SUPPLEMENTAL FIGURES, TABLES, AND TEXTS

Figure S1. Representation of the different A) risk and B) age groups, as well as their contact patterns. A) Female sex workers (FSW) only have sexual contacts with their clients but can retire from sex work, in which case FSW integrate the group of high-risk women. CFSW have sexual contacts with FSW and both low- and high-risk women. Women in the low- and high-risk groups have sexual contacts with all members of the opposite sex, apart from exclusive men who have sex with men (MSM). Exclusive MSM have sexual contacts with both exclusive MSM and MSM who engage in bisexual practices. B) With regards to age, sexual debut is either at 15 or 20 years of age for both men and women. Immigration is allowed in the 25-49 year's age group. All age groups have sexual contacts with other age groups, except for women in the 50-59 age group who do not have sexual contacts with men younger than 25 years of age. This mixing by age was informed by data from Demographic and Health Survey (DHS). DHS data was also used to inform mixing between low- and high-risk groups of men and women.

In the remaining of this appendix, risk groups are indexed from 1 to 9 by subscript *k.* Low-risk women correspond to *k*=1; high-risk women *k*=2; FSW *k*=(3,4) (*k*=4 denotes those engaging in anal intercourse); low-risk men *k*=5; high-risk men *k*=6; CFSW *k*=7; MSM who engage in bisexual practices *k*=8; and exclusive MSM *k*=9. Age groups are indexed from 1 to 4 by subscript *i.* For 15-19 years old *i*=1; 20-24 years old *i*=2; 25-49 years old *i*=3; and 50-59 years old $i=4$.

Figure S2. Model structure of HIV transmission, disease progression, and continuum of care. Where *Γki(t)* is the rate of entry in the sexually active population for members of risk group *k* and age class *i* (i.e., men start their sexually active life later than women and this proportion changes with time); μ_i is the HIV-unrelated background mortality rate for age class *i*; $\lambda_{ki}(t)$ is the force of infection at time *t* for individuals in risk group *k* and age class *i*; *γ^s* is the disease progression rate for the different stages of infections (defined by the CD4 levels and indexed by *s*); τ ^{*s*}_{*ki*}(*t*) is the testing rate that varies with time, stage of infection, risk group, and age class; ρ _{*s*}(*t*) is the ART recruitment rate which is a function of time and disease stage; ϑ is the rate at which individuals on ART become virally suppressed; l_k is the ART discontinuation rate; φ is the rate of therapeutic failures among virally suppressed individuals; and *ω^s* is the HIV-related mortality rate of individuals on ART that varies by disease stage.

In the remaining of this appendix, the different stages of infection are indexed by "*a"* and "*s"*. The superscript "*a*" denotes the testing and antiretroviral therapy (ART) status: *"0"* for undetected individuals, *"1"* for those tested but untreated, *"2"* for those treated with a detectable viral load, *"3"* for those virally suppressed, and *"4"* for those experiencing therapeutic failures. The second superscript "*s*" described the disease progression according to CD4 cell counts: *"1"* for acute infection, *"2"* for CD4>500 cells/µL, *"3"* for CD4 between 350 and 500 cells/µL, *"4"* for CD4 between 200 and 349 cells/µL, and *"5"* for CD4 <200 cells/µL.

Figure S3. Decision tree used to estimate vertical transmission of HIV. Green boxes represent prevention of mother-to-child transmission (PMTCT) interventions. HIV positive women become pregnant at a rate $\gamma_i(t)$ that varies as a function of time *t* and age *i*. A proportion $\Psi(t)$ of these women - as determined by the dynamic transmission model - will already be on antiretroviral treatment (ART). Other women can benefit from PMTCT if they attend and are tested during antenatal care (ANC) visits, according to a proportion $\delta_i(t)$ that varies with time and age *i* (women not accessing ANC or not being tested for HIV do not benefit from PMTCT). This proportion was informed by self-reported ANC attendance and testing data from Demographic and Health Surveys (DHS). Among women tested for HIV during ANC, some will not receive any prophylaxis $(I-\kappa(t)-\phi(t))$, others will receive ART prophylaxis $(\kappa(t))$ for them and their babies or receive ART for life $(\phi(t))$. These proportions were derived from programmatic data reported by Côte d'Ivoire. The probability of mother-to-child transmission also depends on the proportion (*A*) of infants that will be breastfeed and the mother's CD4 cell count at delivery (not shown on figure). Breastfeeding parameters were informed by DHS data.

AI=anal intercourse; FSW=female sex worker; MSM=men who have sex with men; *T*(m,a,b)=Triangular distribution (m=mode, a=minimum, b=maximum); *U*(a,b)=Uniform distribution (a=minimum, b=maximum).

*The proportion of clients of FSW was indirectly estimated using the multiplier method, balancing the partner change rate reported by FSW and by clients of FSW. The proportion of clients of FSW was constrained to be less\ than 20%.

†Parameter informed by the analysis of primary data of respondent-driven sampling surveys conducted in five cities of Côte d'Ivoire (unpublished).

AI=anal intercourse; CFSW=clients of female sex workers; DHS=Demographic and Health Survey; FSW=female sex worker; MSM=men who have sex with men; $U(a,b)$ =Uniform distribution (a=min, b=max).

*Based on the assumption that FSW engage in sex work 4 days per week, 50 weeks per year.

†CFSW were assumed to drive demand and FSW were de facto assumed to adjust their partner change rate in consequence. For MSM, we assumed that the balance parameter is equal to 0.5.

‡ Parameter informed by the analysis of primary data of respondent-driven sampling surveys conducted in five cities of Côte d'Ivoire (unpublished).

Table S3. Biological and treatment parameters.

AI=anal intercourse; ART=antiretroviral therapy; FSW=female sex worker; MSM=men who have sex with men; RR=relative risk; *T*(m,a,b)=Triangular distribution (m=mode, a=minimum, b=maximum); *U*(a,b)=Uniform distribution (a=minimum, b=maximum); VI=vaginal intercourse. *HIV-related mortality (ω_s) for those on treatment is calculated as followed: $\omega_s = (\Sigma \gamma_s)/RR_\omega$.

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ART=antiretroviral therapy; *U*(a,b)=Uniform distribution (a=minimum, b=maximum); WHO=World Health Organization.

Table S5. Parameters used to estimate historical trends in the proportion of sex acts protected by a condom.

AI=anal intercourse; CFSW=client of female sex worker; FSW=female sex worker; MSM=men who have sex with men; RR=relative risk; *T*(m,a,b)=Triangular distribution (m=mode, a=minimum, b=maximum); *U*(a,b)=Uniform distribution (a=minimum, b=maximum); VI=vaginal intercourse.

*The minimum of the range corresponds to the lower confidence bound of the estimate reported by females and the maximum to the upper confidence bound of the estimate reported by males.

†Only the 2005 AIDS Indicator Survey collected information on men aged 50-59 years old. When unavailable, estimates for this age class were approximated by extrapolating those of the 45-49 years old age group.

‡The estimates of the proportion of sex acts protected by a condom are sampled for each year using the same percentile of the distribution to obtain temporally consistent estimates.

§Parameter informed by the analysis of primary data of respondent-driven sampling surveys conducted in five cities of Côte d'Ivoire (unpublished).

Table S6. Parameters used to estimate historical trends in HIV testing.

ANC=antenatal care; FSW=female sex worker; MSM=men who have sex with men; RR=relative risk;

T(m,a,b)=Triangular distribution (m=mode, a=minimum, b=maximum); *U*(a,b)=Uniform distribution (a=minimum, b=maximum); VI=vaginal intercourse.

