Title Page

Title: HIV serosorting as a harm reduction strategy: Evidence from Seattle, Washington
Short title *(40 characters max)*: HIV serosorting as a harm reduction strategy

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Abstract

Objective: The objective of our work is to estimate how serosorting may affect population-level HIV prevalence among MSM in Seattle, Washington, and how the results vary under different assumptions of HIV testing frequency, heterogeneity in sexual behavior, and condom use.

Methods: We developed a deterministic mathematical model of HIV transmission dynamics to study how serosorting affects HIV prevalence within the population. Data from the 2003 random digit dial study of MSM conducted in Seattle, Washington (n = 400) are used to parameterize the model.

Results: Predicted population-level HIV prevalence as well as an individual's risk of HIV acquisition decreases when the odds of serosorting are increased in the mathematical model. In our baseline model with serosorting, HIV prevalence is 16.0%, and increased to 24.5% without reported levels of serosorting. However, our findings depend on rates of condom use, mean anal sex contact rates, and HIV testing in the population.

Conclusions: Under realistic scenarios of sexual behavior and testing frequency for MSM in the US, serosorting can be an effective harm reduction strategy.

Key words: HIV/AIDS, mathematical modeling, homosexual men, HIV testing

Introduction

Hardest hit by the HIV epidemic in the United States and other nations, men who have sex with men (MSM) have employed innovative strategies to reduce their risk of HIV infection (1). In the early 1990s, prevention scientists reported that MSM used condoms more often with partners of discordant HIV status than with partners of concordant HIV status (2-4). Subsequent reports demonstrate that both HIV-positive and HIV-negative men are much more likely to have sex with partners of the same HIV status (5-12). In a recent clinic-based sample of HIV-positive MSM, 24% reported that they decided not to have sex with another man in the last year because that potential partner was HIV-negative, while 31% reported that a potential partner decided not to have sex with them because they were HIV-positive (13).

These documented practices of preferentially selecting sex partners of the same HIV status or preferentially using condoms with partners of different HIV status have been termed serosorting (14). Researchers in the San Francisco, Seattle, London, and Sydney suggest that serosorting may be increasing (15-18), and some investigators have posited that this increase in serosorting has averted increases in HIV infection despite dramatic increases in bacterial sexually transmitted infections among MSM (19).

Despite the potential benefits of serosorting, cohort and case-control studies have revealed associations between unprotected anal intercourse (UAI) with men believed to be HIV-negative (20, 21), UAI with casual partners believed to be HIV-negative (22), and the number of HIV-negative partners a participant reports (23) with HIV acquisition. In one study, 32% of men testing HIV positive acknowledged UAI with an HIV-negative partner as their highest risk behavior (17). Further, two recent reports suggest that because of the high risk of HIV transmission during the acute HIV infection, choosing to have sex with a presumptively negative partner may carry a greater risk of HIV infection than sex with a person with known, established HIV infection (24, 25). However, recent analyses suggest that men who practice higher levels of serosorting are at decreased risk of HIV infection (26) and that the risk of HIV acquisition in men who practice serosorting in the context of negotiated safety is not different than men who do not engage in UAI (21).

Using a deterministic, compartmental mathematical model parameterized with population-based behavioral surveillance data (27), we evaluated the population-level effects of serosorting among MSM in Seattle, WA under varying contexts of partner selection, condom use, and HIV testing.

Methods

We developed a deterministic, continuous-time compartmental mathematical model of HIV transmission. In this framework, the population is divided into compartments (Figure 1) whose size through time is specified using a system of ordinary differential equations. The structure of these equations incorporates information about many behavioral and biological factors affecting HIV transmission and progression. We explain the general structure for our model and the assumptions underlying it here, and provide more complete technical detail in the online appendix.

Data sources: Data from the 2003 random digit dial (RDD) study of MSM conducted in Seattle, Washington (n = 400) are used to parameterize the model, such as levels of condom use and number of contacts/year (27). The response rate for the RDD was 46% (28), comparable to the 2003 Washington State Behavioral Risk Factor Surveillance System survey (29).

Model framework: The compartments for this model are defined by anal sexual activity level (low, high, none) and true HIV serostatus (negative, positive). HIV-infected men are further subdivided by stage of disease and by detection of HIV infection by two tests, 2nd generation enzyme immunoassay (EIA) and RNA amplification testing (NAAT); the five resulting categories are acute NAAT-/EIA-; early NAAT+/EIA-; early NAAT+/EIA+; chronic; and AIDS. Men in the last four of these stages of disease are also categorized by diagnosis status (undiagnosed, diagnosed), and diagnosed men are further subdivided by antiretroviral therapy status (untreated, treated). While men who do not have anal sex do not comprise a separate compartment, they are included in the denominator of summary measures of HIV prevalence. Together these attributes define 24 compartments. These compartments can be grouped into three types, which will play distinct roles in serosorting risk: true negatives, undiagnosed positives and diagnosed positives. We refer to men's "true serostatus" ("true negative" vs. "true positive") and "apparent serostatus" ("apparent negative" (AN) vs. "diagnosed positive" (DP), where "apparent negative" includes both true negatives and undiagnosed positives).

