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The Epidemiological Impact of an HIV Vaccine on the HIV/AIDS Epidemic in Southern India

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Abstract

The potential epidemiological impact of preventive HIV vaccines on the HIV epidemic in Southern India is examined using a mathematical deterministic dynamic compartmental model. Various assumptions about the degree of protection offered by such a vaccine, the extent of immunological response of those vaccinated, and the duration of protection afforded are explored. Alternative targeting strategies for HIV vaccination are simulated and compared with the impact of conventional prevention interventions in high-risk groups and the general population. The impact of disinhibition (increased risk behavior due to the presence of a vaccine) is also considered.

Vaccines that convey a high degree of protection in a share of or all of those immunized and that convey life-long immunity are the most effective in curbing the HIV epidemic. Vaccines that convey less than complete protection may also have substantial public health impact, but disinhibition can easily undo their effects and they should be used combined with conventional prevention efforts. Conventional interventions that target commercial sex workers and their clients to increase condom use can also be highly effective and can be implemented immediately, before the arrival of vaccines.

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Acronyms

ART	Anti-retroviral therapy
CSW	Commercial sex worker
HRG	High-risk group
IDU	Intravenous drug user
MSM	Men who have sex with men
MTCT	Mother-to-child transmission
NACO	National AIDS Control Organisation
STI	Sexually-transmitted infections

1. Introduction

India's current HIV-1 seroprevalence rate of slightly less than 1 percent of adults, or approximately 4 million HIV infected individuals, is bound to increase. In the Southern states of India (Andhra Pradesh, Karnataka, Maharashtra and Tamil Nadu), adult HIV-1 prevalence of approximately 2 percent is already observed (National AIDS Control Organisation website). In some districts, it is already over 4 percent. The four states together account for over 75 percent of all Indian HIV infections, even though they have less than 30 percent of the adult population (Government of India 2002). The engine of the Indian epidemic is almost certainly heterosexual transmission from vulnerable groups, chiefly commercial sex workers (CSWs) and their clients.

HIV infection in vulnerable groups has grown rapidly in India, where control of HIV and sexually transmitted infections (STI) used to be poor. The conditions for further rapid growth are also in place: paid sex is common, rates of STI are high, male mobility is high, rates of condom use in risky sex are low, and rates of male circumcision—a presumed protective factor—are low. Even an increase to a modest 5 percent infection level in India, the lower end of the African epidemics, in 2025, would represent 25-30 million infected adults and, over the next 25 years, approximately 50 million cumulative HIV-1 infections and 40 million cumulative deaths. This is twice the cumulative number of global deaths due to HIV/AIDS over the past two decades.

This paper models the potential epidemiological impact of preventive HIV vaccines on the HIV/AIDS epidemic in Southern India, using a mathematical deterministic dynamic compartmental model. In the second section, we describe the basic assumptions and workings of the model and the characteristics of the vaccines and targeting strategies for an HIV vaccination campaign that are modeled. Various assumptions about the degree of protection offered by such a vaccine, the extent of immunological response in those vaccinated, and the duration of protection afforded are explored. In the third section, we present the results, comparing the epidemiological impact of alternative vaccines and targeting strategies to that of conventional prevention strategies aimed at raising condom use among high-risk groups (sex workers and their client) and improving the syndromic treatment of STIs. The impact of disinhibition (increased risk behavior due to the presence of a vaccine) is also considered. The final section summarizes the results and points to implications for HIV/AIDS prevention policies.

The best long-term hope for control of the HIV epidemic may be a preventive HIV vaccine. Until one is developed, scaling up high impact preventive interventions can reduce the growth of the epidemic. There is an urgent need to develop new candidate vaccines, but also a need to plan the considerable program requirements in introducing new vaccines and in fitting them into other prevention strategies. An AIDS vaccine will greatly help to reduce HIV/AIDS, but it will not be a panacea. Because of the possibility of behavior reversals and an imperfect vaccine (e.g., one that confers

only partial protection, or no protection at all in some of those vaccinated), other preventive efforts must be continued, if not expanded.

2. Methods

This paper complements a recent paper by Stover and others (2002) in which two HIV/AIDS epidemiological models (the Imperial College Model and IwgAIDS) were used to explore the effects of a potential vaccine in Thailand, Uganda, and Zimbabwe. We extend a mathematical model of HIV-1 transmission in Southern India, using methods previously developed for Working Group Five of the Commission on Macroeconomics and Health (Nagelkerke and others 2002). We use the *ModelMaker* program, version 3.0.3 (AP Benson 1993-97), for implementing our model.

The model

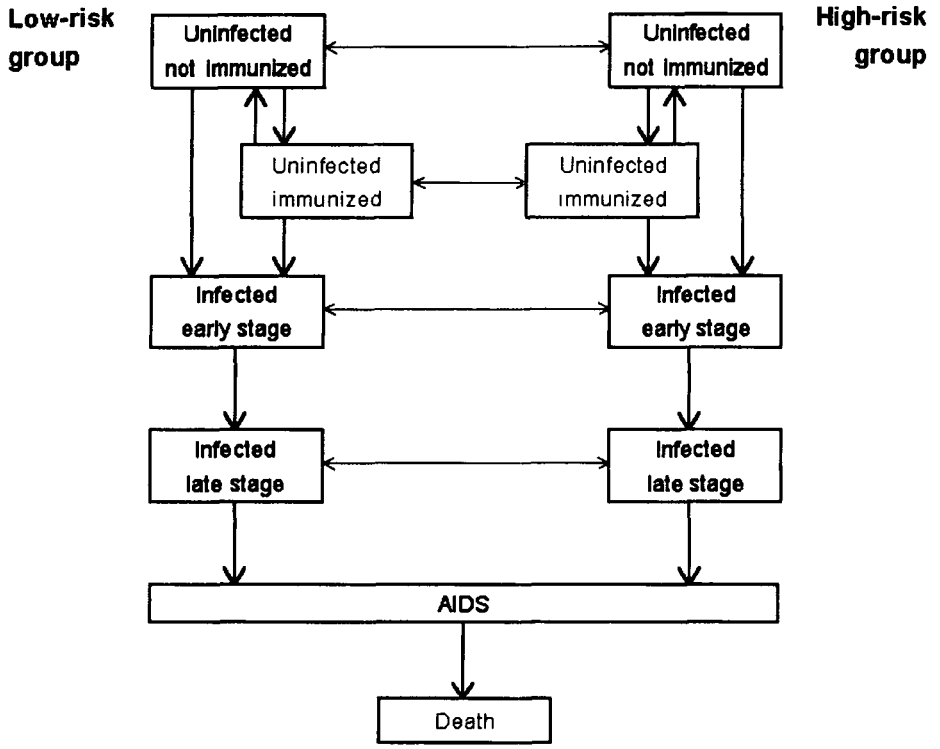
The model is a dynamic, deterministic compartmental model. The main features of the model that are pertinent to the vaccine exercise are described in Figure 1. The boxes represent compartments, or states, that individuals can be in and the arrows show the flow of individuals between compartments. Each compartment shown has been implemented in duplicate, for men and women separately. Individuals move between gender-specific compartments. For example, for women there are two groupings, CSW and low-risk women. Each of these two groups is split into several groups based on their infection and immunization status:

- those uninfected and immunized,
- those uninfected and not immunized, and
- those who are infected (in three sub-groups, early stage, late state, and AIDS).

There is no age structure in the model as used for the purpose of projecting the impact of an HIV vaccine, with the exception that the model only concerns the sexually active adult population.[1] It assumes that the epidemic is primarily heterosexual, driven by commercial sex, and that unsafe sex work is widespread and contributes substantially to the spread of the infection. CSW and their clients were assigned separate compartments to reflect this assumption. In India, approximately 80 percent of STI are first-generation infections derived from sex work, so this seems reasonable (Rodrigues and others 1995). Early female HIV infections occurred predominantly in CSW; infection in monogamous women is probably linked to their husbands having visited a CSW (Gangakhedkar and others 1997, Pais 1996).

1 The model also incorporates mother-to-child transmission (MTCT, "vertical transmission") and an intervention ("nevirapine") to reduce this. However, as we are mainly interested in adult prevalence, which is not affected by MTCT, this is not considered here. A formal description of an earlier and more complete version of the model is available on the Internet (Nagelkerke and others 2001).

Figure 1. Structure of the model



Individuals are "born into" the low-risk uninfected category of their gender and may move to and from high-risk groups (female CSWs and their male clients). In addition to dying from AIDS, they may die from other causes or "age" out of the sexually active age group. High-risk groups may infect low-risk groups (e.g., their current steady partners), including newly acquired low-risk (steady partners), for example, when they get married. The HIV disease process is broken down into three stages: early, late, and AIDS.[2]

The model includes neither transmission between men who have sex with men (MSM) nor transmission due to intravenous drug use (IDU). Both occur in India, but it is believed that they account for a minority of all transmissions. The role of MSM and IDU transmission and interaction with the heterosexual epidemic may be small, although IDU is an important mode of HIV transmission in the Northeastern state of Manipur (the main exception). Appendix 1 provides a formal technical description of the model, including a graphical representation.

2 The three stages of AIDS in the model facilitate modeling the impact of anti-retroviral therapy (ART) targeted at patients in different stages of HIV disease progression. ART is not among the interventions considered in this study, however.

Parameters

In setting the parameters of the model, demographic data from South India were used where available; otherwise data from whole of India were used. A recent study conservatively estimates that there are at least 2 million CSW in India, each having on average—very conservatively—two clients per day, and that their clients number approximately 30 million (i.e., slightly over 10 percent of the adult male population) (Venkataramana and Sarada 2001). This suggests that each client may have some 50 CSW contacts annually. Based on the results of the recent nationwide behavioral survey commissioned by the National AIDS Control Organization (NACO), on others' estimates that over 40 percent of all CSWs work in the four Southern states, and on the higher prevalence of HIV in Southern India, we assumed that approximately 20 percent of all adult males are clients of CSW at any one time (NACO 2002).

Estimates of the rates of becoming a high-risk individual and transitioning back to the low-risk category are not available. From the fact that most studies find the mean age (and age-range) of sex workers to be low (often around 23 years), it follows that the rate of leaving the profession must be high. We chose 20 percent annually for this rate. We took half this value for the transition rate of from client to non-client. Rates of becoming a client can then be derived from the number of clients and the rate of becoming low-risk. For women we also introduced a demand factor, i.e., low-risk women's rate of becoming a CSW was modeled as a function of the demand for sex work.

