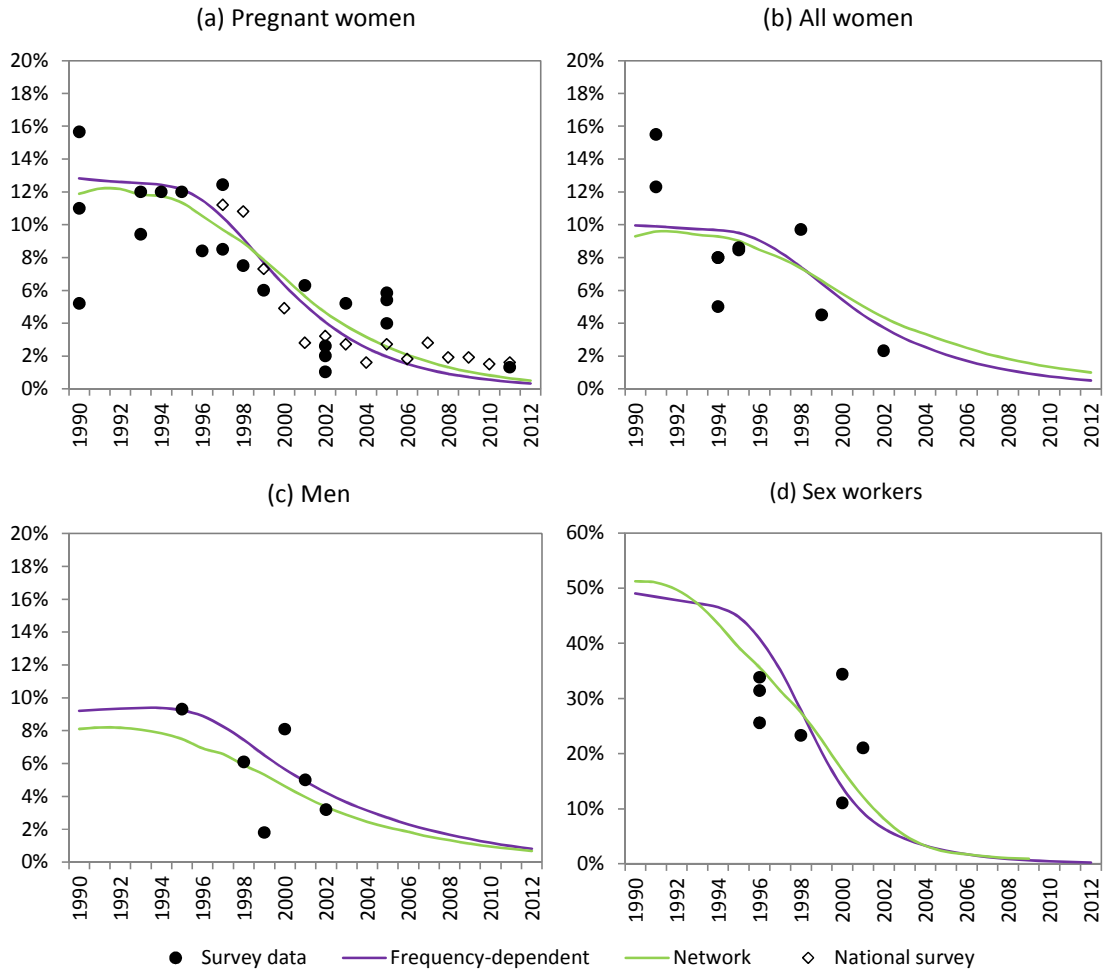

Figure S13: Seroprevalence of syphilis

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Model estimates of seroprevalence include individuals in the immune states but exclude individuals in the incubation phase, to reflect the delay in the development of syphilis antibodies and antibody clearance following resolution of syphilis. Model estimates in panels (b) and (c) are for the 15-49 age group. Closed circles represent data used in the likelihood definition, while open diamonds represent data not used in the likelihood definition. Survey data in panel (b) include data from household surveys and surveys of women attending family planning clinics.

Figure S14 compares the model estimates of gonorrhoea prevalence. The frequency-dependent model appears to provide a better fit to the data than the network model. In women aged 15-49, the network model predicts a steeper decline in gonorrhoea prevalence than

suggested by the data, although there are very few recent studies, and the only two studies conducted since 2002 were conducted in sexually-active schoolgirls and pregnant women – groups known to be at a relatively high risk of gonorrhoea [278]. In sex workers the network model tends to over-estimate gonorrhoea prevalence. This might be because asymptomatic gonorrhoea in sex workers often resolves due to treatment for other STIs, and this dynamic has not been modelled accurately in either model.

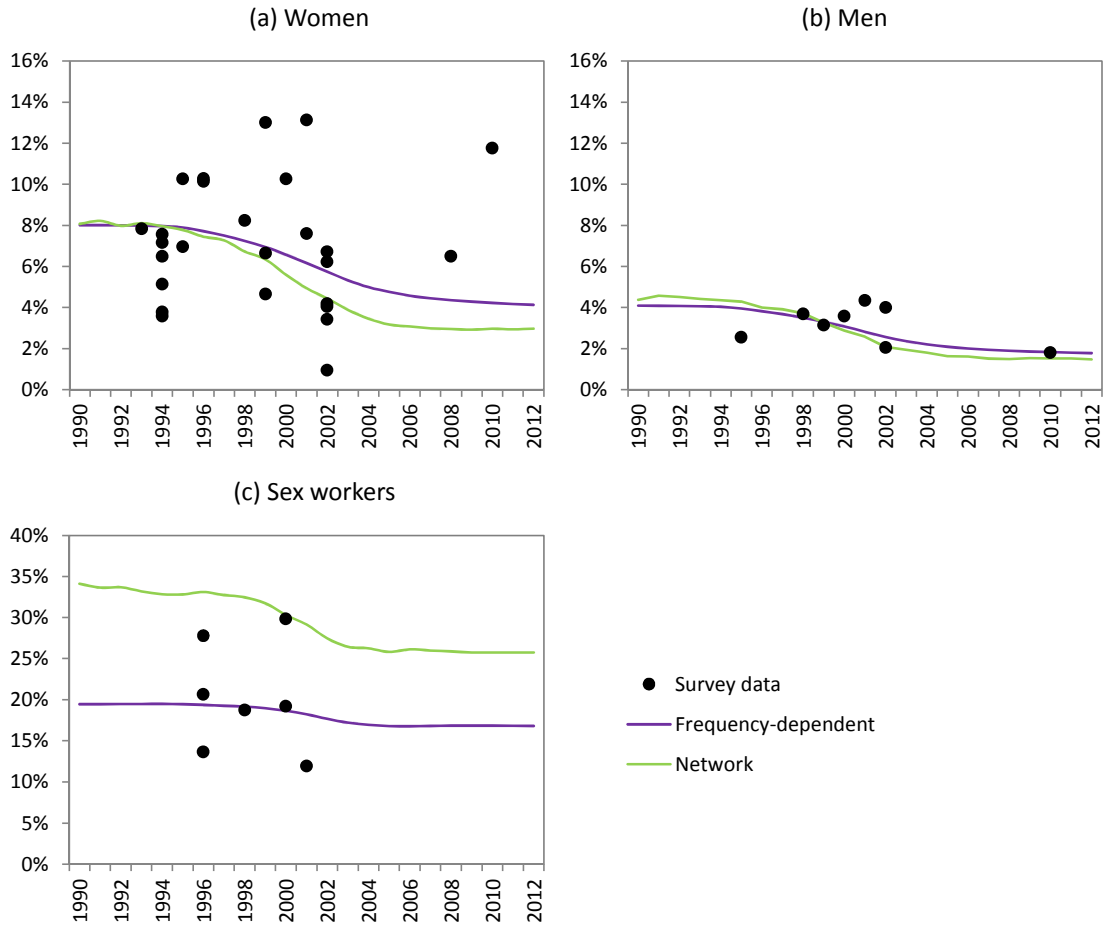

Figure S14: Prevalence of gonorrhoea
Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Model estimates in panels (a) and (b) are for the 15-49 age group. Empirical estimates (closed circles) have been adjusted to reflect the expected sensitivities and specificities of the diagnostics used in the different studies (see Tables S19 and S23). Survey data in panel (a) include data from household surveys, antenatal surveys and surveys of women attending family planning clinics.

Figure S15 shows the estimated prevalence of chlamydia. The two models produce similar fits to the survey data. However, neither model is consistent with the very high levels of chlamydia prevalence measured in recent surveys of women (panel a). This might be because the three most recent data points are from samples of pregnant women and sexually-active schoolgirls, and chlamydia prevalence is known to be particularly high in such samples of young women [278, 279]. Given the lack of recent data, it is difficult to argue with confidence that there has been a real increase in chlamydia prevalence over the last decade.

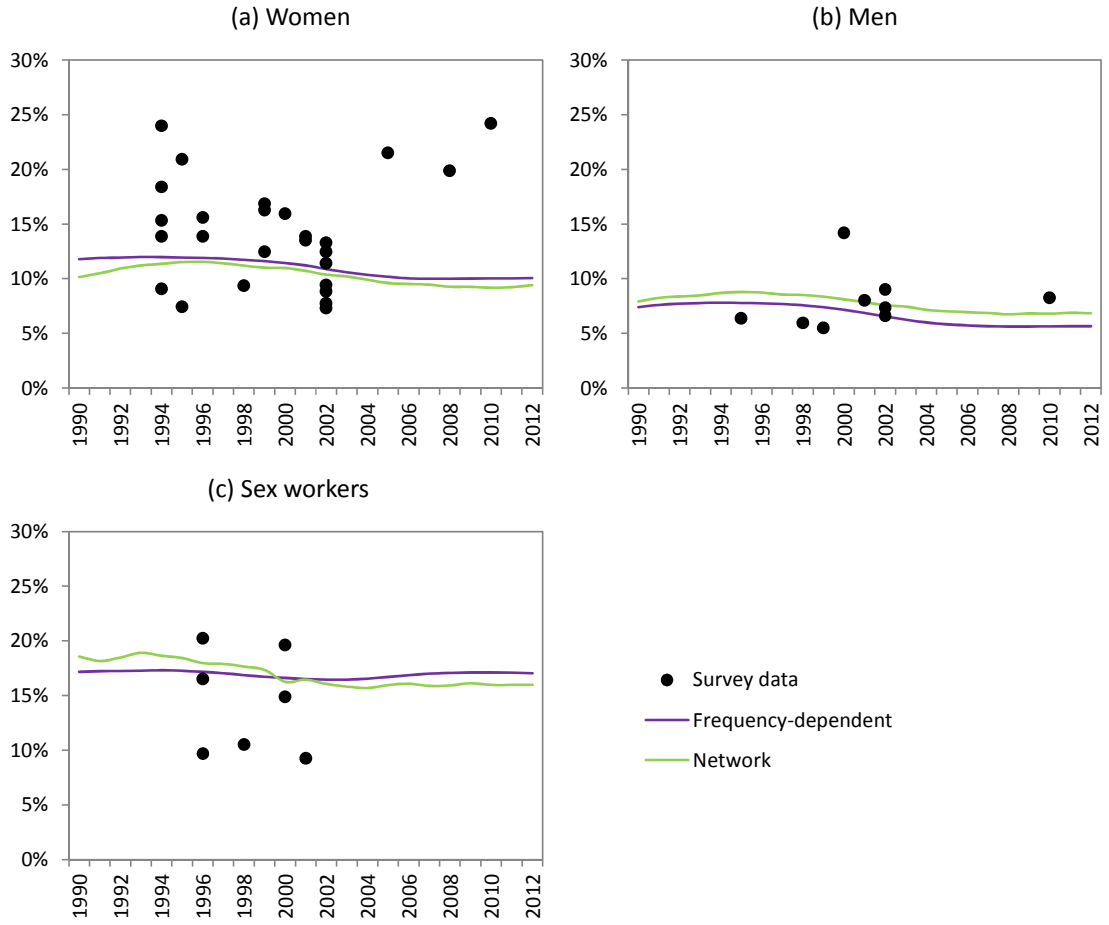

Figure S15: Prevalence of chlamydia

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Model estimates in panels (a) and (b) are for the 15-49 age group. Empirical estimates (closed circles) have been adjusted to reflect the expected sensitivities and specificities of the diagnostics used in the different studies (see Tables S20 and S23). Survey data in panel (a) include data from household surveys, antenatal surveys and surveys of women attending family planning clinics.

Figure S16 shows the estimated prevalence of trichomoniasis. Consistent with the female prevalence data, both models estimate a steady decline in the prevalence of trichomoniasis from 1990 to 2010. Few studies have estimated the prevalence of trichomoniasis in men in the general population and in sex workers, but the two models produce similar prevalence estimates in both cases.

