Influence of Structural Assumptions in Compartmental Models of Heterosexual HIV Transmission

by

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Abstract

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Table 1: Acronyms

Acronym	Definition
HIV	human immunodeficiency virus
AIDS	acquired immunodeficiency syndrome
CD4	cluster of differentiation 4 (lymphocyte type)
ART	antiretroviral therapy
PrEP	pre-exposure prophylaxis
VMMC	voluntary medical male circumcision
FSW	female sex worker
MSM	men who have sex with men
AGYW	adolescent girls and young women
STI	sexually transmitted infection
GUD	genital ulcer disease
p12m	past 12 months
CI	credible interval

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Chapter 1

Model Structure, Parameterization, & Calibration

Drawing on the insights from Chapter **??**, this chapter details the development of a compartmental model of heterosexual HIV transmission in eSwatini. The model aims to capture key determinants of heterosexual transmission dynamics, including sex work, numbers of sexual partners, levels of condom use, anal sex, and ART scale-up.

The model was implemented in Python v3.8.10 with Numpy v1.22.2, and solved numerically using 4th order Runge-Kutta [1] using a timestep of 0.05 years. Post-hoc analysis was conducted in R v3.6.3. All code and selected results are available on GitHub.[1]

1.1 Model Structure

The model aims to capture heterosexual HIV transmission among the Swazi population aged 15–49. The model stratifies the modelled population along four dimensions: two sexes (s), four activity groups (i), six HIV states (h), and five cascade states (c), summarized in Table 1.1 and Figure 1.1. In total, $2 \times 4 \times (1 + 5 \times 5) = 208$ states are modelled. Two additional "dimensions" help organize: four partnership types (p), and two types of sex acts (a).

Sexual activity groups were defined to reflect persistent differences in HIV incidence and prevalence [2, 3, 4, 5] — reflecting acquisition and/or onward transmission risk — as well as common stratifications in the available data, and epidemiologically relevant sub-populations. The lowest sexual activity group ($i = 1$) comprises individuals who had 0-1 sexual partners in the past 12 months (p12m), but did not engage in sex work. The medium activity group ($i = 2$) similarly comprises individuals who had 2+ sexual partners in p12m but did not engage in formal sex work. The highest two activity groups among women ($i = 3, 4$) comprise lower and higher risk FSW (see § 1.2.1 for more details), and the highest two activity groups among men ($i = 3, 4$) likewise comprise lower and higher risk clients of FSW.

[1] github.com/mishra-lab/hiv-fsw-art

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Table 1.1: Overview of model dimensions and stratifications

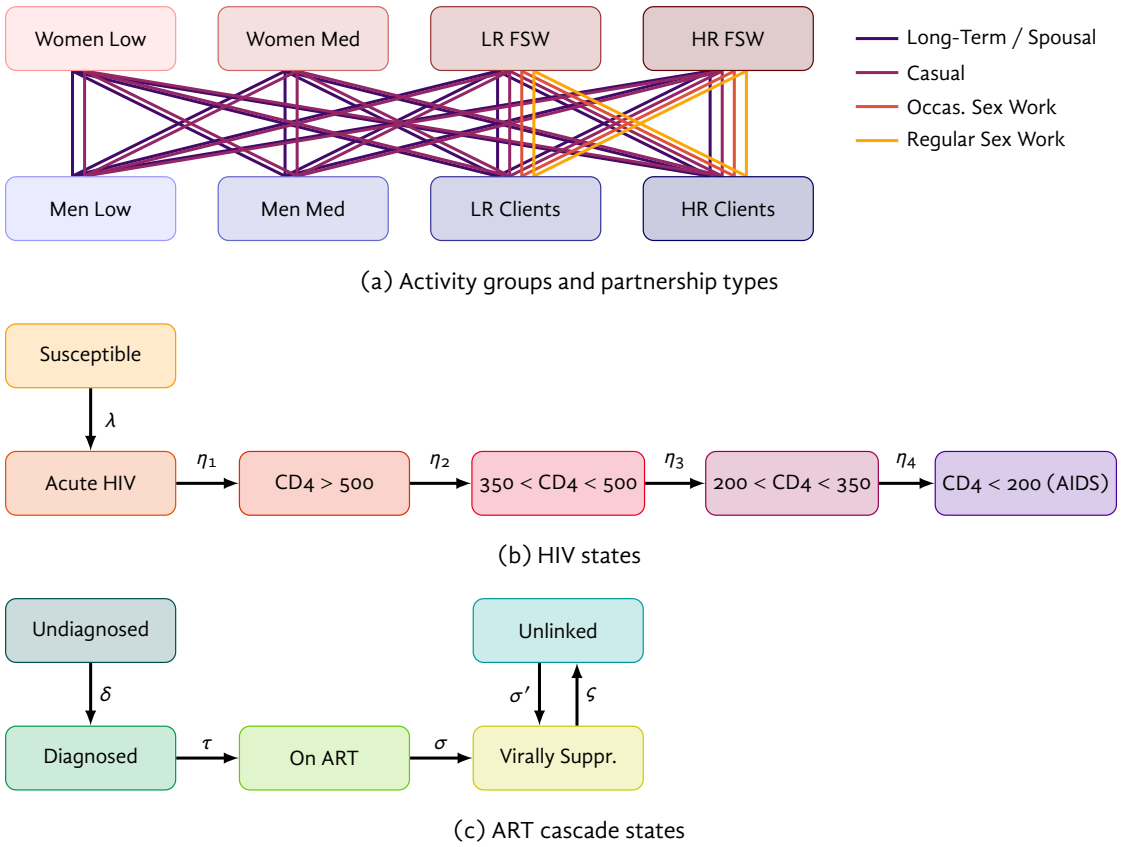
Dimension	Index		Strata
Sex	(s)	1	Heterosexual Women
		2	Heterosexual Men
Activity group	(i)	1	Lowest Activity
		2	Medium Activity
		3	Lower Risk Sex Work
		4	Higher Risk Sex Work
HIV status	(h)	1	Susceptible
		2	Acute HIV
		3	CD4 > 500
		4	350 < CD4 < 500
		5	200 < CD4 < 350
		6	CD4 < 200 (AIDS)
ART cascade	(c)	1	Undiagnosed
		2	Diagnosed
		3	On ART
		4	Virally Suppressed
		5	Unlinked from Care
Partnership types	(p)	1	Main / Spousal
		2	Casual
		3	Occasional Sex Work
		4	Regular Sex Work
Sex act types	(a)	1	Vaginal
		2	Anal

Four types of sexual partnerships are modelled, with different levels of condom use and expected durations: long-term/spousal partnerships (p = 1, lowest condom use, 12–25 years [**??**]); short-term partnerships (p = 2, medium condom use, 1–6 months [**??**]); one-off new/occasional sex work partnerships (p = 3, highest condom use, 1 sex act); and regular sex work partnerships (p = 4, medium condom use, 2–24 months [assumed]). Figure 1.1a illustrates the modelled activity groups and possible partnership types between them.

HIV infection is stratified into acute-HIV and stages defined by CD4 count (Figure 1.1b) to reflect changes in mortality [6], historical ART eligibility [7, 8], and, with CD4 as a proxy for viral load, infectiousness [9]. The modelled ART cascade (Figure 1.1c) includes the major steps associated with the "90-90-90" targets, plus a generic "unlinked" state reflecting any combination of treatment failure, discontinuation, or loss to follow-up.

1.1.1 Initialization & Solving

The first cases of HIV and AIDS in eSwatini were diagnosed in 1986 and 1987, respectively [10], although HIV may have been present several years earlier [11]. As such, I initialize the model in 1980 with no HIV, and simulate introduction of HIV at a random year between 1980 and 1985 (uniform prior). HIV



(a) Activity groups and partnership types

(b) HIV states

(c) ART cascade states

Figure 1.1: Model structure and transitions

Low: lowest activity; Med: medium activity; LR/HR: lower/higher risk; FSW: female sex workers; Clients: of FSW; CD4: CD4+ T-cell count per mm^3; Not shown: turnover amongst activity groups in (a).

introduction is modelled as exogenous infection of 0.01% (~ 24) individuals in the model,[2] distributed across activity groups in proportion to their size, comprising: 5% acute HIV ($h = 2$), 65% with CD4 > 500 ($h = 3$) and 30% with 350 < CD4 < 500 ($h = 4$), all undiagnosed ($c = 1$).[3] The population size of Swazi individuals aged 15–49 in 1980 was defined as 243,000 from [12].