The average duration from infection to AIDS is the sum of the average duration of the early and late stages; we assumed four years in each of the two stages, resulting in an average duration of HIV infection of eight years.

Modeling by the National Intelligence Council (2002) has suggested that the HIV/AIDS epidemic in India, would result in up to 25 million people living with HIV/AIDS by 2010, i.e., an adult HIV prevalence of approximately 5 percent. However, as Southern India appears to be the worst-hit part of the subcontinent, our model corresponds to a scenario in which prevalence grows from its current level of approximately 2 percent of the sexually active population to an equilibrium prevalence of almost 8 percent.

Table 1 gives the (baseline) parameters used for the model: parameters were chosen to reflect conditions in the four Southern Indian states of Andhra Pradesh, Karnataka, Maharashtra, and Tamil Nadu combined. With this choice of parameters, a 2 percent HIV adult prevalence in 2001 was obtained. This prevalence would gradually increase to 7.5 percent in 2033 in the absence of any interventions. While not comparable to the prevalence encountered in many parts of Sub-Saharan Africa, this size of the epidemic would have devastating effects on Indian society and its socio-economic development.

Table 1. Model parameters (no interventions active)

Parameter	*Description (where relevant)*	*Value*
aidsrate	Annual rate of developing AIDS from late stage HIV	0.25
Brate	Birth rate	0.085
cr_before	Contact rate between clients and CSW	50
Cust	Rate at which low-risk men become CSW-clients	0.04
Femgr	Rate of growth adult female population	0.021
Fmrisk	Female to male HIV transmission risk during CSW-client contact	0.0036
HIVprog	Rate of HIV progression from early to late stage	0.25
init_frac_cli	Initial (1998) fraction of adult men who are CSW clients	0.2
init_frac_csw	Initial (1998) fraction of adult women who are CSW	0.013
init_inf_cli	Initial (1998) clients HIV prevalence	0.07
init_inf_csw	Initial (1998) CSW HIV prevalence	0.25
init_inf_fem	Initial (1998) low-risk adult female HIV prevalence	0.005
init_inf_men	Initial (1998) low risk adult males HIV prevalence	0.005
init_pop_female	Initial female adult population	70,000,000
init_pop_male	Initial male adult population	70,000,000
Leak	Transmission parameter high-risk to low-risk	0.075
loss_immun	Rate (annual) of loss of vaccine induced immunity	0
Malegr	Rate of growth of adult male population	0.022
Marrate	Marriage rate	0.058
Mfrisk	Male to female HIV transmission risk during CSW-client contact	0.0052
Muaids	Death rate AIDS patients	1
Muhiv_kids	Death rate HIV infected children	0.25
Muneg	Death rate HIV- adults	0.026
Mupos	Death rates (non-HIV) HIV+ adults	0.028
Prof	Parameter controlling proclivity of low risk women to become CSW	0.004
stabfactor	Parameter on transmission between newly wed discordant couples	100
Startyr_condom_CSW	Startyear (+1998)focussed intervention among CSW (use condoms)	35
Startyr_std	Startyear (+1998) Mwanza style STI control	35
Startyr_vaccin	Startyear vaccine intervention	35
STD_effect	Effect of STI control on transmission (1=no effect, 0=transmission interrupted)	0.7
Uncust	Rate CSW clients become low-risk men	0.1
Unprof	Rate CSW become low-risk women	0.2
Unprot_after	Level of CSW-client non-use of condoms after focused intervention	0.25
Unprot_before	Same. Before intervention	0.5
Vaceff	Level of vaccine protection, 0=100%, 1= 0%	0
Vactake	Proportion of vaccinated who respond	0.5
Vtrate	Mother-to-child (i.e. vertical) HIV transmission rate	0.33
w1	Relative infectiousness early stage HIV+	1
w2	Relative infectiousness late stage HIV+	1

Note: The model has additional parameters for estimating the impact of anti-retroviral therapy; these are not presented, as they are not relevant to modeling the impact of an AIDS vaccine.

Vaccine characteristics

We compare the epidemiological impact over the period 1998-2033 of four different vaccines, defined by the levels of two parameters:

- *Level of protection.* The reduction in HIV susceptibility in those giving an effective immunological response to the vaccine. We consider two levels—50 percent and 100 percent.
- *Level of immune response.* The percentage of those vaccinated who have an immunological response to the vaccine. We consider two levels—50 percent and 95 percent—of those vaccinated. The vaccine has no protective effect on the remaining 50 percent (or 5 percent) receiving the vaccine who have no immune response to it.

We use the term "vaccine efficacy" to mean the product of the level of protection and level of immune response—that is, the average protection afforded to an average vaccinated person. Thus, a vaccine of 50 percent average efficacy in the population could be defined as either: (a) a vaccine conveying 100 percent protection to half of those vaccinated (50 percent immune response); or (b) 50 percent protection to everyone who is vaccinated (100 percent immune response) or (c) some other combination of protection and immune response that yields an average efficacy of 50 percent.[3]

We assume that vaccines would become available in 2008, which is the earliest time vaccines would become available for general use if current development efforts prove successful, and that vaccines would provide (for those conferred any protection) immunity for at least 25 years (or vaccine recipients would be revaccinated sufficiently often to simulate this duration of immunity). In addition, we explored the effects of waning of vaccine efficacy by showing the impact of the "best" vaccine considered here (100 percent protection conferred to 95 percent of those vaccinated), if protection lasted on average 3 years. Following loss of vaccine-induced immunity, vaccine recipients move back to the compartments of susceptibles, from where they may be recruited for vaccination again.

Targeting strategies

We examine the impact of these vaccines of differing levels of efficacy using two different targeting scenarios with different assumptions on coverage:

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3 Stover and others (2002) refer to vaccines that convey 100 percent protection to a share of those vaccinated as "take" vaccines (example (a) in the text) and those that convey partial protection to all who are vaccinated "degree" vaccines (example (b) in the text). They show that the epidemiological impact of a vaccine with a given average efficacy in a population is highly dependent on whether efficacy is achieved through "take" or "degree." This distinction between "take" and "degree" type effectiveness is not relevant for a vaccine with 100 percent efficacy (that is, complete protection of all who are vaccinated), as they are equivalent. Stover and others do not model the impact of vaccines with both partial protection and partial immune response, such as example (c) in the text, though their impact presumably would be somewhere between the impact of "take" and "degree" type vaccines for a given level of effectiveness.

High risk group targeting (HRG). Both CSWs and their clients are targeted. Annually, 75 percent of those eligible (i.e. belonging to the target population and not yet immunized) would be vaccinated. This would result in an average coverage rate (proportion of the groups vaccinated) of approximately 90 percent.

Population targeting (POP). Every sexually active adult is equally targeted regardless of behavioral risk group. At the time the vaccine becomes available, a 2-year vaccination campaign is launched that succeeds in reaching 25 percent of the target population of susceptibles (HIV negative, not immune) annually. This is followed by an indefinite period during which 5 percent of the target population is vaccinated annually. This leads to an equilibrium situation in which approximately 50 percent of the sexually active population has been vaccinated. The initial 2-year vaccination campaign is included to reach that 50 percent coverage level quickly.

Conventional interventions for comparison

As an HIV vaccine has not yet been developed and as a point of comparison, we also modeled the epidemiological impact of two conventional HIV prevention interventions:

A focused CSW intervention. The objective of this intervention is to increase condom use in CSW-client contacts. Focused interventions have proven to be very effective in increasing condom use in this context. This reduces HIV transmission among sex workers and clients, but also in the general population, because of the "core" role of these high-risk groups in spreading infection to the rest of the population (Hethcote and York 1984, Jha and others 2001, World Bank 1997). Many peer-mediated CSW intervention programs in India and Africa have shown increases in condom use of 80 percent or more among those reached (Bhave and others 1995, Jana and others 1998, Jana and Singh 1995, Moses and others 1991). We conservatively assumed that the intervention reduces the percentage of unprotected contacts from 50 to 25 percent. We were also conservative in not assuming an additional reduction in the risk of transmission per CSW-client contact through a reduction in STI prevalence, although this may well be the case.

Syndromic treatment of STIs. Epidemiological studies support the hypothesis that STIs are associated with increased HIV susceptibility and infectiousness. However, confounding makes it difficult to reliably estimate these cofactor effects from observational studies (Korenromp and others 2001). Three experimental studies have been carried out to date, one in Tanzania and two in Uganda. STI management, through improved treatment of patients with symptomatic STI infections, has proven to be effective in a controlled community trial in Mwanza, Tanzania, with an approximate 40 percent reduction in HIV transmission (Grosskurth and others 1995). STI management was based on a syndromic approach to symptomatic cases. It was applied to a rural area in a non-targeted way. People with asymptomatic infections were not treated. However, the failure of a similar intervention in a trial in Masaka, Uganda, to replicate this success (reported at the AIDS conference in Barcelona), and the lack of success in

Rakai, Uganda, to reduce HIV transmission through a program that offered mass treatment for STIs to the population has sparked debate about the efficacy of such interventions in slowing HIV transmission (Gray and others 1999, Hitchcock and Fransen 1999, Hudson 2001, Korenromp and others 2000, Kvale 1999, Matthys and Boelaert 1999, Nicoll and others 1999). We nevertheless assumed for the purposes of this modeling exercise that HIV transmission would decrease by 30 percent across the board (males, females, high-risk, low-risk). Arguably, this is a strong simplification of reality and requires averaging over partnerships with and without STI. In reality, the average effect of the intervention may also vary among risk categories (e.g., CSW and other women), depending on factors that are largely unknown, such as the uptake of the intervention. No effects of the intervention on sexual behavior were assumed. Note that the way this 30 percent reduction is achieved is irrelevant for our predictions. Increasing condom use in the general population could be equally effective. As both interventions use existing technologies, they were assumed to start in 2003.

Disinhibition (increase in risky behavior)

We explore whether disinhibition—that is, an increase in risky behavior associated with the availability of an HIV vaccine—could nullify or reverse the impact of the vaccine. Disinhibition has been observed in high-risk gay men in response to the availability of anti-retroviral therapy (Katz and others 2002, Ostrow and others 2002, Stolte and others 2001, Stolte and Coutinho 2002). All models were run in the presence of a strong disinhibition effect, that is, assuming that condom use between CSWs and clients dropped from 50 percent (assumed to have increased already from very low levels in response to other prevention efforts) prior to the availability of vaccines to nil (0 percent). For comparison (adult) HIV prevalence in 2033 (the last year of the simulation) was used.