*Probabilities of being tested during last 12 months was converted to rate (year⁻¹) using Rate = -ln(1-Probability) †Only the 2005 AIDS Indicator Survey collected information on men aged 50-59 years old. When unavailable, estimates for this age class were approximated by extrapolating those of the 45-49 years old age group. **‡**Women aged 50-59 years old are assumed to have a fertility rate of zero.

§The estimates for HIV testing rate and proportion of pregnant women tested as part of their ANC are sampled for each year using the same percentile of the distribution to obtain temporally consistent estimates.

Table S7. Parameters used to estimate historical trends in the availability of interventions for the prevention of mother-to-child transmission.

ANC=antenatal care; ART=antiretroviral therapy; PMTCT=prevention of mother-to-child transmission. *The lower bound of the confidence interval corresponds to the proportion of infants receiving prophylaxis and the upper bound to the proportion of mothers receiving prophylaxis.

 \dagger The bound of the prior distribution were constructed by varying point estimates by \pm 10%.

‡The estimates for proportion of pregnant women testing positive for HIV receiving prevention of mother-to-child transmission interventions are sampled for each year using the same percentile of the distribution to obtain temporally consistent estimates.

Parameters	Symbols	Prior distributions	References
Recruitment rate into ART when symptomatic $(<200$ CD4 cells/ μ L) (years ⁻¹)	ρ_5	U(0.5, 4)	Assumption
Slope cofactor to define linear relation between CD4 stages and recruitment into ART		U(0, 1)	Assumption

Table S8. Parameters used to estimate historical trends in antiretroviral therapy coverage.

ART=antiretroviral treatment; *U*(a,b)=Uniform distribution (a=minimum, b=maximum).

Table S9. List of outcomes that the mathematical model was fitted to.

Men who have sex with men

*95% CI: 95% confidence intervals.

£The overall likelihood of the model is calculated by summing the binomial log-likelihoods of the relevant model's outcomes and assigning a likelihood of zero if any of the model-predicted outcomes fall outside of the pre-specified constraints.

†Surveys were adjusted for imperfect sensitivity and specificity of the diagnostic assays (see Text S6).

‡Some age categories in the surveys do not exactly match the ones in the model. For the 1989 survey the 25-49 age category corresponds to prevalence of the 25-54 years old and the 50-59 age category corresponds to prevalence in the 55-64 years old. For the 2005 AIDS Indicator Survey, the 50-59 age category corresponds to prevalence from the 45-49 years old. For the 2011-12 Demographic and Health Survey, the 50-59 age category for females corresponds to prevalence in females aged 45-49 years old.

¶ Parameter informed by the analysis of primary data of respondent-driven sampling surveys conducted in five cities of Côte d'Ivoire. The city-specific prevalence estimates were pooled by weighting each city by its total population. ††These prevalence estimates are from first time attendees of the Clinique de Confiance in Abidjan. A design effect of 5

was applied to obtain an effective sample size in the likelihood calculations to take into account the clustered nature of these observations.

‡‡Confidence intervals were adjusted for the clustered design of the survey by using the number of clusters (n=84) as the effective sample size.

§ART coverage was not included in the binomial likelihood because this was derived from programmatic data on the number of individuals on ART (numerator) and the number of HIV positive individuals in Côte d'Ivoire estimated by UNAIDS. As such, there was no sample size to use in the binomial likelihood.

Text S1. Details on sexual mixing patterns.

Sexual mixing

Sexual mixing is a function of age, sex, and the risk category of individuals. The probability *pkijl* of a sexual contact between someone of risk category *k* and age *i* with an individual of risk category *j* and age *l* is estimated using the following equation:

The binary *WMW* matrix described the types of contacts allowed in the model for the following risk groups: low-risk female (F_{LR}) , high-risk female (F_{HR}) , female sex worker (F_{FSW}) , female sex worker that practice anal intercourse (F_{FSWA}) , low-risk males (M_{LR}) , high-risk males (M_{HR}) , clients of female sex workers (M_{CFSW}) , men who have sex with men who engage in bisexual practices (M_{BI}) , and exclusive men who have sex with men (M_{MSM}) .

The sizes of the low- and high-risk groups were defined using data from the 2011-12 Demographic and Health Survey (DHS) based on the number of sexual partners during the last 12 months (F_{HR} defined as >1 partner year⁻¹; M_{HR} defined as >2 partners year⁻¹; excluding those that reported selling or buying sex) [6]. Mixing by risk groups was calculated using these individuallevel records for low- and high-risk males and females. Due to data limitation, mixing by risk groups was only available for couples living in the same household who both agreed to be interviewed and reported complete data on their sexual partners. Because some unions in Côte d'Ivoire are polygamous, mixing by risk group was analyzed from the female perspective. This DHS-reported matrix *M* was expanded to include the other risk groups and their associated

parameters: *Bi_{Pref}* being the fraction of partnerships that are with females for MSM engaging in bisexual practices, Pr_{Bi} the fraction of MSM who are bisexual (implicitly assuming proportional mixing between exclusive MSM and MSM who engage in bisexual practices), and $CFSW_{Mix}$ the fraction of partnerships that are with FSW for CFSW ($CFSW_{Mix} = c\frac{r}{c_7+c_7b}$).

Mixing patterns by age (*Λ*) were informed using data from the 2011-12 DHS [6] for the general population. The survey data refers to the reported age of the most recent sexual partner among the population that was sexually active in the 12 months preceding the interview. The age mixing matrices differ for males and females. For sex workers and clients, it was assumed that age mixing would correspond to that reported by males in the general population. For MSM, it was assumed that age mixing would be somewhere between completely assortative and proportional mixing using the tuning parameter *MSMAgeMix*, which was given a non-informative distribution.

$$
\Lambda_{kil} = \begin{cases}\n0.130 & 0.130 & 0.719 & 0.021 \\
0.130 & 0.130 & 0.719 & 0.021 \\
0.003 & 0.003 & 0.753 & 0.241 \\
0 & 0 & 0.187 & 0.813\n\end{cases}; \quad \text{for } k=1,2 \text{ (}i \text{ indexes row and } l \text{ columns)}
$$
\n
$$
\Lambda_{kil} = \begin{cases}\n0.483 & 0.483 & 0.035 & 0 \\
0.483 & 0.483 & 0.035 & 0 \\
0.194 & 0.194 & 0.609 & 0.004 \\
0.125 & 0.125 & 0.808 & 0.167\n\end{cases}; \quad \text{for } k=3,4,5,6,7
$$
\n
$$
(1 - MSM_{AgelMix}) \begin{pmatrix}\n1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & 0 & 1 & 0 \\
0 & 0 & 0 & 1\n\end{pmatrix} + MSM_{AgelMix} \begin{pmatrix}\n0.152 & 0.188 & 0.553 & 0.107 \\
0.152 & 0.188 & 0.553 & 0.107 \\
0.152 & 0.188 & 0.553 & 0.107 \\
0.152 & 0.188 & 0.553 & 0.107\n\end{pmatrix}; \quad \text{for } k=8,9
$$

The probability of a sexual contact is calculated separately for each type of partnerships (*kijl)*. Imbalances between demand for sexual partnerships by one group and offer by another group are thus possible. Balance between supply of and demand for sexual partnership was achieved by altering the partner change rate (c_{kijl}^*) using the methods described by Garnett and Anderson [20] and is described below.

$$
\Delta_{kijl} = \frac{c_{jl} p_{jlki} N_{jl}}{c_{ki} p_{kijl} N_{ki}}
$$

$$
c_{kijl}^* = c_{ki} \Delta_{kijl} \frac{\eta_k}{\eta_k}
$$

$$
c_{jlki}^* = c_{jl} \Delta_{kijl} \frac{-(1-\eta_k)}{\eta_k}
$$

where *Δkijl* measures the degree of initial imbalance between supply and demand for sexual partnerships of type $kijl$ and η_k is the balance parameter that determines the degree to which partners are submissive (i.e., the degree to which they alter their demand/offer of sexual partnerships). For clients and FSW, we assumed that clients would drive demand. For MSM, the balance parameter was assumed to be equal to 0.5.