1.2 Parameterization

As described in § **??**, model parameterization involves specification of model parameter values, such as proportions, probabilities, and rates, including stratified values to reflect heterogeneity, and sampling distributions to reflect uncertainty.

[2] No further import/export of HIV to/from eSwatini is considered thereafter in the model. HIV transmission between eSwatini and neighbouring countries, including South Africa and Mozambique, has likely continued throughout the epidemic due to labour migration and other factors [11]. However, I assume that such transmissions have low overall influence on epidemic dynamics.

[3] In compartmental models, the numbers of individuals in each state (compartment) need not be whole numbers.

1.2.1 Risk Heterogeneity Among FSW

HIV transmission models which include FSW rarely sub-stratify this population, such as to reflect differential HIV risk or distinct typologies of sex work [13, 14]; yet such heterogeneities likely influence transmission dynamics. Among the studies identified in Chapter **??**, only two sub-stratified FSW by risk-related factors: Low et al. [15] distinguished between occasional and full-time FSW, while Shannon et al. [16] sub-stratified FSW by work environment, violence exposure, and context-specific structural factors. Seven other studies, reflecting two unique models [17, 18], employed age stratification of all activity groups, including FSW; these models had several risk-related parameters which varied by age.

The model structure here (Figure 1.1a) was designed to capture *within*-FSW risk heterogeneity. The objective of the following analysis was therefore to parameterize lower versus higher risk FSW. I sought to define these groups based on biobehavioural and/or contextual factors which are demonstrably associated with HIV risk, and which can be mechanistically incorporated into a transmission model — *i.e.,* through the force of infection equation. Later, the parameterization of these groups was validated through model fitting to relative differences in HIV prevalence § **??**.

Many cross-sectional studies of HIV among FSW quantify the association of risk factors with HIV serostatus [19, 20, 14, 21]. However, serostatus reflects cumulative risk exposure, whereas sexual risk behaviour is dynamic [22, 23], as is use of prevention resources [24]. For example, while HIV prevalence often increases with age, HIV incidence among women can peak shortly after sexual debut [25]. Thus, risk factors associated with HIV serostatus are not necessarily mechanistically related to HIV acquisition. Indeed, FSW may reduce risk behaviours in response to seroconversion [26]. Cohort studies that measure incidence can help identify risk factors for HIV acquisition [27, 28], but large sample sizes are often required to accurately estimate overall incidence rate, let alone risk factors [29].

1.2.1.1 FSW Survey Data

Three surveys, in 2011 [30] (N = 325), 2014 [31] (N = 781), and 2021 [32] (N = 676) provide HIV and biobehavioural data on FSW in eSwatini. The 2011 survey employed respondent driven sampling (RDS, details in [33]), as did the 2021 survey. The 2014 survey employed venue-based snowball sampling, based on the Priorities for Local AIDS Control Efforts (PLACE) methodology, which aims to identify areas of higher incidence [34]. I analyzed the individual-level data from 2011 and 2014 (data from 2021 not yet available) to explore the potential association of biobehavioural factors with HIV risk, so that such factors could then be used to distinguish between lower risk versus higher risk FSW.

1.2.1.2 HIV Status

Only the 2011 and 2021 studies included serologic testing for HIV. Among those tested in 2011 (N = 317, 98%), 70% were HIV+, yielding RDS-adjusted prevalence estimate of 61% (CI: 51–71%) [30]. Among serologically HIV–, 11% self-reported HIV+ status (false positive), and among serologically HIV+, 26% self-reported HIV– status (false negative or undiagnosed). Overall, self-reported HIV status underestimated HIV prevalence in 2011 by a factor of approximately 0.78 (55 vs 70%). Unadjusted HIV prevalence in 2021 was 58.8%, with 88% (363/411) reporting previous awareness of HIV+ status.

Figure 1.2: Illustration of time-to-event analysis framework for cross-sectional FSW survey data

×: HIV infection; SW: time of sex work debut; Dx: time of HIV diagnosis.

In 2014, self-reported HIV prevalence was 38% among respondents who reported (85%). This 38% is surprisingly low considering that the PLACE methodology explicitly aimed to sample venues with higher HIV incidence [34], and 2014 versus 2011 respondents were older (median 27 vs 25 years), had been selling sex longer (median 5 vs 4 years), and tested more frequently (87 vs 75% tested at least once in the past year, 82 vs 63% among self-reported HIV−). Perhaps the differences are attributable to the sampling methodology. Among respondents who self-reported HIV+ status, the 2014 survey also asked for age of HIV diagnosis (6% missing). Age of HIV diagnosis supports crude time-to-event analysis (next section), which can account for confounding by age and censoring, as compared to logistic regression on HIV status, keeping in mind the limitations of self-reported HIV status.

1.2.1.3 Risk Factors

Next, I explored the potential association of risk factors with HIV via the following three models:[4]

1. Logistic regression on serologic HIV status (2011 data)
2. Logistic regression on self-reported HIV status (2014 data)
3. Cox proportional hazards for interval-censored time to HIV infection, with interval from self-reported sex work debut to either self-reported time of HIV diagnosis or survey date (2014 data); Figure 1.2 illustrates the four potential censoring cases in this framework.

An important limitation to all models is that risk factors reported by FSW at the time of survey are assumed to be fixed characteristics of the respondents, rather than dynamic characteristics that vary over time. Additionally, respondents with any missing variables for each individual model were excluded from that model.

Risk factors were selected based on prior knowledge of plausible mechanistic influence on HIV incidence and/or prevalence. The risk factors explored are summarized in Table 1.2, including univariate and multivariate association under each model. Variable selection for multivariate models was performed using backward selection as described by Lawless and Singhal [35], using a $p \leq 0.1$ (per variable) threshold for stepwise variable retention. Estimated conditional effects of variables retained in the multivariate logistic regression models are illustrated in Figure 1.3.

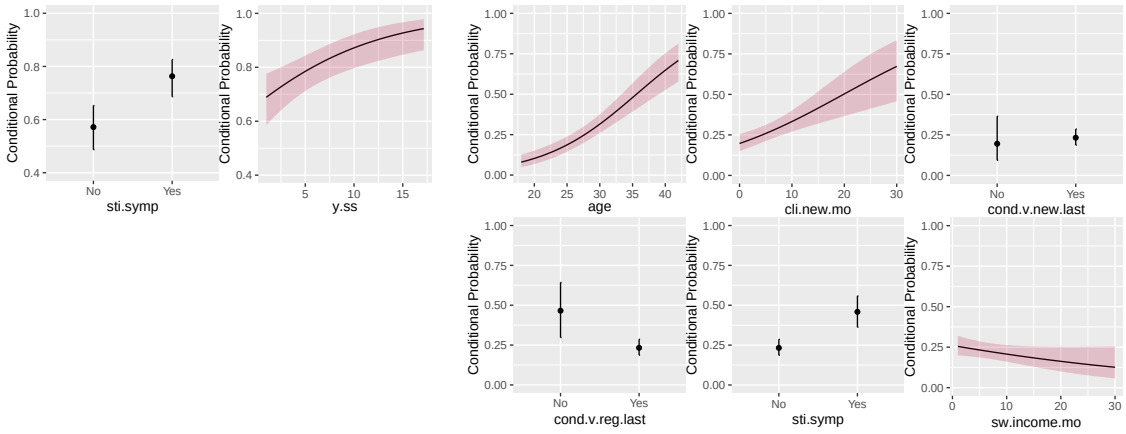
Following variable selection, each multivariate model was used to predict the total HIV+ status odds ratio (logistic) or HIV incidence hazard ratio (Cox) for each respondent in the respective survey — *i.e.,* $e^{X_i \beta}$ for respondent i — representing an overall "risk score" under each model. Respondents were then stratified into the top 20% and bottom 80% by these risk scores. The values of each variable were compared between these two strata using a test for the ratio of the means [36] to support model parameterization; these ratios are summarized in Table 1.3, and the distributions of variable values are illustrated in Figures A.1 and A.2.

[4] Logistic regression models were implemented using `lrm` from: cran.r-project.org/package=rms.
Cox proportional hazards models were implemented using `coxaalen` from: cran.r-project.org/package=coxinterval.