Transition from true negative to undiagnosed positive. Transition from true negative into the acute HIV NAAT-/EIA- stage occurs through transmission, which is a potential consequence of anal sex with a true positive. We define "act" as an instance of anal sex between two men, "contact" as the set of all acts between two men, "contact rate" as the rate at which new contacts occur, and "partner" as either member of a contact. The contact rate between true negatives and true positives is determined by the overall contact rates for apparent negatives and apparent positives (which determine how many contacts occur), the level of serosorting (which determines how many of these are apparently serodiscordant), and the fraction of apparent negatives who are true negatives. We derive these values from our data on respondent's activity level, their apparent serostatus, and their partner's perceived serostatus.

The 2003 Seattle MSM random digit dial study asked men to enumerate their contacts in the prior year. We defined low-activity men as those who reported 1 contact in the prior year and high-activity men as those who had >1 contact in the prior year. The group of men who reported >1 contact had a mean of 10 contacts in the prior year. Therefore, in our model, low-activity men have an average of 1 new contact per year, while high-activity men have 10 contacts per year. We do not have data on the partner's activity level, so we assume random mixing by activity level. We also assume that men tell the truth as they know it; i.e. those who have ever tested positive report themselves to be positive, and those who have never tested positive report themselves to be negative.

In our baseline run, we assume levels of serosorting within our various types of contacts that are consistent with our data, as measured by two odds ratios: (1) the odds that a low-activity DP man's partner is DP is 68 times greater than the odds that a low-activity AN man's partner is DP; and (2) the odds that a high-activity DP man's partner is DP is 12 times greater than the odds that a high-activity AN man's partner is DP. (See the appendix for additional explanation). While

population size and composition change over time (as a result of differential rates of infection among high- and low-activity men), our approach ensures that these two components of serosorting for which we have direct measures remain constant, as do the overall contact rates for every compartment. By measuring serosorting based on the counts in a table of unprotected anal sex contacts by serostatus, we are thus including three separate processes within our definition of serosorting: (1) men selecting sex partners in general based on serostatus; (2) men choosing sex act (anal vs. other) based on serostatus; and (3) men deciding on condom use based on serostatus.

Within serodiscordant contacts, the probability of transmission is a function of the seropositive's stage and treatment status, the number of acts per contact, the role of each partner within those acts (insertive vs. receptive anal intercourse), and condom use. Since estimates of transmission probabilities by stage of infection have not be published for anal sex, we derived overall probabilities of transmission per serodiscordant act by stage and role (insertive, receptive) by taking the stage-specific numbers for heterosexuals from Wawer and colleagues (30) and scaling them all according to Vittinghoff and colleagues (31). The number of acts per contact and sexual role during those acts are derived from participant reports in the 2003 Seattle RDD (Table 1).

Transition through stages of disease. In accordance with the window periods of NAAT and 2nd generation EIA, we assume that it takes 7 days for an acutely infected individual to develop detectable HIV RNA (32, 33) and an additional 35 days to develop detectable anti-HIV antibody (34). Primary infection concludes on average 48 days later, for a total duration of heightened infectiousness of 3 months (30). In the absence of treatment, chronically infected individuals develop AIDS after 10 years (30, 35-38). With treatment, the rate of progression to AIDS is reduced by 0.6 (36, 38, 39).

Transition from apparent negative to diagnosed positive. In our base model, we assume that low-activity men test for HIV by EIA on average once per year, while high-activity men test twice per year (27).

Transition between treated and untreated. In the 2003 Seattle RDD, 67% of HIV-positive participants reported taking antiretroviral medications (27). We assume that men discontinue ARVs because of treatment failure or side effects at a rate of 0.02/year (40-42). Given these two quantities, we derived a rate of treatment initiation for men with chronic infection or AIDS of 0.15/year.

Entry and exit. Men who remained true negatives were assumed to exit the sexually active population either through sexual retirement or death on average 50 years after entering it. Upon a diagnosis of AIDS, we assume men die, on average, within 2 years if not on treatment. Treatment reduces AIDS-related mortality by 0.6. For the sake of convenience, the entrance rate is set equal to the exit rate, and is partitioned among activity classes by the same proportion as the exits occur.

Parameterization. We ran the model under a variety of parameter values as informed by our data and by the hypothetical scenarios we wished to explore. These include a number of testing scenarios (decreases in average inter-test interval, combination of EIA and NAAT testing), and levels of condom use and sexual behavior. Population risk is estimated by examining

equilibrium HIV prevalence in the population as a whole across multiple scenarios with different levels of serosorting.

The system of equations that results from this model is expressed in full in the Appendix. The system is coded and solved using Berkeley Madonna 8.3.14 (Berkeley Madonna, Inc., University of California, Berkeley). This research was approved by the University of Washington Human Subjects Division (07-9056-X/C).

Results

Our baseline model replicates the epidemic using our observed data, including observed levels of serosorting. In this run, endemic prevalence is 16.0%, well within the range of prevalence estimated for Seattle MSM of 13% (95% confidence interval [CI]: 10%-17%). In comparison, by eliminating serosorting (i.e. maintaining the same overall contact rates for both AN and DP, but making contact random by apparent serostatus), endemic prevalence rises to 24.5%. A number of other qualitative differences emerge between the two baseline models, as a result of both the lack of serosorting and simply increased prevalence. In the baseline model with serosorting, 22% of contacts are apparently serodiscordant, whereas without serosorting 50% are. The proportion of contacts between two AN men in which one man is an undiagnosed positive is lower in the baseline model (2.3%) compared to the model without serosorting (4.8%). However, the proportion of infections resulting from all undiagnosed, infected men and undiagnosed, recently infected men (<3 months) is higher in the baseline model with serosorting (48.1% and 30.6% respectively) compared to the model without serosorting (26.0% and 16.5% respectively). In all the models, serosorting tends to increase the proportion of infections stemming from undiagnosed men, including undiagnosed men with primary stage HIV infection.