Text S2. HIV transmission model's ordinary differential equations.

The dynamic system formed by the different model's compartments can be represented through 22 differential equations. All susceptible individuals at time *t* in risk category *k* and age *i* are represented by $S_{ki}(t)$. The different stages of infection are indexed by I_{ki}^{as} . The superscript "*a*" denotes the testing and antiretroviral therapy (ART) status: *"0"* for undetected individuals, *"1"* for those tested but untreated, *"2"* for those treated with a detectable viral load, *"3"* for those virally suppressed, and *"4"* for those experiencing therapeutic failures. The second supersript "*s*" described the disease progression according to CD4 cell counts: *"1"* for acute infection, *"2"* for CD4>500 cells/µL, *"3"* for CD4 between 350 and 500 cells/µL, *"4"* for CD4 between 200 and 349 cells/µL, and *"5"* for CD4 $<$ 200 cells/ μ L.

1. Susceptible

$$
\frac{dS_{ki}(t)}{dt} = E_{ki}(t) + W_i^1 - S_{ki}(t) \sum_j (\lambda_{kij}(t)) - S_{ki}(t) (\mu_i + Aging_i)
$$
\n
$$
\frac{dE_{ki}(t)}{\sum_{s=2}^{5} I_{ki}^{2s} (t) \omega_{s-1} + \sum_{s=2}^{5} I_{ki}^{3s} (t) \omega_{s-1} + \varepsilon \sum (I_{ki}^{as}(t) + S_{ki}(t)) + V_k(t)) + \frac{1}{2} \left(1 - Vir_k(t) \right); \text{ if } i = 15 - 19 \text{ years old}
$$
\n
$$
\frac{dE_{ki}(t)}{dt} = \begin{cases} \sum_i \mu_i V_k(t) + S_{ki}(t) \omega_{s-1} + \sum_{s=2}^{5} I_{ki}^{3s} (t) \omega_{s-1} + \varepsilon \sum (I_{ki}^{as}(t) + S_{ki}(t) + V_k(t)) + V_k(t)) + \frac{1}{2} (1 - Vir_k(t)); \text{ if } i = 15 - 19 \text{ years old} \\ \sum_i (I_{ki}^{as}(t)) ; \text{ if } i = 20 - 24 \text{ years old} \\ \sum_i (I_{ki}^{as}(t)) ; \text{ if } i = 25 - 49 \text{ years old} \end{cases}
$$
\n
$$
\frac{dV_k(t)}{dt} = \begin{cases} \sum_i \mu_i V_k(t) + \sum_i (\mu_i (I_{ki}^{as}(t) + S_{ki}(t))) + \gamma_5 (I_{ki}^{0.5}(t) + I_{ki}^{1.5}(t) + I_{ki}^{4.5}(t)) + \\ \sum_{s=2}^{5} I_{ki}^{2s} (t) \omega_{s-1} + \sum_{s=2}^{5} I_{ki}^{3s} (t) \omega_{s-1} + \varepsilon \sum (I_{ki}^{as}(t) + S_{ki}(t) + V_k(t)) + V_k(t) + V_k(t) (I_{ki} + Aging_1) \end{cases}
$$

 $E_{ki}(t)$ represents individuals of category *k* and age class *i* entering the susceptible compartment at time *t*. These individuals are those lost by the system at time *t-1* as a result of background mortality, HIV-related mortality, and exit from sexually active life. Immigration occurs at a rate *χ* and immigrants are assumed to be aged between 25 and 49 years old. The population of Côte d'Ivoire grows at a rate *ε*. It should be noted that individuals enter the sexual active life either at 15 or 20 years old, according to the proportion $Vir_k(t)$, and $V_k(t)$ is the number of individuals 15-19 years of age who are not yet sexually active at time *t*. They do not get infected and cannot die of AIDS but are subjected to the same background mortality rate. Aging rates are stored in the homonymous vector and the aging process is defined the following way:

$$
\frac{dA \text{ged}_{ki}^{a,s}(t)}{dt} = \begin{cases} 0; & \text{if } i = 15-19 \text{ years old} \\ I_{k(i-1)}^{as}(t) \text{Aging}_{(i-1)}; & \text{if } i \neq 15-49 \text{ years old} \\ \frac{1}{5}, \frac{1}{5}, \frac{1}{5}, \frac{1}{25}, \frac{1}{10} \end{cases}
$$

Female sex workers (FSW) will be involved in sex work for a certain period of time before exiting this practice and returning to the group composed of high-risk females. This turnover (symbol in tables: *tur*) is denoted by W_{ki}^c (where *c* indexes the compartment's number that varies from 1 to 22, as in the equations presented here). It occurs independently of HIV status and disease progression. An

equal number of high-risk females will then enter the FSW risk group to maintain stable the size of the FSW population. The decision tree used to model mother-to-child transmission also calculates the number of HIV positive pregnant women that will be tested at time *t* or be given antiretroviral treatment. These women are then attributed to the relevant compartment using the symbol $P_{ki}^{c}(t)$. Finally, immigration into the 25-49 years old age group of infected individuals (assumed undetected and untreated) is denoted by U_{ki}^{as} . These immigrants are assumed to have the same HIV prevalence as observed in Côte d'Ivoire at time *t*.

2. Primary Infection
\n
$$
\frac{dI_{ki}^{0,1}(t)}{dt} = Aged_{ki}^{0,1}(t) + S_{ki}(t) \sum_{jl} (\lambda_{kijl}(t)) + W_{ki}^{2}(t) + U_{ki}^{0,1}(t) - I_{ki}^{0,1}(t)(\gamma_{1} + \mu_{i} + Aging_{i})
$$
\n
$$
U_{ki}^{0,1}(t) = \begin{cases} \chi(\sum_{a} I_{ki}^{a,1}(t)); & \text{if } i=25-49 \text{ years old} \\ 0; & \text{if } i \neq 25-49 \text{ years old} \end{cases}
$$
\n3. Undetected HIV positive with CD4>500 cell/µL
\n
$$
\frac{dI_{ki}^{0,2}(t)}{dt} = Aged_{ki}^{0,2}(t) + I_{ki}^{0,1}(t)\gamma_{1} + W_{ki}^{3}(t) + U_{ki}^{0,2}(t) - I_{ki}^{0,2}(t)\left(\tau_{ki}^{1}(t) + \gamma_{2} + \mu_{i} + Aging_{i}\right) - P_{ki}^{3}(t)
$$
\n
$$
U_{ki}^{0,2}(t) = \begin{cases} \chi(\sum_{a} I_{ki}^{a,2}(t)); & \text{if } i=25-49 \text{ years old} \\ 0; & \text{if } i \neq 25-49 \text{ years old} \end{cases}
$$
\n4. Undetected HIV positive with CD4 between 350 and 500 cells/µL

$$
\frac{dI_{ki}^{0,3}(t)}{dt} = Aged_{ki}^{0,3}(t) + I_{ki}^{0,2}(t)\gamma_2 + W_{ki}^4(t) + U_{ki}^{0,3}(t) - I_{ki}^{0,3}(t)\left(\tau_{ki}^1(t) + \gamma_3 + \mu_i + Aging_i\right) - P_{ki}^4(t)
$$

$$
U_{ki}^{0,3}(t) = \begin{cases} \chi(\sum_a I_{ki}^{a,3}(t)); & \text{if } i=25-49 \text{ years old} \\ 0; & \text{if } i \neq 25-49 \text{ years old} \end{cases}
$$