Table 1.2: Risk factors explored for association with HIV+ status among FSW in eSwatini

	2011 LR				2014 LR				2014 CPH			
	Univar		Multivar		Univar		Multivar		Univar		Multivar	
Factor	OR	p	OR	p	OR	p	OR	p	HR	p	HR	p
Age [a]	1.11	<0.001*	—	—	1.14	<0.001*	1.15	<0.001*	1.09	<0.001*	1.09	<0.001*
Years selling sex [a]	1.13	<0.001*	1.13	<0.001*	1.12	<0.001*	—	—	1.08	<0.001*	—	—
Monthly sex work income [b]	0.98	0.155	—	—	0.98	0.097	0.97	0.084	0.98	0.019*	0.97	0.001*
Non-paying partners [c]	0.88	0.307	—	—	1.07	0.233	—	—	1.05	0.312	—	—
Monthly new clients [c]	1.01	0.412	—	—	1.05	<0.001*	1.07	<0.001*	1.04	<0.001*	1.04	<0.001*
Monthly regular clients [c]	1.01	0.351	—	—	1.03	0.002	—	—	1.02	<0.001*	1.02	0.034*
Non-paying condom use [d]	0.90	0.703	—	—	0.90	0.673	—	—	0.92	0.677	—	—
New client condom use [d]	0.60	0.100	—	—	0.48	0.006*	1.25	0.599	0.56	0.004*	—	—
Regular client condom use [d]	0.58	0.110	—	—	0.39	<0.001*	0.35	0.004*	0.49	<0.001*	0.50	<0.001*
Any anal sex past month	0.97	0.896	—	—	1.89	0.015*	—	—	1.57	0.015*	1.27	0.260
Any STI symptoms past year	2.29	<0.001*	2.41	<0.001*	2.75	<0.001*	2.80	<0.001*	2.17	<0.001*	2.05	<0.001*

[a] OR per year; [b] OR per Swazi lilangeni per month; [c] OR per partner; [d] 2011: always vs not always, 2014: at last sex. — indicates variable was not selected in the multivariate model. LR: logistic regression on HIV+/− status; CPH: Cox proportional hazards on time to self-reported HIV seroconversion. OR: odds ratio; HR: hazard ratio; p: p-value. 2011 data based on serologic HIV test; 2014 data based on self-reported HIV status, age of sex work debut, and age of HIV diagnosis.



(a) 2011 (serologic HIV status) (b) 2014 (self-reported HIV status)

Figure 1.3: Predicted conditional effects (probability) of significant variables in multivariate logistic regression models from 2011 and 2014 surveys

sti.symp: any STI symptoms past year; y.ss: years selling sex; cli.new.mo: monthly new clients; sw.income.mo: monthly sex work income. Conditional probabilities shown for fixed covariates at arbitrary values.

Table 1.3: Ratios of HIV risk factor variables among higher vs lower risk FSW in eSwatini

	2011 LR		2014 LR		2014 CPH	
Factor	High / Low	Ratio (95% CI)	High / Low	Ratio (95% CI)	High / Low	Ratio (95% CI)
Age	31.8 / 24.7	1.29 (1.22, 1.36)*	32.6 / 26.2	1.24 (1.20, 1.28)*	33.5 / 26.6	1.26 (1.21, 1.31)*
Years selling sex	11.3 / 4.03	2.81 (2.41, 3.25)*	10.0 / 5.47	1.83 (1.64, 2.03)*	10.2 / 5.83	1.75 (1.54, 1.98)*
Monthly sex work income [a]	15.1 / 15.2	1.00 (0.86, 1.15)	6.77 / 7.06	0.96 (0.82, 1.11)	6.32 / 7.28	0.87 (0.73, 1.02)
Non-paying partners	1.42 / 1.43	0.99 (0.81, 1.19)	1.56 / 1.11	1.40 (1.11, 1.72)*	1.53 / 1.19	1.29 (0.98, 1.62)
Monthly new clients	5.50 / 6.98	0.79 (0.49, 1.15)	8.39 / 4.15	2.02 (1.63, 2.44)*	8.36 / 4.41	1.90 (1.43, 2.39)*
Monthly regular clients	9.35 / 9.05	1.03 (0.69, 1.42)	11.1 / 8.25	1.35 (1.13, 1.57)*	12.4 / 8.61	1.44 (1.18, 1.71)*
Non-paying condom use [bc]	0.26 / 0.35	0.73 (0.40, 1.11)	0.77 / 0.81	0.95 (0.84, 1.06)	0.76 / 0.81	0.95 (0.81, 1.08)
New client condom use [bc]	0.68 / 0.76	0.89 (0.73, 1.06)	0.79 / 0.91	0.86 (0.79, 0.94)*	0.74 / 0.94	0.79 (0.69, 0.88)*
Regular client condom use [bc]	0.38 / 0.46	0.83 (0.45, 1.28)	0.67 / 0.91	0.74 (0.65, 0.82)*	0.60 / 0.92	0.65 (0.55, 0.75)*
Any anal sex past month	0.59 / 0.41	1.41 (1.06, 1.84)*	0.17 / 0.07	2.43 (1.47, 3.85)*	0.23 / 0.07	3.24 (1.95, 5.34)*
Any STI symptoms past year [c]	0.79 / 0.43	1.86 (1.54, 2.25)*	0.59 / 0.15	3.94 (3.15, 5.03)*	0.61 / 0.17	3.67 (2.87, 4.79)*
HIV prevalence [d]	0.94 / 0.64	1.46 (1.30, 1.63)*	0.66 / 0.29	2.29 (1.92, 2.75)*	0.71 / 0.31	2.32 (1.94, 2.80)*

High / Low: mean variable value among higher / lower risk groups, as defined by the top 20% / bottom 80% in multivariate model-predicted risk score: odds ratio from logistic regression (LR); hazards ratio from Cox proportional hazards (CPH). [a] Swazi lilangeni per month; [b] 2011: always vs not always, 2014: did use condom at last sex; [c] proportion of respondents; [d] 2011: serologic HIV status; 2014: self-reported HIV status; * statistically significant, $p < 0.05$.

1.2.1.4 Discussion

TODO

1.2.2 Probability of HIV Transmission

I parameterized the overall probability of transmission per sex act β as the product of a base rate β_0, and independent relative effects corresponding to multiple factors. Such factors (indexed k) included: sex act type a, condom use, prevalence of circumcision among susceptible men, partner HIV infection stage h' and viral suppression via ART c', as well as prevalence of STI co-infection/symptoms among both partners. Thus, β was defined as:

$$\beta_{asis'i'h'c'} = \beta_0 R_{\beta,k_1} \ldots R_{\beta,k_N} \tag{1.1}$$

The impact of each factor (except ART) on the probability of HIV transmission is described in the following subsections, while the prevalence of each factor is given in § 1.2.3. The impact of ART on transmission is described in § 1.2.5.1.

1.2.2.1 HIV Infection Stage

Boily et al. [9] synthesized per-act transmission probability in the absence of ART from 43 studies in 25 populations. Among 7 studies reporting stage of HIV infection (early, asymptomatic, late), infection stage explained 95% of variance in per-act probability of transmission in [9]. Such differences in transmission are most likely due to differences in viral load, which is associated with HIV stage [37, 38]. The probability of transmission during the middle asymptomatic period, was reported as mean (95% CI)

0.072 (0.053, 0.097)% per act, which was used to define a skewnormal prior distribution for β_0. This probability was assumed to apply to vaginal intercourse, based on the studies considered.

For early infection ($h = 2$), Boily et al. [9] estimated the relative infectiousness of the first 5 months of infection as 9.2 (4.5, 18.8) times higher than the asymptomatic period. However, both the duration and infectiousness of the acute phase have been long debated [39, 40, 41]. In a recent reanalysis of the Rakai cohort data, Bellan et al. [42] estimate a much smaller contribution of the acute phase to overall infection, summarized as 8.4 (0, 63) "excess hazard-months" (EHM). This excess risk represents the joint uncertainty and collinearity in the estimated duration of 1.7 (.55, 6.8) months and relative infectiousness of 5.3 (.79, 57). Thus, I sampled the duration $\delta_{h=2}$ from a gamma prior with mean (95% CI) 1.7 (.55, 6) months, and relative infectiousness $R_{\beta,h'=2}$ from a gamma prior with 5.3 (1, 15) times the asymptomatic period (confidence intervals were adjusted to fit the gamma distributions, and to ensure $1 < \text{EHM} < 63$).