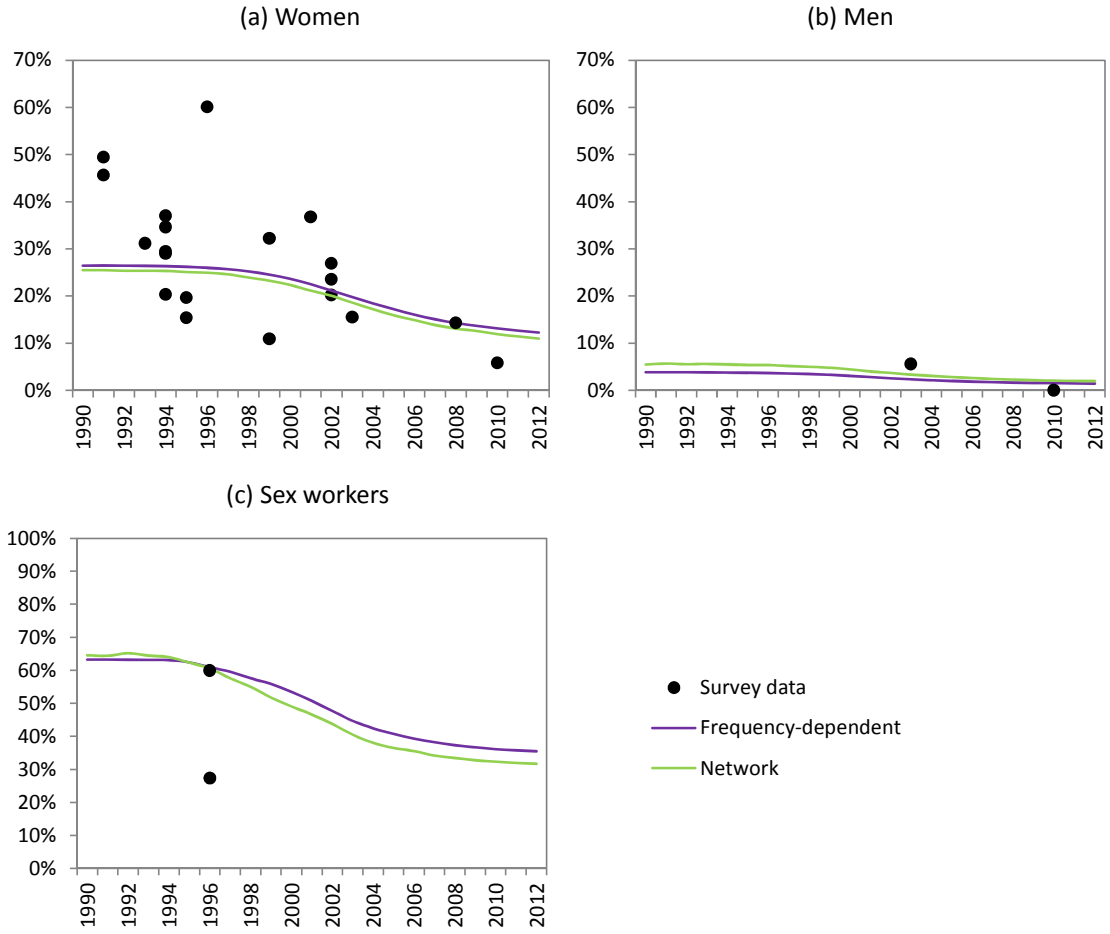

Figure S16: Prevalence of trichomoniasis

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Model estimates in panels (a) and (b) are for the 15-49 age group. Empirical estimates (closed circles) have been adjusted to reflect the expected sensitivities and specificities of the diagnostics used in the different studies (see Tables S21 and S23). Survey data in panel (a) include data from household surveys, antenatal surveys and surveys of women attending family planning clinics.

Figure S17 shows the model estimates of the prevalence of HSV-2. Although the frequency-dependent model provides a slightly better fit to the empirical data when considering young women (panel c), the network model produces a better fit to the empirical data when considering men aged 30-49 (panel f). Although it is concerning that there is little HSV-2 prevalence data and that most of the data come from a single community (Orange Farm in the Gauteng province), it is reassuring that both models produce an HSV-2 prevalence estimate consistent with that measured in a national survey of antenatal clinics [201], even though the national data have not been included in the definition of the likelihood function (panel a).

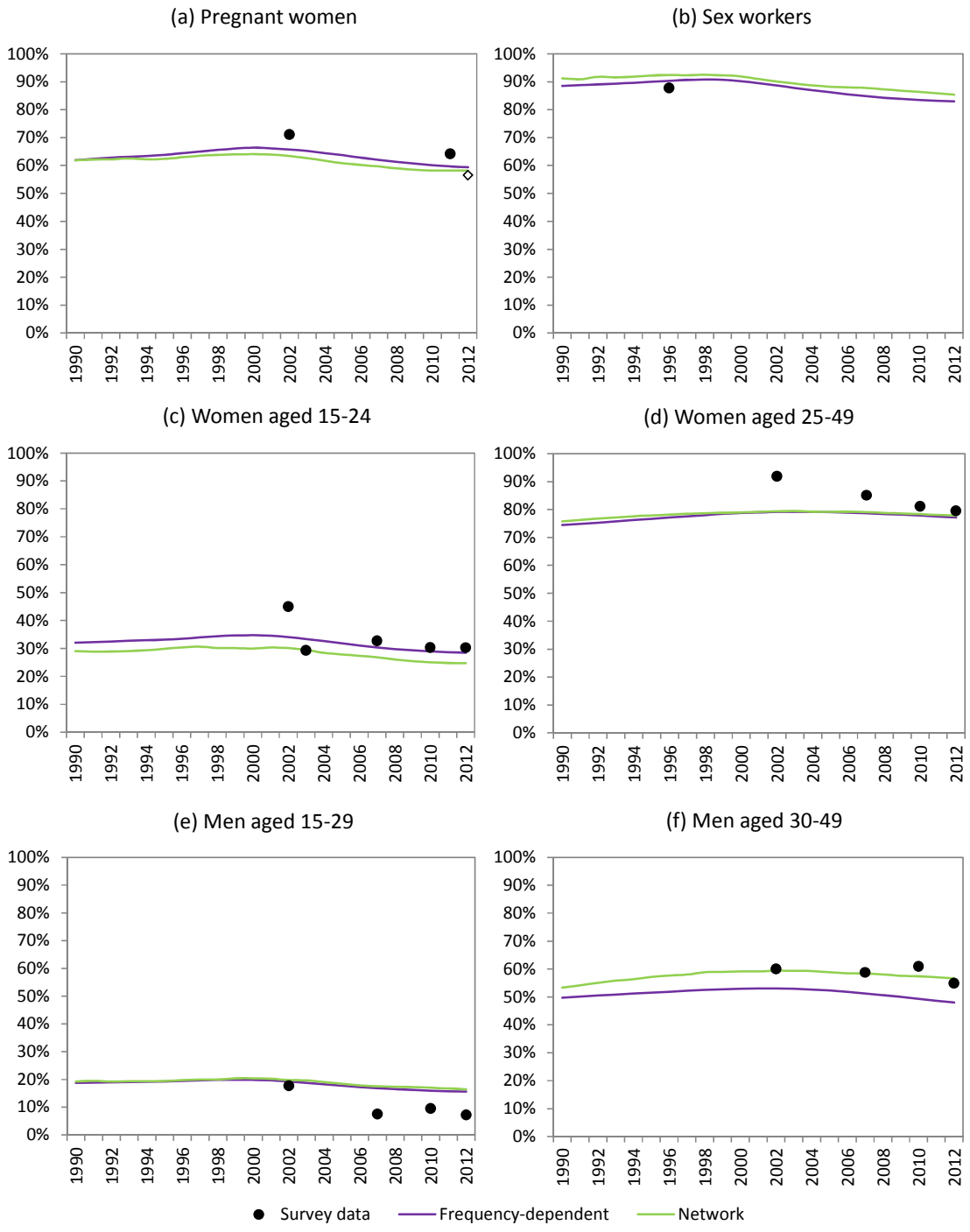

Figure S17: Prevalence of genital herpes (HSV-2)

Solid lines represent the average results obtained using the 100 best-fitting parameter combinations. Model estimates in panels (b) and (c) are for the 15-49 age group. Closed circles represent data used in the likelihood definition, while open diamonds represent data not used in the likelihood definition. All survey estimates have been adjusted to reflect the expected sensitivities and specificities of the diagnostics used in the different studies (see Tables S22 and S23).

5. Correlates of model outputs

Table S24 shows the extent to which the differences between the network and frequency-dependent model outputs are explained by individual parameters. The second column shows the results of a multivariate regression model fitted to the endemic prevalence ratio (the 100 parameter combinations used in this analysis are the same as those used to generate the results in Table 2 and Figure 2 of the main text). When considering the predictors of the ratio of the endemic STI prevalence in the network model to that in the frequency-dependent model, a number of points emerge:

- For all STIs with the exception of HIV, the endemic prevalence ratio is positively associated with the STI transmission probabilities (or not significantly associated). This is in contrast to findings from simpler models, in which there is assumed to be no heterogeneity in sexual behaviour and no STI immunity [280]. We speculate that when there is heterogeneity in behaviour and STI immunity, this creates a virtual threshold above which STI prevalence is unlikely to rise, and because this virtual threshold is similar for the two models, the ratio of the network model to the frequency-dependent model prevalence estimates increases towards 1 as the STI transmission probabilities increase. In the case of HIV the thresholds are less significant, in part because there is assumed to be no HIV immunity and in part because the transmission probabilities in long-term relationships are relatively high (when compared with genital herpes), so that the proportion of the population that can potentially be infected is much higher.
- The level of immunity following STI recovery is in most cases negatively associated with the endemic prevalence ratio. This is because higher levels of immunity following recovery imply a lower likelihood that an individual can reinfect the partner from whom they acquired an STI (which in turn implies greater bias due to the frequency-dependent assumption that newly-infected individuals can transmit their infection while they remain in contact with the partner from whom they acquired the infection).
- For most STIs, the average duration of the infectious period is positively associated with the endemic prevalence ratio. This is because longer durations of infectiousness imply lower early transmission fractions and hence less bias associated with the frequency-dependent assumption.
- For most STIs, the fraction of STIs that are correctly treated is negatively associated with the endemic prevalence ratio. This is because a greater treatment effectiveness implies a higher early transmission fraction, and hence a greater bias due to the frequency-dependent assumption.
- A higher level of infectiousness in the acute stage of HIV infection is associated with a lower endemic prevalence ratio. This is to be expected, since a higher acute infectivity parameter is associated with a greater early transmission fraction and hence a greater bias due to the frequency-dependent assumption. However, it is unexpected that the relative level of infectiousness in the advanced stage of HIV disease is also negatively associated with the endemic prevalence ratio.

Table S24: Factors affecting differences between the network and frequency-dependent models

Parameters	Endemic prevalence ratio	Reduction in incidence due to 50% less commercial sex			Reduction in incidence due to 50% less unprotected sex in spousal relationships		
		Network	Frequency-dependent	Difference	Network	Frequency-dependent	Difference
HIV							
Transmission probability per sex act							
M-to-F, non-spousal	-1.87 (-2.77; -0.96)**	-13.7	-18.4	4.7 (-19.5; 28.9)	-9.4	-3.0	-6.4 (-20.1; 7.3)
F-to-M, non-spousal	12.7 (9.90-15.5)**	-80.7	-10.2	-70.5 (-153.0; 12.1)	-47.2	-10.5	-36.7 (-83.4; 10.0)
M-to-F, spousal	-11.1 (-18.6; -3.56)**	-0.9	-0.6	-0.3 (-40.1; 39.5)	16.1	18.6	-2.9 (-25.1; 20.1)
F-to-M, spousal	-3.53 (-13.1; 6.00)	8.6	-2.6	11.2 (-38.8; 61.2)	16.0	30.5	-14.5 (-42.9; 13.9)
Relative infectiousness, acute HIV	-0.002 (-0.003; -0.001)**	-0.0047	-0.0046	0.000 (-0.007; 0.007)	-0.0044	-0.0005	-0.004 (-0.008; 0.000)*
Relative infectiousness, AIDS	-0.004 (-0.007; -0.002)**	0.031	0.009	0.022 (-0.005; 0.050)	0.017	0.001	0.016 (0.000; 0.032)
Initial prevalence in high-risk women	1.38 (0.41; 2.35)*	-11.9	-2.92	-8.98 (-14.29; -3.67)**	1.17	-0.13	1.30 (-1.72; 4.32)
Bias in self-reported condom use	0.00 (-0.02; 0.02)	0.156	0.067	0.089 (-0.193; 0.371)	-0.094	0.001	-0.095 (-0.255; 0.064)
Genital herpes							
Transmission probability per sex act							
M-to-F, non-spousal	3.58 (2.58; 4.57)**	-0.45	-1.45	1.01 (-1.47; 3.48)	0.17	-0.11	0.29 (-0.34; 0.92)
F-to-M, non-spousal	7.36 (6.08; 8.64)**	-10.85	-10.00	-0.85 (-11.41; 9.71)	2.12	0.98	1.14 (-1.56; 3.83)
M-to-F, spousal	-5.04 (-13.4; 3.30)	1.59	-0.28	1.87 (-16.31; 20.04)	-3.32	-0.01	-3.30 (-7.94; 1.22)
F-to-M, spousal	9.15 (-53.5; 71.8)	-66.8	-3.4	-63.4 (-189.8; 63.1)	41.3	16.7	24.7 (-7.6; 56.9)
Client-to-SW	-3.67 (-11.1; 3.77)	-0.11	2.17	-2.28 (-19.06; 14.51)	1.73	-0.04	1.77 (-2.50; 6.04)
% of cases that become symptomatic	0.08 (-0.02; 0.17)	-0.132	-0.018	-0.114 (-0.251; 0.023)	-0.015	-0.002	-0.013 (-0.048; 0.022)
Annual # reactivations: males	0.000 (-0.004; 0.004)	-0.0003	-0.0003	-0.000 (-0.007; 0.007)	-0.001	0.000	-0.001 (-0.002; 0.001)
Annual # reactivations: females	-0.001 (-0.008; 0.007)	-0.0081	-0.0003	-0.008 (-0.021; 0.006)	-0.002	0.000	-0.002 (-0.006; 0.001)
Relative infectiousness, symptomatic	0.000 (-0.001; 0.000)	-0.0013	-0.0003	-0.001 (-0.002; 0.001)	0.000	0.000	0.000 (-0.0004; 0.0004)
Annual decline in reactivations	-0.09 (-0.31; 0.13)	0.012	0.019	-0.007 (-0.371; 0.357)	0.028	0.000	0.028 (-0.065; 0.121)

* Significant at p <0.05 level. ** Significant at p <0.005 level. 95% confidence intervals around coefficients are shown in brackets.
F-to-M = female-to-male; M-to-F = male-to-female; SM = syndromic management; SW = sex worker.