5. Undetected HIV positive with CD4 between 200 and 349 cells/µL

$$
\frac{dI_{ki}^{0,4}(t)}{dt} = Aged_{ki}^{0,4}(t) + I_{ki}^{0,3}(t)\gamma_3 + W_{ki}^5(t) + U_{ki}^{0,4}(t) - I_{ki}^{0,4}(t)\left(\tau_{ki}^1(t) + \gamma_4 + \mu_i + Aging_i\right) - P_{ki}^5(t)
$$
\n
$$
U_{ki}^{0,4}(t) = \begin{cases} \chi(\sum_a I_{ki}^{a,4}(t)); & \text{if } i=25-49 \text{ years old} \\ 0; & \text{if } i \neq 25-49 \text{ years old} \end{cases}
$$

6. Undetected HIV positive with CD4<200 cells/µL

$$
\frac{dI_{ki}^{0.5}(t)}{dt} = Aged_{ki}^{0.5}(t) + I_{ki}^{0.4}(t)\gamma_4 + W_{ki}^6(t) + U_{ki}^{0.5}(t) - I_{ki}^{0.5}(t)\left(\tau_{ki}^2(t) + \gamma_5 + \mu_i + Aging_i\right) - P_{ki}^6(t)
$$
\n
$$
U_{ki}^{0.5}(t) = \begin{cases} \chi(\sum_a I_{ki}^{a.5}(t)); & \text{if } i = 25-49 \text{ years old} \\ 0; & \text{if } i \neq 25-49 \text{ years old} \end{cases}
$$
\n7. Dateded/untracted, HIV positive with CD4>500, cells/uJ

7. Detected/untreated HIV positive with CD4>500 cells/µL

$$
\frac{dI_{ki}^{1,2}(t)}{dt} = Aged_{ki}^{1,2}(t) + I_{ki}^{0,2}(t)\tau_{ki}^{1}(t) + I_k(I_{ki}^{2,2}(t) + I_{ki}^{3,2}(t)) + W_{ki}^{7}(t) + P_{ki}^{7}(t) - I_{ki}^{1,2}(t)\left(\rho_2(t) + \gamma_2 + \mu_i + Aging_i\right)
$$

8. Detected/untreated HIV positive with CD4 between 350 and 500 cells/µL

$$
\frac{dI_{ki}^{1,3}(t)}{dt} = A \text{ge} d_{ki}^{1,3}(t) + I_{ki}^{1,2}(t) \gamma_{2} + I_{ki}^{0,3}(t) \tau_{ki}^{1}(t) + I_{k} (I_{ki}^{2,3}(t) + I_{ki}^{3,3}(t)) + W_{ki}^{8}(t) + P_{ki}^{8}(t) - I_{ki}^{1,3}(t) (\rho_{3}(t) + \gamma_{3} + \mu_{i} + A \text{ging}_{i})
$$
\n9. *Detected/untreated HIV positive with CD4 between 200 and 349 cells/µL*\n
$$
\frac{dI_{ki}^{1,4}(t)}{dt} = A \text{ge} d_{ki}^{1,4}(t) + I_{ki}^{1,3}(t) \gamma_{3} + I_{ki}^{0,4}(t) \tau_{ki}^{1}(t) + I_{ki} (I_{ki}^{2,4}(t) + I_{ki}^{3,4}(t)) + W_{ki}^{0}(t) - I_{ki}^{1,4}(t) (\rho_{4}(t) + \gamma_{4} + \mu_{i} + A \text{ging}_{i})
$$
\n10. *Detected/untreated HIV positive with CD4 < 200 cells/µL*\n
$$
\frac{dI_{ki}^{1,5}(t)}{dt} = A \text{ge} d_{ki}^{1,5}(t) + I_{ki}^{1,4}(t) \gamma_{4} + I_{ki}^{0,5}(t) \tau_{ki}^{1}(t) + I_{ki}^{1,5}(t) + W_{ki}^{1,6}(t) + W_{ki}^{10}(t) + I_{ki}^{1,5}(t) (\rho_{4}(t) + \gamma_{5} + \mu_{i} + A \text{ging}_{i})
$$
\n11. *Treated HIV positive, with detectable viral load and CD4 > 500 cells/µL*\n
$$
\frac{dI_{ki}^{2,2}(t)}{dt} = A \text{ge} d_{ki}^{2,2}(t) + \rho_{2}(t) (I_{ki}^{1,2}(t) + I_{ki}^{4,3}(t)) + W_{ki}^{1,1}(t) - I_{ki}^{2,2}(t) (I_{k} + \theta + \alpha_{1} + \mu_{i} + A \text{ging}_{i})
$$
\n12. *Treated HIV positive, with detectable viral load and CD4 between 350 and 500 cells/µL*\n<

$$
\frac{dI_{ki}^{3,2}(t)}{dt}=A\text{gcd}_{ki}^{3,2s}(t)+I_{ki}^{2,2}(t)\mathcal{G}+W_{ki}^{15}-I_{ki}^{3,2}(t)\big(l_k+\varphi+\omega_1+\mu_i+A\text{ging}_i\big)
$$

16. Treated HIV positive, with undetectable viral load and CD4 between 350 and 500 cells/µL

$$
\frac{dI_{ki}^{3,3}(t)}{dt} = Aged_{ki}^{3,3}(t) + I_{ki}^{2,3}(t) \mathcal{G} + W_{ki}^{16}(t) - I_{ki}^{3,3}(t) (l_k + \varphi + \omega_2 + \mu_i + Aging_i)
$$

17. Treated HIV positive, with undetectable viral load and CD4 between 200 and 349 cells/µL

$$
\frac{dI_{ki}^{3,4}(t)}{dt} = Aged_{ki}^{3,4}(t) + I_{ki}^{2,4}(t) \mathcal{G} + W_{ki}^{17}(t) - I_{ki}^{3,4}(t) (l_k + \varphi + \omega_3 + \mu_i + Aging_i)
$$

18. Treated HIV positive, with undetectable viral load and CD4<200 cells/µL

$$
\frac{dI_{ki}^{3,5}(t)}{dt} = Aged_{ki}^{3,5}(t) + I_{ki}^{2,5}(t)\mathcal{G} + W_{ki}^{18}(t) - I_{ki}^{3,5}(t)(l_k + \varphi + \omega_4 + \mu_i + Aging_i)
$$

19. HIV positive experiencing treatment failure and with CD4>500 cells/µL

4,2

$$
\frac{dI_{ki}^{4,2}(t)}{dt}=A\text{ge}d_{ki}^{4,2}(t)+I_{ki}^{3,2}(t)\varphi+W_{ki}^{19}(t)-I_{ki}^{4,2}(t)(\gamma_2+\rho_2(t)+\mu_i+A\text{ging}_i)
$$