Individual-level risk of acquiring HIV from an unprotected anal sex act with an AN man is also lower in the baseline model with serosorting compared to the no-serosorting scenario. The probability of HIV transmission during unprotected receptive anal intercourse (URAI) and unprotected insertive anal intercourse (UIAI) with an AN man in the serosorting scenario is equal to 0.0002 and 0.00005, respectively. The rate is nearly twice as high for both URAI and UIAI in the no-serosorting scenario. UAI with an AN, according to our model, is not as risky as UAI with a DP. In the serosorting scenario, the probability of acquiring HIV from URAI and UIAI is equal to 0.005 and 0.00047, respectively. The rates did not change in the no-serosorting scenario.

Figure 2 shows endemic prevalence for a variety of contact rates (produced by increasing the contact rate of each group by the same fraction), with and without condom use in perceived seroconcordant contacts. In the baseline model, the mean contact rate equals 3.76, and as seen in Figure 2 (panel A) the difference in endemic prevalence between the serosorting and no-serosorting scenario is 8.5% (24.5% - 16.0%). Endemic prevalence is lower in serosorting scenarios compared to equivalent no-serosorting scenarios when contact rates are below 7.0 contacts per year. The difference in prevalence becomes smaller as mean contact rates increase. When the mean contact rate exceeds 7.0 contacts per year, the relationship is reversed and

endemic HIV prevalence is higher under the serosorting scenarios. In other words, men would need an 86% increase in contact rate to abrogate the protective effects of serosorting.

Butler et al. only considered a scenario in which there was no condom use for perceived concordant contacts, and the prevalence of acute-stage positives among all sexually active AN men was 4% (43). Although our data do not reflect either of these processes, we conducted additional simulations incorporating each of these two assumptions for the sake of comparison (see Figure 2, panel B). We consider the epidemic resulting from observed serosorting level vs. no serosorting under a variety of mean contact rates in order to achieve different proportions of acute-stage positives among AN men; in addition, we assume no condom use within perceived concordant contacts. Again, endemic prevalence is lower in the serosorting scenarios compared to equivalent no-serosorting scenarios when the mean contact rate is less than 4.9, but the relationship is reversed when the mean contact rate exceeds 4.9 contacts per year. Thus, if men did not use condoms within perceived concordant contacts, the model suggests that men would need a 30% increase in contact rate to abrogate the protective effects of serosorting. In order to achieve the scenario that Butler et al. consider (4% of AN men actually recently infected within 6 months) we need to increase contact rates by 90% over observed contact rates for our population. At this unrealistic level, serosorting appears to be detrimental; prevalence is 3% higher in the serosorting scenario compared to the equivalent no-serosorting scenario. Individual-level risk of acquiring HIV per unprotected anal sex act with an AN man is also higher in the serosorting scenario compared with the no-serosorting scenario (0.001 vs. 0.0009 for URAI, 0.0003 vs. 0.0002 for UIAI). No difference is observed in individual-level risk of HIV from UAI with a DP man in the two serosorting scenarios.

Next, we consider the effect of observed serosorting levels vs. no serosorting under various levels of condom use in perceived discordant contacts (Figure 3a). In the baseline model, 31% of sexual acts were protected with condoms in perceived discordant contacts. Endemic prevalence is consistently higher in the no-serosorting scenarios for all levels of condom use in perceived discordant contacts, by around 8%. At no point does the effect from serosorting reverse direction. Under reported contact rates, were men not to serosort, they would need to increase the mean proportion of acts in perceived discordant contacts that are protected by condoms by 50% in order to achieve the prevalence observed in the base serosorting scenario.

For all levels of condom use in perceived concordant contacts, endemic prevalence is higher given baseline levels of serosorting compared to equivalent no-serosorting scenarios (see Figure 3b). However, the difference in prevalence decreases as the proportion of acts protected by condoms decreases, from a 12% difference at 90% coverage, to about 5% difference at 10% condom coverage in perceived concordant contacts. Thus, the more condoms are used in perceived concordant contacts, the bigger the protective effect of serosorting. Under reported contact rates, no increase in the mean proportion of acts in perceived concordant contacts that are protected by condoms would allow a population without serosorting to achieve the prevalence observed in the base serosorting scenario.

Figure 4 yields five observations of the effect of testing frequency on the relationship between HIV endemic prevalence and serosorting. First, the addition of testing to the model results in a large decrease in endemic prevalence. Second, endemic prevalence declines as testing frequency

increases. However, within each scenario the reduction in the endemic prevalence with each increase in testing frequency decreases as high-activity men test more frequently. Third, endemic prevalence is consistently lower under assumptions of serosorting compared with no serosorting under all testing frequency assumptions. Fourth, the magnitude of the difference in prevalence between the serosorting and no-serosorting scenarios increases with more frequent testing. Finally, test type leads to differential effects on endemic prevalence in models with and without serosorting behavior. Under serosorting, the addition of NAAT to EIA testing decreases the equilibrium prevalence by 1.8% when both low- and high-activity men test once per year and by 2.4% when low-activity men test once per year and high-activity men test 4 times per year. Under no serosorting, these estimates are 0.6% and 1.3%, respectively. We note that our estimates of endemic prevalence would be similar if we assumed that low-activity men tested twice per year instead of once per year (data not shown).