For late-stage disease, defined as 6-15 months before death in [9], Boily et al. estimated the relative rate of transmission as 7.3 (4.5, 11.9). However, I defined later HIV stages by CD4 count, including $200 <$ CD4 < 350 ($h = 5$) and CD4 < 200 ($h = 6$, AIDS), which reflects closer to 50 and 18 months before death in the absence of ART, respectively. Therefore, I combined estimates from several sources [43, 9, 38] to define two gamma prior distributions with mean (95 CI%) 1.6 (1.3, 1.9) and 8.3 (4.5, 13), for the relative rate of HIV transmission in these two stages ($h = 5, 6$), respectively. For CD4 > 350 ($h = 3, 4$), I assumed no change from the baseline probability β_0.

1.2.2.2 Sex Act Types

The model considers vaginal and anal intercourse, further stratified by sex (male-to-female/insertive vs female-to-male/receptive). For vaginal intercourse, evidence for differential risk by sex is mixed, with some studies reporting no difference [43, 44], and others reporting up to 2-times higher male-to-female ($s' = 2, s = 1$) transmission vs female-to-male ($s' = 1, s = 2$) [9]. To reflect this uncertainty, I defined a gamma prior distribution for the relative rate of male-to-female vs female-to-male transmission with 95% CI: (1, 2); in applying this relative rate, both male-to-female and female-to-male transmission probabilities were adjusted such that the overall mean was preserved.

Baggaley et al. [45] synthesized the per-act transmission probability for anal intercourse, with most data from MSM studies. Analyses in [45] were not stratified by HIV stage, so I assumed the same relative rates derived in § 1.2.1 applied equally to vaginal and anal intercourse. Overall female-to-male (insertive) per-act transmission probabilities were similar for anal intercourse [46] (without ART): 0.14 (0.04, 0.29)% vs vaginal intercourse [9] (without commercial sex exposure): 0.164 (0.056, 0.481)%; thus I assumed that female-to-male (insertive) transmission probabilities for anal vs vaginal intercourse were equal. By contrast, male-to-female (receptive) per-act transmission probabilities were approximately 10 higher in anal intercourse [45] (without ART): 1.67 (0.44, 3.67)% vs vaginal intercourse [9] (without commercial sex exposure): 0.143 (0.088, 0.233)%; thus I assumed a fixed 10-fold increase in male-to-female transmission probability for anal vs vaginal intercourse. See § **??** for sex act frequency within each partnership type.

1.2.2.3 Circumcision

Relative risk in per-act HIV female-to-male transmission for circumcised vs uncircumcised men via vaginal intercourse has consistently been estimated as approximately 0.50, with 95% CI spanning (0.29, 0.96) [9, 44, 47]. Since circumcision status is unrelated to the research question, I fixed this effect at 50% relative risk. For anal intercourse, Wiysonge et al. [48] estimated that circumcision resulted in .27 (.17, .44) the odds of HIV acquisition for the insertive partner. It can be shown that relative reduction in incidence represents a lower bound on relative reduction in per-act transmission probability.[5] Thus, for anal intercourse, I similarly fixed the per-act effect at 27%. Finally, there is inconclusive evidence to suggest that circumcision status affects male-to-female/receptive transmission [49, 48], so I assumed no effect. See § 1.2.3.1 for prevalence of circumcision in eSwatini over time.

1.2.2.4 Condoms

The most recent meta-analysis of condom effectiveness in heterosexual couples by Giannou et al. [50] estimated a relative risk of approximately 0.26 (0.13, 0.43). No significant differences were noted between female-to-male vs male-to-female transmission. A recent study among men who have sex with men found a similar effect for anal sex [51]. Thus, condom effectiveness was fixed at 74%. See § 1.2.3.2 for levels of condom use in eSwatini over time.

1.2.2.5 Genital Ulcer Disease

Genital ulcer disease (GUD) is another another established risk factor for HIV transmission [52, 53]. Some, but not all GUD is associated with sexually transmitted infections (STIs), and some, but not all STIs can cause GUD [53]. GUD is thought to increase both HIV susceptibility and infectiousness through a variety of mechanisms [53, 54, 55], but HIV may also facilitate transmission of various STIs through immunosuppression [56]. The meta-analysis by Boily et al. [9] found that presence of STI alone was not associated with increased HIV transmission: RR 1.11 (0.30, 4.14), but GUD was: RR 5.29 (1.43, 19.6), with most studies examining GUD among the HIV-susceptible partner. One study [57] estimated RR 2.58 (1.03, 5.69) of transmission for GUD among the HIV-positive partner. Most studies defined GUD status as any experience of symptoms during the study period (*e.g.,* past 12 months, p12m), since precise delineation of GUD episodes is challenging. Thus, the true effect of GUD on HIV transmission during GUD episodes may be larger. However, if estimates of GUD prevalence and GUD effect (on HIV transmission) use consistent definitions (*e.g.,* any GUD in p12m), then the time-averaged effect can be applied without need to estimate GUD episode duration. As such, I applied factors for increased susceptibility and infectiousness due to GUD in accordance with group-specific p12m GUD prevalence (see § 1.2.3.3), with 95% CI (1.4, 19.5) and (1.03, 5.69), respectively.

[5] See § 1.2.1 for more discussion.

1.2.3 Prevalence of Transmission Modifiers

1.2.3.1 Circumcision

Traditional (non-medical) circumcision in eSwatini is rare, reported as approximately 0.7% of men aged 15-49 in 2016 [5]. Voluntary medical male circumcision (VMMC) increased circumcision coverage to 8.2% by 2007, following demand for mainly hygienic reasons [2]. In 2007, the government further increased scale-up of VMMC services as part of HIV prevention efforts [2], leading to 17.1% coverage in 2011 [58], 30.0% in 2017 [5], and 37% in 2021 [59]. Since VMMC continues to be a key element of eSwatini's HIV response [59], I assumed that coverage could reach and plateau at 50–90% (95% CI) by 2050. There is minimal evidence of differential condom use by circumcision status [58], so I assumed no differences. Similarly, while circumcision differed by union status in [5] (*e.g.,* 22.1% circumcised among men in a union vs 31.7% among men not in a union), differences did not persist after re-stratifying these men into groups with 0-1 vs 2+ partners per year, as described in § **??**. In Zambia, circumcision status was not associated with paying for sex [60].

1.2.3.2 Condom Use

Condom use is typically reported as either categorical for a recent period, usually 30 days, *e.g.,* never, rarely, sometimes, often, always; or binary for the most recent sex act. Both report types may be subject to reporting bias [61], but the "last sex" more directly translates into a proportion of sex acts. Table 1.4 summarizes the available condom use data for eSwatini.

Main/Spousal & Casual. No direct estimates of condom use in main/spousal partnerships are available; condom use at last sex (with a non-paying partner) was either reported overall or for casual partners only.[6] However, the proportions of individuals with various relationship statuses (*e.g.,* polygynous union, non-polygynous union, not in a union, see § **??**) can be used to back-calculate condom use in main/spousal partnerships for both 2006 [2] and 2016 [5]. To do so, I assumed whether "last sex" among individuals in unions with 2+ partners was with their main/spousal partner or with a casual partner; or more generally, what proportion of most recent sex acts was with a casual partner. I repeated the back-calculation assuming 5% and 95%, yielding the confidence intervals shown in Table 1.4. Estimates of condom use in non-paying partners were lower among FSW vs the wider population in 2011 (20.8% vs ~32% "always"), but higher in 2014-16 (80.1% vs ~55.7% "last sex"). Therefore, I assumed no differences in condom use among FSW vs the wider population for main/spousal or casual partnerships.

Sex Work. All data on sex work partnerships in eSwatini is from FSW (*i.e.,* not their clients). A 2001 study in Ghana [64] suggested that FSW were more likely than their clients to report having used a condom. As such, I adjusted the lower bound of 95% CI for condom use in sex work partnerships ($p = 3, 4$) as either 75% of the reported lower bound, or the lowest reported region-specific estimate. Estimates for 2002 [63] were obtained from two major cities only (Manzini and Mbambane); since early condom availability was mainly urban, treated these estimates as 95% CI upper-bounds, and defined the lower bound as 20% of the reported values.