20. HIV positive experiencing treatment failure and with CD4 between 350 and 500 cells/µL

$$
\frac{dI_{ki}^{4,3}(t)}{dt} = Aged_{ki}^{4,3}(t) + I_{ki}^{3,3}(t)\varphi + I_{ki}^{4,2}(t)\gamma_2 + W_{ki}^{20}(t) - I_{ki}^{4,3}(t)(\gamma_3 + \rho_3(t) + \mu_i + Aging_i)
$$

21. HIV positive experiencing treatment failure and with CD4 between 200 and 349 cells/µL

$$
\frac{dI_{ki}^{4,4}(t)}{dt} = Aged_{ki}^{4,4}(t) + I_{ki}^{3,4}(t)\varphi + I_{ki}^{4,3}(t)\gamma_3 + W_{ki}^{21}(t) - I_{ki}^{4,4}(t)(\gamma_4 + \rho_4(t) + \mu_i + Aging_i)
$$

22. HIV positive experiencing treatment failure and with CD4<200 cells/µL

$$
\frac{dI_{ki}^{4,5}(t)}{dt}=A\text{g}ed_{ki}^{4,5}(t)+I_{ki}^{3,5}(t)\varphi+I_{ki}^{4,4}(t)\gamma_4+W_{ki}^{22}(t)-I_{ki}^{4,5}(t)(\gamma_5+\rho_5(t)+\mu_i+A\text{ging}_i)
$$

Text S3. Equations for the force of infection.

The force of infection (FOI) is defined as the annual probability of HIV transmission from an individual of risk group *j* and age class *l* to an individual of risk group *k* and age class *i* [54]. Five equations are used to describe the force of infection, depending on partnership and type of sexual contacts in the partnership: 1) vaginal female-to-male or 2) vaginal male-to-female, 3) receptive and insertive anal male-to-male, 4) vaginal and anal female-to-male, 4) vaginal and anal male-to-female. The *λkijl(t)* matrix contains the FOI for each partnership combination of category *k/j* and age *i/l*, where the susceptible partner is the one indexed by *ki*.

1) Force of infection for HIV transmission between a female (j) and a male (k).

$$
\lambda_{kijl}(t) = c_{_{kijl}}^{*} p_{kijl} \left[\sum_{a} s \left(\left(\frac{I_{jl}^{as}(t)}{\sum_{a} I_{jl}^{as}(t) + S_{jl}(t)} \right) \left(1 - \left(\left(1 - \beta_{fm} R_{ji}^{as} \right)^{\alpha_{kijl}(1 - v_{ki}(t))} \left(1 - \beta_{fm} R_{ji}^{as}(1 - \zeta) \right)^{\alpha_{kijl}(v_{ki}(t))} \right) \right) \right]
$$

(2) Force of infection for HIV transmission between a male (j) and a female (k).

$$
\lambda_{kijl}(t) = c_{_{kijl}}^{*} p_{kijl} \left[\sum_{a} \left(\left(\frac{I_{jl}^{as}(t)}{\sum_{a} I_{jl}^{as}(t) + S_{jl}(t)} \right) \left(1 - \left(\left(1 - \beta_{mf} R_{ji}^{as} \right)^{\alpha_{kijl}(1 - v_{ki}(t))} \left(1 - \beta_{mf} R_{ji}^{as}(1 - \zeta) \right)^{\alpha_{kijl}(v_{ki}(t))} \right) \right) \right]
$$

(3) Force of infection for HIV transmission between a male (j) and a male (k).

$$
\lambda_{kijl}(t) = c_{_{kijl}}^{*} p_{kijl} \left[\sum_{a} \left(\frac{I_{jl}^{as}(t)}{\sum_{a} I_{jl}^{as}(t) + S_{jl}(t)} \right) \left(1 - \left(\left(1 - \beta_{fm} Z_{ji}^{as} \right)^{\alpha_{kijl}(1 - v_{ki}(t))^{*0.5}} \left(1 - \beta_{fm} Z_{ji}^{as}(1 - \zeta) \right)^{\alpha_{kijl}(v_{ki}(t))^{*0.5}} \right) \right) \right) \right]
$$

(4) Force of infection for HIV transmission between a female (j) and a male (k) who engage in both anal and vaginal intercourse.

$$
\lambda_{kijl}(t) = c_{_{kijl}}^{*} p_{kijl} \left[\sum_{a} \left(\left(\frac{I_{jl}^{as}(t)}{\sum_{a} I_{jl}^{as}(t) + S_{jl}(t)} \right) \left(1 - \left(\left(1 - \beta_{mf} R_{ji}^{as} \right)^{VI_{kijl}(1 - v_{ki}(t))} \left(1 - \beta_{mf} R_{ji}^{as}(1 - \zeta) \right)^{VI_{kijl}(v_{ki}(t))} \right) \right) \right) \right]
$$

(5) Force of infection for HIV transmission between a male (j) and a female (k) who engage in both anal and vaginal intercourse.

$$
\lambda_{kijl}(t) = c_{_{kijl}}^{*} p_{kijl} \left[\sum_{a} \left(\left(\frac{I_{jl}^{as}(t)}{\sum_{a} I_{jl}^{as}(t) + S_{jl}(t)} \right) \left(1 - \left(\left(1 - \beta_{mf} R_{ji}^{as} \right)^{VI_{kijl}(1 - v_{ki}(t))} \left(1 - \beta_{mf} R_{ji}^{as}(1 - \zeta) \right)^{VI_{kijl}(v_{ki}(t))} \right) \right) \right) \right]
$$

Where c^*_{kijl} is the balanced partner change rate and p_{kijl} is the probability of a sexual contact between an individual of risk category *k* and age class *i* with an individual of risk category *j* and age class *l*. The terms *βmf* and *βfm* represent the per act probability of HIV transmission from an infected partner with whom receptive vaginal intercourse (β_{m} *f)* and insertive vaginal intercourse (β_{fm}) is performed, respectively. The matrix R_{ji}^{as} contains the coefficients for the relative risk of HIV

transmission associated with different stages of infection (indexed by superscript *a*), treatment (indexed by superscript *s*), or susceptibility of young women (*i*).

For the FOI between two males (Equation 3), two matrices of coefficients are introduced and they contain the relative risk estimates specific to anal intercourse. Z_{ji}^{as} is similar to R_{ji}^{as} , except that it includes relative risks that take into account the increased probability of HIV transmission for receptive anal intercourse. Similarly, the matrix L_i^{as} contains the relative risks that take into account the increased probability of HIV transmission resulting from insertive anal intercourse. It was assumed that, within each MSM partnership, half of sex acts would be performed as the receptive partner and the other half as the insertive partner. When both anal and vaginal intercourse are involved in a partnership (Equations 4 and 5), it was assumed that each sexual contact would either involve anal intercourse or vaginal intercourse - but not both.

Condom efficacy was modeled using the term ζ , which reduces the probability of HIV transmission. The proportion of sex acts protected by a condom is equal to $v_{ki}(t)$ and the number of sex acts between individuals *ki* and *jl* is *αkijl*. When both vaginal and anal sex acts are involved in a partnership, we divided these sex acts between those that are vaginal $(VI_{kijl} = \alpha_{kijl} * (1 - Pr_{\text{ActsAI}}))$ and anal $(AI_{kijl} = \alpha_{kijl} * Pr_{ActsAI})$. It should be noted that the additive scale was used to calculate the effect of multiple risk factors in matrices R_{ji}^{as} , L^{as} , and Z_{ji}^{as} . This was necessary because the multiplicative risk scale resulted, for some combination of parameters, in per act transmission probability greater than one. This suggested interaction of some risk factors on the multiplicative scale and provided evidence that the additive scale was more appropriate. When the effect was protective (reducing transmission), however, we used the multiplicative scale as not to underestimate the impact of potential interventions. This matrix was calculated as follows:

$$
R_{ij} = \begin{cases} RR_{k} = 0; \\ \text{R} = 1 + (RR_{joumgFem}) \\ \text{R} = 0; \\ \text{R} = 2; \\ \text{R} = 2; \\ \text{R} = 1 + (RR_{joumgFem}) \end{cases}
$$
\n
$$
R_{ij} = \begin{cases} \frac{1}{2} \cdot (RR_{joungFem} - 1) + (RR_{joumgFem} - 1); \\ \text{R} = 2; \\ \text
$$

Text S4. Equations for the vertical transmission of HIV.