Table S24 (continued)

Parameters	Endemic prevalence ratio	Reduction in incidence due to 50% less commercial sex			Reduction in incidence due to 50% less unprotected sex in spousal relationships		
		Network	Frequency-dependent	Difference	Network	Frequency-dependent	Difference
Syphilis							
Transmission probability per sex act							
M-to-F	0.67 (0.35; 0.98)**	-2.60	-0.52	-2.08 (-3.35; -0.81)**	0.37	0.05	0.32 (-0.96; 1.60)
F-to-M	0.91 (0.35; 1.47)**	-3.30	-1.16	-2.14 (-4.07; -0.20)*	-0.55	-0.11	-0.44 (-2.41; 1.54)
Average duration (in years)							
Primary infection (untreated)	-0.28 (-0.94; 0.39)	0.91	-0.31	1.21 (-0.24; 2.66)	0.92	0.13	0.79 (-0.70; 2.28)
Secondary infection (untreated)	0.67 (0.27; 1.07)**	-1.27	-0.45	-0.81 (-1.71; 0.08)	0.07	0.09	-0.02 (-0.94; 0.91)
Latent infection	0.005 (-0.006; 0.016)	-0.020	0.005	-0.025 (-0.049; -0.001)*	-0.003	-0.001	-0.002 (-0.027; 0.023)
Immunity following primary/2ndary	-0.43 (-0.69; -0.18)**	0.42	-0.10	0.52 (0.09; 0.96)*	0.06	0.02	0.04 (-0.41; 0.49)
Immunity following latency	0.06 (-0.05; 0.17)	0.028	0.017	0.01 (-0.13; 0.16)	-0.03	0.00	-0.03 (-0.18; 0.12)
% symptoms correctly treated pre-SM	-0.60 (-0.89; -0.32)**	1.68	0.19	1.49 (0.68; 2.29)**	0.10	0.05	0.05 (-0.78; 0.87)
% reduction in 2ndary health seeking	0.28 (0.17; 0.38)**	-1.03	-0.02	-1.02 (-5.33; 3.30)	-0.12	-0.03	-0.09 (-0.50; 0.32)
% not immune after treatment of primary infection	0.26 (-0.10; 0.61)	0.19	0.03	0.15 (-0.38; 0.69)	-0.18	-0.02	-0.15 (-0.70; 0.40)
Gonorrhoea							
Transmission probability per sex act							
M-to-F	0.03 (-0.09; 0.15)	-0.05	-0.03	-0.02 (-0.10; 0.06)	-0.01	-0.06	0.05 (0.02; 0.08)**
F-to-M	0.35 (0.13; 0.56)**	-0.14	-0.09	-0.05 (-0.23; 0.13)	-0.06	-0.17	0.11 (0.05; 0.17)**
% of cases that become symptomatic							
Male	-0.03 (-0.23; 0.18)	0.12	0.04	0.08 (-0.07; 0.23)	0.04	0.06	-0.02 (-0.07; 0.02)
Female	-0.15 (-0.23; -0.07)**	0.08	0.01	0.07 (0.01; 0.14)*	0.00	0.02	-0.02 (-0.04; 0.00)
Average duration (in years)							
Male infection (untreated)	0.10 (0.03; 0.16)**	-0.05	-0.01	-0.04 (-0.08; 0.01)	-0.01	-0.02	0.01 (0.00; 0.03)
Female infection (untreated)	0.25 (0.17; 0.33)**	-0.13	-0.03	-0.10 (-0.17; -0.03)**	0.00	-0.05	0.05 (0.02; 0.08)**
Immunity	-0.07 (-0.10; -0.05)**	0.000	0.000	0.000 (0.000; 0.001)	0.000	0.000	0.000 (0.000; 0.000)
% immune after treatment cure	-0.14 (-0.18; -0.10)**	0.032	-0.014	0.046 (0.001; 0.091)*	-0.018	-0.015	-0.003 (-0.018; 0.012)
% symptoms correctly treated pre-SM	-0.06 (-0.20; 0.07)	0.03	0.02	0.01 (-0.07; 0.09)	0.00	0.03	-0.04 (-0.06; -0.01)*

* Significant at p <0.05 level. ** Significant at p <0.005 level. 95% confidence intervals around coefficients are shown in brackets.
F-to-M = female-to-male; M-to-F = male-to-female; SM = syndromic management; SW = sex worker.

Table S24 (continued)

Parameters	Endemic prevalence ratio	Reduction in incidence due to 50% less commercial sex			Reduction in incidence due to 50% less unprotected sex in spousal relationships		
		Network	Frequency-dependent	Difference	Network	Frequency-dependent	Difference
Chlamydia							
Transmission probability per sex act							
M-to-F	0.21 (-0.01; 0.43)	-0.04	-0.01	-0.02 (-0.11; 0.06)	-0.01	-0.08	0.07 (0.04; 0.10)**
F-to-M	0.42 (0.30; 0.54)**	-0.01	-0.06	0.05 (-0.04; 0.15)	-0.02	-0.21	0.18 (0.14; 0.23)**
% of cases that become symptomatic							
Male	-0.22 (-0.30; -0.15)**	0.0004	-0.0002	0.00 (-0.06; 0.06)	-0.005	0.008	-0.01 (-0.03; 0.01)
Female	-0.11 (-0.28; 0.05)	0.0031	-0.0005	0.00 (-0.10; 0.10)	0.023	0.003	0.02 (-0.01; 0.05)
Average duration (in years)							
Symptomatic infection (untreated)	-0.05 (-0.14; 0.03)	0.011	0.000	0.01 (-0.02; 0.04)	0.000	0.001	-0.001 (-0.012; 0.010)
Asymptomatic infection (untreated)	0.21 (0.15; 0.27)**	-0.008	0.001	-0.01 (-0.05; 0.03)	0.008	-0.005	0.014 (0.000; 0.027)*
Immunity	-0.001 (-0.004; 0.003)	-0.0006	-0.0002	0.000 (-0.006; 0.005)	-0.0022	-0.0012	-0.001 (-0.003; 0.001)
% immune after treatment cure	-0.04 (-0.09; 0.00)	-0.010	-0.003	-0.007 (-0.035; 0.022)	-0.003	0.000	-0.003 (-0.012; 0.007)
% symptoms correctly treated pre-SM	-0.08 (-0.23; 0.06)	-0.011	0.002	-0.013 (-0.061; 0.036)	0.002	0.007	-0.004 (-0.020; 0.012)
Trichomoniasis							
Transmission probability per sex act							
M-to-F	-0.08 (-0.29; 0.14)	-0.06	-0.12	0.06 (-0.01; 0.13)	-0.04	-0.11	0.08 (0.03; 0.12)**
F-to-M	2.52 (1.73; 3.31)**	-0.84	-1.48	0.64 (0.17; 1.12)*	-0.57	-1.69	1.12 (0.80; 1.45)**
% of cases that become symptomatic							
Male	-0.38 (-0.53; -0.23)**	0.06	0.03	0.03 (-0.03; 0.08)	0.03	0.03	0.00 (-0.03; 0.03)
Female	-0.07 (-0.25; 0.10)	0.03	0.00	0.03 (-0.02; 0.08)	0.01	0.02	-0.01 (-0.04; 0.01)
Average duration (in years)							
Symptomatic infection (untreated), M	1.62 (0.31; 2.93)*	-0.16	-0.11	-0.06 (-0.36; 0.25)	0.06	-0.08	0.14 (-0.01; 0.29)
Symptomatic infection (untreated), F	-0.06 (-0.29; 0.17)	-0.04	0.00	-0.04 (-0.07; 0.00)*	0.004	-0.004	0.01 (-0.01; 0.03)
Asymptomatic infection (untreated), M	0.38 (0.24; 0.52)**	-0.08	-0.03	-0.04 (-0.09; 0.01)	-0.05	-0.04	-0.01 (-0.04; 0.01)
Asymptomatic infection (untreated), F	0.05 (0.03; 0.06)**	-0.012	0.001	-0.013 (-0.02; -0.007)**	-0.001	-0.002	0.001 (-0.002; 0.004)
Immunity	-0.05 (-0.09; 0.00)*	-0.0002	-0.0004	0.000 (-0.0002; 0.0006)	-0.0005	-0.0004	0.000 (-0.0003; 0.0001)
% immune after treatment cure	-0.08 (-0.14; -0.02)*	-0.020	-0.014	-0.006 (-0.023; 0.12)	0.002	-0.005	0.007 (-0.002; 0.016)
% symptoms correctly treated pre-SM	-0.18 (-0.30; -0.06)**	-0.018	-0.004	-0.014 (-0.040; 0.012)	0.005	0.007	-0.001 (-0.014; 0.011)

* Significant at p <0.05 level. ** Significant at p <0.005 level. 95% confidence intervals around coefficients are shown in brackets.
F-to-M = female-to-male; M-to-F = male-to-female; SM = syndromic management; SW = sex worker.

The third and fourth columns show the results of multivariate regression models fitted to the predicted reduction in STI incidence due to a 50% reduction in sex worker contact, for the network and frequency-dependent models respectively. Each regression model is fitted using the 100 parameter combinations that yield the best fit to the STI prevalence data, and the parameter combinations are therefore not the same for the network and frequency-dependent models. It is therefore not possible to fit one regression model to assess the effect of individual parameters on the difference between the network and frequency-dependent models. However, it is possible to calculate the difference between the coefficients of the two regression models and to assess the statistical significance of these differences (shown in the fifth column). Since the network model tends to predict a greater impact of a reduction in commercial sex than the frequency-dependent model (Figure 3a of the main text), a positive difference in coefficients indicates a parameter that is positively associated with the difference between the two models. Most of the differences in coefficients are not statistically significant, but a few parameters are significant:

- The duration of infectiousness tends to be negatively associated with the model difference (particularly in the case of gonorrhoea and trichomoniasis). This is because a greater duration of infectiousness is associated with a lower early transmission fraction and hence less bias due to the frequency-dependent assumption. The smaller the bias due to the frequency-dependent assumption, the smaller the difference between the two models in the predicted impact of the behaviour change.
- The fraction of female gonorrhoea cases that become symptomatic and the fraction of syphilis symptoms that are correctly treated are both positively associated with the model difference. This is probably because both parameters are positively associated with the early transmission fraction and thus are associated with greater bias due to the frequency-dependent assumption.
- Immunity exacerbates the bias due to the frequency-dependent assumption, because in the absence of immunity a newly-infected individual is more likely to reinfect the partner from whom they acquired their infection, and there is thus less transmission potential ‘wasted’ on the partner from whom they acquired the infection. The immunity parameters for gonorrhoea and syphilis are thus positively associated with the difference between the two models.
- For both models, and for all STIs, transmission probabilities tend to be negatively associated with the predicted impact of a reduction in sex worker contact. This is because in order to maintain the same goodness of fit to the STI prevalence data, any increase in transmission probability must be compensated for by an increase in heterogeneity in STI risk, and increases in heterogeneity imply reductions in intervention impacts [38, 281]. However, the extent of the negative association tends to be greater for the network model than the frequency-dependent model, especially in the case of syphilis. This is because commercial sex accounts for a greater fraction of STI transmission in the network model than the frequency-dependent model, and hence the network model is more sensitive to changes in the frequency of sex worker contact.

The sixth and seventh columns of the table show the results of multivariate regression models fitted to the predicted reduction in STI incidence due to a 50% reduction in unprotected sex in long-term partnerships, for the network and frequency-dependent models respectively. The approach adopted is the same as in the previous analysis, except that the predictor in the regression is different. The final column of the table shows the difference in the regression coefficients between the two models (the network coefficient less the frequency-dependent

coefficient). Since the network model tends to predict a smaller impact of a reduction in unprotected spousal sex than the frequency-dependent model (Figure 3c of the main text), a positive difference in coefficients indicates a parameter that is *negatively* associated with the difference between the two models (i.e. increasing that parameter reduces the extent of the model difference on average). Most of the differences in coefficients are not statistically significant, but a few parameters are significant:

- As in the previous analysis, the duration of infectiousness tends to be negatively associated with the model difference, particularly in the case of the gonorrhoea and chlamydia. Again, this is because a longer duration of infectiousness is associated with a lower early transmission fraction and thus less bias due to the frequency-dependent assumption.
- The relative level of infectiousness in the acute stage of HIV infection is positively associated with the model difference. This might be because this parameter is positively associated with the early transmission fraction and thus increases the bias due to the frequency-dependent assumption.
- For gonorrhoea, chlamydia and trichomoniasis, transmission probabilities are negatively associated with the model difference. As explained previously, higher transmission probabilities are associated with greater heterogeneity in STI risk and thus lower intervention impacts. Because the frequency-dependent model estimates a greater fraction of STI transmission occurring in long-term partnerships than the network model, it is more sensitive to the effect of increased condom use in long-term relationships than the network model, and hence an increase in transmission probabilities is likely to reduce the extent of the difference between the models.