[6] "Higher risk" partners were defined in [2] as: "*Sexual intercourse with a partner who was neither a spouse nor lived with the respondent*", effectively matching the model definition of "casual" partnerships.

Table 1.4: Estimates of condom use in eSwatini

Partnership Type	Year	Population	Type	%	(95% CI)	Ref	Notes
Main	2006	Women	last sex	23.5	(23.2, 23.9)	[2]	a
		Men	last sex	23.1	(19.4, 26.9)	[2]	a
	2016	Women	last sex	52.7	(52.5, 52.9)	[5]	a
		Men	last sex	33.7	(30.8, 36.7)	[5]	a
Main or Casual	1988	Women	currently	0.6	(0.4, 1.3)	[62]	b
		Men	currently	7.3	(5.9, 12.1)	[62]	b
	2002	FSW	last sex	60	—	[63]	cd
			always	45.8	—	[63]	cd
	2006	Women	last sex	36.5	—	[2]	
		Men	last sex	47.2	—	[2]	
	2011	Women	always	30	—	[2]	
		Men	always	34	—	[2]	
		FSW	last sex	51.1	(41.8, 60.4)	[30]	de
			always	20.8	(14.7, 26.9)	[30]	de
	2014	FSW	last sex	80.6	(64.7, 89.6)	[31]	g
	2016	Women	last sex	58.3	—	[5]	
		Men	last sex	53.1	—	[5]	
Casual	2006	Women	last sex	53.5	—	[2]	
		Men	last sex	66.0	—	[2]	
	2016	Women	last sex	64.9	—	[5]	
		Men	last sex	73.7	—	[5]	
Sex Work Unspecified	2002	FSW	last sex	90	—	[63]	d
			always	74.4	—	[63]	d
	2020	FSW	always	50	—	[32]	
New Sex Work	2011	FSW	last sex	84.8	(57.9, 92.4)	[30]	ef
			always	56.7	(47.8, 65.6)	[30]	d
	2014	FSW	last sex	88.5	(54.9, 95.9)	[31]	g
Regular Sex Work	2011	FSW	last sex	82.9	(56.8, 90.0)	[30]	ef
			always	38.6	(29.5, 47.7)	[30]	e
	2014	FSW	last sex	85.6	(47.9, 95.0)	[31]	g

[a] Back-calculated as described in § 1.2.3.2; [b] 95% CI from urban & rural data; [c] Described as "non-paying partners" in the survey; [d] Two major cities only (Manzini & Mbambane); [e] RDS-adjusted; [f] 95% CI lower bound reduced by 25% due to possible reporting bias; [g] 95% CI bounds from regions with lowest and highest reported condom use.

Anal Sex. Owen et al. [65] estimate that among FSW globally, condom use in anal sex is approximately 79 (66, 94)% that of condom use in vaginal sex.[7] In eSwatini [30, 31], relative condom use in anal sex vs vaginal sex ranged from 44% among new clients in 2011 to 88% among regular clients in 2014. So, I sampled relative condom use in anal vs vaginal sex from a beta prior distribution with 95% CI: (50, 95)%.

Sampling & Trends. While levels of condom use reported by men and women do not always agree, the levels should agree in simulated partnerships. To reflect uncertainty due to the discrepancy, I sampled condom use for each year and partnership type from beta prior distributions having 95% CI that spans the range of estimates from men and women (where applicable), including the widest points of all confidence intervals. I assume that condom use was effectively zero in 1980 [62]. I also assume andd enforce two conditions that: condom use must be monotonic increasing over time; and condom use must be highest in new sex work partnerships, and lowest in main partnerships, for all sampled parameter values. For each available year, I simultaneously sample condom use for all partnership types, and samples failing the condition are discarded. As illustrated in § A.2.2, this sampling strategy minimizes differences between the prior and posterior (sampled) distributions. For each partnership type, I then smoothly interpolate between sampled levels of condom use over the available years using monotone piecewise cubic interpolation [66].

1.2.3.3 Genital Ulcer Disease

Self-reported prevalence of GUD in p12m among sexually active women and men aged 15–49 was approximately 7% in 2006 [2, Table 13.14]; this prevalence was not stratified by numbers of partners, so I assumed it was equal across sexually active individuals in lowest and medium risk groups. However, approximately 40% of the lowest risk group reported being not sexually active during p12m [2, 5] (see § **??**); thus I reduced GUD prevalence by 40% among this group.

The 2011 and 2014 FSW surveys did not ask respondents about GUD specifically, but about any STI symptoms in p12m.[8] In the wider population [2], approximately 60% of women self-reporting any STI symptoms specifically reported GUD in p12m; thus, self-reported STI symptoms among FSW may overestimate p12m GUD prevalence. Approximately 50% and 25% of FSW reported STI symptoms in 2011 and 2014, respectively. Reflecting uncertainty related to self-reported estimates, STI vs GUD, and sampling bias, I sampled p12m GUD prevalence among lower risk FSW from a beta distribution with 95% CI (10, 40)%. Per analysis in § 1.2.1, I assumed that STI (and thus GUD) prevalence was approximately 3 (1.5, 5) times higher among higher risk FSW (gamma prior), with an upper bound of 100%. FSW data also suggest declining STI prevalence between 2011 and 2014. However, STI prevalence among Swazi youth in 2017–18 remained high [67]. Thus, to reflect uncertainty in STI/GUD prevalence trends, I sampled a relative reduction in GUD prevalence for all populations by 2050 from a uniform distribution spanning [0.2, 1].

Finally, no eSwatini-specific data are available for clients of FSW, but studies in Zimbabwe [68], Senegal [69] and Zambia [60] have found 2.5–3.7 (95% CI span 1.4–5.0) the odds of STI symptoms during the past 6–12 months among clients versus non-clients. Yet, I assumed that even higher risk clients could

[7] I integrated the reported confidence intervals using the delta method after assuming binomial-distributed proportions.
[8] The survey question about STI symptoms was: "*In the last 12 months, have you had symptoms of a sexually transmitted infection including discharge from your vagina or sores on or around your vagina or anus*".

not have greater GUD prevalence than lower risk FSW. Thus, I sampled higher risk client GUD prevalence uniformly between 7% and that of lower risk FSW, and sampled lower risk client GUD prevalence uniformly between 7% and that of higher risk clients.

1.2.4 HIV Progression & Mortality

1.2.4.1 HIV Progression

The length of time spent in each HIV stage is related to rates of progression between stages η_h, rates of additional HIV-attributable mortality by stage $\mu_{\text{HIV},h}$, and treatment via antiretroviral therapy (ART). Lodi et al. [70] estimate median times from seroconversion to CD4 < 500, < 350, and < 200 cells/mm^3, while Mangal [6] directly estimate the rates of progression between CD4 states η_h in a simple compartmental model. Based on these data, I modelled mean durations ($1/\eta_h$) of:[9] 0.142 years in acute infection ($h = 2$, from § 1.2.2.1); 3.35 years in CD4 > 500 ($h = 3$); 3.74 years in 350 < CD4 < 500 ($h = 4$); and 5.26 years in 200 < CD4 < 350 ($h = 5$); plus the remaining time until death in CD4 < 200 ($h = 6$, AIDS). Since the duration in acute infection ($h = 2$) is randomly sampled, the remaining duration in CD4 > 500 ($h = 3$) is adjusted accordingly.

1.2.4.2 HIV Mortality

Mortality rates by CD4-count in the absence of ART were estimated in multiple African studies [71, 72, 6]; based on these data, I estimated yearly HIV-attributable mortality rates $\mu_{\text{HIV},h}$ as: 0 during acute phase ($h = 2$); 0.4% during CD4 > 500 ($h = 3$); 2% during 350 < CD4 < 500 ($h = 4$); 4% during 200 < CD4 < 350 ($h = 5$); and 20% during CD4 < 200 ($h = 6$, AIDS).