3. Results

Table 2 shows the effect of the different interventions on long run (2033) adult HIV prevalence. Conventional prevention programs begin in 2003 and vaccine interventions in 2008.

All vaccine scenarios show a decline in HIV prevalence. Generally, a high degree of protection appears to be more important than a high "take" rate. Disinhibition (i.e. condoms are no longer used during CSW-clients) has the potential of undoing much of the vaccine benefits, and may even aggravate the epidemic. However, our assumed extreme disinhibition effect of total abandonment of condom use, may be unlikely to happen, as condoms also provide protection against conventional sexually transmitted infections, an advantage that CSWs may be keen to keep. Targeting high-risk groups tends to be more effective than targeting the general population (at least at the levels considered). For the best vaccine considered (scenario 4), the impact appears to be similar, perhaps due to "over-vaccination" of high-risk groups (both clients and CSWs have a high vaccine coverage).

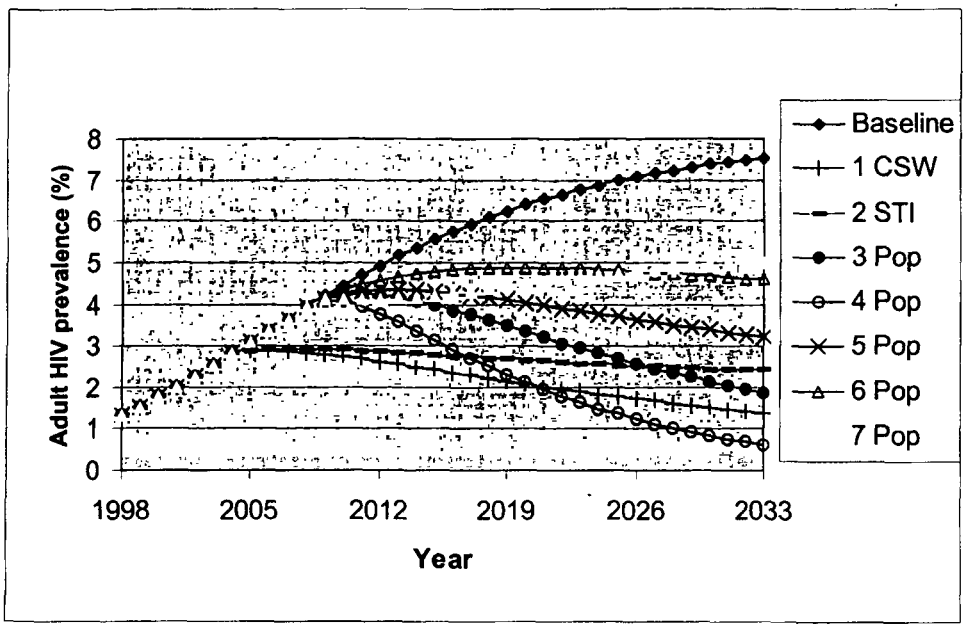
Table 2. Adult HIV prevalence in 2033 under seven scenarios, with and without disinhibition[a]

	Scenario		Adult HIV prevalence in 2033 (percent)			
0	Baseline		7.5			
	Conventional interventions	**Protection (percent)**				
1	CSW condom intervention	75[b]	1.4			
2	STI syndromic treatment	30[c]	2.4			
	Vaccine scenarios	**Efficacy (percent)**	**Targeted to high-risk groups (HRG)**		**Targeted to general population (POP)**	
			No disinhibition	**Disinhibition**	**No disinhibition**	**Disinhibition**
3	100% protection, 50% response, ***25 years duration***	50	1.0	3.3	1.9	4.8
4	100% protection, 95% response, ***25 years duration***	95	0.6	1.4	0.6	2.1
5	50% protection, 95% response, ***25 years duration***	47.5	2.9	9.5	3.2	8.5
6	50% protection, 50% response, ***25 years duration***	25	3.7	10.3	4.6	10.0
7	100% protection, 95% response, ***3 years duration***	95	1.5	5.7	5.0	10.5

a. Decline in condom use in commercial sex from 50% to zero.
b. Increase in protection from 50% to 75% among CSW-client contacts.
c. Reduction in transmission probability.

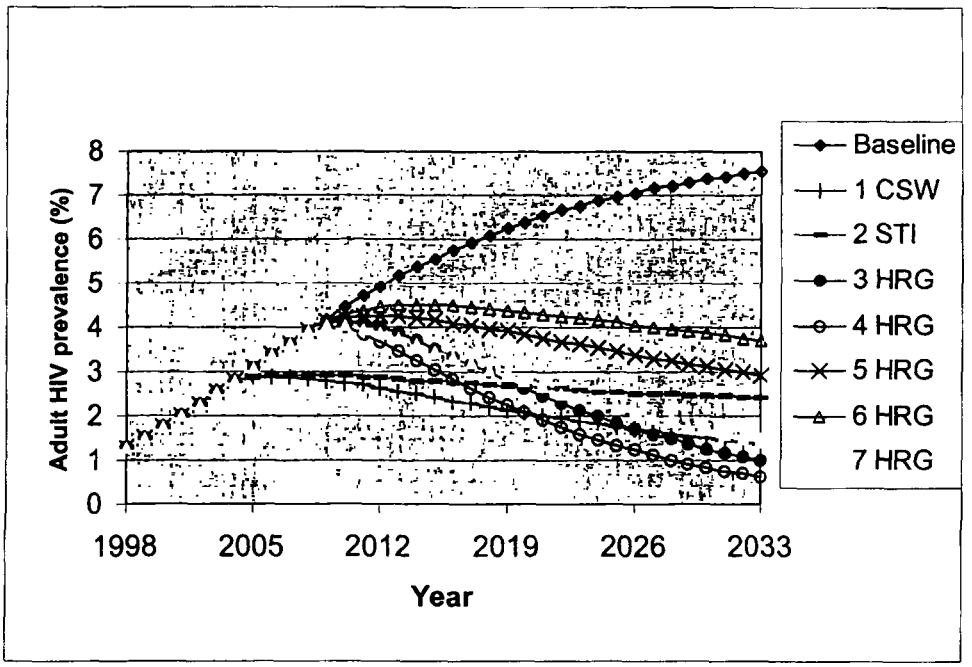
Figures 2a and 2b show the impact of the different interventions (without disinhibition) on HIV prevalence over the period 2003-2033. Note that a highly effective vaccine (scenario 4, 100 percent protection for 95 percent of those vaccinated) appears to be by far the most effective method to bring down HIV prevalence quickly, much faster than conventional prevention programs. The latter, however, have the advantage that their implementation could start immediately.

Figure 2a. Epidemiological impact of targeting a preventive HIV vaccine to the general population, compared with CSW and STI interventions, South India



Note: Vaccines target approximately 50 percent of the general population; scenarios defined in Table 2.

Figure 2b. Epidemiological impact of targeting a preventive HIV vaccine to the high-risk population, compared with CSW and STI interventions, South India



Note: Vaccines target approximately 90 percent of CSWs and clients; scenarios defined in Table 2.

Vaccine, condom, and drug requirements

Figures 3a and 3b show the cumulative number of vaccinations needed under different strategies, assuming that those who are already HIV-positive are not vaccinated. Targeting high-risk groups typically requires substantially fewer vaccine doses than targeting the general population, at a similar or higher impact. Thus, unless the costs of targeting high risk groups are extremely high, targeting high-risk groups should be typically several times more cost-effective than targeting the general population. Also, to achieve reasonably high vaccination coverage for the general population, we assumed that a very intensive two-year vaccination campaign would "kick-start" vaccination coverage. This would put enormous strains on production facilities and other infrastructure and may be difficult to implement. The annual number of vaccinations implied by each of the targeted and general population strategies are in Table 3—including both the "kick-start" phase and the "maintenance" phase.

Costs follow from the costs to set up and maintain the infrastructure and of course the cost per vaccination, at present unknown. Even targeting only high-risk populations may require hundreds of millions of vaccine doses over a 25-year period. However, this is less than the number of childhood vaccinations given over that period and, unless the vaccine is very expensive (over US$100, say), this would definitely seem affordable.

The number of condoms needed for a focused intervention for high-risk groups is easy to calculate. We assumed that approximately 20 percent of sexually active adult men would be clients, and that they have—on average—50 CSW contacts annually. We (optimistically, but based on NACO's sexual behavior surveys) assumed that approximately half of these contacts are already protected by condoms obtained from other sources (NACO 2002). Thus, to increase condom use to 75 percent, 13 additional condoms per client, or approximately 3 per adult male, would be required annually.

The amount of drugs and their costs for an STI control program are hard to predict. In addition to current prevalence and incidence of STIs, knock-on effects in terms of a reduction in transmission and a consequent decline in incidence may yield long-term savings in costs. For Mwanza, the annual per-capita costs of running am STI program were estimated to be $0.39 (Gilson and others 1997).

Figure 3a. Cumulative number of vaccinations required for targeting the general population, South India

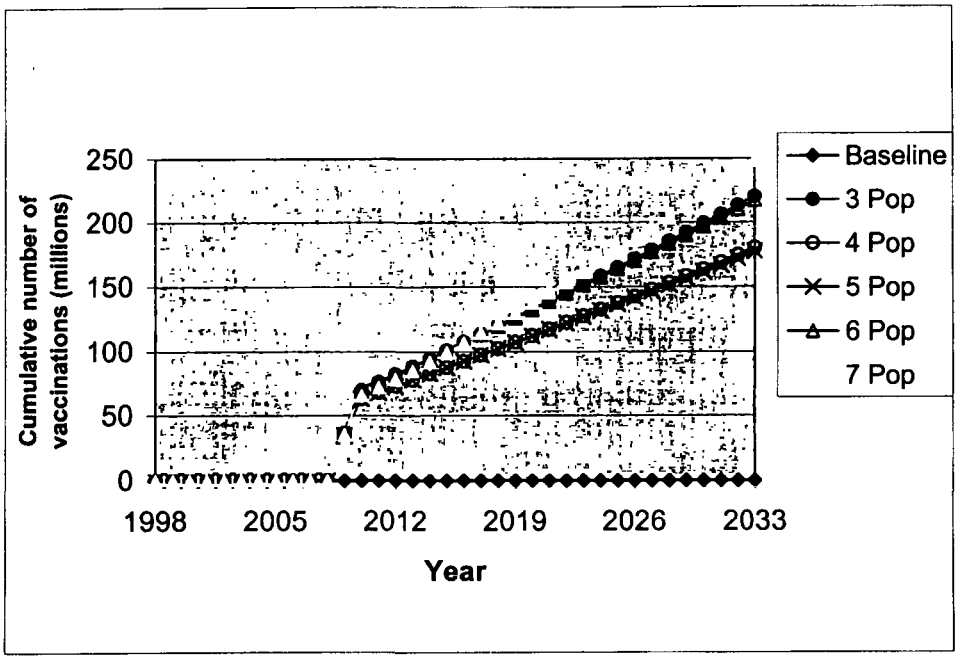


Figure 3b. Cumulative number of vaccinations required for targeting high-risk groups, South India