References

1. Johnson LF, Dorrington RE, Bradshaw D, Pillay-Van Wyk V, Rehle TM. Sexual behaviour patterns in South Africa and their association with the spread of HIV: insights from a mathematical model. *Demographic Research* 2009; **21**:289-340.
2. Budlender D, Chobokoane N, Simelane S. Marriage patterns in South Africa: Methodological and substantive issues. *Southern African Journal of Demography* 2004; **9**:1-26.
3. Dunkle KL, Jewkes RK, Brown HC, Gray GE, McIntryre JA, Harlow SD. Transactional sex among women in Soweto, South Africa: prevalence, risk factors and association with HIV infection. *Social Science and Medicine* 2004; **59**:1581-1592.
4. Jewkes RK, Nduna M, Jama PN, Dunkle KL, Levin JB. Steadys, roll-ons and hit and runs: using indigenous typology to measure number of sexual partners [Abstract TuPpE2069]. *14th International AIDS Conference*. Barcelona, Spain; 2002.
5. Shisana O, Rehle T, Simbayi LC, Parker W, Zuma K, Bhana A, *et al.* South African National HIV Prevalence, HIV Incidence, Behaviours and Communication Survey, 2005. Cape Town: HSRC Press; 2005. Available: http://www.hsrcpress.ac.za. Accessed 1 Dec 2005
6. Department of Health. National HIV and Syphilis Prevalence Survey, South Africa 2005. Pretoria: Directorate of Epidemiology and Surveillance; 2006. Available: http://www.doh.gov.za/docs/reports-f.html. Accessed 6 Aug 2010
7. Jewkes R, Vundule C, Maforah F, Jordaan E. Relationship dynamics and teenage pregnancy in South Africa. *Social Science and Medicine* 2001; **52**:733-744.
8. Ross A, Van der Paal L, Lubega R, Mayanja BN, Shafer LA, Whitworth J. HIV-1 disease progression and fertility: the incidence of recognized pregnancy and pregnancy outcome in Uganda. *AIDS* 2004; **18**:799-804.
9. Terceira N, Gregson S, Zaba B, Mason PR. The contribution of HIV to fertility decline in rural Zimbabwe, 1985-2000. *Population Studies* 2003; **57**:149-164.
10. Hankins C, Tran T, Lapointe N. Sexual behaviour and pregnancy outcome in HIV-infected women. *Journal of Acquired Immune Deficiency Syndromes* 1998; **18**:479-487.
11. Greenblatt RM, Bacchetti P, Barkan S, Augenbraun M, Silver S, Delapenha R, *et al.* Lower genital tract infections among HIV-infected and high-risk uninfected women: findings of the Women's Interagency HIV Study (WIHS). *Sexually Transmitted Diseases* 1999; **26**:143-151.
12. Moatti JP, Prudhomme J, Traore DC, Juillet-Amari A, Akribi HA, Msellati P. Access to antiretroviral treatment and sexual behaviours of HIV-infected patients aware of their serostatus in Côte d'Ivoire. *AIDS* 2003; **17** (Suppl 3):S69-77.
13. Bunnell R, Ekwaru JP, Solberg P, Wamai N, Bikaako-Kajura W, Were W, *et al.* Changes in sexual behavior and risk of HIV transmission after antiretroviral therapy and prevention interventions in rural Uganda. *AIDS* 2006; **20**:85-92.
14. Mpofu E, Flisher AJ, Bility K, Onya H, Lombard C. Sexual partners in a rural South African setting. *AIDS and Behavior* 2006; **10**:399-404.
15. Pettifor AE, van der Straten A, Dunbar MS, Shiboski SC, Padian NS. Early age of first sex: a risk factor for HIV infection among women in Zimbabwe. *AIDS* 2004; **18**:1435-1442.

16. Department of Health. South Africa Demographic and Health Survey 1998: Full Report. 1999.
17. Williams B, Gilgen D, Campbell C, Taljaard D, MacPhail C. *The natural history of HIV / AIDS in South Africa: A biomedical and social survey in Carletonville*. Johannesburg: Council for Scientific and Industrial Research; 2000.
18. Hallman K. Socioeconomic Disadvantage and Unsafe Sexual Behaviors among Young Women and Men in South Africa. New York: Population Council, Policy Research Division; 2004.
19. Kelly K. Communicating for action: A contextual evaluation of youth responses to HIV/AIDS. Department of Health; 2000. Available: http://www.cadre.org.za. Accessed 12 October 2006
20. Nnko S, Boerma JT, Urassa M, Mwaluko G, Zaba B. Secretive females or swaggering males? An assessment of the quality of sexual partnership reporting in rural Tanzania. *Social Science and Medicine* 2004; **59**:299-310.
21. Ferry B, Caraël M, Buvé A, Auvert B, Laourou M, Kanhonou L, *et al.* Comparison of key parameters of sexual behaviour in four African urban populations with different levels of HIV infection. *AIDS* 2001; **15**:S41-50.
22. Statistics South Africa. Marriages and divorces, 2004. Pretoria; 2006. Available: http://www.statssa.gov.za/publications/Report-03-07-01/Report-03-07-012004.pdf. Accessed 2 Aug 2008
23. Konstant TL, Rangasami J, Stacey MJ, Stewart ML, Nogoduka C. Estimating the number of sex workers in South Africa: rapid population size estimation. *AIDS and Behavior* 2015; **19** (Suppl 1):S3-15.
24. Varga CA. The condom conundrum: barriers to condom use among commercial sex workers in Durban, South Africa. *African Journal of Reproductive Health* 1997; **1**:74-88.
25. Abdool Karim QA, Abdool Karim SS, Soldan K, Zondi M. Reducing the risk of HIV infection among South African sex workers: socioeconomic and gender barriers. *American Journal of Public Health* 1995; **85**:1521-1525.
26. Ramjee G, Abdool Karim SS, Sturm AW. Sexually transmitted infections among sex workers in KwaZulu-Natal, South Africa. *Sexually Transmitted Diseases* 1998; **25**:346-349.
27. Dunkle KL, Beksinska ME, Rees VH, Ballard RC, Htun Y, Wilson ML. Risk factors for HIV infection among sex workers in Johannesburg, South Africa. *International Journal of STD and AIDS* 2005; **16**:256-261.
28. Gould C, Fick N. *Selling Sex in Cape Town: Sex Work and Human Trafficking in a South African City*: Institute for Security Studies; 2008.
29. Peltzer K, Seoka P, Raphala S. Characteristics of female sex workers and their HIV/AIDS/STI knowledge, attitudes and behaviour in semi-urban areas in South Africa. *Curationis* 2004; March 2004:4-11.
30. van Loggerenberg F, Mlisana K, Williamson C, Auld SC, Morris L, Gray CM, *et al.* Establishing a cohort at high risk of HIV infection in South Africa: challenges and experiences of the CAPRISA 002 acute infection study. *PLoS One* 2008; **3**:e1954.
31. Delva W, Richter M, De Koker P, Chersich M, Temmerman M. Sex work during the 2010 FIFA World Cup: results from a three-wave cross-sectional survey. *PLoS One* 2011; **6**:e28363.
32. Caraël M, Slaymaker E, Lyerla R, Sarkar S. Clients of sex workers in different regions of the world: hard to count. *Sexually Transmitted Infections* 2006; **82** (Suppl 3):iii26-33.

33. Rees H, Beksinska ME, Dickson-Tetteh K, Ballard RC, Htun Y. Commercial sex workers in Johannesburg: risk behaviour and HIV status. *South African Journal of Science* 2000; **96**:283-284.
34. Wingood GM, Reddy P, Lang DL, Saleh-Onoya D, Braxton N, Sifunda S, *et al.* Efficacy of SISTA South Africa on sexual behavior and relationship control among isiXhosa women in South Africa: results of a randomized-controlled trial. *Journal of Acquired Immune Deficiency Syndromes* 2013; **63** (Suppl 1):S59-65.
35. Harling G, Newell ML, Tanser F, Kawachi I, Subramanian S, Bärnighausen T. Do age-disparate relationships drive HIV incidence in young women? Evidence from a population cohort in rural KwaZulu-Natal, South Africa. *Journal of Acquired Immune Deficiency Syndromes* 2014; **66**:443-451.
36. Johnson S, Kincaid DL, Figueroa ME, Delate R, Mahlasela L, Magni S. The Third National HIV Communication Survey, 2012. Pretoria: Johns Hopkins Health and Education in South Africa; 2013. Available: http://jhhesa.org/sites/default/files/hiv_survey.pdf. Accessed 19 April 2014
37. Katz I, Low-Beer D. Why has HIV stabilized in South Africa, yet not declined further? Age and sexual behavior patterns among youth. *Sexually Transmitted Diseases* 2008; **35**:837-842.
38. Johnson LF, Hallett TB, Rehle TM, Dorrington RE. The effect of changes in condom usage and antiretroviral treatment coverage on HIV incidence in South Africa: a model-based analysis. *Journal of the Royal Society Interface* 2012; **9**:1544-1554.
39. Department of Health. South Africa Demographic and Health Survey 2003: Preliminary Report. Pretoria; 2004. Available: http://www.doh.gov.za/docs/reports/2003/sadhs2003/part2.pdf. Accessed 6 Jan 2012
40. Kaufman CE. The politics and practice of reproductive control in South Africa: a multilevel analysis of fertility and contraceptive use [Doctoral thesis]. Ann Arbor: University of Michigan; 1996.
41. Jochelson K, Mothibeli M, Leger JP. Human immunodeficiency virus and migrant labor in South Africa. *International Journal of Health Services* 1991; **21**:157-173.
42. Richter ML, Chersich M, Temmerman M, Luchters S. Characteristics, sexual behaviour and risk factors of female, male and transgender sex workers in South Africa. *South African Medical Journal* 2013; **103**:246-251.
43. Sex Worker Education and Advocacy Taskforce. Beginning to build the picture: South African national survey of sex worker knowledge, experiences and behaviour. 2013.
44. Human Sciences Research Council. South African national HIV prevalence, behavioural risks and mass media household survey 2002. 2002. Available: http://www.hsrcpress.ac.za. Accessed 18 Feb 2009
45. Reproductive Health Research Unit. HIV and sexual behaviour among young South Africans: a national survey of 15-24 year olds. Joint publication of Reproductive Health Research Unit and loveLife; 2004. Available: www.rhru.co.za/images/Docs/national%20survey%20RHRU.pdf. Accessed 8 May 2004
46. Shisana O, Rehle T, Simbayi LC, Zuma K, Jooste S, Pillay-van Wyk V, *et al.* South African national HIV prevalence, incidence, behaviour and communication survey, 2008: A turning tide among teenagers? Cape Town: Human Sciences Research Council; 2009. Available: http://www.hsrcpress.ac.za. Accessed 9 June 2009
47. Boily MC, Asghar Z, Garske T, Ghani AC, Poulin R. Influence of selected formation rules for for finite population networks with fixed macrostructures: implications for

individual-based model of infectious diseases. *Mathematical Population Studies* 2007; **14**:237-267.
48. Johnson LF, Alkema L, Dorrington RE. A Bayesian approach to uncertainty analysis of sexually transmitted infection models. *Sexually Transmitted Infections* 2010; **86**:169-174.
49. Johnson LF, Dorrington RE, Bradshaw D, Coetzee DJ. The effect of syndromic management interventions on the prevalence of sexually transmitted infections in South Africa. *Sexual and Reproductive Healthcare* 2011; **2**:13-20.
50. Johnson LF, Dorrington RE, Bradshaw D. The role of immunity in the epidemiology of gonorrhoea, chlamydial infection and trichomoniasis: insights from a mathematical model. *Epidemiology and Infection* 2011; **139**:1875-1883.
51. Johnson LF, Dorrington RE, Bradshaw D, Coetzee DJ. The role of sexually transmitted infections in the evolution of the South African HIV epidemic. *Tropical Medicine and International Health* 2012; **17**:161-168.
52. Plummer FA, Simonsen JN, Chubb H, Slaney L, Kimata J, Bosire M, *et al.* Epidemiologic evidence for the development of serovar-specific immunity after gonococcal infection. *Journal of Clinical Investigation* 1989; **83**:1472-1476.
53. Moodley P, Martin IM, Ison CA, Sturm AW. Typing of *Neisseria gonorrhoeae* reveals rapid reinfection in rural South Africa. *Journal of Clinical Microbiology* 2002; **40**:4567-4570.
54. Schmidt KA, Schneider H, Lindstrom JA, Boslego JW, Warren RA, Van de Verg L, *et al.* Experimental gonococcal urethritis and reinfection with homologous gonococci in male volunteers. *Sexually Transmitted Diseases* 2001; **28**:555-564.
55. Garnett GP, Mertz KJ, Finelli L, Levine WC, St Louis ME. The transmission dynamics of gonorrhoea: modelling the reported behaviour of infected patients from Newark, New Jersey. *Philosophical Transactions of the Royal Society of London. Series B* 1999; **354**:787-797.
56. Harrison WO, Hooper RR, Wiesner PJ, Campbell AF, Karney WW, Reynolds GH, *et al.* A trial of minocycline given after exposure to prevent gonorrhea. *New England Journal of Medicine* 1979; **300**:1074-1078.
57. Buimer M, van Doornum GJ, Ching S, Peerbooms PG, Plier PK, Ram D, *et al.* Detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* by ligase chain reaction-based assays with clinical specimens from various sites: implications for diagnostic testing and screening. *Journal of Clinical Microbiology* 1996; **34**:2395-2400.
58. Price MA, Miller WC, Kaydos-Daniels SC, Hoffman IF, Chilongozi D, Martinson FE, *et al.* Trichomoniasis in men and HIV infection: data from 2 outpatient clinics at Lilongwe Central Hospital, Malawi. *Journal of Infectious Diseases* 2004; **190**:1448-1455.
59. Joyner JL, Douglas JM, Jr., Ragsdale S, Foster M, Judson FN. Comparative prevalence of infection with *Trichomonas vaginalis* among men attending a sexually transmitted diseases clinic. *Sexually Transmitted Diseases* 2000; **27**:236-240.
60. Hobbs MM, Kazembe P, Reed AW, Miller WC, Nkata E, Zimba D, *et al.* *Trichomonas vaginalis* as a cause of urethritis in Malawian men. *Sexually Transmitted Diseases* 1999; **26**:381-387.
61. Platt R, Rice PA, McCormack WM. Risk of acquiring gonorrhea and prevalence of abnormal adnexal findings among women recently exposed to gonorrhea. *Journal of the American Medical Association* 1983; **250**:3205-3209.