1.2.5 Antiretroviral Therapy

Viral suppression via antiretroviral therapy (ART) influences the probability of HIV transmission, as well as rates of HIV progression and HIV-related mortality. The model considers individuals on ART before ($c = 4$) and after ($c = 5$) achieving full viral load suppression (VLS), as defined by undetectable HIV RNA in blood samples. Among retained patients initiating ART, time to VLS is usually described as "within 6 months" [73]. More specifically, Mujugira et al. [74] estimate the median time to VLS as 3 months, yielding an estimated *mean* duration for $c = 4$ of 4.3 months (see § A.2.3).

1.2.5.1 Probability of HIV Transmission

All available evidence suggests that viral suppression by ART to undetectable levels prevents HIV transmission, *i.e.,* undetectable = untransmittable ("U=U") [75]. Thus, I assumed zero HIV transmission from individuals with VLS ($c = 5$). However, HIV transmission may still occur during the period between ART initiation to viral suppression ($c = 4$) [74]. Donnell et al. [38] estimate an adjusted incidence ratio of 0.08 (0.0, 0.57) for all individuals on ART. However, in [38] and [76], the 1 and 4 (respectively) genetically

[9] Assuming exponential distributions for durations in each CD4 state (see § A.2.3 for more details).

linked infections from individuals on ART all occurred within 90 days of ART initiation, suggesting that risk of transmission only persists before viral suppression. Adjusting the incidence denominator (person-time) to 90 days per individual who initiated ART in [38] results in approximately 3.13 times higher estimated incidence ratio: 0.25 for this specific period.[10] Thus, I sampled relative infectiousness on ART but before viral suppression ($c = 4$) from a beta distribution with mean (95% CI) of 0.25 (0.01, 0.67).

1.2.5.2 HIV Progression & Mortality

Effective ART stops CD4 cell decline and results in some CD4 recovery [77, 78]. Most CD4 recovery occurs within the first year of treatment [77]. Due to the limited number of modelled treatment states, I model this initial recovery to be associated with the 4.3-month pre-VLS ART state ($c = 4$). Lawn et al. [78] and Gabillard et al. [79] estimate an increase of between 25–39 cells/mm^3 per month during the first 3 months of treatment. Since HIV states $h = 4, 5, 6$ correspond to 150, 150, and 200-wide CD4 strata, I model rates of movement along $h = 6 \rightarrow 5 \rightarrow 4 \rightarrow 3$ during pre-VLS ART ($c = 4$) as 0.20, 0.20, 0.17 per month, respectively. After initial increases, CD4 recovery is modest and plateaus. Battegay et al. [77] report approximate increases of 22.4 cells/mm^3 per year between years 1 and 5 on ART. Thus, I model rates of movement along $h = 6 \rightarrow 5 \rightarrow 4 \rightarrow 3$ after VLS ($c = 5$) as 0.15 per year.

Since higher CD4 states are modelled to have lower mortality rates (see § 1.2.4.2), the modelled recovery of CD4 cells via ART described above implicitly affords a mortality benefit. However, HIV infection is associated with increased risk of death by non-AIDS causes — *i.e.,* unrelated to CD4 count — including cardiovascular disease and renal disease [80]. Lundgren et al. [81] estimated 61% reduction in non-AIDS life-threatening events due to ART. For the same CD4 strata, Gabillard et al. [79] also report approximately 2-times higher mortality rates within the first year of ART versus thereafter, suggesting that VLS is associated with 50% mortality reduction independent of CD4 increase. Thus, I modelled an additional 50% reduction in mortality among individuals with VLS ($c = 5$), and half this (25%) reduction before achieving VLS ($c = 4$).

[10] In [38], individuals who initiated ART contributed approximately 9.4 months per-person (273 persons / 349 person-years, Tables 2 and 3); thus the first 3 months of each individual represent 3/9.4 = 0.319 fewer person-months of follow-up.

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Appendix A

Supplement to Chapter 1

A.1 FSW Data

A.1.1 FSW Risk Factor Variable Distributions

Figures A.1 and A.2.

A.1.2 Duration in Sex Work

Figure A.3.

A.2 Supporting Mathematics

A.2.1 Continuous Approximation of the Binomial Distribution

Numerous model parameters and calibration targets represent population proportions. Such proportions can be estimated as $\rho = n/N$, where N is the sample size and n is the number of individuals with the characteristic of interest. The uncertainty around n is then given by the binomial distribution:

$$p(n) = \binom{N}{n} \rho^n (1-\rho)^{N-n} \tag{A.1}$$

However, Eq. (A.1) is only defined for discrete values of n. It is more convenient to have a continuous distribution for ρ, for sampling parameters and evaluating the likelihood of calibration targets. For this purpose, I use a beta approximation of the binomial distribution:

$$p(\rho) = \frac{\Gamma(\alpha+\beta)}{\Gamma(\alpha)\,\Gamma(\beta)} \rho^{\alpha-1}(1-\rho)^{\beta-1} \tag{A.2}$$

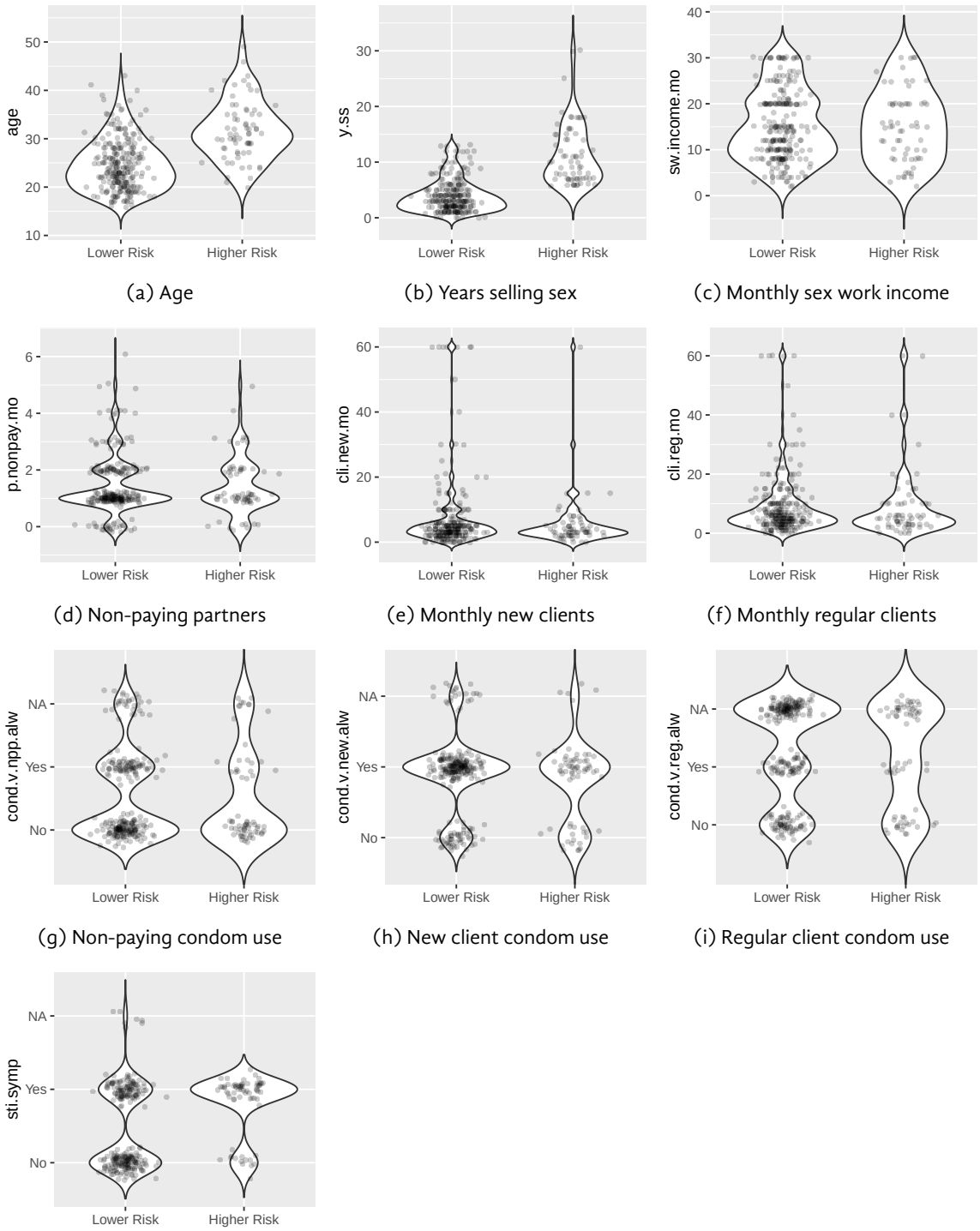
with $\alpha = N\rho$ and $\beta = N(1-\rho)$; Unlike the approximation by a normal distribution, the beta distribution ensures that $\rho \in [0, 1]$. Figure A.4 illustrates the approximation for $N = \{10, 20, 40\}$ and $\rho = \{0.01, 0.1, 0.5\}$.

