MGH
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DRIVE CERVICAL (Data-driven Recommendations for Interventions against Viral infection)

Massachusetts General Hospital

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Version Table

Version	Date	Notes
1.0.00	2025-09-30	Major update
HI.001.03202020.9999	2020-03-20	Historical release

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Readers Guide

Reader's Guide

Core Profile Documentation

These topics will provide an overview of the model without the burden of detail. Each can be read in about 5-10 minutes. Each contains links to more detailed information if required.

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Model Purpose
This document describes the primary purpose of the model.

Model Overview
This document describes the primary aims and general purposes of this modeling effort.

Assumption Overview
An overview of the basic assumptions inherent in this model.

Parameter Overview
Describes the basic parameter set used to inform the model, more detailed information is available for each specific parameter.

Component Overview
A description of the basic computational building blocks (components) of the model.

Output Overview
Definitions and methodologies for the basic model outputs.

Results Overview
A guide to the results obtained from the model.

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Model Purpose

Summary

The purpose of this mathematical model is to study the impact of HPV vaccination, cervical cancer screening and treatment, accounting for HIV infection dynamics including HIV disease progression by CD4 count and antiretroviral therapy (ART) on cervical cancer outcomes. We created a model that simulates heterosexual transmission of oncogenic HPV (high-risk HPV;hrHPV) and heterosexual HIV transmission. The model is parameterized to KwaZulu-Natal, South Africa (KZN), a region with high HIV prevalence.

Purpose

The model reproduces population-level HIV and HPV disease dynamics and stratifies the population by age, gender, and sexual risk. We model HIV progression by CD4+ T-cell (CD4) count and HIV RNA concentration (viral load). The impact of ART scale-up targeted to HIV-positive persons is also modelled starting in 2004.

HPV progression is modelled by progression through a precancer pathway that leads to cervical cancer. The interaction between HPV and HIV in coinfected subpopulations is modelled by accounting for the increased risk of HPV transmission to an HIV-positive person and the accelerated disease progression of cervical lesions in HIV-positive women.

Using demographic data from the population under study, the model is calibrated to recapitulate observed patterns of HIV and HPV disease. The population-level impact of HPV vaccination is then assessed by comparing health outcomes in vaccine vs. non-vaccine scenarios.

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Summary

There are three key components to the model: 1) Dynamic HPV and HIV transmission, 2) HIV progression and ART scale up, and 3) HPV-related pre-cancer/cancer progression.

Purpose

Our dynamic transmission compartmental model examines the impact of HPV vaccination on HIV-positive and HIV-negative women in a high HIV-prevalence setting. Model parameters on transition rates for HIV and HPV disease states were derived by synthesizing relevant findings in the literature. Parameter calibration was performed to enhance the model's ability to reflect observed disease patterns.

Background

HPV and HIV infections can interact to increase cervical cancer (CC) risk. The 9-valent HPV (9vHPV) vaccine has high demonstrated effectiveness against HPV types causing 90% of CC [1]. Additionally, one dose of the 9vHPV vaccine has the potential to achieve greater coverage at lower costs than a two-dose schedule. However, the potential impact of single-dose 9vHPV vaccine accounting for HPV-HIV interactions has not been estimated. This model adapts a previously published dynamic compartmental HIV transmission model.

Reference List

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References

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Assumption Overview

Summary

This section describes the basic assumptions made by MGH's HIV-HPV coinfection model.

Background

Compartmental models divide the population under study into various compartments that are characterized by demographic and health-related features. In the present model, this is achieved by stratifying groups according to HIV disease state, HIV Viral Load, Vaccine type HPV disease state, non-vaccine disease state, cervical cancer or hysterectomy status, vaccination and screening history, gender, age, and risk based on sexual activity.

Maximum likelihood-based calibration is used to calibrate model parameters to various data sources and to infer the value of parameters that cannot be obtained through direct observation.