Discussion

Our model suggests that serosorting is protective at both the population level and the individual level under a variety of scenarios. This includes the scenario representing the current behavior of MSM in Seattle, for whom current serosorting patterns are highly protective as compared to the alternative of no serosorting *ceteris paribus*. To place serosorting in context, if Seattle MSM were to abandon serosorting as a practice and choose partners randomly by serostatus instead, they would need to either reduce their contact rate by 30% or increase their condom use with discordant contacts by 50% in order to prevent an increase in transmission. This 30% contact rate reduction can be conceived of as either a 30% reduction in sex partners altogether or as substituting 30% of anal sex partners for oral sex partners, since the latter act carries little to no HIV transmission risk (44-50) . The magnitude of the benefit depends on numerous behavioral determinants, including condom use and the sexual behavior of recently HIV infected, undiagnosed individuals.

Our results are consistent with both case-control and prospective longitudinal studies of MSM evaluating risks for HIV acquisition have reported higher odds ratios for having unprotected sex with known HIV positive partners than for having unprotected sex with partners thought to be HIV negative (51-53). Our work does differ slightly from some previous findings, however. Butler et al. found that serosorting would be detrimental when the prevalence of recent (<6 months) seroconverters among all negative disclosers was 4% (43). We find that such a percentage would be unrealistic for the population we modeled; in our models, the figure is typically around 1.2% if we assumed a 6 month primary stage, or about 0.5% given our assumption of a 3 month primary stage. Direct evidence on the question is sparse. For instance, McKellar et al. found that 7.4% (80/1075) of the men disclosing as negative in their study were positive and in any stage of infection (54). They did not provide an estimate of the number who were positive and in the acute stage, although a minimum value from their data would be 2.8% (30/1075) of the men disclosing as negative, i.e. the number who were positive and had tested negative < 6 months ago. This figure is likely to be too high for the general MSM population, since the study was targeting young, high-risk men through venue-based sampling (55, 56). Nonetheless, in our model we found that if indeed 4% of seronegative disclosures actually were in the acute stage, serosorting would be detrimental.

It is imperative to remember that our scenarios considered serosorting relative to not serosorting, with all other behaviors being held equal. That is, for an HIV-negative man to choose an apparently HIV-negative partner for UAI is generally highly protective relative to choosing one who is known to be HIV-positive. However, choosing UAI with an apparently HIV-negative man may be more risky than either PAI or oral sex with an apparently HIV-positive man. Our results also showed that the relative protective effect of serosorting grew as condom use grew within perceived seroconcordant contacts. Although condoms provide the greatest protection against HIV transmission, some MSM will practice UAI. Serosorting is one strategy that MSM employ to minimize (but not eliminate) the risk of acquiring or transmitting HIV and still attain sexual satisfaction and intimacy (57). Some men in primary relationships with men they know to be seroconcordant may practice UAI within the relationship, but not outside of the relationship (58-61). In a recent report, HIV-negative men who practiced this strategy, known as negotiated safety, were not at higher risk of HIV infection compared to men who did not practice UAI (21). Serosorting outside the context of negotiated safety and with casual partners carried a higher risk of infection than not practicing UAI but a lower risk than having UAI with known HIV-positive partners (21). Knowing that the risk of HIV transmission associated with insertive UAI is less that associated with receptive UAI, some HIV-negative men only practice insertive UAI and some HIV-positive men only practice receptive UAI (21, 26, 62). HIV-negative men who only practiced insertive UAI have a risk of HIV infection smilar to those who did not practice UAI (21). Finally, HIV-negative men who have receptive UAI and HIV-positive men who have insertive UAI may practice withdrawal before ejaculation (62). For HIV-negative men, this appears to be the least effective harm reduction strategy as it is practiced mainly with HIV-positive partners (21). Therefore, any attempt to promote serosorting as a harm reduction strategy must be seen as one piece of a combination of strategies that considers a hierarchy of risk and acknowledges an individual's current place in that hierarchy.

Our model is agnostic as to the exact mechanisms behind serosorting. That is, is the process being driven primarily by the decisions of diagnosed seropositives, apparent seronegatives, or a mix of the two? As members of one group began to say no to sexual contact with members of the other, did the second group replace those lost contacts with other ones, or simply have less sex? We did not seek to separate out these pieces, since our data did not allow us to. Moreover, the impacts of positive-driven serosorting vs. negative-driven serosorting may not be as different as it initially seems. If one group begins to drive the process of serosorting, a smaller fraction of their contacts will be with members of the other group. If the second group does not reduce their activity rate but simply seeks out new partners, then the overall proportion of their contacts that are within-serostatus will also go up. Regardless of which group initiates the process, both groups end up seeing an increase in the proportion of their contacts that are seroconcordant. Thus, efforts to encourage serosorting among positives should filter into protecting seronegatives, and may avoid many of the ethical issues involved in promoting serosorting among seronegatives, even as a risk-reduction strategy only.

Although serosorting appears to generally reduce the level of HIV infections, it also strongly increases the proportion of new HIV infections stemming from all undiagnosed individuals, including from those recently infected. Typically such infections are the most difficult to prevent, since attempts to prevent transmission that are based on knowledge of positive status (so-called “prevention with positives”) are not available. Other interventions capable of reducing

infectivity without requiring knowledge of status (e.g. pre-exposure prophylaxis, or future vaccines) might be relatively more important in the presence of strong serosorting than they would otherwise. In general, efforts to consider the potential impact of interventions among MSM that rely on solid estimates of the population-attributable risk of acute infection need to consider serosorting and likely other behavioral factors common among MSM.