A.2.2 Joint Sampling with Relational Constraints

Figure A.5 illustrates the posterior (sampled) distributions for variables X_1, X_2, X_3, having uniform priors but subject to $X_1 < X_2 < X_3$. Three approachs to enforcing $X_1 < X_2 < X_3$ were explored:

- **joint:** sample X_1, X_2, X_3 simultaneously; then discard any samples failing $X_1 < X_2 < X_3$.
- **forward:** sample X_1; then sample X_2 until $X_1 < X_2$; then sample X_3 until $X_2 < X_3$.
- **backward:** sample X_3; then sample X_2 until $X_2 < X_3$; then sample X_1 until $X_1 < X_2$.

All three methods result in a different posterior versus the prior, but the forward and backward methods severely distort the distributions for X_3 and X_1, respectively, while leaving the distributions for X_1 and X_3 unchanged. By contrast, the joint method influences the posterior distributions of each variable in a more "equitable" way, which is preferred.



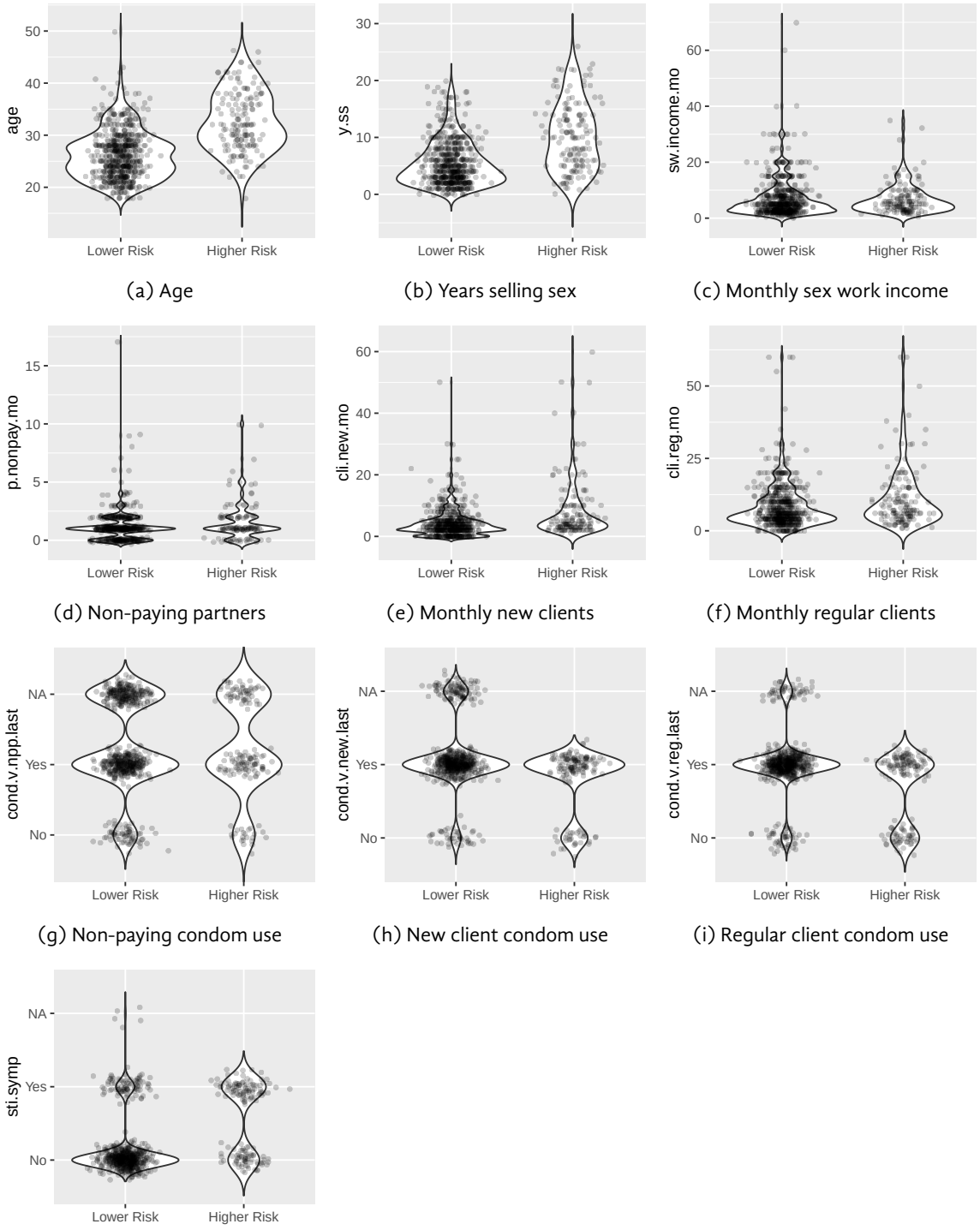
(a) Age (b) Years selling sex (c) Monthly sex work income

(d) Non-paying partners (e) Monthly new clients (f) Monthly regular clients

(g) Non-paying condom use (h) New client condom use (i) Regular client condom use

(j) Any STI symptoms past year

Figure A.1: HIV risk factor variables among higher vs lower risk FSW in eSwatini, as estimated by multivariate logistic regression model for serologic HIV status (2011)



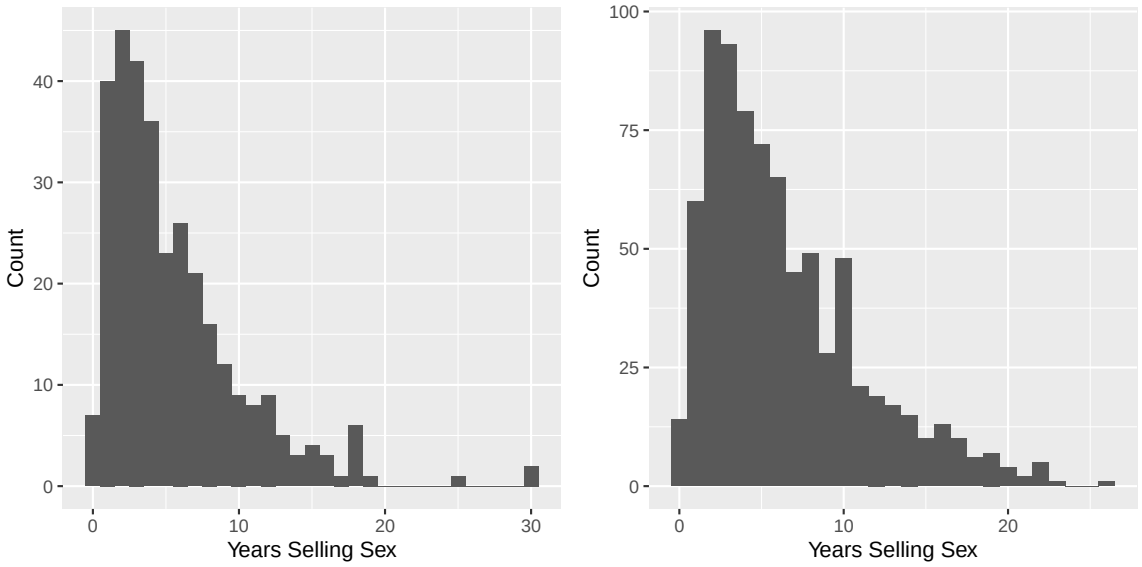
(a) Age (b) Years selling sex (c) Monthly sex work income

(d) Non-paying partners (e) Monthly new clients (f) Monthly regular clients

(g) Non-paying condom use (h) New client condom use (i) Regular client condom use

(j) Any STI symptoms past year

Figure A.2: HIV risk factor variables among higher vs lower risk FSW in eSwatini, as estimated by multivariate logistic regression model for self-reported HIV status (2014)