The decision tree describing mother-to-child transmission of HIV can be translated into the following set of equations. The number of babies, born to HIV positive mothers at time *t*, who became infected is equal to $BB_{HIV}(t)$. Note that the equations below only apply for females (*k=1,2,3,4*).

$$
BB_{HIV}(t) = \sum_{i=1}^{3} \left(\Upsilon_{i}(t) \sum I_{ki}^{as}(t)\right) \begin{pmatrix} \left(\left(1-\delta_{i}(t)-\Psi(t)\right)+\delta_{i}(t)\left(1-\kappa(t)-\phi(t)\right)\right)MTCT(t)+\\ \left(\delta_{i}(t)\kappa(t)\right)PMTCT_{Px}(t)+\\ \left(\delta_{i}(t)\phi(t)\right)PMTCT_{Tx}(t)+\\ \Psi(t)ART\end{pmatrix}
$$

where $\Upsilon_i(t) = ASFR_i(t)^*RR_{Fert}$

The age-specific fertility rates of HIV positive women at time *t* (*ϒi(t)*) are used to estimate the number of expected pregnancies. All pregnancies are assumed to be singleton. The number of pregnancies from HIV positive women is then multiplied by peripartum and postpartum transmission probabilities for the different interventions and distribution of women among the different model's compartments: women not tested or that did not access antenatal care $[(1-\delta_i(t))$ $\psi(t)$ + $\delta_i(t)$ (1- $\kappa(t)$ – $\phi(t)$))MTCT, those that are tested and received ART prophylaxis $[(\delta_i(t)\kappa(t))PMTCT_{Px}(t))]$, those that are tested and received ART treatment $[(\delta_i(t)\phi(t))PMTCT_{Tx}(t))]$, and those already receiving ART treatment before becoming pregnant [*ψ(t)ART*]. The transmission probabilities are described below.

$$
MTCT(t) = A \begin{pmatrix} \left(\beta_{<200}^{0} + (1 - (1 - \beta_{BF<350}^{0})^{D} \right) \frac{\sum_{i=1}^{3} I_{ki}^{a5}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} + \\ \left(\beta_{<350}^{0} + (1 - (1 - \beta_{BF<350}^{0})^{D} \right) \frac{\sum_{i=1}^{3} I_{ki}^{a5}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} + \\ \left(\beta_{\ge350}^{0} + (1 - (1 - \beta_{BF\ge350}^{0})^{D} \right) \frac{\sum_{i=1}^{3} \sum_{s=1}^{3} I_{ki}^{a5}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} + \\ + (1 - A) \left(\beta_{<200}^{0} \frac{\sum_{i=1}^{3} I_{ki}^{a5}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} + \beta_{<350}^{0} \frac{\sum_{i=1}^{3} I_{ki}^{a4}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} + \beta_{\ge350}^{0} \frac{\sum_{i=1}^{3} \sum_{s=1}^{3} I_{ki}^{a4}(t)}{\sum_{i=1}^{3} I_{ki}^{a5}(t)} \right)
$$

The proportion of infant being breastfed is equal to *A* and the average duration of any breastfeeding is equal to *D* months. Women receiving PMTCT were assumed not to reduce their breastfeeding period, in line with qualitative data on the topic [55]. The vector β_x^0 contains the peripartum transmission probabilities in the absence of prevention interventions. Similarly, the vector β_{BFx}^0 has the monthly postpartum probability of HIV transmission from any breastfeeding in the absence of prevention interventions. Because the probability of transmission is a function of CD4 count concentrations, the *x* subscript is associated with the following categories of CD4 cell counts: less than 200 CD4 cells/ μ L ($\beta_{<200}^{0}$), between 200 and 349 CD4 cells/ μ L ($\beta_{<350}^{0}$), and >350 CD4 cells/ μ L (β _{>350}⁰). These probabilities are then added and weighted according to the distribution of women in the different stages of infection at time *t*.

$$
PMTCT_{Px}(t) = A \left(\frac{(\beta_{Px}^t + (1 - (1 - \beta_{BF<350}^t)^D)) \frac{\sum_{i=1}^3 \sum_{s=4}^5 I_{ki}^{as}(t)}{\sum_{i=1}^3 I_{ki}^{as}(t)} + (1 - A)(\beta_{Px}^t)}{(\beta_{Px}^t + (1 - (1 - \beta_{BF\ge350}^t)^D)) \frac{\sum_{i=1}^3 \sum_{s=1}^3 I_{ki}^{as}(t)}{\sum_{i=1}^3 I_{ki}^{as}(t)}} \right) + (1 - A)(\beta_{Px}^t)
$$

PMTCT interventions evolved in time and the vector β_{Px} ^{*t*} contains the peripartum probability of transmission for the type of interventions available at time *t* (see Table S4 for details). These probabilities do not depend on CD4 count concentrations. The vector β_{BFx}^t has the monthly probabilities of HIV transmission due to breastfeeding. These probabilities differ according to CD4 cell counts, except when the available intervention is the Option A/B in which case the same probabilities are used regardless of CD4 cell counts.

$$
PMTCT_{Tx}(t) = A\left(\beta_{Tx} + \left(\left(1 - \left(1 - \beta_{BFT_X}\right)^D\right)\right) + \left(1 - A\right)\left(\beta_{Tx}\right)\right)
$$

When a woman is put on lifelong ART during her pregnancy, the probability of peripartum HIV transmission is β_{Tx} . During breastfeeding, infants are still protected by their mothers' intake of ART, and the monthly probability of postpartum transmission is equal to β_{BFTx} .

$$
ART = A \left(\beta_{\scriptscriptstyle ARV} + \left(\left(1 - \left(1 - \beta_{\scriptscriptstyle BFART} \right)^D \right) \right) \right) + \left(1 - A \right) \left(\beta_{\scriptscriptstyle ART} \right)
$$

Similarly, when a woman is already on ART before getting pregnant, the probability of peripartum HIV transmission is *βARV*. Finally, the monthly probability of postpartum transmission due to breastfeeding is equal to *βBFARV*.

Text S5. Details on the estimation of historical trends in the proportion of sex acts protected by a condom, HIV testing, prevention of mother-to-child transmission, and antiretroviral therapy coverage.