62. Korenromp EL, Sudaryo MK, de Vlas SJ, Gray RH, Sewankambo NK, Serwadda D, *et al.* What proportion of episodes of gonorrhoea and chlamydia becomes symptomatic? *International Journal of STD and AIDS* 2002; **13**:91-101.
63. Handsfield HH, Lipman TO, Harnisch JP, Tronca E, Holmes KK. Asymptomatic gonorrhea in men. Diagnosis, natural course, prevalence and significance. *New England Journal of Medicine* 1974; **290**:117-123.
64. Lin JS, Donegan SP, Heeren TC, Greenberg M, Flaherty EE, Haivanis R, *et al.* Transmission of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* among men with urethritis and their female sex partners. *Journal of Infectious Diseases* 1998; **178**:1707-1712.
65. Orroth KK, Freeman EE, Bakker R, Buvé A, Glynn JR, Boily M, *et al.* Understanding differences between contrasting HIV epidemics in East and West Africa: results from a simulation model of the Four Cities Study. *Sexually Transmitted Infections* 2007; **83** (Suppl 1):i5-16.
66. Bracher M, Santow G, Watkins SC. 'Moving' and marrying: modelling HIV infection among newly-weds in Malawi. *Demographic Research* 2003; Special Collection 1:207-246.
67. Korenromp EL, Van Vliet C, Grosskurth H, Gavyole A, Van der Ploeg CPB, Fransen L, *et al.* Model-based evaluation of single-round mass treatment of sexually transmitted diseases for HIV control in a rural African population. *AIDS* 2000; **14**:573-593.
68. Ghani AC, Aral SO. Patterns of sex worker-client contacts and their implications for the persistence of sexually transmitted infections. *Journal of Infectious Diseases* 2005; **191** Suppl 1:S34-41.
69. Holmes KK, Johnson DW, Trostle HJ. An estimate of the risk of men acquiring gonorrhea by sexual contact with infected females. *American Journal of Epidemiology* 1970; **91**:170-174.
70. Hooper RR, Reynolds GH, Jones OG, Zaidi A, Wiesner PJ, Latimer KP, *et al.* Cohort study of veneral disease. I: The risk of gonorrhea transmission from infected women to men. *American Journal of Epidemiology* 1978; **108**:136-144.
71. Mathews C, van Rensburg A, Schierhout G, Coetzee N, Lombard CJ, Fehler HG, *et al.* An assessment of care provided by a public sector STD clinic in Cape Town. *International Journal of STD and AIDS* 1998; **9**:689-694.
72. Somsé P, Mberyo-Yaah F, Morency P, Dubois MJ, Grésenguet G, Pépin J. Quality of sexually transmitted disease treatments in the formal and informal sectors of Bangui, Central African Republic. *Sexually Transmitted Diseases* 2000; **27**:458-464.
73. Buvé A, Changalucha J, Mayaud P, Gavyole A, Mugeye K, Todd J, *et al.* How many patients with a sexually transmitted infection are cured by health services? A study from Mwanza region, Tanzania. *Tropical Medicine and International Health* 2001; **6**:971-979.
74. Chabikuli N, Schneider H, Blaauw D, Zwi AB, Brugha R. Quality and equity of private sector care for sexually transmitted diseases in South Africa. *Health Policy and Planning* 2002; **17**:40-46.
75. Fraser C, Hollingsworth TD, Chapman R, de Wolf F, Hanage WP. Variation in HIV-1 set-point viral load: epidemiological analysis and an evolutionary hypothesis. *Proceedings of the National Academy of Sciences U S A* 2007; **104**:17441-17446.
76. Brunham RC, Kimani J, Bwayo J, Maitha G, Maclean I, Yang C, *et al.* The epidemiology of *Chlamydia trachomatis* within a sexually transmitted diseases core group. *Journal of Infectious Diseases* 1996; **173**:950-956.

77. Arno JN, Katz BP, McBride R, Carty GA, Batteiger BE, Caine VA, *et al.* Age and clinical immunity to infections with *Chlamydia trachomatis*. *Sexually Transmitted Diseases* 1994; **21**:47-52.
78. Golden MR, Schillinger JA, Markowitz L, St Louis ME. Duration of untreated genital infections with chlamydia trachomatis: a review of the literature. *Sexually Transmitted Diseases* 2000; **27**:329-337.
79. Schachter J, Cles LD, Ray RM, Hesse FE. Is there immunity to chlamydial infections of the human genital tract? *Sexually Transmitted Diseases* 1983; **10**:123-125.
80. Katz BP, Batteiger BE, Jones RB. Effect of prior sexually transmitted disease on the isolation of *Chlamydia trachomatis*. *Sexually Transmitted Diseases* 1987; **14**:160-164.
81. Brunham RC, Pourbohloul B, Mak S, White R, Rekart ML. The unexpected impact of a *Chlamydia trachomatis* infection control program on susceptibility to reinfection. *Journal of Infectious Diseases* 2005; **192**:1836-1844.
82. Rekart ML, Brunham RC. Epidemiology of chlamydial infection: are we losing ground? *Sexually Transmitted Infections* 2008; **84**:87-91.
83. Su H, Morrison R, Messer R, Whitmire W, Hughes S, Caldwell HD. The effect of doxycycline treatment on the development of protective immunity in a murine model of chlamydial genital infection. *Journal of Infectious Diseases* 1999; **180**:1252-1258.
84. Krieger JN, Jenny C, Verdon M, Siegel N, Springwater R, Critchlow CW, *et al.* Clinical manifestations of trichomoniasis in men. *Annals of Internal Medicine* 1993; **118**:844-849.
85. Parks KS, Dixon PB, Richey CM, Hook EWI. Spontaneous clearance of *Chlamydia trachomatis* infection in untreated patients. *Sexually Transmitted Diseases* 1997; **24**:229-235.
86. Rahm VA, Gnarpe H, Odlind V. *Chlamydia trachomatis* among sexually active teenage girls. Lack of correlation between chlamydial infection, history of the patient and clinical signs of infection. *British Journal of Obstetrics and Gynaecology* 1988; **95**:916-919.
87. Molano M, Meijer CJ, Weiderpass E, Arslan A, Posso H, Franceschi S, *et al.* The natural course of *Chlamydia trachomatis* infection in asymptomatic Colombian women: a 5-year follow-up study. *Journal of Infectious Diseases* 2005; **191**:907-916.
88. Morré SA, van den Brule AJ, Rozendaal L, Boeke AJ, Voorhorst FJ, de Blok S, *et al.* The natural course of asymptomatic *Chlamydia trachomatis* infections: 45% clearance and no development of clinical PID after one-year follow-up. *International Journal of STD and AIDS* 2002; **13** (Suppl 2):12-18.
89. Lyng J, Christensen J. A double-blind study of the value of treatment with a single dose tinidazole of partners to females with trichomoniasis. *Acta Obstetricia et Gynecologica Scandinavica* 1981; **60**:199-201.
90. Ackers JP. Immunologic aspects of human trichomoniasis. In: *Trichomonads Parasitic in Humans*. Edited by Honigberg BM. New York: Springer-Verlag; 1990. pp. 36-52.
91. Watson-Jones D, Mugeye K, Mayaud P, Ndeki L, Todd J, Mosha F, *et al.* High prevalence of trichomoniasis in rural men in Mwanza, Tanzania: results from a population based study. *Sexually Transmitted Infections* 2000; **76**:355-362.
92. Wølner-Hanssen P, Krieger JN, Stevens CE, Kiviat NB, Koutsky L, Critchlow C, *et al.* Clinical manifestations of vaginal trichomoniasis. *Journal of the American Medical Association* 1989; **261**:571-576.

93. Thomas T, Choudhri S, Kariuki C, Moses S. Identifying cervical infection among pregnant women in Nairobi, Kenya: limitations of risk assessment and symptom-based approaches. *Genitourinary Medicine* 1996; **72**:334-338.
94. Price MA, Zimba D, Hoffman IF, Kaydos-Daniels SC, Miller WC, Martinson F, *et al.* Addition of treatment for trichomoniasis to syndromic management of urethritis in Malawi: a randomized clinical trial. *Sexually Transmitted Diseases* 2003; **30**:516-522.
95. Weston TET, Nicol CS. Natural history of trichomonal infection in males. *British Journal of Venereal Diseases* 1963; **39**:251-257.
96. Krieger JN, Verdon M, Siegel N, Holmes KK. Natural history of urogenital trichomoniasis in men. *Journal of Urology* 1993; **149**:1455-1458.
97. Ipsen J, Feigl P. A biomathematical model for prevalence of *Trichomonas vaginalis*. *American Journal of Epidemiology* 1970; **91**:175-184.
98. Bowden FJ, Garnett GP. *Trichomonas vaginalis* epidemiology: parameterizing and analyzing a model of treatment interventions. *Sexually Transmitted Infections* 2000; **76**:248-256.
99. White RG, Orroth KK, Korenromp EL, Bakker R, Wambura M, Sewankambo NK, *et al.* Can population differences explain the contrasting results of the Mwanza, Rakai, and Masaka HIV/sexually transmitted disease intervention trials?: A modeling study. *Journal of Acquired Immune Deficiency Syndromes* 2004; **37**:1500-1513.
100. Ganesan A, Mesner O, Okulicz JF, O'Bryan T, Deiss RG, Lalani T, *et al.* A single dose of benzathine penicillin G is as effective as multiple doses of benzathine penicillin G for the treatment of HIV-infected persons with early syphilis. *Clinical Infectious Diseases* 2015; **60**:653-660.
101. Schober PC, Gabriel G, White P, Felton WF, Thin RN. How infectious is syphilis? *British Journal of Venereal Diseases* 1983; **59**:217-219.
102. Garnett GP, Aral SO, Hoyle DV, Cates W, Anderson RM. The natural history of syphilis: implications for the transmission dynamics and control of infection. *Sexually Transmitted Diseases* 1997; **24**:185-200.
103. Rolfs RT, Joesoef MR, Hendershot EF, Rompalo AM, Augenbraun MH, Chiu M, *et al.* A randomized trial of enhanced therapy for early syphilis in patients with and without human immunodeficiency virus infection. *New England Journal of Medicine* 1997; **337**:307-314.
104. Schroeter AL, Lucas JB, Price EV, Falcone VH. Treatment for early syphilis and reactivity of serologic tests. *Journal of the American Medical Association* 1972; **221**:471-476.
105. Hutchinson CM, Hook EW, 3rd, Shepherd M, Verley J, Rompalo AM. Altered clinical presentation of early syphilis in patients with human immunodeficiency virus infection. *Annals of Internal Medicine* 1994; **121**:94-100.
106. Orle KA, Gates CA, Martin DH, Body BA, Weiss JB. Simultaneous PCR detection of Haemophilus ducreyi, Treponema pallidum, and Herpes simplex virus types 1 and 2 from genital ulcers. *Journal of Clinical Microbiology* 1996; **34**:49-54.
107. Morse SA, Trees DL, Htun Y, Radebe F, Orle KA, Dangor Y, *et al.* Comparison of clinical diagnosis and standard laboratory and molecular methods for the diagnosis of genital ulcer disease in Lesotho: association with human immunodeficiency virus infection. *Journal of Infectious Diseases* 1997; **175**:583-589.
108. Schroeter AL, Turner RH, Lucas JB, Brown WJ. Therapy for incubating syphilis: effectiveness of gonorrhea treatment. *Journal of the American Medical Association* 1971; **218**:711-713.

109. Korenromp EL, Bakker R, de Vlas SJ, Gray RH, Wawer MJ, Serwadda D, *et al.* HIV dynamics and behaviour change as determinants of the impact of sexually transmitted disease treatment on HIV transmission in the context of the Rakai trial. *AIDS* 2002; **16**:2209-2218.
110. Chilongozi DA, Daly CC, Franco L, Liomba NG, Dallabetta G. Sexually transmitted diseases: a survey of case management in Malawi. *International Journal of STD and AIDS* 1996; **7**:269-275.
111. Wald A, Zeh J, Selke S, Warren T, Ryncarz AJ, Ashley R, *et al.* Reactivation of genital herpes simplex virus type 2 infection in asymptomatic seropositive patients. *New England Journal of Medicine* 2000; **342**:844-850.
112. Wald A, Zeh J, Selke S, Ashley RL, Corey L. Virologic characteristics of subclinical and symptomatic genital herpes infections. *New England Journal of Medicine* 1995; **333**:770-775.
113. Straus SE, Seidlin M, Takiff HE, Rooney JF, Felser JM, Smith HA, *et al.* Effect of oral acyclovir treatment on symptomatic and asymptomatic virus shedding in recurrent genital herpes. *Sexually Transmitted Diseases* 1989; **16**:107-113.
114. Harger JH, Amortegui AJ, Meyer MP, Pazin GJ. Characteristics of recurrent genital herpes simplex infections in pregnant women. *Obstetrics and Gynecology* 1989; **73**:367-372.
115. LeGoff J, Weiss HA, Gresenguet G, Nzambi K, Frost E, Hayes RJ, *et al.* Cervicovaginal HIV-1 and herpes simplex virus type 2 shedding during genital ulcer disease episodes. *AIDS* 2007; **21**:1569-1578.
116. Augenbraun M, Feldman J, Chirgwin K, Zenilman J, Clarke L, DeHovitz J, *et al.* Increased genital shedding of herpes simplex virus type 2 in HIV-seropositve women. *Annals of Internal Medicine* 1995; **123**:845-847.
117. Ghys PD, Diallo MO, Ettiègne-Traoré V, Yeboué KM, Gnaoré E, Lorougnon F, *et al.* Genital ulcers associated with human immunodeficiency virus-related immunosuppression in female sex workers in Abidjan, Ivory Coast. *Journal of Infectious Diseases* 1995; **172**:1371-1374.
118. McFarland W, Gwanzura L, Bassett MT, Machekano R, Latif AS, Ley C, *et al.* Prevalence and incidence of herpes simplex virus type 2 infection among male Zimbabwean factory workers. *Journal of Infectious Diseases* 1999; **180**:1459-1465.
119. Fleming DT, McQuillan GM, Johnson RE, Nahmias AJ, Aral SO, Lee FK, *et al.* Herpes simplex virus type 2 in the United States, 1976 to 1994. *New England Journal of Medicine* 1997; **337**:1105-1111.
120. Koutsky LA, Ashley RL, Holmes KK, Stevens CE, Critchlow CW, Kiviat N, *et al.* The frequency of unrecognized type 2 herpes simplex virus infection among women. Implications for the control of genital herpes. *Sexually Transmitted Diseases* 1990; **17**:90-94.
121. Cowan FM, Johnson AM, Ashley R, Corey L, Mindel A. Relationship between antibodies to herpes simplex virus (HSV) and symptoms of HSV infection. *Journal of Infectious Diseases* 1996; **174**:470-475.
122. Koelle DM, Benedetti J, Langenberg A, Corey L. Asymptomatic reactivation of herpes simplex virus in women after the first episode of genital herpes. *Annals of Internal Medicine* 1992; **116**:433-437.
123. Corey L, Adams HG, Brown ZA, Holmes KK. Genital herpes simplex virus infections: clinical manifestations, course, and complications. *Annals of Internal Medicine* 1983; **98**:958-972.