(a) 2011 [1] (b) 2014 [2]

Figure A.3: Duration in sex work among FSW in eSwatini

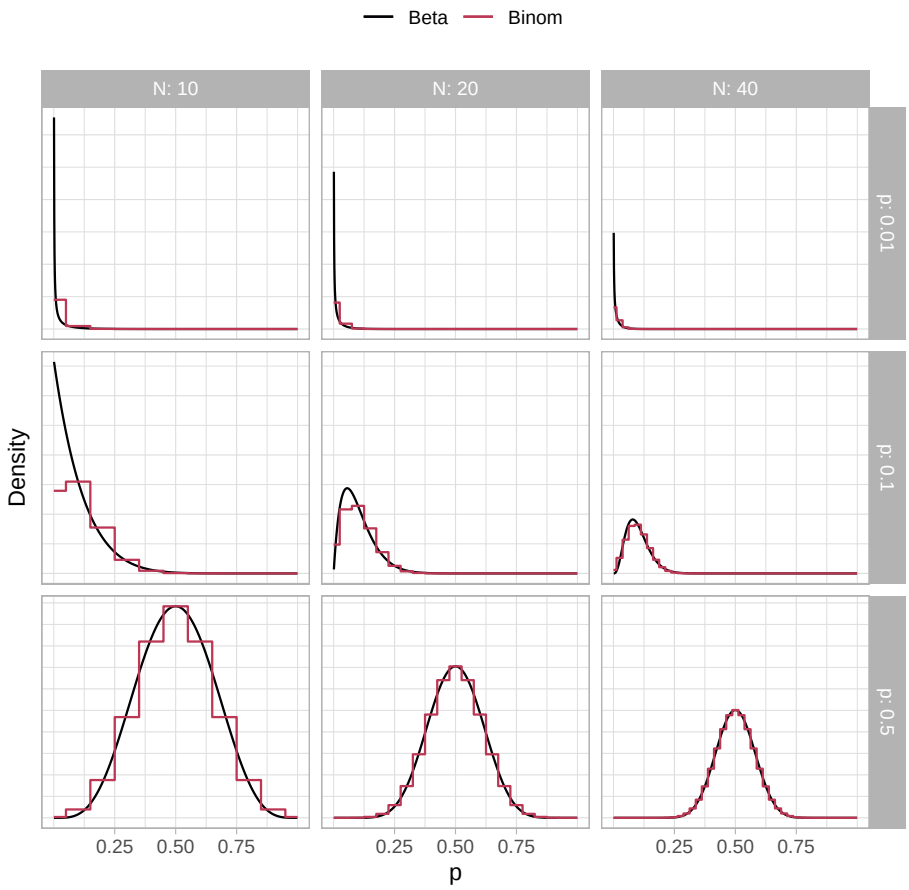


Figure A.4: Approximation of the binomial distribution with the beta distribution

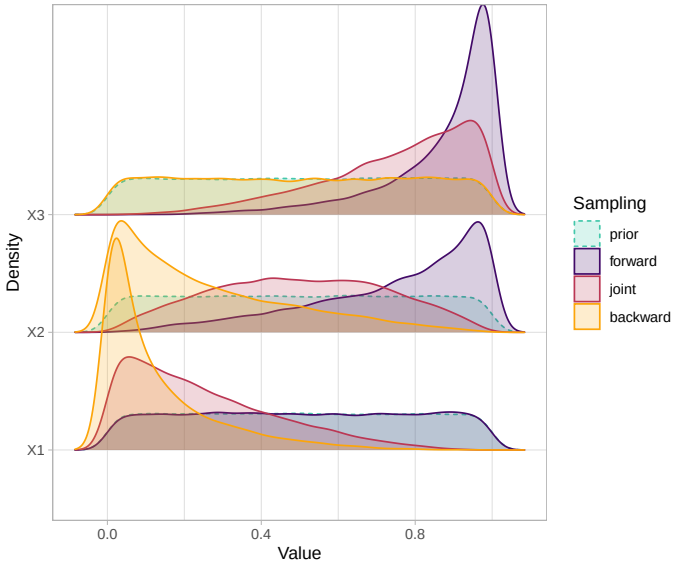


Figure A.5: Illustration of different sampling biases when enforcing $X_1 < X_2 < X_3$

A.2.3 Properties of Compartments with Fixed Exit Rates

Let λ be the fixed exit rate from compartment A, which is assumed to be homogeneous. Then $\delta \sim \lambda e^{-\lambda\delta}$ is the exponentially distributed duration time in the group.

Mean & Median Duration. The mean duration is $1/\lambda$ and the median is $\log(2)/\lambda$. Thus, if 50% of individuals progress from compartment A to B by time τ (median duration), the exit rate λ is given by $\log(2)/\tau$.

Collapsing Compartments in Series. Let compartments A and B be in series, with exit rates λ_A and λ_B respectively. Collapsing A and B into AB will sum the mean durations: $1/\lambda_A + 1/\lambda_B$; thus, the exit rate from AB will be $\lambda_{AB} = 1/(1/\lambda_A + 1/\lambda_B)$.

Collapsing Compartments in Parallel. Let compartments A and B be in parallel, with exit rates λ_A and λ_B respectively. Collapsing A and B into AB will sum the exit rates: $\lambda_A + \lambda_B$; thus, the mean duration in AB will be $\delta_{AB} = 1/(\lambda_A + \lambda_B)$.

A.2.4 Estimating Duration in Sex Work from Cross Sectional Data

Cross sectional sex work surveys will often ask respondents about their duration in sex work. These durations might then be taken to be the average durations in sex work; however, this will be an underestimate, because respondents will continue selling sex after the survey.[1]

Figure A.6 illustrates a steady-state population with 7 women selling sex at any given time. The steady-state assumption implies that a women leaving sex work after δ years will be immediately replaced by a women entering sex work whose eventual duration will also be δ years. Let δ be this true duration, and δ_s be the duration reported in the survey. If we assume that the survey reaches women at a random time point during the duration δ, then $\delta_s \sim \text{Unif}(0, \delta)$, and the mean reported duration is $E(\delta_s) = \frac{1}{2}E(\delta)$. Thus, $E(\delta) = 2E(\delta_s)$ would be an estimate of the true mean duration from the sample. In reality, sex work surveys may be more likely to reach women who have already been selling sex for several months or years, due to delayed self-identification as sex worker [3]. Thus, we would expect that $E(\delta)/E(\delta_s) \in (1, 2)$, which we can use to compute the mean exit rate as described in § A.2.3.

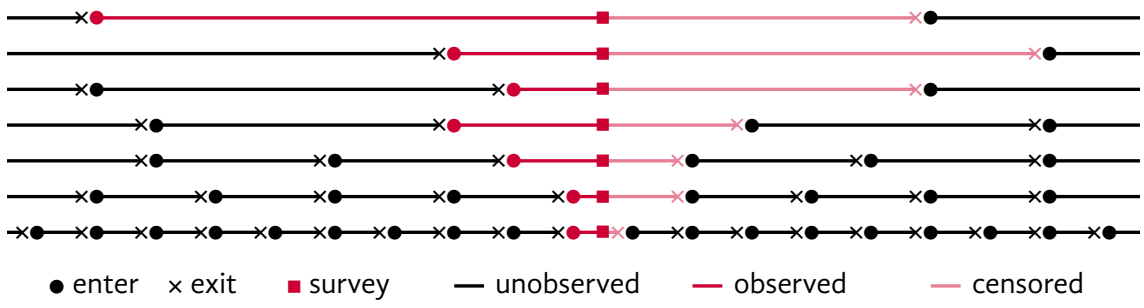


Figure A.6: Illustrative steady-state population of 7 FSW, with varying true durations in sex work δ, versus the observed durations in sex work δ_s via cross-sectional survey.

Another observation we can make from Figure A.6 is that women who sell sex longer are more likely to be

[1] An alternate example would be to take the mean age of a population as the life expectancy! Thanks to Saulius Simcikas and Dr. Jarle Tufto for help identifying and discussing this bias: stats.stackexchange.com/questions/298828.

captured in the survey. That is, while the sampled durations are representative of women who *currently* sell sex, these durations are biased high versus the population of women who *ever* sell sex. It's not clear whether this observation is widely understood and kept in mind when interpreting sex work survey data.

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