Our work relies on self-reports of perceived partner status. Unlike many other cases where self-reports are inherently limiting, in this case it is appropriate because serosorting occurs on the basis of individuals' beliefs about their potential partners' serostatus rather than the actual status. We did not, however, include the possibility that individuals might knowingly misrepresent their serostatus (13). This misrepresentation would undoubtedly reduce the protective effect of serosorting, but in the 2003 RDD only 2 of 37 (5%) HIV-positive men and 4 of 189 (2%) HIV-negative men reporting anal sex in the prior year reported misrepresenting their HIV status.

Our work included more detail about testing types and testing frequency than had previous work, a key addition since the gap between perceived and actual status is key in determining whether or not serosorting is an HIV preventative behavior. However, we did not consider the use of 3rd and 4th generation EIA technologies and p24 antigen testing which have much shorter window periods compared to 2nd generation EAI (63, 64). The use of these tests would likely enhance the effectiveness of serosorting. Furthermore, we also did not consider the motivation for testing: while some MSM test on a regular basis regardless of risk, others may be spurred to test by specific risk events and symptoms of acute HIV infection (65). In our model, we only consider the former; future work should consider the latter as well. Doing so might require a move to more agent-based modeling tools that allow for a wider range of hazard functions for phenomena such as testing (other than the exponential that is implicit in compartment modeling) that can be more closely tuned to data.

Despite the paucity of data that exist on the benefits of serosorting, some public health programs and community-based organizations are now promoting the practice (66). Our data and model suggest that under realistic scenarios of sexual behavior and testing frequency for MSM in the US, serosorting can be an effective harm reduction strategy.

Table 1. Model inputs, Seattle MSM serosorting model.

Parameter	Value	Source
Population Dynamics		
Proportion of men with no anal sex contacts in the prior year	0.39	Derived from(27)
Proportion of low-activity men	0.26	Derived from(27)
Proportion of high-activity men	0.35	Derived from(27)
Duration of sexual activity ($1/\mu$ years)	50	
HIV Disease Dynamics		
Duration of acute RNA-/Ab- infection ($1/\upsilon$ days)	7	(30, 35-38, 69)
Duration of acute RNA+/Ab- infection ($1/\rho$ days)	35	(30, 35-38, 69)
Duration of early RNA+/Ab+ infection ($1/\kappa$ days)	48	(30, 35-38)
Duration of chronic infection ($1/\gamma$ years)	10	(30, 35-38)
Duration of AIDS ($1/\alpha$ years)	2	(30, 35-38, 70)
HIV Treatment		
Proportion of men with chronic infection or AIDS who are on treatment	0.63	Derived from(27)
Rate of withdrawal from treatment (ω yr^{-1})	0.02	(40-42)
Reduction in rate of disease progression from chronic infection to AIDS as a result of ARV (ε_P)	0.6	(36, 38, 39)
Reduction in rate of death from AIDS as a result of ARV (ε_M)	0.6	(36-38, 70)
Reduction in infectiousness as a result of ARV (ε_T)	0.6	(71-74)
HIV Transmission		
Transmission probability per receptive UAI act, primary infection ($\beta_{A\,rec}^{1}$)	0.02	(30, 31, 75-79)
Transmission probability per receptive UAI act, chronic infection ($\beta_{A\,rec}^{2}$)	0.008	(30, 31, 75-78)
Transmission probability per receptive UAI act, AIDS ($\beta_{A\,rec}^{3}$)	0.01	(30, 31, 75-78)
Transmission probability per insertive UAI act, primary infection ($\beta_{A\,ins}^{1}$)	0.008	(30, 31, 75-79)
Transmission probability per insertive UAI act, chronic infection ($\beta_{A\,rec}^{2}$)	0.0007	(30, 31, 75-78)
Transmission probability per insertive UAI act, AIDS ($\beta_{A\,rec}^{3}$)	0.001	(30, 31, 75-78)
Sexual Risk Behavior		
Mean number of anal sex contacts per year (c), base model		
Low-activity men (c_L)	1	Derived from(27)
High-activity men (c_H)	10	Derived from(27)
Mean number of anal sex acts per perceived concordant anal sex contacts reported by HIV-negative men, base model[a]		