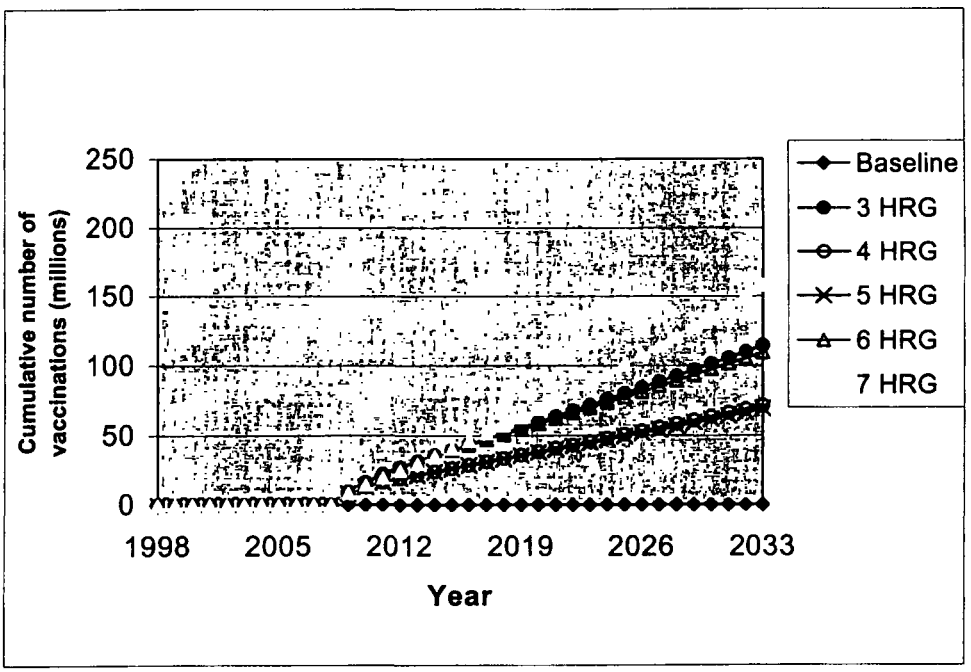


Table 3. Annual number of vaccinations (millions), by vaccination scenario

Year	*3Pop*	*4Pop*	*5Pop*	*6Pop*	*7Pop*	*3HRG*	*4HRG*	*5HRG*	*6HRG*	*7HRG*
2008	0	0	0	0	0	0	0	0	0	0
2009	36.75584	34.83045	34.83009	36.75562	35.24657	9.458652	8.191432	8.190107	9.4577	8.463783
2010	32.95754	28.07567	28.07135	32.95466	30.39358	7.024372	4.732682	4.723124	7.015272	5.880448
2011	6.244858	5.029587	5.026452	6.242568	6.04508	5.64389	3.323605	3.303129	5.61899	5.199646
2012	6.2351	4.984517	4.977201	6.229522	6.485979	4.86343	2.748514	2.718075	4.819448	5.084898
2013	6.231736	4.949949	4.936769	6.221292	6.808229	4.42484	2.514796	2.475336	4.3611	5.139177
2014	6.234492	4.924991	4.904447	6.217654	7.051646	4.181922	2.421921	2.374015	4.09868	5.24384
2015	6.243104	4.908842	4.879604	6.218406	7.242737	4.051666	2.387704	2.331829	3.949434	5.36314
2016	6.257324	4.900801	4.861685	6.223352	7.399158	3.986756	2.378304	2.314969	3.866054	5.486258
2017	6.276922	4.900245	4.850188	6.232322	7.53268	3.96011	2.379766	2.309487	3.821432	5.609946
2018	6.30168	4.906626	4.844663	6.245144	7.651148	3.95619	2.386378	2.309613	3.8	5.733307
2019	6.331402	4.919453	4.844703	6.261668	7.7598	3.966054	2.395833	2.312953	3.792784	5.856183
2020	6.3659	4.938289	4.849935	6.281744	7.862127	3.984578	2.407233	2.318507	3.794608	5.978658
2021	6.40501	4.962744	4.860026	6.305238	7.960466	4.008822	2.42026	2.325846	3.802472	6.100907
2022	6.448568	4.992467	4.874671	6.33202	8.056381	4.037122	2.434812	2.334781	3.814642	6.223128
2023	6.496434	5.027143	4.893588	6.36197	8.150922	4.068544	2.450875	2.345216	3.830102	6.345532
2024	6.548474	5.066488	4.916528	6.394974	8.244798	4.10257	2.468455	2.357095	3.848254	6.468314
2025	6.604564	5.110247	4.94326	6.430926	8.338489	4.138926	2.487564	2.370379	3.868744	6.591671
2026	6.66459	5.158189	4.973572	6.469724	8.432323	4.177472	2.508202	2.385034	3.891352	6.715784
2027	6.728448	5.210106	5.007272	6.511276	8.526526	4.21814	2.530365	2.401028	3.915942	6.840825
2028	6.796044	5.265813	5.044189	6.555494	8.621255	4.260894	2.554045	2.418331	3.942418	6.966954
2029	6.867288	5.325138	5.084162	6.602292	8.716624	4.30573	2.579222	2.436913	3.970718	7.094314
2030	6.942102	5.387928	5.127048	6.6516	8.812714	4.352646	2.605879	2.456742	4.000782	7.223042
2031	7.02041	5.454048	5.172717	6.703342	8.909587	4.401634	2.633994	2.477794	4.032574	7.353264
2032	7.102148	5.523376	5.221045	6.75745	9.007293	4.4527	2.66354	2.500041	4.066054	7.485094
2033	7.18726	5.595799	5.271928	6.813868	9.105866	4.505834	2.694497	2.523456	4.101184	7.618637

4. Discussion

The HIV/AIDS epidemic in Southern India is more serious than that in most other parts of India. Current adult HIV prevalence approximates 2 percent, and in the absence of any intervention our model predicts a 7.5 percent adult HIV seroprevalence in 2033. Unfortunately, this prediction cannot be very precise as many model parameters are only approximately known.

Our model suggests that all interventions considered are potentially able to substantially dent the HIV epidemic. The effect of both highly effective vaccines and of a focused CSW intervention, based on condom promotion in which unprotected sex is reduced by 50 percent, is impressive. In the presence of a CSW intervention, prevalence would decline to 1.4 percent by 2033, less than the prevalence in 2001. This is consistent with the finding (Gangakhedkar and others, 1997) that infections among monogamous women in Pune (Maharashtra state) arise mostly from their husband's unprotected contact with sex workers. Even in mature epidemics, sex work is a key source of new infections. For example, adult prevalence in Cotonou, Benin, has exceeded 3 percent for the last decade or more. Careful work by Lowndes and others (2002) has concluded that virtually all of the ongoing HIV-1 transmission is related to

infection of female sex workers, male clients of female sex workers, and the other non-regular sexual partners of those men.

Syndromic treatment of STIs would reduce HIV prevalence to 2.4 percent by 2033—not as impressive as a CSW intervention, but still important. It needs to be stressed however, that the empirical basis for the impact of syndromic treatment of STIs in India is less solid or well understood than that for focused CSW interventions, especially since preliminary results from the trial in Masaka, Uganda, have come out.

Preventive HIV vaccines could be highly effective in controlling the epidemic. Early understanding of the immunology correlates of HIV-1 protection, and the genetic variability and rapid mutations of the HIV virus all suggest that a high efficacy vaccine is unlikely at the outset, but could develop with continuous testing (Esparza 2001, Plummer and others 2001). A vaccine that conveys substantially less than full protection to those who are immunized will not prevent sex workers from getting infected, but would delay infection. Thus, targeting vaccines with low protection to high-risk groups is less effective than providing them with highly effective vaccines or condom-based programs. Moreover, sustained condom use among high-risk groups reduces transmission of STIs other than HIV.

Given a specified average vaccine efficacy, vaccines would be most effective if providing near 100 percent protection in to those who are immunized, even if not everyone vaccinated has an immunological response—in the terminology of Stover and others (2002), "take"-type efficacy. Vaccines that confer the same average partial level of protection to all vaccine recipients ("degree"-type efficacy) have less of an epidemiological impact. This makes sense, as partial protection may be insufficient to protect individuals with high-risk behaviors, although it would delay their infection. This is consistent with findings by Stover and others (2002).

A vaccine that confers 100 percent protection in 95 percent of all vaccine recipients could almost eradicate HIV within 25 years. Irrespective of the targeting strategy (the general population or high-risk groups), adult HIV prevalence would shrink to a mere 0.6 percent in 2033 and would subsequently decline even further. More importantly, this vaccine would still have a substantial impact, even if CSW-client condom use were to drop to zero (disinhibition). If high-risk populations were targeted with this highly effective vaccine and condom use were to drop, adult HIV prevalence in 2033 would be 1.4 percent; if the general population were targeted with this vaccine and with disinhibition, HIV prevalence would reach 2.1 percent.

The vaccine that would have the least effect is the vaccine that confers 50 percent protection to 50 percent of recipients—an average efficacy of only 25 percent. Adult HIV prevalence would rise to 3.7 percent in 2033 if high risk groups are targeted, while it would rise to 4.6 percent if the general population is targeted. The effects of this vaccine could be reversed by disinhibition, with adult HIV prevalence in 2033 of 10.3 percent (if high-risk groups are targeted) and 10.0 percent (population targeting), respectively. In other words, in the presence of disinhibition, HIV prevalence in 2033

would be 2.5-2.8 percentage points *higher* than the projected baseline, which reaches 7.5 percent in that time frame. This is also broadly consistent with findings by Stover and others: "a vaccine with low efficacy and low duration could have negative impact on public health if its implementation were accompanied by widespread reversion to riskier sexual behaviors" (p. 29). They conclude that "with low efficacy vaccines it will be very important to support the vaccination program with efforts to combat any reversal to riskier sex. If efforts to maintain safer sex behaviors are not successful, then behavioral reversals could eliminate most of the benefits of the vaccine. In some cases the effect could be to increase HIV incidence" (p. 29).