124. Benedetti JK, Zeh J, Corey L. Clinical reactivation of genital herpes simplex virus infection decreases in frequency over time. *Annals of Internal Medicine* 1999; **131**:14-20.
125. Benedetti J, Corey L, Ashley R. Recurrence rates in genital herpes after symptomatic first-episode infection. *Annals of Internal Medicine* 1994; **121**:847-854.
126. Obasi A, Mosha F, Quigley M, Sekirassa Z, Gibbs T, Munguti K, *et al.* Antibody to herpes simplex virus type 2 as a marker of sexual risk behavior in rural Tanzania. *Journal of Infectious Diseases* 1999; **179**:16-24.
127. Schacker T, Zeh J, Hu H, Hill E, Corey L. Frequency of symptomatic and asymptomatic herpes simplex virus type 2 reactivations among human immunodeficiency virus-infected men. *Journal of Infectious Diseases* 1998; **178**:1616-1622.
128. Brown ZA, Vontver LA, Benedetti J, Critchlow CW, Hickok DE, Sells CJ, *et al.* Genital herpes in pregnancy: risk factors associated with recurrences and asymptomatic viral shedding. *American Journal of Obstetrics and Gynecology* 1985; **153**:24-30.
129. Rattray MC, Corey L, Reeves WC, Vontver LA, Holmes KK. Recurrent genital herpes among women: symptomatic v. asymptomatic viral shedding. *British Journal of Venereal Diseases* 1978; **54**:262-265.
130. Ramjee G, Williams B, Gouws E, Van Dyck E, De Deken B, Abdool Karim S. The impact of incident and prevalent herpes simplex virus-2 infection on the incidence of HIV-1 infection among commercial sex workers in South Africa. *Journal of Acquired Immune Deficiency Syndromes* 2005; **39**:333-339.
131. Wald A, Krantz E, Selke S, Lairson E, Morrow RA, Zeh J. Knowledge of partners' genital herpes protects against herpes simplex virus type 2 acquisition. *Journal of Infectious Diseases* 2006; **194**:42-52.
132. Schiffer JT, Mayer BT, Fong Y, Swan DA, Wald A. Herpes simplex virus-2 transmission probability estimates based on quantity of viral shedding. *Journal of the Royal Society Interface* 2014; **11**:20140160.
133. Wald A, Langenberg AGM, Link K, Izu AE, Ashley R, Warren T, *et al.* Effect of condoms on reducing the transmission of herpes simplex virus type 2 from men to women. *Journal of the American Medical Association* 2001; **285**:3100-3106.
134. Wawer MJ, Gray RH, Sewankambo NK, Serwadda D, Li X, Laeyendecker O, *et al.* Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *Journal of Infectious Diseases* 2005; **191**:1403-1409.
135. Johnson LF, Dorrington RE. Modelling the demographic impact of HIV/AIDS in South Africa and the likely impact of interventions. *Demographic Research* 2006; **14**:541-574.
136. Johnson LF, Dorrington RE, Matthews AP. An investigation into the extent of uncertainty surrounding estimates of the impact of HIV/AIDS in South Africa. *South African Journal of Science* 2007; **103**:135-140.
137. Johnson LF, Mossong J, Dorrington RE, Schomaker M, Hoffmann CJ, Keiser O, *et al.* Life expectancies of South African adults starting antiretroviral treatment: Collaborative analysis of cohort studies. *PLoS Medicine* 2013; **10**:e1001418.
138. Hollingsworth TD, Anderson RM, Fraser C. HIV-1 transmission, by stage of infection. *Journal of Infectious Diseases* 2008; **198**:687-693.
139. Cohen MS, Dye C, Fraser C, Miller WC, Powers KA, Williams BG. HIV treatment as prevention: debate and commentary - will early infection compromise treatment-as-prevention strategies? *PLoS Medicine* 2012; **9**:e1001232.

140. Boily MC, Baggaley RF, Wang L, Masse B, White RG, Hayes RJ, *et al.* Heterosexual risk of HIV-1 infection per sexual act: systematic review and meta-analysis of observational studies. *Lancet Infectious Diseases* 2009; **9**:118-129.
141. Ramjee G, Gouws E. Prevalence of HIV among truck drivers visiting sex workers in KwaZulu-Natal, South Africa. *Sexually Transmitted Diseases* 2002; **29**:44-49.
142. Ramjee G, Weber AE, Morar NS. Recording sexual behavior: comparison of recall questionnaires with a coital diary. *Sexually Transmitted Diseases* 1999; **26**:374-380.
143. Hayes RJ, Schulz KF, Plummer FA. The cofactor effect of genital ulcers on the per-exposure risk of HIV transmission in sub-Saharan Africa. *Journal of Tropical Medicine and Hygiene* 1995; **98**:1-8.
144. Auvert B, Ballard R, Campbell C, Caraël M, Carton M, Fehler G, *et al.* HIV infection in a South African mining town is associated with herpes simplex virus-2 seropositivity and sexual behaviour. *AIDS* 2001; **15**:885-898.
145. Pettifor AE, Hudgens MG, Levandowski BA, Rees HV, Cohen MS. Highly efficient HIV transmission to young women in South Africa. *AIDS* 2007; **21**:861-865.
146. Mahiane SG, Legeai C, Taljaard D, Latouche A, Puren A, Peillon A, *et al.* Transmission probabilities of HIV and herpes simplex virus type 2, effect of male circumcision and interaction: a longitudinal study in a township of South Africa. *AIDS* 2009; **23**:377-383.
147. Baeten JM, Richardson BA, Lavreys L, Rakwar JP, Mandaliya K, Bwayo JJ, *et al.* Female-to-male infectivity of HIV-1 among circumcised and uncircumcised Kenyan men. *Journal of Infectious Diseases* 2005; **191**:546-553.
148. Gray R, Wawer M, Brookmeyer R, Sewankambo N, Serwadda D, Wabwire-Mangen F, *et al.* Probability of HIV-1 transmission per coital act in monogamous, heterosexual, HIV-1-discordant couples in Rakai, Uganda. *Lancet* 2001; **357**:1149-1153.
149. Allen S, Tice J, Van de Perre P, Serufilira A, Hudes E, Nsengumuremyi F, *et al.* Effect of serotesting with counselling on condom use and seroconversion among HIV discordant couples in Africa. *British Medical Journal* 1992; **304**:1605-1609.
150. Hughes JP, Baeten JM, Lingappa JR, Magaret AS, Wald A, de Bruyn G, *et al.* Determinants of per-coital-act HIV-1 infectivity among African HIV-1-serodiscordant couples. *Journal of Infectious Diseases* 2012; **205**:358-365.
151. Department of National Health and Population Development. First national HIV survey of women attending antenatal clinics, South Africa, October/November 1990. *Epidemiological comments.* 1991; **18**:35-45.
152. Montana LS, Mishra V, Hong R. Comparison of HIV prevalence estimates from antenatal care surveillance and population-based surveys in sub-Saharan Africa. *Sexually Transmitted Infections* 2008; **84** (Suppl 1):i78-i84.
153. Williams BG, Gouws E, Colvin M, Sitas F, Ramjee G, Abdool Karim SS. Patterns of infection: using age prevalence data to understand the epidemic of HIV in South Africa. *South African Journal of Science* 2000; **96**:305-312.
154. Nugent RP, Krohn MA, Hillier SL. Reliability of diagnosing bacterial vaginosis is improved by a standardized method of Gram stain interpretation. *Journal of Clinical Microbiology* 1991; **29**:297-301.
155. Hillier SL, Krohn MA, Nugent RP, Gibbs RS. Characteristics of three vaginal flora patterns assessed by Gram stain among pregnant women. *American Journal of Obstetrics and Gynecology* 1992; **166**:938-944.
156. Smart S, Singal A, Mindel A. Social and sexual risk factors for bacterial vaginosis. *Sexually Transmitted Infections* 2004; **80**:58-62.

157. Ness RB, Kip KE, Soper DE, Stamm CA, Rice P, Richter HE. Variability of bacterial vaginosis over 6- to 12-month intervals. *Sexually Transmitted Diseases* 2006; **33**:381-385.
158. Bradshaw CS, Morton AN, Garland SM, Morris MB, Moss LM, Fairley CK. Higher-risk behavioral practices associated with bacterial vaginosis compared with vaginal candidiasis. *Obstetrics and Gynecology* 2005; **106**:105-114.
159. Shoubnikova M, Hellberg D, Nilsson S, Mardh PA. Contraceptive use in women with bacterial vaginosis. *Contraception* 1997; **55**:355-358.
160. Yen S, Shafer MA, Moncada J, Campbell CJ, Flinn SD, Boyer CB. Bacterial vaginosis in sexually experienced and non-sexually experienced young women entering the military. *Obstetrics and Gynecology* 2003; **102**:927-933.
161. Amsel R, Totten PA, Spiegel CA, Chen KCS, Eschenbach D, Holmes KK. Nonspecific vaginitis. Diagnostic criteria and microbial and epidemiologic associations. *American Journal of Medicine* 1983; **74**:14-22.
162. Eschenbach DA, Hillier S, Critchlow C, Stevens C, DeRouen T, Holmes KK. Diagnosis and clinical manifestations of bacterial vaginosis. *American Journal of Obstetrics and Gynecology* 1988; **158**:819-828.
163. Wilkinson D, Ndovela N, Harrison A, Lurie M, Connolly C, Sturm AW. Family planning services in developing countries: an opportunity to treat asymptomatic and unrecognized genital tract infections? *Genitourinary Medicine* 1997; **73**:558-560.
164. Calderone RA, Fonzi WA. Virulence factors of *Candida albicans*. *Trends in Microbiology* 2001; **9**:327-335.
165. Sobel JD, Wiesenfeld HC, Martens M, Danna P, Hooton TM, Rompalo A, *et al.* Maintenance fluconazole therapy for recurrent vulvovaginal candidiasis. *New England Journal of Medicine* 2004; **351**:876-883.
166. El-Din SS, Reynolds MT, Ashbee HR, Barton RC, Evans EG. An investigation into the pathogenesis of vulvo-vaginal candidosis. *Sexually Transmitted Infections* 2001; **77**:179-183.
167. Sobel JD. Candidal vulvovaginitis. *Clinical Obstetrics and Gynecology* 1993; **36**:153-165.
168. Geiger AM, Foxman B, Gillespie BW. The epidemiology of vulvovaginal candidiasis among university students. *American Journal of Public Health* 1995; **85**:1146-1148.
169. Ferrer J. Vaginal candidosis: epidemiological and etiological factors. *International Journal of Gynaecology and Obstetrics* 2000; **71** (Suppl 1):S21-27.
170. Ohmit SE, Sobel JD, Schuman P, Duerr A, Mayer K, Rompalo A, *et al.* Longitudinal study of mucosal *Candida* species colonization and candidiasis among human immunodeficiency virus (HIV)-seropositive and at-risk HIV-seronegative women. *Journal of Infectious Diseases* 2003; **188**:118-127.
171. Daus AD, Hafez ES. *Candida albicans* in women. *Nursing Research* 1975; **24**:430-433.
172. McClelland RS, Lavreys L, Katingima C, Overbaugh J, Chohan V, Mandaliya K, *et al.* Contribution of HIV-1 infection to acquisition of sexually transmitted disease: a 10-year prospective study. *Journal of Infectious Diseases* 2005; **191**:333-338.
173. Duerr A, Sierra MF, Feldman J, Clarke LM, Ehrlich I, DeHovitz J. Immune compromise and prevalence of Candida vulvovaginitis in human immunodeficiency virus-infected women. *Obstetrics and Gynecology* 1997; **90**:252-256.
174. Rugpao S, Nagachinta T, Wanapirak C, Srisomboon J, Suriyanon V, Sirirojn B, *et al.* Gynaecological conditions associated with HIV infection in women who are partners