Low-activity men (n_L^1)	32	Derived from(27)
High-activity men (n_H^1)	22	Derived from(27)
Mean number of anal sex acts per perceived discordant anal sex contacts reported by HIV-negative men, base model[a]		
Low-activity men (n_L^0)	14	Derived from(27)
High-activity men (n_H^0)	6	Derived from(27)
Proportion of anal sex acts in which the negative partner is insertive, for perceived discordant contact, base model[a]		
Low-activity men ($p_{ins\,L}^{\ 0}$)	0.64	Derived from(27)
High-activity men ($p_{ins\,H}^{\ 0}$)	0.41	Derived from(27)
Proportion of acts of insertive anal sex with perceived concordant partners during which HIV-negative men use condoms, base model [a]		
Low-activity men ($C_{ins\,L}^{\ 1}$)	0.54	Derived from(27)
High-activity men ($C_{ins\,H}^{\ 1}$)	0.34	Derived from(27)
Proportion of acts of insertive anal sex with perceived disconcordant partners during which HIV-negative men use condoms, base model [a]		
Low-activity men ($C_{ins\,L}^{\ 0}$)	0.36	Derived from(27)
High-activity men ($C_{ins\,H}^{\ 0}$)	0.14	Derived from(27)
Proportion of acts of receptive anal sex with perceived concordant partners during which HIV-negative men use condoms, base model[a]		
Low-activity men ($C_{rec\,L}^{\ 1}$)	0.54	Derived from(27)
High-activity men ($C_{rec\,H}^{\ 1}$)	0.34	Derived from(27)
Proportion of acts of receptive anal sex with perceived disconcordant partners during which HIV-negative men use condoms, base model[a]		
Low-activity men ($C_{rec\,L}^{\ 0}$)	0.29	Derived from(27)
High-activity men ($C_{rec\,H}^{\ 0}$)	0.22	Derived from(27)
Effectiveness of condoms (ε_C)	0.8	(80)
HIV Testing Frequency		
Second generation enzyme immunoassay (EIA), base model (range)		
Low-activity men (θ_L yr^{-1})	1 (1-2)	Derived from(27)
High-activity men (θ_H yr^{-1})	2 (1-4)	Derived from(27)

Nucleic acid amplification testing (NAAT), base model (range)		
Low-activity men (τ_L yr^{-1})	0 (0-2)	
High-activity men (τ_H yr^{-1})	0 (0-4)	
Sexual Mixing		
Serosorting odds ratio derived from reports by low-activity men (OR_L)	68	Derived from(27)
Serosorting odds ratio derived from reports by high-activity men (OR_H)	12	Derived from(27)
Serosorting odds ratio in contacts between low-activity and high-activity men ($OR_{L,H}$)	60	

[a]For these parameters, our data provide us with two estimates: one for low-activity men and high-activity men. However, our model requires three parameters defined on activity levels for actor pairs (HH, HL, LL). The method we use to derive these three parameters from the two observed data points is identical for each parameter class and is described in the online appendix.

Figure 1: Flow chart of mathematical model

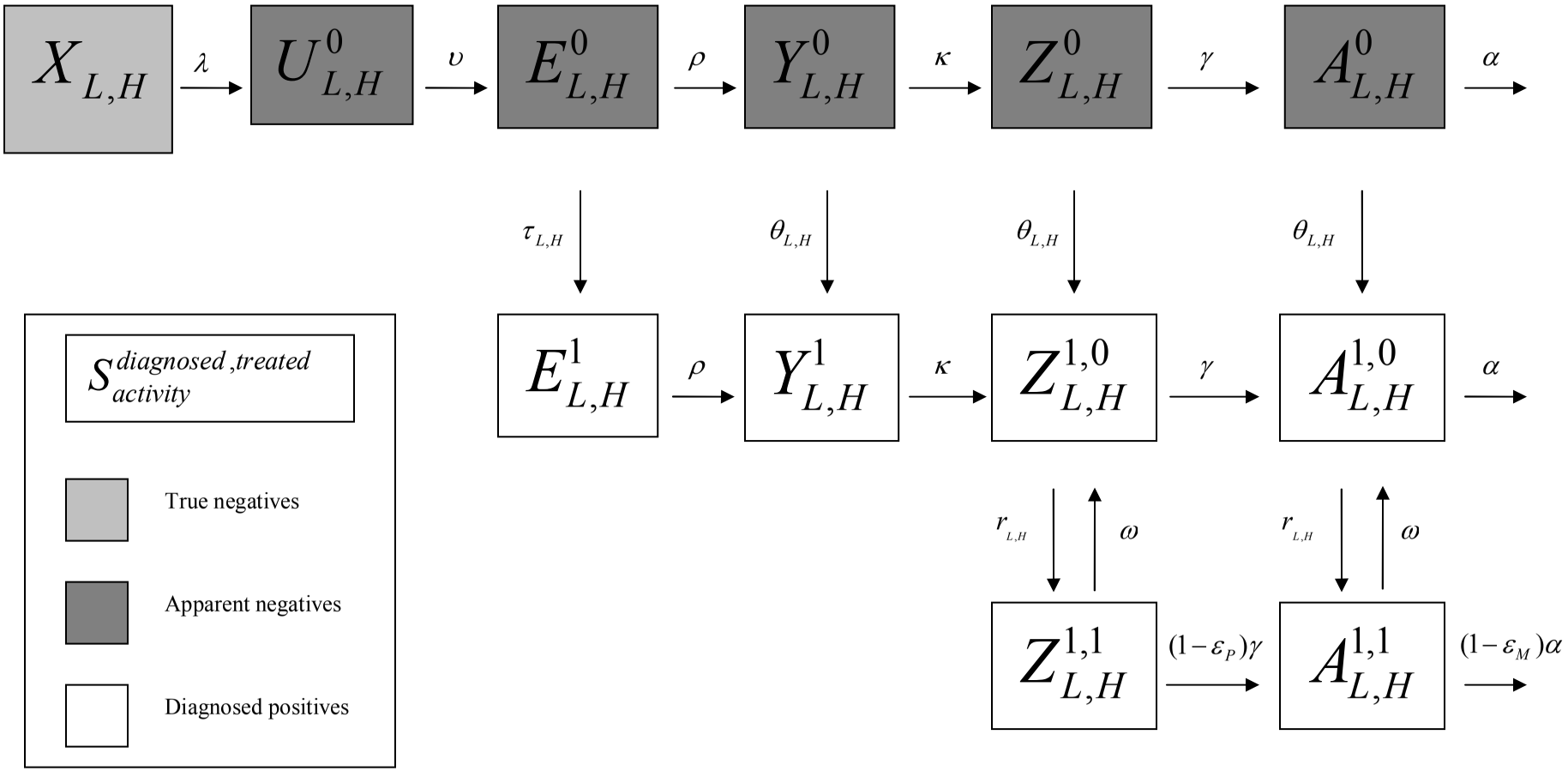


Figure 2: Equilibrium HIV prevalence for a variety of mean anal sex contact rates, with and without condom use in perceived seroconcordant contacts