Assumption Listing

In compartmental models, it is assumed that the members within each compartment are homogeneous in nature. As such, the model is not designed to answer questions pertaining to individual-level interventions or health outcomes. However, in the absence of granular individual-level data, the assumption of homogeneity provides for an economical model that can be used to determine the impact of population-level interventions and health outcomes.

Susceptible females can acquire high-risk (HR) HPV infection from a male sexual partner and progress to precancerous lesions categorized as cervical intraepithelial neoplasia, grades 1, 2, or 3 (CIN 1, 2, or 3). HPV infection and CIN1, CIN2, and CIN3 lesions can regress to normal over time and females with CIN3 can develop cervical cancer (categorized in 3 stages: local, regional, and distant). Given the strong connection between HPV infection and cervical cancer incidence, we assume that the pathogenesis of all cervical cancers begins with HPV infection. We assume females who clear their HPV infection can develop low-level natural immunity while males who clear HPV infection do not develop natural immunity. The model estimates the force of HPV infection as a function of sexual mixing (by age and sexual activity), proportion of HPV infected individuals of the opposite sex, and HPV transmission probability, which depends on HIV status and CD4 count if HIV-positive. Once females are infected, the probability of HPV disease progression is governed by age, HIV-status, and CD4 count if infected with HIV.

HIV-positive females have a higher risk of HPV acquisition and CIN1-2 progression and a lower probability of disease regression and infection clearance. HPV disease progression is inversely related to CD4 count; women at the lowest category CD4 counts are least likely to clear and more likely to experience disease progression.

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Parameter Overview

Summary

This section contains the parameters used to inform the natural history model.

Background

The MGH natural history model is based on data published in the literature. HIV transition rates by CD4 count and viral load level are informed by previous clinical studies. Transition rates for HPV infection and CIN were also informed by existing literature. The transition from CIN3 to cervical cancer was inferred through calibration as this quantity could not be obtained through direct observation.

Parameter Listing Overview

The parameters used to inform the model follow below:

Population Demographics

- Population Size – based on the United Nations Population estimates, and KZN population estimates.
- South Africa's age-specific mortality before 1950, values are based on United Nations 2019 Population prospects. After the start of the generalized HIV epidemic, values are based on IHME Global Burden of Disease Study[1,2].
- Fertility rates are based on the United Nations Population Division total fertility estimates, and KZN population estimates. Future simulations model fertility rates from 2020 to 2035 to match the projected United Nations Population estimates for population size, age distribution and fertility[1,3,4,5].
- Fertility rate by age and HIV status. Females on ART are assumed to have equal fertility to HIV-negative females[6,7].

Sexual History and Sexual Mixing

- Sexual risk distribution by age and sex. Values are based on Africa Centre cohort partnership data from KwaZulu Natal, South Africa[8]. Risk distribution derived from male partner data is used for both men and women.
- Annual number of sexual partnerships by age, gender, and sexual risk group. Values are based on Africa Centre cohort partnership data from KwaZulu Natal, South Africa[8].
- The number of coital acts per partnership by sex, age, and sexual risk group. Values are calibrated to fit age-specific HIV and HPV prevalence data.
- Sexual mixing by age and sexual risk group. The mixing parameter varies from random to assortative, calibrated to fit age-specific HIV incidence and prevalence data[9].

HIV

- HIV prevalence and incidence by gender and age over time in KZN. Values are based on Africa Centre cohort partnership data from KwaZulu Natal, South Africa[8,10].
- HIV-associated mortality. Values are estimates are from observational studies of untreated HIV-positive persons. Persons age 0 to 4 and older than 50 are assumed to have greater mortality as observed[11-14].
- Risk multipliers for HIV transmission by viral load[15-19].
- The duration of time in each CD4 and viral load stage by sex and age[17,20,21,22].
- Proportion of births from HIV-positive females that results in mother-to-child transmission. The rate decreases linearly from 2004 to 2005 and from 2005 to 2008[9,23,24].
- Proportion of persons living with HIV on ART and virally suppressed coverage over time[25].