of HIV-positive Thai blood donors. *International Journal of STD and AIDS* 1998; **9**:677-682.
175. Minkoff HL, Eisenberger-Matityahu D, Feldman J, Burk R, Clarke L. Prevalence and incidence of gynecologic disorders among women infected with human immunodeficiency virus. *American Journal of Obstetrics and Gynecology* 1999; **180**:824-836.
176. Brown D, Henzl MR, LePage ME, Tsao L, Izu AE, Walker KAM, *et al.* Butoconazole vaginal cream in the treatment of vulvovaginal candidiasis. *Journal of Reproductive Medicine* 1986; **31**:1045-1048.
177. Dladla-Qwabe N, Govinden R, Ramjee G, Masse B, Kelly C. HIV and STI prevalence and incidence in urban and rural KwaZulu-Natal [Abstract P203]. *Second South African AIDS Conference*. Durban, South Africa; 2005.
178. Barbone F, Austin H, Louv WC, Alexander WJ. A follow-up study of methods of contraception, sexual activity, and rates of trichomoniasis, candidiasis, and bacterial vaginosis. *American Journal of Obstetrics and Gynecology* 1990; **163**:510-514.
179. Fennema JS, van Ameijden EJ, Coutinho RA, van den Hoek AA. HIV, sexually transmitted diseases and gynaecologic disorders in women: increased risk for genital herpes and warts among HIV-infected prostitutes in Amsterdam. *AIDS* 1995; **9**:1071-1078.
180. Fleming DT, Leone P, Esposito D, Heitman CK, Justus S, Chin S, *et al.* Herpes virus type 2 infection and genital symptoms in primary care patients. *Sexually Transmitted Diseases* 2006; **33**:416-421.
181. Foxman B, Barlow R, D'Arcy H, Gillespie B, Sobel JD. Candida vaginitis: self-reported incidence and associated costs. *Sexually Transmitted Diseases* 2000; **27**:230-235.
182. Wilkinson D, Connolly A, Harrison A, Lurie M, Abdool Karim SS. Sexually transmitted disease syndromes in rural South Africa: results from health facility surveillance. *Sexually Transmitted Diseases* 1998; **25**:20-23.
183. O'Farrell N, Hoosen AA, Coetzee KD, Van den Ende J. Genital ulcer disease in men in Durban, South Africa. *Genitourinary Medicine* 1991; **67**:327-330.
184. O'Farrell N, Hoosen AA, Coetzee KD, Van den Ende J. Genital ulcer disease in women in Durban, South Africa. *Genitourinary Medicine* 1991; **67**:322-326.
185. Reddy SP, Panday S, Swart D, Jinabhai CC, Amosun SL, James S, *et al.* Umthenthe Uhlaba Usamila - The South African Youth Risk Behaviour Survey 2002. Cape Town: South African Medical Research Council; 2003. Available: http://www.mrc.ac.za/healthpromotion/healthpromotion.htm. Accessed 15 Dec 2003
186. Lewis JJ, Garnett GP, Nyamukapa CA, Donnelly CA, Mason PR, Gregson S. Patterns of uptake of treatment for self reported sexually transmitted infection symptoms in rural Zimbabwe. *Sexually Transmitted Infections* 2005; **81**:326-332.
187. Wilkinson D, Wilkinson N. HIV infection among patients with sexually transmitted diseases in rural South Africa. *International Journal of STD and AIDS* 1998; **9**:736-739.
188. Wilson T, Strebel A, Simbayi L, Andipatin M, Potgieter C, Ratele K, *et al.* Health care-seeking behaviour for sexually transmitted diseases in South Africa. *Journal of Psychology in Africa* 2000; **10**:101-121.
189. Ndhlovu L, Searle C, Van Dam J, Mzaidume Y, Rasego B, Moema S. Reducing the transmission of HIV and sexually transmitted infections in a mining community: Findings from the Carletonville Mothusimpilo intervention project: 1998 to 2001.

Washington, DC: Population Council; 2005. Available: http://www.popcouncil.org/pdfs/horizons/crltnvll.pdf. Accessed 1 Dec 2006
190. Eaton JW, Johnson LF, Salomon JA, Bärnighausen T, Bendavid E, Bershteyn A, *et al.* HIV treatment as prevention: systematic comparison of mathematical models of the potential impact of antiretroviral therapy on HIV incidence in South Africa. *PLoS Medicine* 2012; **9**:e1001245.
191. Lewis DA, Scott L, Slabbert M, Mkhonto S, van Zijl A, Ntuli D, *et al.* Escalation in the prevalence of ciprofloxacin resistant gonorrhoea in men presenting with urethritis to public health facilities in two South African cities [Abstract P-449]. *17th International Society for STD Research World Congress*. Seattle, USA; 2007.
192. Black V, Magooa P, Radebe F, Myers M, Pillay C, Lewis DA. The detection of urethritis pathogens among patients with the male urethritis syndrome, genital ulcer syndrome and HIV VCT clients: should South Africa's syndromic management approach be revised? *Sexually Transmitted Infections* 2008; **84**:254-258.
193. Moodley P, Sturm AW. Ciprofloxacin-resistant gonorrhoea in South Africa. *Lancet* 2005; **366**:1159.
194. Department of Health. First Line Comprehensive Management and Control of Sexually Transmitted Infections (STIs). Pretoria; 2009. Available: http://www.nicd.ac.za/units/stirc. Accessed 10 July 2009
195. Harrison A, Wilkinson D, Lurie M, Connolly AM, Abdool Karim S. Improving quality of sexually transmitted disease case management in rural South Africa. *AIDS* 1998; **12**:2329-2335.
196. Dartnall E, Schneider H, Hlatswayo Z, Clews F. STD management in the private sector: a national evaluation. Centre for Health Policy; 1997.
197. Pick W, Fisher B, Kowo H, Conway S, Kgosidintsi N, Weiner R. Measuring quality of care in South African clinics and hospitals. (Technical report to chapter 14 of the South African Health Review 1998). Department of Community Health, University of Witwatersrand; 1998. Available: http://www.hst.org.za/uploads/files/care_98.pdf. Accessed 6 Aug 2010
198. Schneider H, Chabikuli N, Blaauw D, Funani I, Brugha R. Sexually transmitted infections - factors associated with quality of care among private general practitioners. *South African Medical Journal* 2005; **95**:782-785.
199. Ramkissoon A, Kleinschmidt I, Beksinska M, Smit J, Hlazo J, Mabude Z. National Baseline Assessment of Sexually Transmitted Infection and HIV Services in South African Public Sector Health Facilities. Durban: Reproductive Health Research Unit; 2004. Available: http://www.rhru.co.za. Accessed 13 February 2004
200. Reagon G, Irlam J, Levin J. The National Primary Health Care Facilities Survey 2003. Durban: Health Systems Trust; 2004. Available: http://www.hst.org.za/publications/617. Accessed 6 Aug 2010
201. Department of Health. The 2012 National Antenatal Sentinel HIV and Herpes Simplex Type-2 Prevalence Survey in South Africa. 2014. Available: http://www.health.gov.za/reports.php. Accessed 14 May 2014
202. Shisana O, Rehle T, Simbayi LC, Zuma K, Jooste S, Zungu N, *et al.* South African National HIV Prevalence, Incidence, and Behaviour Survey, 2012. Cape Town: Human Sciences Research Council; 2014. Available: http://www.hsrc.ac.za/en/research-outputs/view/6871. Accessed 16 April 2014
203. Coetzee N. Syphilis in women attending antenatal clinics in the Western Cape: a pilot investigation into the use of a laboratory based surveillance scheme. University of Cape Town Department of Community Health; 1994.

204. Opai-Tetteh ET, Hoosen AA, Moodley J. Re-screening for syphilis at the time of delivery in areas of high prevalence. *South African Medical Journal* 1993; **83**:725-726.
205. Bam RH, Cronje HS, Muir A, Griessel DJ, Hoek BB. Syphilis in pregnant patients and their offspring. *International Journal of Gynaecology and Obstetrics* 1994; **44**:113-118.
206. Hoosen A, Nteta C, Moodley J, Sturm A. Diagnosis of bacterial vaginosis and its effect on pregnancy outcome: a preliminary study. *Southern African Journal of Epidemiology and Infection* 1996; **11**:104-106.
207. Qolohle DC, Hoosen AA, Moodley J, Smith AN, K.P. M. Serological screening for sexually transmitted infections in pregnancy: is there any value in re-screening for HIV and syphilis at the time of delivery? *Genitourinary Medicine* 1995; **71**:65-67.
208. Govender L, Hoosen AA, Moodley J, Moodley P, Sturm AW. Bacterial vaginosis and associated infections in pregnancy. *International Journal of Gynecology and Obstetrics* 1996; **55**:23-28.
209. Kharsany ABM, Hoosen AA, Moodley J. Bacterial vaginosis and lower genital tract infections in women attending out-patient clinics at a tertiary institution serving a developing community. *Journal of Obstetrics and Gynaecology* 1997; **17**:171-175.
210. Sturm A, Moodley P, Sturm P, Karim F, Khan N. Trends in the prevalence of sexually transmitted infections and HIV in pregnant women in KwaZulu/Natal from 1995 to 2002. *South African AIDS Conference*. Durban, South Africa; 2003.
211. Sturm AW, Wilkinson D, Ndovela N, Bowen S, Connolly C. Pregnant women as a reservoir of undetected sexually transmitted diseases in rural South Africa: implications for disease control. *American Journal of Public Health* 1998; **88**:1243-1245.
212. Mashiane Y, Chephe JH, Hoosen AA. Syphilis in pregnancy at Ga-Rankuwa Hospital: high prevalence and the need for intervention. *Southern African Journal of Epidemiology and Infection* 1999; **14**:13-15.
213. Dawadi BR, Joubert G, Hiemstra LA. Syphilis serology testing of pregnant women in Whittlesea district, Eastern Cape province, South Africa. *Southern African Journal of Epidemiology and Infection* 2001; **16**:117-121.
214. Myer L, Wilkinson D, Lombard C, Zuma K, Rotchford K, Abdool Karim SS. Impact of on-site testing for maternal syphilis on treatment delays, treatment rates, and perinatal mortality in rural South Africa: a randomised control trial. *Sexually Transmitted Infections* 2003; **79**:208-213.
215. Bronzan RN, Mwesigwa-Kayongo DC, Narkunas D, Schmid GP, Neilsen GA, Ballard RC, *et al.* Onsite rapid antenatal syphilis screening with an immunochromatographic strip improves case detection and treatment in rural South African clinics. *Sexually Transmitted Diseases* 2007; **34**:S55-60.
216. Mothupi R, Mtezuka L, Bereda E, Mbelle N, Moodley S, Buchmann EJ. A retrospective study of the seroprevalence of syphilis in antenatal admissions at Chris Hani Baragwanath Hospital [Abstract 64]. *2nd Joint Congress of the Federation of Infectious Diseases Societies of Southern Africa*. Cape Town, South Africa; 2007.
217. Frohlich JA, Abdool Karim Q, Mashego MM, Sturm AW, Abdool Karim SS. Opportunities for treating sexually transmitted infections and reducing HIV risk in rural South Africa. *Journal of Advanced Nursing* 2007; **60**:377-383.
218. Sebitloane HM, Moodley J, Esterhuizen TM. Pathogenic lower genital tract organisms in HIV-infected and uninfected women, and their association with postpartum infectious morbidity. *South African Medical Journal* 2011; **101**:466-469.

219. Dinh TH, Kamb ML, Msimang V, Likibi M, Molebatsi T, Goldman T, *et al.* Integration of preventing mother-to-child transmission of HIV and syphilis testing and treatment in antenatal care services in the Northern Cape and Gauteng provinces, South Africa. *Sexually Transmitted Diseases* 2013; **40**:846-851.
220. Devjee J, Moodley J, Singh M. Syphilis in pregnancy - prevalence at different levels of health care in Durban. *South African Medical Journal* 2006; **96**:1182-1184.
221. Perti T, Nyati M, Gray G, De Bruyn G, Selke S, Magaret A, *et al.* Frequent genital HSV-2 shedding among women during labor in Soweto, South Africa. *Infectious Diseases in Obstetrics and Gynecology* 2014; **2014**:258291.
222. Steen R, Vuylsteke B, DeCoito T, Ralepeli S, Fehler G, Conley J, *et al.* Evidence of declining STD prevalence in a South African mining community following a core-group intervention. *Sexually Transmitted Diseases* 2000; **27**:1-8.
223. Williams BG, Taljaard D, Campbell CM, Gouws E, Ndhlovu L, van Dam J, *et al.* Changing patterns of knowledge, reported behaviour and sexually transmitted infections in a South African gold mining community. *AIDS* 2003; **17**:2099-2107.
224. Vickerman P, Terris-Prestholt F, Delany S, Kumaranayake L, Rees H, Watts C. Are targeted HIV prevention activities cost-effective in high prevalence settings? Results from a sexually transmitted infection treatment project for sex workers in Johannesburg, South Africa. *Sexually Transmitted Diseases* 2006; **33**:S122-132.
225. Schneider H, Coetzee DJ, Fehler HG, Bellingan A, Dangor Y, Radebe F, *et al.* Screening for sexually transmitted diseases in rural South African women. *Sexually Transmitted Infections* 1998; **74**:S147-S152.
226. Hoosen AA, Moodley J, Maitin P, Sturm AW. Bacterial vaginosis in symptomatic women attending a gynaecology outpatient clinic. *Southern African Journal of Epidemiology and Infection* 1997; **12**:119-121.
227. Fehler HG, Lyall M, Htun Y, Muiznieks S, Khoza BD, Ballard RC. Genital tract infections among women attending an urban family planning clinic. *Southern African Journal of Epidemiology and Infection* 1998; **13**:79-82.
228. Cronje HS, Joubert G, Muir A, Chapman RD, Divall P, Bam RH. Prevalence of vaginitis, syphilis and HIV infection in women in the Orange Free State. *South African Medical Journal* 1994; **84**:602-605.
229. Colvin M, Abdool Karim SS, Connolly C, Hoosen AA, Ntuli N. HIV infection and asymptomatic sexually transmitted infections in a rural South African community. *International Journal of STD and AIDS* 1998; **9**:548-550.
230. Auvert B, Males S, Puren A, Taljaard D, Carael M, Williams B. Can highly active antiretroviral therapy reduce the spread of HIV? A study in a township of South Africa. *Journal of Acquired Immune Deficiency Syndromes* 2004; **36**:613-621.
231. Rours GIJG, Verkooyen RP, Ye H, Radebe F, Rothberg AD, Cooper PA, *et al.* Sexually transmitted infections in pregnant urban South African women: socio-economic characteristics and risk factors. *Southern African Journal of Epidemiology and Infection* 2006; **21**:14-19.
232. Sturm PD, Connolly C, Khan N, Ebrahim S, Sturm AW. Vaginal tampons as specimen collection device for the molecular diagnosis of non-ulcerative sexually transmitted infections in antenatal clinic attendees. *International Journal of STD and AIDS* 2004; **15**:94-98.
233. Odendaal HJ, Schoeman J, Grové D, de Jager M, Theron GB, Orth H, *et al.* The association between Chlamydia trachomatis genital infection and spontaneous preterm labour. *South African Journal of Obstetrics and Gynaecology* 2006; **12**:146-149.