Whether disinhibition is a likely scenario is unknown. It seems to be largely based on the experience with anti-retroviral therapy. While a vaccine may have the same effect, a vaccination campaign may also raise HIV awareness in the population and increase a sense of vulnerability in unvaccinated individuals. A sense of *in*vulnerability in vaccinated individuals would only be a problem in partially effective vaccines.

Aside from effectiveness, there is the issue of the cost and feasibility of interventions. Interventions share infrastructure costs (e.g., surveillance costs would be used for both types of program). Large population laboratories are needed to support new generations of vaccine testing and newer intervention research on interventions for high-risk groups. These costs are often of the nature of joint costs. Costs for preventing HIV growth have to be integrated with costs of other interventions. For example, outreach campaigns for vaccines would probably aim to deliver several vaccines, including those for childhood vaccine preventable diseases.

The assumed vaccination coverage rates, while not 100 percent, even if integrated in existing structures, would still require substantial efforts and costs, with tens to hundreds of millions of vaccines administered over a 25-year period. A major advantage of a preventive HIV vaccine, which it shares with CSW and STI interventions, is that a potential recipient is not required to take an HIV test as a prerequisite for receiving a vaccine. While vaccines given to HIV-positive adults are clearly wasted (in the case of high-risk-group targeting this can be substantial), a policy of non-testing may be more efficient than one in which individuals are tested and counseled. Nevertheless, our estimates of the required number of vaccines only include vaccines for those who are HIV-negative. For population targeting, the wastage of vaccinating everybody is small. For high-risk populations, with a higher HIV prevalence, the wastage may be more substantial, at least in relative terms. Targeting high-risk groups is much more cost-effective than targeting the entire adult population. Using approximately one third of the number of vaccines, a higher reduction in prevalence is achieved. Although we did not explore this scenario, highly effective vaccines could be targeted to CSW only (i.e., excluding their clients), as in the long run this would be almost as effective as protecting both CSW and their clients. Vaccinating both CSWs and their clients would in the long run lead to substantial redundancy in

prevention efforts. By contrast, for vaccines conferring partial protection or with a low "take" rate it would seem sensible to vaccinate both CSWs and their clients.

Conventional HIV prevention programs, especially those targeting CSWs (focused interventions) and using existing low-tech methods, may achieve results that are similar to reasonably effective vaccines and are probably less sensitive to disinhibition effects. It would therefore seem wise not to wait for the arrival of a vaccine, but to implement and expand focused CSW prevention programs as early and vigorously as possible. This will also create the infrastructure for effectively introducing HIV vaccines into these groups as soon as vaccines become available, and for scaling up vaccination campaigns. Such programs, however, require the political will to initiate and sustain them. Political support for vaccination campaigns, even for partially effective vaccines, may come easy, perhaps more so than for programs seemingly focusing on marginal groups such as CSWs. In sum, for the next few years expanding coverage of vulnerable group interventions while accelerating vaccine research and strengthening capacity for both with surveillance, human resource development, and operations research are the best strategies to contain the Indian HIV-1 epidemic. When vaccines become available and particularly if efficacy or coverage is not perfect (most likely they are not!), then "other prevention programs should continue in conjunction with vaccination programs in order to reduce HIV infections to the lowest possible levels and maintain the other health benefits, such as prevention of sexually transmitted diseases" (Stover and others 2002, p. 30).

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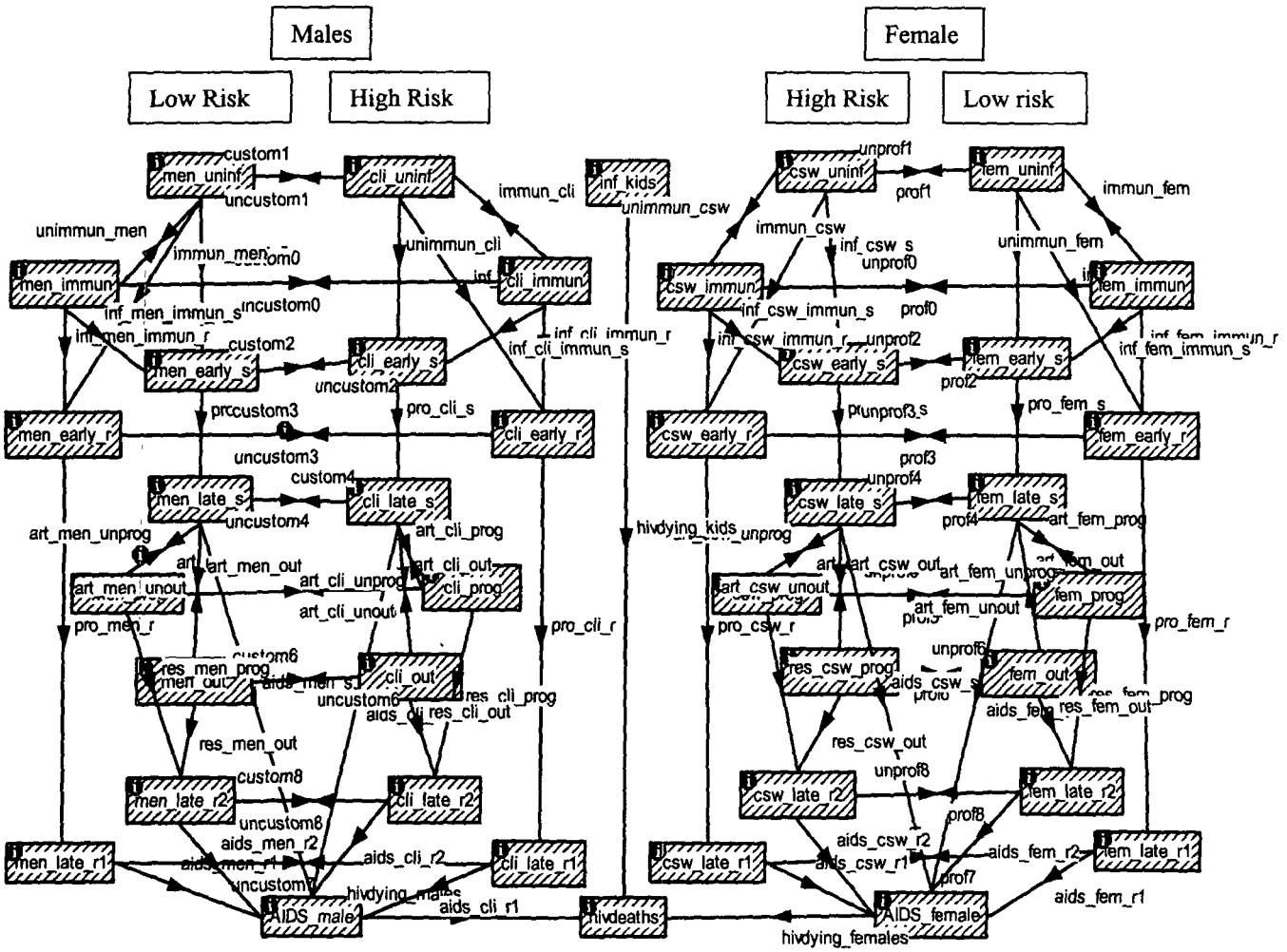
Annex 1. Description of the model, including a graphical representation.



COMPARTMENTS
Differential equations and initial values

AIDS_female
dAIDS_female/dt =
+aids_csw_s+aids_csw_r1+aids_fem_r1+aids_fem_s-
hivdying_females-
mupos*AIDS_female+aids_csw_r2+aids_fem_r2
Initial Value = 0.0

AIDS_male
dAIDS_male/dt = -
hivdying_males+aids_cli_r1+aids_men_r1+aids_men_s+ai
ds_cli_s-mupos*AIDS_male+aids_men_r2+aids_cli_r2
Initial Value = 0.0

cli_early_r
dcli_early_r/dt = -mupos*cli_early_r+custom3-
uncustom3+inf_cli_r-pro_cli_r+inf_cli_immun_r
Initial Value = 0.0

cli_early_s
dcli_early_s/dt = -mupos*cli_early_s+custom2-
uncustom2+inf_cli_s-pro_cli_s+inf_cli_immun_s
Initial Value = init_pop_male*init_frac_cli*init_inf_cli*(1-
hivprog/(aidsrate+hivprog)*hivprog/(hivprog+uncust))

cli_immun
dcli_immun/dt = -muneg*cli_immun+custom0-
uncustom0+immun_cli-inf_cli_immun_r-inf_cli_immun_s-
unimmun_cli
Initial Value = 0.0

cli_late_r1
dcli_late_r1/dt = -mupos*cli_late_r1+custom7-
uncustom7+pro_cli_r-aids_cli_r1
Initial Value = 0.0

cli_late_r2
dcli_late_r2/dt = -mupos*cli_late_r2+custom8-
uncustom8+res_cli_prog+res_cli_out-aids_cli_r2
Initial Value = 0.0

cli_late_s
dcli_late_s/dt = -mupos*cli_late_s+custom4-
uncustom4+pro_cli_s-art_cli_prog+art_cli_unprog-
art_cli_out+art_cli_unout-aids_cli_s
Initial Value =
init_pop_male*init_frac_cli*init_inf_cli*hivprog/(aidsrate+
hivprog)*hivprog/(hivprog+uncust)

cli_out
dcli_out/dt = -mupos*cli_out+custom6-
uncustom6+art_cli_out-art_cli_unout-res_cli_out
Initial Value = 0.0

cli_prog
dcli_prog/dt = -mupos*cli_prog+custom5-
uncustom5+art_cli_prog-art_cli_unprog-res_cli_prog
Initial Value = 0.0

cli_uninf
dcli_uninf/dt = -muneg*cli_uninf+custom1-uncustom1-
inf_cli_r-inf_cli_s-immun_cli+unimmun_cli
Initial Value = init_pop_male*init_frac_cli*(1-init_inf_cli)

csw_early_r
dcsw_early_r/dt = -mupos*csw_early_r+prof3-
unprof3+inf_csw_r-pro_csw_r+inf_csw_immun_r
Initial Value = 0.0

csw_early_s
dcsw_early_s/dt = -mupos*csw_early_s+prof2-
unprof2+inf_csw_s-pro_csw_s+inf_csw_immun_s
Initial Value =
init_pop_female*init_frac_csw*init_inf_csw*(1-
hivprog/(aidsrate+hivprog)*hivprog/(hivprog+unprof))