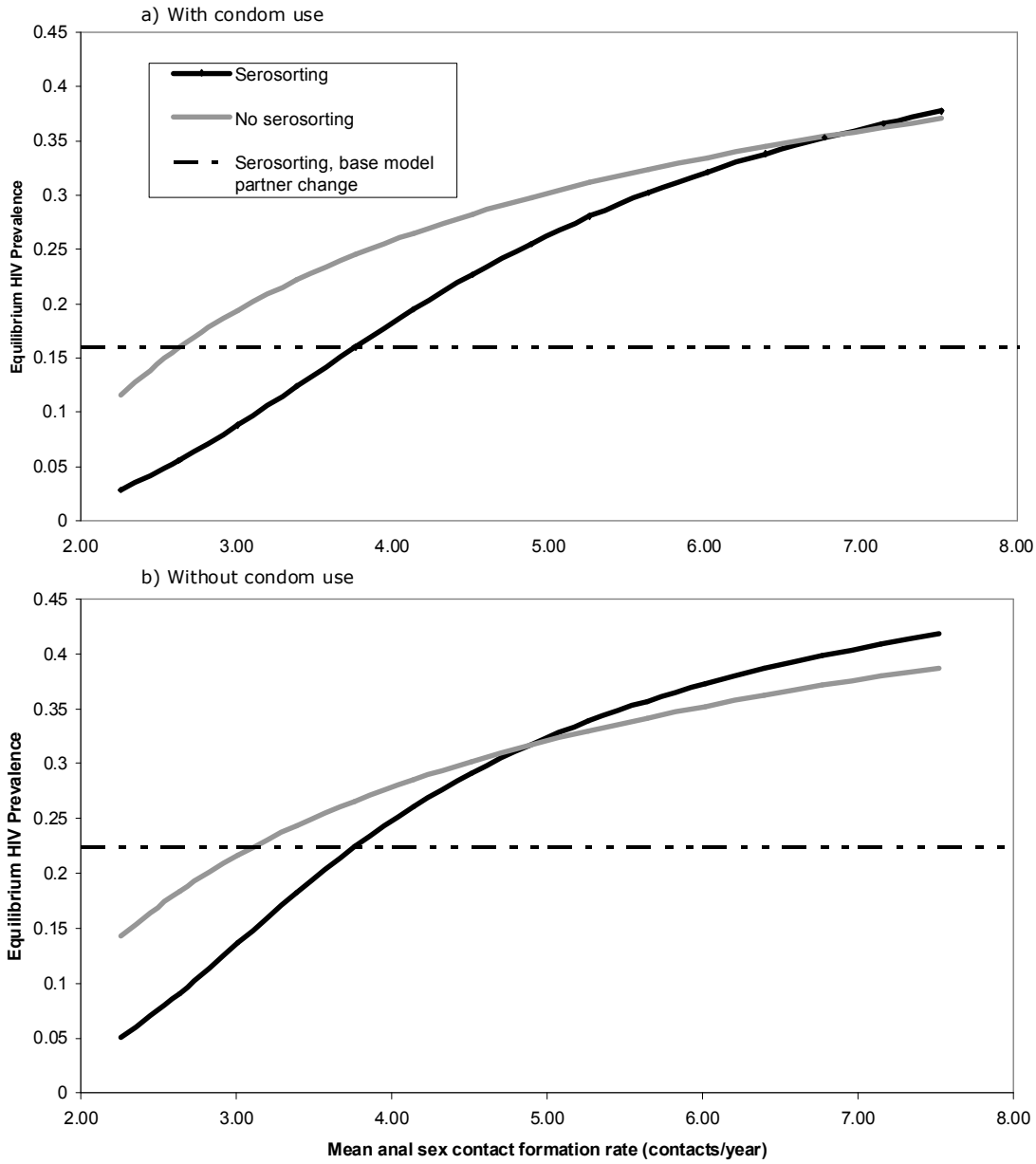


Figure 3: Equilibrium HIV prevalence for various levels of condom use in perceived serodiscordant and perceived seroconcordant contacts.