234. Moodley D, Moodley P, Sebitloane M, Soowamber D, McNaughton-Reyes HL, Groves AK, *et al.* High prevalence and incidence of asymptomatic sexually transmitted infections during pregnancy and postdelivery in KwaZulu Natal, South Africa. *Sexually Transmitted Diseases* 2015; **42**:43-47.
235. Kleinschmidt I, Rees H, Delany S, Smith D, Dinat N, Nkala B, *et al.* Injectable progestin contraceptive use and risk of HIV infection in a South African family planning cohort. *Contraception* 2007; **75**:461-467.
236. Pettifor AE, Kleinschmidt I, Levin J, Rees HV, MacPhail C, Madikizela-Hlongwa L, *et al.* A community-based study to examine the effect of a youth HIV prevention intervention on young people aged 15-24 in South Africa: results of the baseline survey. *Tropical Medicine and International Health* 2005; **10**:971-980.
237. Hurkchand HP, Levine JB, Makuluma H, Molefe N. Measuring the impact of HIV and STIs in a cluster designed household prevalence survey in a coal mining community, eMbalenhle, Mpumalanga, South Africa [Abstract ThPeC7319]. *15th International AIDS Conference*. Bangkok, Thailand; 2004.
238. O'Leary A, Jemmott JB, Jemmott LS, Teitelman A, Anita Heeren G, Ngwane Z, *et al.* Associations between psychosocial factors and incidence of sexually transmitted disease among South african adolescents. *Sexually Transmitted Diseases* 2015; **42**:135-139.
239. Govender S, Theron GB, Odendaal HJ, Chalkley LJ. Prevalence of genital mycoplasmas, ureaplasmas and *chlamydia* in pregnancy. *Journal of Obstetrics and Gynaecology* 2009; **29**:698-701.
240. Funk M, Pistorius LR, Pattison RC. Antenatal screening for bacterial vaginosis using the amine test. *Southern African Journal of Epidemiology and Infection* 1996; **11**:74-76.
241. Charumilind S, Abdolrazoli A, Oliff M, Veary J, Moyo W, Andersen A, *et al.* *Trichomonas vaginalis* infection is associated with HIV in high-risk migrant men in Johannesburg [Abstract 560]. *3rd South African AIDS Conference*. Durban, South Africa; 2007.
242. Sturm AW. Incidence of sexually transmitted infections in pregnant women in rural KwaZulu/Natal. *South African AIDS Conference*. Durban, South Africa; 2003.
243. Mlaba N, Akpomiemie G, Readhead A, Moyes J, Delany-Moretlwe S. Herpes simplex virus type 2 (HSV-2) is associated with age, migration and HIV in women in inner-city Johannesburg [Abstract 555]. *3rd South African AIDS Conference*. Durban, South Africa; 2007.
244. Jewkes R, Nduna M, Levin J, Jama N, Dunkle K, Puren A, *et al.* Impact of Stepping Stones on incidence of HIV and HSV-2 and sexual behaviour in rural South Africa: cluster randomised controlled trial. *British Medical Journal* 2008; **337**:a506.
245. Jean K, Puren A, Cutler E, Singh B, Masseko V, Bouscaillou J, *et al.* The role of male circumcision and antiretroviral drugs in the evolution of the HIV and HSV-2 epidemics in Orange Farm (South Africa) between 2002 and 2012 [MOPEC439]. *8th International AIDS Society Conference on HIV Pathogenesis, Treatment and Prevention*. Vancouver, Canada; 2015.
246. Bruisten SM, Cairo I, Fennema H, Pijl A, Buimer M, Peerbooms PG, *et al.* Diagnosing genital ulcer disease in a clinic for sexually transmitted diseases in Amsterdam, The Netherlands. *Journal of Clinical Microbiology* 2001; **39**:601-605.
247. Risbud A, Chan-Tack K, Gadkari D, Gangakhedkar RR, Shepherd ME, Bollinger R, *et al.* The etiology of genital ulcer disease by multiplex polymerase chain reaction and

relationship to HIV infection among patients attending sexually transmitted disease clinics in Pune, India. *Sexually Transmitted Diseases* 1999; **26**:55-62.
248. Behets FMT, Brathwaite AR, Hylton-Kong T, Chen CY, Hoffman I, Weiss JB, *et al.* Genital ulcers: etiology, clinical diagnosis, and associated human immunodeficiency virus infection in Kingston, Jamaica. *Clinical Infectious Diseases* 1999; **28**:1086-1090.
249. Castro R, Prieto ES, Santo I, Azevedo J, Exposto Fda L. Evaluation of an enzyme immunoassay technique for detection of antibodies against Treponema pallidum. *Journal of Clinical Microbiology* 2003; **41**:250-253.
250. Hooper N, Malloy D, Passen S. Evaluation of a *Treponema pallidum* enzyme immunoassay as a screening test for syphilis. *Clinical and Diagnostic Laboratory Immunology* 1994; **1**:477-481.
251. West B, Walraven G, Morison L, Brouwers J, Bailey R. Performance of the rapid plasma reagin and the rapid syphilis screening tests in the diagnosis of syphilis in field conditions in rural Africa. *Sexually Transmitted Infections* 2002; **78**:282-285.
252. Orroth KK, Korenromp EL, White RG, Changalucha J, de Vlas S, Gray RH, *et al.* Comparison of STD prevalences in the Mwanza, Rakai, and Masaka trial populations: the role of selection bias and diagnostic errors. *Sexually Transmitted Infections* 2003; **79**:98-105.
253. van Doornum GJ, Schouls LM, Pijl A, Cairo I, Buimer M, Bruisten S. Comparison between the LCx Probe system and the COBAS AMPLICOR system for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* infections in patients attending a clinic for treatment of sexually transmitted diseases in Amsterdam, The Netherlands. *Journal of Clinical Microbiology* 2001; **39**:829-835.
254. Cook RL, Hutchison SL, Østergaard L, Braithwaite RS, Ness RB. Systematic review: noninvasive testing for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. *Annals of Internal Medicine* 2005; **142**:914-925.
255. Fontana C, Favaro M, Cicchetti O, Minelli S, Pistoia ES, Favalli C. Performance of Strand Displacement Amplification assay in the detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. *Japanese Journal of Infectious Diseases* 2005; **58**:283-288.
256. Van Der Pol B, Ferrero DV, Buck-Barrington L, Hook E, Lenderman C, Quinn T, *et al.* Multicenter evaluation of the BDProbeTec ET System for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in urine specimens, female endocervical swabs, and male urethral swabs. *Journal of Clinical Microbiology* 2001; **39**:1008-1016.
257. Van Dyck E, Ieven M, Pattyn S, Van Damme L, Laga M. Detection of Chlamydia trachomatis and Neisseria gonorrhoeae by enzyme immunoassay, culture, and three nucleic acid amplification tests. *Journal of Clinical Microbiology* 2001; **39**:1751-1756.
258. Chernesky M, Jang D, Gilchrist J, Hatchette T, Poirier A, Flandin JF, *et al.* Head-to-head comparison of second-generation nucleic acid amplification tests for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* on urine samples from female subjects and self-collected vaginal swabs. *Journal of Clinical Microbiology* 2014; **52**:2305-2310.
259. Mushanski LM, Brandt K, Coffin N, Levett PN, Horsman GB, Rank EL. Comparison of the BD Viper System with XTR Technology to the Gen-Probe APTIMA COMBO 2 Assay using the TIGRIS DTS system for the detection of *Chlamydia trachomatis*

and *Neisseria gonorrhoeae* in urine specimens. *Sexually Transmitted Diseases* 2012; **39**:514-517.
260. Van Der Pol B, Liesenfeld O, Williams JA, Taylor SN, Lillis RA, Body BA, *et al.* Performance of the cobas CT/NG test compared to the Aptima AC2 and Viper CTQ/GCQ assays for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. *Journal of Clinical Microbiology* 2012; **50**:2244-2249.
261. Thejls H, Gnarpe J, Gnarpe H, Larsson PG, Platz-Christensen JJ, Ostergaard L, *et al.* Expanded gold standard in the diagnosis of Chlamydia trachomatis in a low prevalence population: diagnostic efficacy of tissue culture, direct immunofluorescence, enzyme immunoassay, PCR and serology. *Genitourinary Medicine* 1994; **70**:300-303.
262. Mills RD, Young A, Cain K, Blair TM, Sitorius MA, Woods GL. Chlamydiazyme plus blocking assay to detect Chlamydia trachomatis in endocervical specimens. *American Journal of Clinical Pathology* 1992; **97**:209-212.
263. Lefebvre J, Laperrière H, Rousseau H, Massé R. Comparison of three techniques for detection of Chlamydia trachomatis in endocervical specimens from asymptomatic women. *Journal of Clinical Microbiology* 1988; **26**:726-731.
264. Haugland S, Thune T, Fosse B, Wentzel-Larsen T, Hjelmevoll SO, Myrmel H. Comparing urine samples and cervical swabs for Chlamydia testing in a female population by means of Strand Displacement Assay (SDA). *BMC Women's Health* 2010; **10**:9.
265. Wiese W, Patel SR, Patel SC, Ohl CA, Estrada CA. A meta-analysis of the Papanicolaou smear and wet mount for the diagnosis of vaginal trichomoniasis. *American Journal of Medicine* 2000; **108**:301-308.
266. Wendel KA, Erbelding EJ, Gaydos CA, Rompalo AM. *Trichomonas vaginalis* polymerase chain reaction compared with standard diagnostic and therapeutic protocols for detection and treatment of vaginal trichomoniasis. *Clinical Infectious Diseases* 2002; **35**:576-580.
267. Mayta H, Gilman RH, Calderon MM, Gottlieb A, Soto G, Tuero I, *et al.* 18S ribosomal DNA-based PCR for diagnosis of *Trichomonas vaginalis*. *Journal of Clinical Microbiology* 2000; **38**:2683-2687.
268. Madico G, Quinn TC, Rompalo A, McKee KT, Jr., Gaydos CA. Diagnosis of *Trichomonas vaginalis* infection by PCR using vaginal swab samples. *Journal of Clinical Microbiology* 1998; **36**:3205-3210.
269. Patel SR, Wiese W, Patel SC, Ohl C, Byrd JC, Estrada CA. Systematic review of diagnostic tests for vaginal trichomoniasis. *Infectious Diseases in Obstetrics and Gynecology* 2000; **8**:248-257.
270. Nye MB, Schwebke JR, Body BA. Comparison of APTIMA *Trichomonas vaginalis* transcription-mediated amplification to wet mount microscopy, culture, and polymerase chain reaction for diagnosis of trichomoniasis in men and women. *American Journal of Obstetrics and Gynecology* 2009; **200**:188 e181-187.
271. Schwebke JR, Hobbs MM, Taylor SN, Sena AC, Catania MG, Weinbaum BS, *et al.* Molecular testing for *Trichomonas vaginalis* in women: results from a prospective U.S. clinical trial. *Journal of Clinical Microbiology* 2011; **49**:4106-4111.
272. Eing BR, Lippelt L, Lorentzen EU, Hafezi W, Schlumberger W, Steinhagen K, *et al.* Evaluation of confirmatory strategies for detection of type-specific antibodies against herpes simplex virus type 2. *Journal of Clinical Microbiology* 2002; **40**:407-413.
273. Spiezia KV, Dille BJ, Mushahwar IK, Kifle L, Okasinski GF. Prevalence of specific antibodies to herpes simplex virus type 2 as revealed by an enzyme-linked

immunoassay and western blot analysis. *Advances in Experimental Medicine and Biology* 1990; **278**:231-242.
274. Cusini M, Cusan M, Parolin C, Scioccati L, Decleva I, Mengoli C, *et al.* Seroprevalence of herpes simplex virus type 2 infection among attendees of a sexually transmitted disease clinic in Italy. Italian Herpes Forum. *Sexually Transmitted Diseases* 2000; **27**:292-295.
275. Turner K, Wong E, Kent C, Klausner J. Serologic herpes testing in the real world: validation of new type-specific serologic herpes simplex virus tests in a public health laboratory. *Sexually Transmitted Diseases* 2002; **29**:422-425.
276. Gopal R, Gibbs T, Slomka MJ, Whitworth J, Carpenter LM, Vyse A, *et al.* A monoclonal blocking EIA for herpes simplex virus type 2 antibody: validation for seroepidemiological studies in Africa. *Journal of Virological Methods* 2000; **87**:71-80.
277. Eaton JW, Bacaër N, Bershteyn A, Cambiano V, Cori A, Dorrington RE, *et al.* Assessment of epidemic projections using recent HIV survey data in South Africa: a validation analysis of ten mathematical models of HIV epidemiology in the antiretroviral therapy era. *Lancet Global Health* 2015; **3**:e598-608.
278. Kapiga S, Kelly C, Weiss S, Daley T, Peterson L, Leburg C, *et al.* Risk factors for incidence of sexually transmitted infections among women in South Africa, Tanzania, and Zambia: results from HPTN 055 study. *Sexually Transmitted Diseases* 2009; **36**:199-206.
279. Peters RP, Dubbink JH, van der Eem L, Verweij SP, Bos ML, Ouburg S, *et al.* Cross-sectional study of genital, rectal, and pharyngeal chlamydia and gonorrhea in women in rural South Africa. *Sexually Transmitted Diseases* 2014; **41**:564-569.
280. Lloyd-Smith JO, Getz WM, Westerhoff HV. Frequency-dependent incidence in models of sexually transmitted diseases: portrayal of pair-based transmission and effects of illness on contact behaviour. *Proceedings of the Royal Society of London, Series B.* 2004; **271**:625-634.
281. Althaus CL, Turner KM, Schmid BV, Heijne JC, Kretzschmar M, Low N. Transmission of *Chlamydia trachomatis* through sexual partnerships: a comparison between three individual-based models and empirical data. *Journal of the Royal Society Interface* 2012; **9**:136-146.