csw_immun
dcsw_immun/dt = -muneg*csw_immun-
unprof0+prof0+immun_csw-inf_csw_immun_r-
inf_csw_immun_s-unimmun_csw
Initial Value = 0.0

csw_late_r1
dcsw_late_r1/dt = -mupos*csw_late_r1+prof7-
unprof7+pro_csw_r-aids_csw_r1
Initial Value = 0.0

csw_late_r2
dcsw_late_r2/dt = -mupos*csw_late_r2+prof8-
unprof8+res_csw_out+res_csw_prog-aids_csw_r2
Initial Value = 0.0

csw_late_s
dcsw_late_s/dt = -mupos*csw_late_s+prof4-
unprof4+pro_csw_s-art_csw_prog+art_csw_unprog-
art_csw_out+art_csw_unout-aids_csw_s
Initial Value =
init_pop_female*init_frac_csw*init_inf_csw*hivprog/(aids
rate+hivprog)*hivprog/(hivprog+unprof)

csw_out
dcsw_out/dt = -mupos*csw_out+prof6-
unprof6+art_csw_out-art_csw_unout-res_csw_out
Initial Value = 0.0

csw_prog
dcsw_prog/dt = -mupos*csw_prog+prof5-
unprof5+art_csw_prog-art_csw_unprog-res_csw_prog
Initial Value = 0.0

csw_uninf
dcsw_uninf/dt = -muneg*csw_uninf+prof1-unprof1-
inf_csw_r-inf_csw_s-immun_csw+unimmun_csw
Initial Value = init_pop_female*init_frac_csw*(1-
init_inf_csw)

cum_incidence
dcum_incidence/dt = incidence
Initial Value = 0.0

fem_early_r
dfem_early_r/dt = -mupos*fem_early_r-
prof3+unprof3+inf_fem_r-pro_fem_r+inf_fem_immun_r
Initial Value = 0.0

fem_early_s
dfem_early_s/dt = -mupos*fem_early_s-
prof2+unprof2+inf_fem_s-pro_fem_s+inf_fem_immun_s
Initial Value = init_pop_female*(1-
init_frac_csw)*init_inf_fem*aidsrate/(aidsrate+hivprog)

fem_immun
dfem_immun/dt = -muneg*fem_immun+unprof0-
prof0+immun_fem-inf_fem_immun_r-inf_fem_immun_s-
unimmun_fem
Initial Value = 0.0

fem_late_r1
dfem_late_r1/dt = -mupos*fem_late_r1-
prof7+unprof7+pro_fem_r-aids_fem_r1
Initial Value = 0.0

fem_late_r2

dfem_late_r2/dt = -mupos*fem_late_r2-prof8+unprof8+res_fem_prog+res_fem_out-aids_fem_r2
Initial Value = 0.0

fem_late_s
dfem_late_s/dt = -mupos*fem_late_s-prof4+unprof4+pro_fem_s-art_fem_prog+art_fem_unprog-art_fem_out+art_fem_unout-aids_fem_s
Initial Value = init_pop_female*(1-init_frac_csw)*init_inf_fem*hivprog/(aidsrate+hivprog)

fem_out
dfem_out/dt = -mupos*fem_out-prof6+unprof6+art_fem_out-art_fem_unout-res_fem_out
Initial Value = 0.0

fem_prog
dfem_prog/dt = -mupos*fem_prog-prof5+unprof5+art_fem_prog-art_fem_unprog-res_fem_prog
Initial Value = 0.0

fem_uninf
dfem_uninf/dt = -muneg*fem_uninf-prof1+unprof1-inf_fem_r-inf_fem_s+population*femgr-immun_fem+unimmun_fem
Initial Value = init_pop_female*(1-init_frac_csw)*(1-init_inf_fem)

hivdeaths
dhivdeaths/dt = +hivdying_males+hivdying_females+hivdying_kids
Initial Value = 0.0

inf_kids
dinf_kids/dt = +inf_births-hivdying_kids
Initial Value = 0.0

men_early_r
dmen_early_r/dt = -mupos*men_early_r-custom3+uncustom3+inf_men_r-pro_men_r+inf_men_immun_r
Initial Value = 0.0

men_early_s
dmen_early_s/dt = -mupos*men_early_s-custom2+uncustom2+inf_men_s-pro_men_s+inf_men_immun_s
Initial Value = init_pop_male*(1-init_frac_cli)*init_inf_men*aidsrate/(aidsrate+hivprog)

men_immun
dmen_immun/dt = -muneg*men_immun-custom0+uncustom0+immun_men-inf_men_immun_r-inf_men_immun_s-unimmun_men
Initial Value = 0.0

men_late_r1
dmen_late_r1/dt = -mupos*men_late_r1-custom7+uncustom7+pro_men_r-aids_men_r1
Initial Value = 0.0

men_late_r2
dmen_late_r2/dt = -mupos*men_late_r2-custom8+uncustom8+res_men_out+res_men_prog-aids_men_r2
Initial Value = 0.0

men_late_s
dmen_late_s/dt = -mupos*men_late_s-custom4+uncustom4+pro_men_s-art_men_prog+art_men_unprog-art_men_out+art_men_unout-aids_men_s
Initial Value = init_pop_male*(1-init_frac_cli)*init_inf_men*hivprog/(aidsrate+hivprog)

men_out
dmen_out/dt = -mupos*men_out-custom6+uncustom6+art_men_out-art_men_unout-res_men_out
Initial Value = 0.0

men_prog
dmen_prog/dt = -mupos*men_prog-custom5+uncustom5+art_men_prog-art_men_unprog-res_men_prog
Initial Value = 0.0

men_uninf
dmen_uninf/dt = -muneg*men_uninf-custom1+uncustom1-inf_men_r-inf_men_s+population*malegr-immun_men+unimmun_men
Initial Value = init_pop_male*(1-init_frac_cli)*(1-init_inf_men)

prog_recr
dprog_recr/dt = art_men_prog+art_cli_prog+art_csw_prog+art_fem_prog
Initial Value = 0.0

wild_recr
dwild_recr/dt = art_men_out+art_cli_out+art_csw_out+art_fem_out
Initial Value = 0.0

FLOWS

Movements between compartments

aids_cli_r1
Flow from cli_late_r1 to AIDS_male
aids_cli_r1 = aidsrate * cli_late_r1

aids_cli_r2
Flow from cli_late_r2 to AIDS_male
aids_cli_r2 = aidsrate * cli_late_r2

aids_cli_s
Flow from cli_late_s to AIDS_male
aids_cli_s = aidsrate * cli_late_s

aids_csw_r1
Flow from CSW_late_r1 to AIDS_female
aids_csw_r1 = aidsrate * csw_late_r1

aids_csw_r2
Flow from CSW_late_r2 to AIDS_female
aids_csw_r2 = aidsrate * csw_late_r2

aids_csw_s
Flow from CSW_late_s to AIDS_female
aids_csw_s = aidsrate * csw_late_s

aids_fem_r1
Flow from fem_late_r1 to AIDS_female
aids_fem_r1 = aidsrate * fem_late_r1

aids_fem_r2
Flow from fem_late_r2 to AIDS_female
aids_fem_r2 = aidsrate * fem_late_r2

aids_fem_s
Flow from fem_late_s to AIDS_female
aids_fem_s = aidsrate * fem_late_s

aids_men_r1
Flow from men_late_r1 to AIDS_male
aids_men_r1 = aidsrate * men_late_r1

aids_men_r2

Flow from men_late_r2 to AIDS_male
aids_men_r2 = aidsrate * men_late_r2

aids_men_s
Flow from men_late_s to AIDS_male
aids_men_s = aidsrate * men_late_s

art_cli_out
Flow from cli_late_s to cli_out
art_cli_out = recr_cli_out * cli_late_s

art_cli_prog
Flow from cli_late_s to cli_prog
art_cli_prog = recr_cli_prog * cli_late_s

art_cli_unout
Flow from cli_out to cli_late_s
art_cli_unout = outloss * cli_out

art_cli_unprog
Flow from cli_prog to cli_late_s
art_cli_unprog = progloss * cli_prog

art_csw_out
Flow from csw_late_s to csw_out
art_csw_out = recr_CSW_out * csw_late_s

art_csw_prog
Flow from csw_late_s to csw_prog
art_csw_prog = recr_csw_prog * csw_late_s

art_csw_unout
Flow from CSW_out to csw_late_s
art_csw_unout = outloss * csw_out

art_csw_unprog
Flow from csw_prog to csw_late_s
art_csw_unprog = progloss * csw_prog

art_fem_out
Flow from fem_late_s to fem_out
art_fem_out = recr_fem_out * fem_late_s

art_fem_prog
Flow from fem_late_s to fem_prog
art_fem_prog = recr_fem_prog * fem_late_s

art_fem_unout
Flow from fem_out to fem_late_s
art_fem_unout = outloss * fem_out

art_fem_unprog
Flow from fem_prog to fem_late_s
art_fem_unprog = progloss * fem_prog

art_men_out
Flow from men_late_s to men_out
art_men_out = recr_men_out * men_late_s

art_men_prog
Flow from men_late_s to men_prog
art_men_prog = recr_men_prog * men_late_s

art_men_unout
Flow from men_out to men_late_s
art_men_unout = outloss * men_out

art_men_unprog
Flow from men_prog to men_late_s
art_men_unprog = progloss * men_prog

custom0
Flow from men_immun to cli_immun
custom0 = cust * men_immun

custom1
Flow from men_uninf to cli_uninf
custom1 = cust* men_uninf

custom2
Flow from men_early_s to cli_early_s
custom2 = cust* men_early_s

custom3
Flow from men_early_r to cli_early_r
custom3 = cust * men_early_r

custom4
Flow from men_late_s to cli_late_s
custom4 = cust * men_late_s

custom5
Flow from men_prog to cli_prog
custom5 = cust * men_prog

custom6
Flow from men_out to cli_out
custom6 = cust * men_out

custom7
Flow from men_late_r1 to cli_late_r1
custom7 = cust* men_late_r1

custom8
Flow from men_late_r2 to cli_late_r2
custom8 = cust * men_late_r2

hivdying_females
Flow from AIDS_female to hivdeaths
hivdying_females = round(muaids * AIDS_female)

hivdying_kids
Flow from Inf_kids to hivdeaths
hivdying_kids = round(muhiv_kids * inf_kids)

hivdying_males
Flow from AIDS_male to hivdeaths
hivdying_males = round(muaids* AIDS_male)