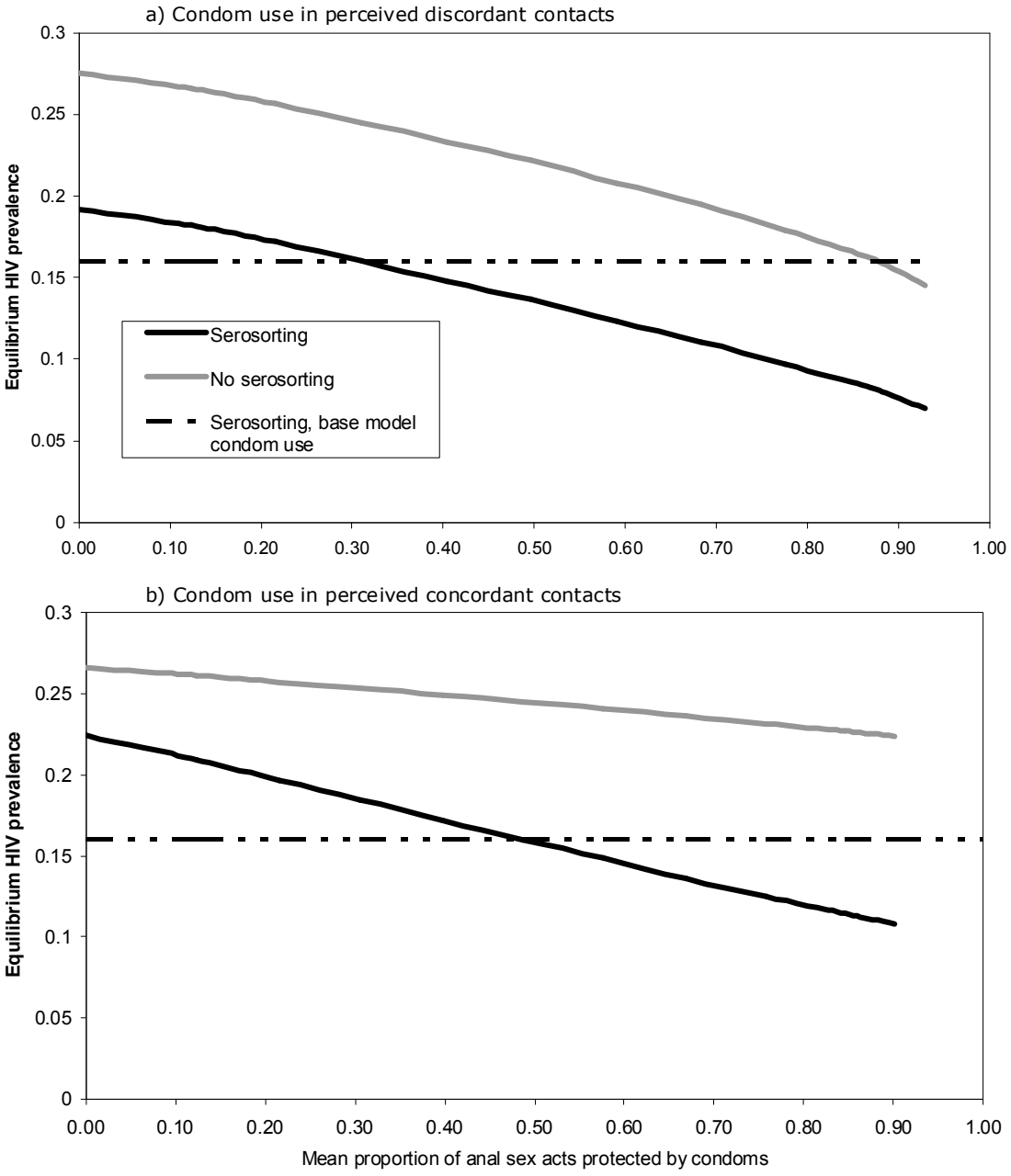
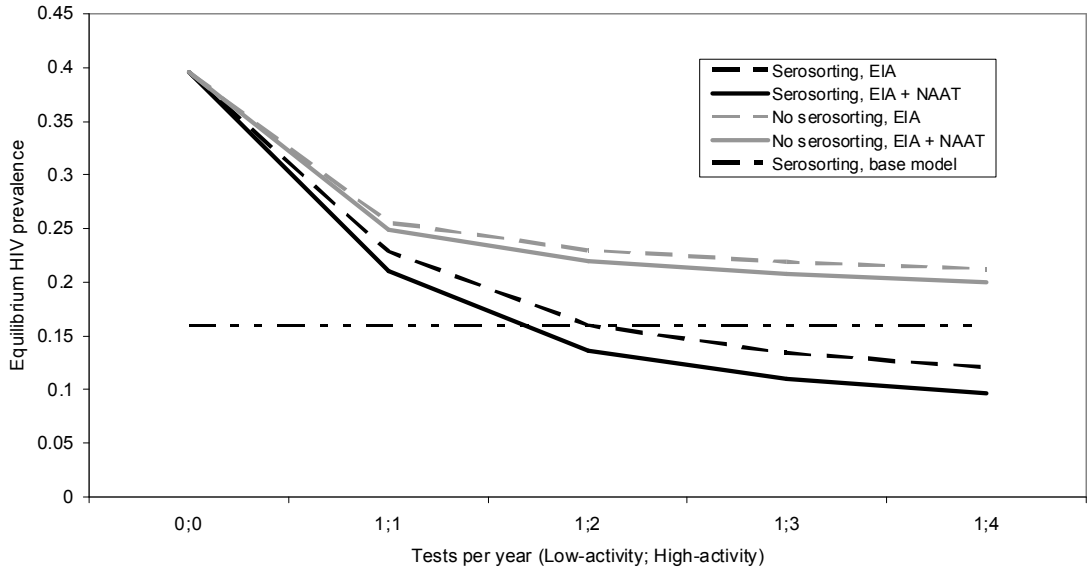


Figure 4: Equilibrium HIV prevalence by testing frequency and type.



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