HPV and Cervical Cancer

- HPV prevalence in women and men in South Africa. Mbulawa et al[26-28].
- HPV prevalence in women without CIN2/3[29].
- CIN2/3 prevalence by HIV status[27,28].

- Cervical cancer mortality by disease stage, CD4 count and HIV status[30,31].
- Cervical cancer incidence based on 2018 GLOBOCAN estimates. Values are adjusted to in KZN rather than South Africa nationally by age to take into account higher HIV prevalence in KZN[32].

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Component Overview

Summary

This section describes the components of our natural history model.

Overview

There are three key components to the model: 1) Dynamic HPV and HIV transmission, 2) HIV progression and ART scale up, and 3) HPV-related pre-cancer/cancer progression.

Component Listing

Dynamic HPV and HIV transmission

Our model simulates the transmission of HPV and HIV across the population, which is divided into various compartments based on health states (ex. HPV-Infected, HIV-positive, CD4 count etc.) and demographic factors (age, gender, sexual risk level). The rate of infection that a susceptible compartment is subject to depends on a) the mixing patterns corresponding to the gender, age, and sexual risk level used to describe the compartment, b) the prevalence of infection in other compartments that interact with the susceptible compartment.

HIV progression and ART scale up

HIV progression is simulated by modelling the rate of progression through HIV disease states as described by CD4 count and viral load level. ART scale-up is modelled by representing the rate of ART uptake as a function of time and CD4 count to reflect the reported clinical criteria for ART uptake. In the model, ART uptake reduces the probability of HIV transmission and attenuates a HIV-positive person's rate of progression through the HPV and cervical pre-cancer pathway.

HPV-related pre-cancer/cancer progression

HPV progression is simulated by modelling the rate of progression through HPV disease states. The HPV disease pathway consists of three cervical pre-cancer lesion stages: CIN1, CIN2, and CIN3. The transitions between these stages are modelled as a reversible process to reflect the possibility of spontaneous pre-cancer lesion clearance. Meanwhile, the transition from CIN3 to cervical cancer (CC) is modelled as an irreversible process. The CC associated mortality rate increases with the severity of the cancer, which is described by local, regional, and distant cervical cancer stages in the model.

Further details about the model can be found below:

HIV Natural History

The natural history of HIV infection is modeled in stages defined by CD4 count and viral load as shown in Figure S1. When a person becomes HIV-infected, s/he enters the acute stage characterized by a short duration and high probability of HIV transmission. The person then progresses through stages of CD4 count and viral load at rates ω^d and l^d, respectively, where d represents the current HIV disease state defined by CD4 count and v represents the current viral load. Transition rates are based on literature describing the average duration in each stage by gender and age[1-4]. The average life expectancy from infection to death for untreated persons is 10.7 years.