immun_cli
Flow from cli_uninf to cli_immun
immun_cli = vactake*vacrate_cli*cli_uninf

immun_csw
Flow from csw_uninf to csw_immun
immun_csw = vactake*vacrate_csw*csw_uninf

immun_fem
Flow from fem_uninf to fem_immun
immun_fem = vactake*vacrate_fem* fem_uninf

immun_men
Flow from men_uninf to men_immun
immun_men = vactake*vacrate_men*men_uninf

inf_cli_immun_r
Flow from cli_immun to cli_early_r
inf_cli_immun_r = vaceff*(cli_immun*cr*fmrisk*unprot * (w1*csw_early_r + w2*csw_late_r1 + w2*counsel_csw*(1-sustrans)*csw_late_r2) / csw)

inf_cli_immun_s
Flow from cli_immun to cli_early_s
inf_cli_immun_s = vaceff*(cli_immun*cr*fmrisk*unprot * (w1*csw_early_s + w2*csw_late_s + w2*counsel_csw*sustrans*csw_late_r2 + w2*resid_infect*csw_out) / csw)

inf_cli_r
Flow from cli_uninf to cli_early_r

inf_cli_r = STD_control*(cli_uninf*cr*fmrisk*unprot * (w1*csw_early_r + w2*csw_late_r1 + w2*counsel_csw*(1-sustrans)*csw_late_r2) / csw)

inf_cli_s
Flow from cli_uninf to cli_early_s
inf_cli_s = STD_control*(cli_uninf*cr*fmrisk*unprot * (w1*csw_early_s + w2*csw_late_s + w2*counsel_csw*sustrans*csw_late_r2 + w2*resid_infect*csw_out) / csw)

inf_csw_immun_r
Flow from csw_immun to csw_early_r
inf_csw_immun_r = vaceff*(csw_immun *annualCSWcontacts*mfrisk*unprot * (w1*cli_early_r + w2*cli_late_r1 +w2*counsel_cli*(1-sustrans)*cli_late_r2) / clients)

inf_csw_immun_s
Flow from csw_immun to csw_early_s
inf_csw_immun_s = vaceff*(csw_immun *annualCSWcontacts*mfrisk*unprot * (w1*cli_early_s + w2*cli_late_s +w2*counsel_cli*sustrans*cli_late_r2 + w2*resid_infect*cli_out) / clients)

inf_csw_r
Flow from csw_uninf to csw_early_r
inf_csw_r = STD_control*(csw_uninf *annualCSWcontacts*mfrisk*unprot * (w1*cli_early_r + w2*cli_late_r1 +w2*counsel_cli*(1-sustrans)*cli_late_r2) / clients)

inf_csw_s
Flow from csw_uninf to csw_early_s
inf_csw_s = STD_control*(csw_uninf *annualCSWcontacts*mfrisk*unprot * (w1*cli_early_s + w2*cli_late_s +w2*counsel_cli*sustrans*cli_late_r2 + w2*resid_infect*cli_out) / clients)

inf_fem_immun_r
Flow from fem_immun to fem_early_r
inf_fem_immun_r = vaceff* (leak*(w1*cli_early_r + w2*cli_late_r1 + w2*counsel_cli*(1-sustrans)*cli_late_r2 + w1*men_early_r + w2*men_late_r1+ w2*counsel_men*(1-sustrans)*men_late_r2) * fem_immun/fem + fem_immun*stabfactor*mfrisk*marrate2 * (w1*men_early_r + w2*men_late_r1+ w2*counsel_men*(1-sustrans)*men_late_r2) / men)

inf_fem_immun_s
Flow from fem_immun to fem_early_s
inf_fem_immun_s = vaceff*(leak*(w1*cli_early_s + w2*cli_late_s + w2*counsel_cli*sustrans*cli_late_r2 + w2*resid_infect*cli_out + w1*men_early_s + w2*men_late_s + w2*counsel_men*sustrans*men_late_r2 + w2*resid_infect*men_out) * fem_immun/fem +fem_immun*stabfactor*mfrisk*marrate2 * (w1*men_early_s + w2*men_late_s + w2*counsel_men*sustrans*men_late_r2 + w2*resid_infect*men_out) / men)

inf_fem_r
Flow from fem_uninf to fem_early_r
inf_fem_r = STD_control*((leak*(w1*cli_early_r + w2*cli_late_r1 + w2*counsel_cli*(1-sustrans)*cli_late_r2 + w1*men_early_r + w2*men_late_r1+ w2*counsel_men*(1-sustrans)*men_late_r2) * fem_uninf/fem + fem_uninf*stabfactor*mfrisk*marrate2 * (w1*men_early_r + w2*men_late_r1+ w2*counsel_men*(1-sustrans)*men_late_r2) / men))

inf_fem_s
Flow from fem_uninf to fem_early_s
inf_fem_s = STD_control*((leak*(w1*cli_early_s + w2*cli_late_s + w2*counsel_cli*sustrans*cli_late_r2 + w2*resid_infect*cli_out + w1*men_early_s + w2*men_late_s + w2*counsel_men*sustrans*men_late_r2 + w2*resid_infect*men_out) * fem_uninf/fem +fem_uninf*stabfactor*mfrisk*marrate2 * (w1*men_early_s + w2*men_late_s + w2*counsel_men*sustrans*men_late_r2 + w2*resid_infect*men_out) / men))

inf_men_immun_r
Flow from men_immun to men_early_r
inf_men_immun_r = vaceff*((leak*(w1*csw_early_r + w2*csw_late_r1 + w2*counsel_csw*(1-sustrans)*csw_late_r2 + w1*fem_early_r + w2*fem_late_r1+ w2*counsel_fem*(1-sustrans)*fem_late_r2) * men_immun/men+ men_immun*stabfactor*fmrisk*marrate * (w1*fem_early_r + w2*fem_late_r1 + w2*counsel_fem*(1-sustrans)*fem_late_r2) / fem))

inf_men_immun_s
Flow from men_immun to men_early_s
inf_men_immun_s = vaceff* (leak*(w1*csw_early_s + w2*csw_late_s + w2*counsel_csw*sustrans*csw_late_r2 + w2*resid_infect*csw_out + w1*fem_early_s + w2*fem_late_s+w2*counsel_fem*sustrans*fem_late_r2 + w2*resid_infect*fem_out) * men_immun/men+ men_immun*stabfactor*fmrisk*marrate * (w1*fem_early_s + w2*fem_late_s +w2*counsel_fem*sustrans*fem_late_r2 + w2*resid_infect*fem_out) / fem)

inf_men_r
Flow from men_uninf to men_early_r
inf_men_r = STD_control*((leak*(w1*csw_early_r + w2*csw_late_r1 + w2*counsel_csw*(1-sustrans)*csw_late_r2 + w1*fem_early_r + w2*fem_late_r1+ w2*counsel_fem*(1-sustrans)*fem_late_r2) * men_uninf/men+ men_uninf*stabfactor*fmrisk*marrate * (w1*fem_early_r + w2*fem_late_r1 + w2*counsel_fem*(1-sustrans)*fem_late_r2) / fem))

inf_men_s
Flow from men_uninf to men_early_s
inf_men_s = STD_control*((leak*(w1*csw_early_s + w2*csw_late_s + w2*counsel_csw*sustrans*csw_late_r2 + w2*resid_infect*csw_out + w1*fem_early_s + w2*fem_late_s+w2*counsel_fem*sustrans*fem_late_r2 + w2*resid_infect*fem_out) * men_uninf/men+ men_uninf*stabfactor*fmrisk*marrate * (w1*fem_early_s + w2*fem_late_s +w2*counsel_fem*sustrans*fem_late_r2 + w2*resid_infect*fem_out) / fem))

pro_cli_r
Flow from cli_early_r to cli_late_r1
pro_cli_r = hivprog * cli_early_r

pro_cli_s
Flow from cli_early_s to cli_late_s
pro_cli_s = HIVprog * cli_early_s

pro_csw_r
Flow from CSW_early_r to CSW_late_r1
pro_csw_r = hivprog * csw_early_r

pro_csw_s
Flow from CSW_early_s to CSW_late_s
pro_csw_s = HIVprog * csw_early_s

pro_fem_r
Flow from fem_early_r to fem_late_r1
pro_fem_r = hivprog * fem_early_r

pro_fem_s
Flow from fem_early_s to fem_late_s

pro_fem_s = HIVprog * fem_early_s

pro_men_r
Flow from men_early_r to men_late_r1
pro_men_r = hivprog * men_early_r

pro_men_s
Flow from men_early_s to men_late_s
pro_men_s = HIVprog * men_early_s

prof0
Flow from fem_immun to csw_immun
prof0 = prof* exp(annualCSWcontacts/1000-1) * fem_immun

prof1
Flow from fem_uninf to csw_uninf
prof1 = prof* fem_uninf*exp(annualCSWcontacts/1000-1)

prof2
Flow from fem_early_s to csw_early_s
prof2 = prof* fem_early_s*exp(annualCSWcontacts/1000-1)

prof3
Flow from fem_early_r to CSW_early_r
prof3 = prof * fem_early_r*exp(annualCSWcontacts/1000-1)

prof4
Flow from fem_late_s to CSW_late_s
prof4 = prof* exp(annualCSWcontacts/1000-1) * fem_late_s

prof5
Flow from fem_prog to CSW_prog
prof5 = prof*exp(annualCSWcontacts/1000-1) * fem_prog

prof6
Flow from fem_out to CSW_out
prof6 = prof*exp(annualCSWcontacts/1000-1) * fem_out

prof7
Flow from fem_late_r1 to CSW_late_r1
prof7 = prof*exp(annualCSWcontacts/1000-1) * fem_late_r1

prof8
Flow from fem_late_r2 to CSW_late_r2
prof8 = prof*exp(annualCSWcontacts/1000-1) * fem_late_r2