Proportion of sex acts protected by a condom

Few estimates on the proportion of sex acts protected by condoms $(\nu_{ki}(t))$ were available during the 1980s. We used data from the 1980-81 World Fertility Survey (WFS) as an approximation of the baseline proportion of condom-protected sex acts [42, 43]. The next estimates available are from the early-to-mid 1990s, depending on the risk groups. The latter are considerably higher than reports found in the 1980-81 WFS. Because of the uncertainties associated with the timing of increases in condom use, the model was parameterized using a uniform prior distribution for the date condom use begun to increase between 1981 and 1990 (*DateCondk*). Independent distributions were used for the following groups: general population, FSW, and MSM. Because each estimate of condom use had its associated uncertainty, and to maintain temporal consistency, the following quantities were computed by uniformly sampling a percentile of the uniform distribution for all time points were data was available. (Figure A).

$$
v_{ki}(t) = \begin{cases} Condom_i(t) * CondPtl; & \text{for } k=(1,2,5,6,7) \\ CondomSW(t) * CondSWPtl; & \text{for } k=(3,4) \\ CondomMSM(t) * CondMSMPtl; & \text{for } k=(8,9) \end{cases}
$$

Date of condom increase during the 1980s v_{ki} (*DateCond*_k) = v_{ki} (1981)

For Anal Intercourse during Sex Work $CondAI(t) = V_4(t)^* RR_{CondAI}$ 100% -Condom_k (2000)

Figure A: Schematic representation of historical trends estimation of the proportion of sex acts protected by condoms. The stars represent data points from which survey estimates are available. These are sampled by using the same percentile (*CondPtli*) of their range. No data points are available from 1981 to 1990s and condom use during that period rose rapidly. The specific date of increase remains unknown, however. This

uncertainty was taken into account by maintaining fixed the proportion of sex acts protected by a condom until a specific date has been reached (*DateCondk*), date after which condom use increased.

Finally, a shape-preserving cubic piecewise interpolation was used to estimate the proportion of sex acts protected by condoms for the years were this information was not available (the figure above represents linear interpolation for ease of visualization). Extrapolation beyond the last available estimate (i.e., the 2011-12 Demographic and Health Survey) was achieved by assuming that this estimate would remain constant.

HIV testing

The first population-based survey that collected information on HIV testing was conducted in 2000 [45]. It only recorded if women reported having ever been tested for HIV and it was not possible to know if it was as part of antenatal care visits (ANC). The survey's results show that 3.9% and 6.0% of women had ever been tested for HIV among the 15-24 and 25-49 years old, respectively. We therefore conservatively assumed testing rates of zero for men and women before 1996. The 2005 AIDS Indicator Survey and 2011-12 Demographic and Health Survey were used to inform testing rates and proportions of pregnant women accessing ANC and being tested [5, 6]. The probabilities of being tested for HIV during the last year were converted to yearly rate using the following formula: *Rate = -ln(1-Prob)/t*. Testing rates for each risk and age categories were computed as followed for each date were data was available:

$$
\tau_{ki}^{0}(TestPtl); \qquad \text{for } k=(1,2,5,6,7) \text{ and } s=(2,3,4)
$$
\n
$$
\tau_{ki}^{0}(Symp_R R_{Test}) Test; \qquad \text{for } k=(1,2,5,6,7) \text{ and } s=5
$$
\n
$$
\tau_{ki}^{0}(FSW_R R_{Test})TestPtl; \qquad \text{for } k=(3,4) \text{ and } s=(2,3,4)
$$
\n
$$
\tau_{ki}^{0}(1+(FSW_R R_{Test}-1)+(Symp_R R_{Test}-1))TestPtl; \qquad \text{for } k=(3,4) \text{ and } s=5
$$
\n
$$
\tau_{ki}^{0}(MSM_R R_{Test})TestPtl; \qquad \text{for } k=(8,9) \text{ and } s=(2,3,4)
$$
\n
$$
\tau_{ki}^{0}(1+(MSM_R R_{Test}-1)+(Symp_R R_{Test}-1))TestPtl; \qquad \text{for } k=(8,9) \text{ and } s=5
$$

HIV testing for pregnant women as part of ANC visits were calculated as followed (this is the marginal probability of ANC attendance and of being tested for HIV):

$$
\delta_i(t) = ANC\tau_i * ANCPtl
$$

HIV testing rates and probability of being tested for pregnant women were both interpolated using a shape-preserving cubic piecewise algorithm. Because programmatic data [34-39, 47] suggested a sustained increased in HIV testing rates beyond 2012, we extrapolated the trends observed during the 2005-2012 period over the 2012-2015 period.

Prevention of mother-to-child transmission

In 2002, only 16 sites were offering prevention of mother-to-child transmission (PMTCT) interventions [56]. It is further believed that, prior to 2005, PMTCT interventions were only available in urban areas of Côte d'Ivoire [46]. Information on the historical trends in coverage of PMTCT activities were abstracted from government reports available from 2002 onwards [34-39, 47]. For each year were such data was available, we computed the following quantities:

$$
\kappa(t) = K(t)(ARTPxPt)
$$

$$
\phi(t) = \Phi(t)(ARTTxPt)
$$

Again, a shape-preserving cubic piecewise interpolation was used to estimate the trends in coverage of PMTCT activities. Extrapolation beyond the last available estimate was achieved by assuming that this estimate would remain constant.

Antiretroviral therapy

The UNAIDS initiative's pilot phase for treatment access was officially launched in Côte d'Ivoire in November of 1997 and was supposed to provide ART to 4,000 individuals [57]. In August of 1998, the Minister of Health announced at a press conference that ART would become effectively accessible at the end of that month. At that time, the only individuals benefiting from free/subsidized ART in Côte d'Ivoire were those enrolled in clinical trials. From August 1998 to March 2000, 422 patients enrolled in the Retro-CI project were able to access ART [57]. In May of 2002, it is reported that 703 patients had obtained ART in Côte d'Ivoire [57]. Ministry of Health data indicated that a total of 3,190 individuals received ART from 1998 to 2002 [56]. For the purpose of the modeling exercise, it was assumed that individuals could receive ART after 2000. Comprehensive data on the number of people receiving ART was abstracted from government reports for the years 2002 and from 2007 to 2014 [34-39, 47].

Precise quantification of ART coverage scale-up and differential recruitment into ART according to CD4 cell counts is difficult. ART eligibility criteria were expanded in 2012 to include all patients with a CD4≤350 cells/µL or with WHO stage 4 clinical disease [58] but the country has yet to adopt the new WHO guidelines [59]. Further, CD4 cell counts are not believed to have been widely available in Côte d'Ivoire [39]. A flexible approach was therefore adopted. Specifically, we assumed that ART coverage would increase at a rate corresponding to the ratio of number of individuals on ART at time *t* to the number of individuals on ART in 2015. The parameter $\sigma(t)$ is used to identify availability of ART at time *t* and linearly varied (as suggested by data on ART coverage) from 0 in 2000, to 0.018 in 2003 (2399/133143), to 1 in 2015. Individuals with low CD4 cell counts (i.e., \leq 200 CD4 cells/ μ L) could be more likely to exhibit clinical symptoms and, thereby, to be recruited on ART. A linear relation was thus assumed between CD4 count stage and recruitment rate using parameter *ι*. This parameter was given a non-informative prior, uniformly distributed between 0 and 1 so that lower CD4 cell counts had higher recruitment rates. The model was calibrated using the <200 CD4 cells/ μ L stage as the referent category. For this stage, it was assumed that the recruitment rate into ART (in 2015) would be between 0.5 and 4 years (ρ_5) . Recruitment rate was hence modeled using the following equation:

$$
\rho_s(t) = \rho_s \sigma(t)
$$

where

$$
m = ((0 - \rho_s)/9.25)t
$$

$$
\rho_4 = 3m + \rho_s
$$

$$
\rho_3 = 6m + \rho_s
$$

$$
\rho_2 = 9m + \rho_s
$$

$$
t \sim U(0,1)
$$

$$
\rho_s \sim U(0.5,4)
$$

Text S6. Statistical adjustments for the imperfect accuracy of diagnostic assays.

The sensitivity and specificity of the diagnostic assays of all HIV surveys used for model fitting were reviewed. It was found that some of those conducted prior to 1990 used an ELISA (Elavia, Diagnostic Pasteur) which has a low specificity of 89% (95% Confidence Interval (CI): 81- 96%) on African sera [60]. Because concerns regarding false positives, these surveys performed Western blots as a confirmatory assay on the positive sera. When the disease is relatively rare, however, an imperfect specificity could overestimate HIV prevalence. Further, despite being described as having high specificity, Western blots are technically challenging to perform, with results that are subjectively assessed and cumbersome to interpret, and the methodology lacks standardization [61, 62]. There is thus the possibility that suboptimal laboratory conditions and/or training of laboratory technicians could have compromised specificity of these confirmatory Western Blots.

The HIV prevalence estimates prior to 1990 were adjusted for potential misclassification using a Bayesian latent class model that accounts for the conditional dependence between the ELISA and the Western Blots [63, 64]. Since these two tests targeted some of the same antibodies, high correlation between the two tests are expected [65]. The model takes the following form:

Likelihood:
\n
$$
y_i \sim binomial(p_i, N_i)
$$
\n
$$
p_i = \pi_i (Se_i Se_2 + \gamma_{se}) + (1 - \pi_i) \Big((1 - Sp_1)(1 - Sp_2) + \gamma_{sp} \Big)
$$

where y_i is the number of individuals with reactive HIV assays in population *i*; N_i is the total number of individuals tested; p_i is the probability of having a reactive assay; π_i is the true (unobserved) probability of being HIV positive; $Se₁$ and $Se₂$ are the sensitivities of the ELISA and Western Blots, respectively; $Sp₁$ and $Sp₂$ are the specificities of the ELISA and Western Blots, respectively; and *γSe* and *γSp* are the conditional covariance for sensitivity and specificity, respectively. The model specification is completed by the following priors:

$$
\pi_{i} \sim \text{dbeta}(a, b) \quad \text{[Pior varies according to population]}
$$
\n
$$
Se_{1} \sim \text{dbeta}(286.83, 1.03) \quad \text{[Corresponds to a 95%CI: 98.7-99.99%]}
$$
\n
$$
Se_{2} \sim \text{dbeta}(94.46, 0.81) \quad \text{[Corresponds to a 95%CI: 96.6-99.99%]}
$$
\n
$$
Sp_{1} \sim \text{dbeta}(54.1, 6.18) \quad \text{[Corresponds to a 95%CI: 81.0-96.0%]}
$$
\n
$$
Sp_{2} \sim \text{dbeta}(154.36, 1.69) \quad \text{[Corresponds to a 95%CI: 96.8-99.9%]}
$$
\n
$$
\rho_{Se} \sim \text{dbeta}(5.97, 1.26) \quad \text{[Corresponds to a 95%CI: 50-99%]}
$$
\n
$$
\rho_{Sp} \sim \text{dbeta}(5.97, 1.26) \quad \text{[Corresponds to a 95%CI: 50-99%]}
$$
\n
$$
\lambda_{Se} = \min(Se_{1}, Se_{2}) - Se_{1}Se_{2}
$$
\n
$$
\lambda_{Sp} = \min(Sp_{1}, Sp_{2}) - Sp_{1}Sp_{2}
$$
\n
$$
\gamma_{Se} = \rho_{Se} \lambda_{Se}
$$
\n
$$
\gamma_{Sp} = \rho_{Sp} \lambda_{Sp}
$$

The priors for the true (unobserved) HIV prevalence varies according to the population under consideration and were specified using beta distributions. For the general population, we assumed that in 1989 the 95% CI of HIV prevalence would lie in the 0.5% to 7.5% interval. A beta distribution was used to model this prior by matching the percentile of this distribution to the beta distribution. For female sex workers surveyed in 1986-1990, we assumed that the 95% CI of HIV

prevalence would be in the 10% to 90% range. The priors for the sensitivity and specificity of the first assay (Elavia) correspond to the sensitivity and specificity estimated using African sera by Van Kerckhoven [60]. Information on the sensitivity and specificity of the Western Blots performed in Côte d'Ivoire prior to 1991 could not be found. Further, the sensitivity and specificity of the Western Blots vary according to laboratory conditions and the technicians' level of expertise. To be conservative, we used the minimum (discarding one extreme value for sensitivity and two extreme values for specificity) and maximum sensitivity and specificity of now discontinued Western blots reported by the World Health Organization [66] as the lower and upper bound of a 95% confidence intervals. Finally, the conditional covariances of the sensitivities and specificities are expressed as a proportion (ρ_{Se} and ρ_{Sp}) of the maximum degree of dependence (λ_{Se} and λ_{Sp}). Because both ELISA and Western Blots targets similar antibodies for reactivity, we assumed a high degree of dependence between the two tests, where the 95% CI for the proportion of dependence lies in the 50-99% interval.

The model was fitted using Markov chain Monte Carlo simulations and the posterior distributions of the parameters were estimated using JAGS [67, 68]. Inferences are based on three chains of 50,000 iterations, after an initial burn-in of 10,000 iterations (totaling 150,000 iterations). Medians and 95% credible intervals are reported as the summary estimates. The results of the unadjusted (frequentist) and adjusted for imperfect sensitivity and specificity are presented in Table 16. When prevalence of HIV is low, the imperfect specificity has a noticeable impact on prevalence estimates. Hence, these adjusted estimates will be used for model fitting.

Table A. Unadjusted and adjusted HIV prevalence estimates for studies conducted prior to 1991 in Côte d'Ivoire.

*Urban and rural areas were combined to provide nationally representative estimate of HIV prevalence in 1989. This was achieved by assuming that 39% of the population lived in urban areas in 1989 [69]. The numerator and denominator of the combined category were obtained by substituting the original sample size for an effective sample size adjusted for the correct number of degrees of freedom [70]. This was achieved by having calculated the average design effect of the cluster-sampling design of the 2005 AIS and 2011-12 DHS (for rural and urban area separately).

Figure S4. Observed HIV prevalence, to which the model was calibrated to (mean and 95% confidence intervals), and predicted HIV prevalence in the general population of Côte d'Ivoire, stratified by age and sex. FSW are not assumed to be sampled in the household-based HIV prevalence surveys and were excluded in calculating model-based overall prevalence estimates. The 1989 survey grouped together individuals aged 25 to 54 years of age. For the 50-59 age groups, the 1989 survey lumped individuals aged 55 to 64 together. The 2005 AIDS Indicator Survey and the 2012-12 Demographic and Health survey did not sample individuals aged above 50 years (except in 2011-12 for men) and prevalence in the 45-49 years old was used as a proxy. Incremental Mixture Importance Sampling (IMIS) resulted in 41,146 unique sets of parameters labelled samples from posterior (grey curves). One thousand of these curves were resampled with probability proportional to their importance weights to derive the median (red) and 95% credible intervals (shaded blue area). The specific references for the data points plotted on the figures can be found in Table S9.

Figure S5. Observed HIV prevalence (points, mean and 95% confidence intervals) and predicted (lines/shaded area) HIV prevalence among men who have sex with men (MSM) in Côte d'Ivoire.

Figure S6. Model predicted ART coverage in Côte d'Ivoire with ART coverage data to which the model was calibrated to. The plotted points refer to UNAIDS estimates of ART coverage which are themselves based on data from the *Direction de l'information, de la planification et de l'évaluation* [34-39, 47] (see Table S9).

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