res_cli_out
Flow from cli_out to cli_late_r2
res_cli_out = outRDR * cli_out

res_cli_prog
Flow from cli_prog to cli_late_r2
res_cli_prog = progRDR * cli_prog

res_csw_out
Flow from csw_out to csw_late_r2
res_csw_out = outRDR * csw_out

res_csw_prog
Flow from csw_prog to csw_late_r2
res_csw_prog = progRDR * csw_prog

res_fem_out
Flow from fem_out to fem_late_r2
res_fem_out = outRDR * fem_out

res_fem_prog
Flow from fem_prog to fem_late_r2
res_fem_prog = progRDR * fem_prog

res_men_out
Flow from men_out to men_late_r2
res_men_out = outRDR * men_out

res_men_prog
Flow from men_prog to men_late_r2
res_men_prog = progRDR * men_prog

uncustom0
Flow from cli_immun to men_immun
uncustom0 = uncust * cli_immun

uncustom1
Flow from cli_uninf to men_uninf
uncustom1 = uncust* cli_uninf

uncustom2
Flow from cli_early_s to men_early_s
uncustom2 = uncust * cli_early_s

uncustom3
Flow from cli_early_r to men_early_r
uncustom3 = uncust * cli_early_r

uncustom4
Flow from cli_late_s to men_late_s
uncustom4 = uncust * cli_late_s

uncustom5
Flow from cli_prog to men_prog
uncustom5 = uncust * cli_prog

uncustom6
Flow from cli_out to men_out
uncustom6 = uncust * cli_out

uncustom7
Flow from cli_late_r1 to men_late_r1
uncustom7 = uncust * cli_late_r1

uncustom8
Flow from cli_late_r2 to men_late_r2
uncustom8 = uncust * cli_late_r2

unimmun_cli
Flow from cli_immun to cli_uninf
unimmun_cli = loss_immun * cli_immun

unimmun_csw
Flow from csw_immun to csw_uninf
unimmun_csw = loss_immun* csw_immun

unimmun_fem
Flow from fem_immun to fem_uninf
unimmun_fem = loss_immun * fem_immun

unimmun_men
Flow from men_immun to men_uninf
unimmun_men = loss_immun * men_immun

unprof0
Flow from csw_immun to fem_immun
unprof0 = unprof * csw_immun

unprof1
Flow from csw_uninf to fem_uninf
unprof1 = unprof* csw_uninf

unprof2
Flow from CSW_early_s to fem_early_s
unprof2 = unprof* csw_early_s

unprof3
Flow from CSW_early_r to fem_early_r

unprof3 = unprof * csw_early_r

unprof4
Flow from CSW_late_s to fem_late_s
unprof4 = unprof * csw_late_s

unprof5
Flow from CSW_prog to fem_prog
unprof5 = unprof * csw_prog

unprof6
Flow from CSW_out to fem_out
unprof6 = unprof * csw_out

unprof7
Flow from CSW_late_r1 to fem_late_r1
unprof7 = unprof * csw_late_r1

unprof8
Flow from CSW_late_r2 to fem_late_r2
unprof8 = unprof * csw_late_r2

VARIABLES
Variables defined in terms of compartments, flows etc in the model

aids_dead
aids_dead =
hivdying_females+hivdying_males+hivdying_kids

annualCSWcontacts
annualCSWcontacts = cr*clients/csw

cli_prop_prog
cli_prop_prog =
res_cli_prog/(res_cli_prog+res_cli_out+0.0001)

clients
clients =
cli_immun+cli_early_s+cli_late_s+cli_uninf+cli_prog+cli_late_r1+cli_late_r2+cli_out+cli_early_r

counsel_cli
counsel_cli = (1-old_cli_pp)+old_cli_pp*counsel

counsel_csw
counsel_csw = (1-old_csw_pp)+old_csw_pp*counselcsw

counsel_fem
counsel_fem = (1-old_fem_pp)+old_fem_pp*counsel

counsel_men
counsel_men = (1-old_men_pp)+old_men_pp*counsel

csw
csw =
csw_immun+csw_early_s+csw_late_s+csw_uninf+csw_prog+csw_late_r1+csw_late_r2+csw_out+csw_early_r

csw_prop_prog
csw_prop_prog =
res_csw_prog/(res_csw_prog+res_csw_out+0.0001)

fem
fem =
fem_immun+fem_early_s+fem_late_s+fem_uninf+fem_late_r1+fem_late_r2+fem_prog+fem_early_r+fem_out

fem_prop_prog
fem_prop_prog =
res_fem_prog/(res_fem_prog+res_fem_out+0.0001)

female_prev_res
female_prev_res =
(csw_late_r1+csw_late_r2+fem_late_r1+fem_late_r2+csw_early_r+fem_early_r)/females

female_prevalence
female_prevalence = (females-fem_uninf-csw_uninf-fem_immun-csw_immun)/females

females
females = fem+csw

In_out1
In_out1 = fem_out+men_out+csw_out+cli_out

In_out2
In_out2 = fem_out+men_out+csw_out+cli_out+(1-old_fem_pp)*fem_late_r2+(1-old_men_pp)*men_late_r2+(1-old_csw_pp)*csw_late_r2+(1-old_cli_pp)*cli_late_r2

In_prog1
In_prog1 = fem_prog+men_prog+csw_prog+cli_prog

In_prog2
In_prog2 =
fem_prog+men_prog+csw_prog+cli_prog+old_fem_pp*fem_late_r2+old_men_pp*men_late_r2+old_csw_pp*csw_late_r2+old_cli_pp*cli_late_r2

incidence
incidence =
round(inf_cli_immun_s+inf_csw_immun_s+inf_men_immun_s+inf_fem_immun_s+inf_cli_immun_r+inf_csw_immun_r+inf_men_immun_r+inf_fem_immun_r+inf_cli_s+inf_csw_s+inf_men_s+inf_fem_s+inf_cli_r+inf_csw_r+inf_men_r+inf_fem_r+inf_births)

inf_births
inf_births = round(brate*vtrate*(w1*csw_early_r+w1*fem_early_r+(w1*csw_early_s+w2*csw_late_s+w1*fem_early_s+w2*fem_late_s)*(nevirapine_rate*nevirapine_effect+(1-nevirapine_rate))+(csw_prog+fem_prog+csw_out+fem_out)*w2*nevirapine_effect+w2*(csw_late_r1+fem_late_r1+csw_late_r2+fem_late_r2)))

male_prev_res
male_prev_res =
(cli_late_r1+cli_late_r2+men_late_r1+men_late_r2+cli_early_r+men_early_r)/males

male_prevalence
male_prevalence = (males-cli_uninf-men_uninf-men_immun-cli_immun)/males

males
males = men+clients

marrate2
marrate2 = marrate*men/fem

men
men =
men_immun+men_early_s+men_late_s+men_uninf+men_late_r1+men_late_r2+men_prog+men_early_r+men_out

men_prop_prog
men_prop_prog =
res_men_prog/(res_men_prog+res_men_out+0.0001)

milpop
milpop = population/1000000

non_vaccinated

non_vaccinated = 1-
(men_immun+cli_immun+csw_immun+fem_immun)/(men
_immun+cli_immun+csw_immun+fem_immun+men_uninf
+cli_uninf+csw_uninf+fem_uninf)

population
population = round(males+females)

prev_CSW
prev_CSW = (csw-csw_uninf-csw_immun)/csw

preval_res
preval_res =
(males*male_prev_res+females*female_prev_res)/(males+f
emales)

prevalence
prevalence =
(males*male_prevalence+females*female_prevalence)/(mal
es+females)

prim_resistant
prim_resistant =
(csw_early_r+csw_late_r1+fem_early_r+fem_late_r1+cli_e
arly_r+cli_late_r1+men_early_r+men_late_r1)/(population*
prevalence)

prop_client
prop_client = clients/males

prop_csw
prop_csw = csw/females

prop_inf_births
prop_inf_births = inf_births/(brate*females)

prop_males
prop_males = males/population

resistant
resistant =
(csw_early_r+csw_late_r1+csw_late_r2+fem_early_r+fem_
late_r1+fem_late_r2+cli_early_r+cli_late_r1+cli_late_r2+m
en_early_r+men_late_r1+men_late_r2)/(population*prevale
nce)

vacrate_cli Conditional
vacrate_cli =
0.75 for t>startyr_vaccin+2
0.75 for t>startyr_vaccin
0 by default

vacrate_csw Conditional
vacrate_csw =
0.75 for t>startyr_vaccin+2
0.75 for t>startyr_vaccin
0 by default

vacrate_fem Conditional
vacrate_fem =
0 for t>startyr_vaccin+2
0 for t>startyr_vaccin
0 by default

vacrate_men Conditional
vacrate_men =
0 for t>startyr_vaccin+2
0 for t>startyr_vaccin
0 by default

year
year = t+1998

DELAYS
Time-lagged variables

old_cli_pp
Delay = 1.5
Initial Value = 0
Maximum Delay = 1.7

old_csw_pp
Delay = 1.5
Initial Value = 0
Maximum Delay = 1.7

old_fem_pp
Delay = 1.5
Initial Value = 0
Maximum Delay = 1.7

old_men_pp
Delay = 1.5
Initial Value = 0
Maximum Delay = 1.7

DEFINE VALUES
Variables influenced by interventions

cr
cr = cr_before

nevirapine_rate
nevirapine_rate = 0

recr_cli_out
recr_cli_out = 0

recr_cli_prog
recr_cli_prog = 0

recr_CSW_out
recr_CSW_out = 0

recr_csw_prog
recr_csw_prog = 0

recr_fem_out
recr_fem_out = 0

recr_fem_prog
recr_fem_prog = 0

recr_men_out
recr_men_out = 0

recr_men_prog
recr_men_prog = 0

STD_control
STD_control = 1

unprot
unprot = unprot_before

INDEPENDENT EVENTS
Interventions

STD_prog
Non-periodic triggers at:
startyr_std
Actions:
STD_control = STD_effect;

Introd_art
Non-periodic triggers at:
startyr_art_intro
Actions:
recr_men_out = art_out_effect;
recr_fem_out = art_out_effect;
recr_csw_out = art_out_effect;
recr_cli_out = art_out_effect;

Inter_HAART_pop
Non-periodic triggers at:
startyr_art_pop
Actions:
recr_cli_prog = recr_cli_prog_effect;
recr_fem_prog=recr_fem_prog_effect;
recr_men_prog = recr_men_prog_effect;
recr_men_out = gen_prog_eff*art_out_effect;
recr_fem_out = gen_prog_eff*art_out_effect;
recr_cli_out = gen_prog_eff*art_out_effect;
cr=cr_after;

Inter_HAART_CSW
Non-periodic triggers at:
startyr_art_csw
Actions:
recr_csw_prog = recr_csw_prog_effect;
recr_csw_out = gen_prog_eff*art_out_effect;

Inter_CSW
Non-periodic triggers at:
startyr_condom_CSW
Actions:
unprot = unprot_after;

Inter_MCT
Non-periodic triggers at:
startyr_MCT
Actions:
nevirapine_rate = nevirapine_prop;

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