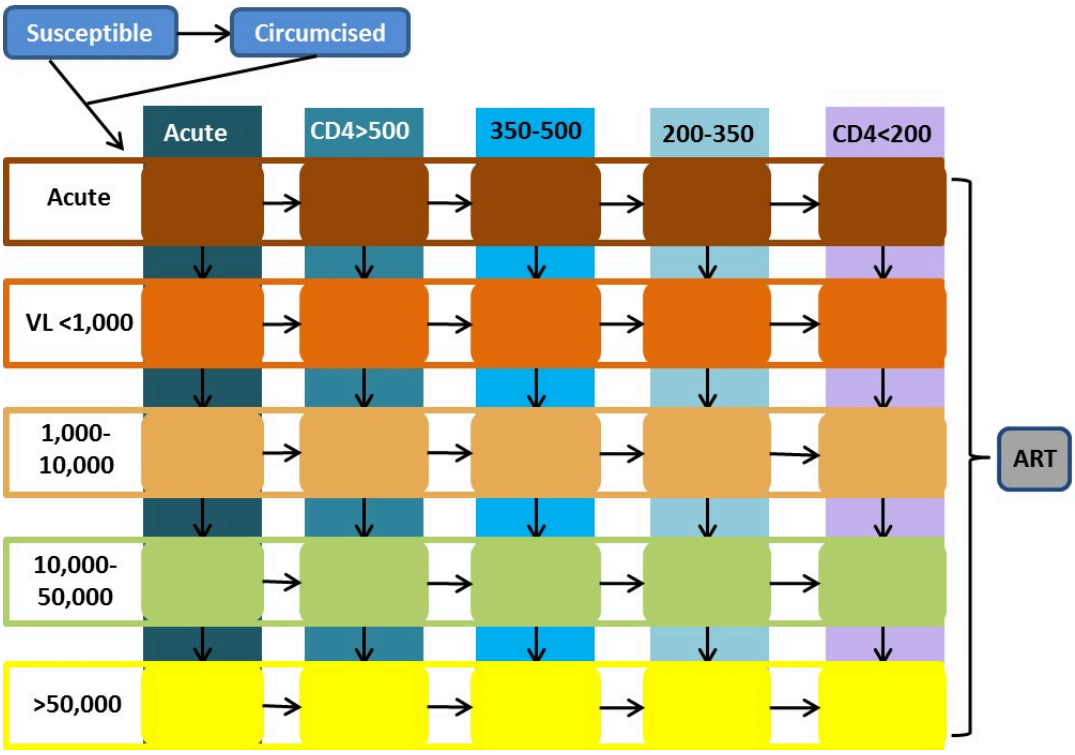

Figure S1. Model transition diagram. A diagram of the natural history of HIV infection. All movement is in one direction except for enrollment in and dropout from interventions from ART.

Ordinary Differential Equations

Throughout each simulation, we track a population demographics in five-year age-groups and the number of individuals with infection, with progressed disease or with preventative or therapeutic treatment capturing vertical transmission and aging. The system of ODE's describes the states $X_{g,a,r}^{d,v,h,s,x,p}(t)$ with the following indices:

- d refers to HIV disease state defined by CD4 cell count, circumcision status, and ART status
  $d = 1$ for HIV-negative, uncircumcised; $d = 2$ for HIV-negative, circumcised; $d = 3$ for HIV-positive, acute infection; $d = 4$ for HIV-positive, CD4 > 500 cells/μL; $d = 5$ for HIV-positive, CD4 350–500 cells/μL; $d = 6$ for HIV-positive, CD4 200-350 cells/μL; $d = 7$ for HIV-positive, CD4 200 £ cells/μL; $d = 8$ for HIV-positive, on ART.

- v refers to disease state defined by HIV viral load
  $v = 1$ if $(2 < d < 8)$, Acute infection, if $(d = 1, 2)$, HIV-negative: $VL = 0$; $v = 2$ for Asymptomatic: $VL = 3.0 - 4.5log10$; $v = 3$ for Pre-AIDS symptomatic: $VL = 4.0 - 5.5log10$; $v = 4$ for AIDS: $= 5.5 - 7.0log10$; $v = 5$ for Late-stage; $v = 6$ for on ART and virally suppressed: $VL = 0.0$

- h refers to vaccine-type HPV precancer or disease state
  $h = 1$ for Susceptible; $h = 2$ for Infected; $h = 3$ for CIN1; $h = 4$ for CIN2; $h = 5$ for CIN3; $h = 6$ for Cervical Cancer or hysterectomy; $h = 7$ for Immune

- s refers to non-vaccine-type HPV precancer or disease state
  $s = 1$ for Susceptible; $s = 2$ for Infected; $s = 3$ for CIN1; $s = 4$ for CIN2; $s = 5$ for CIN3; $s = 6$ for Cervical Cancer or hysterectomy; $s = 7$ for Immune

- x refers to cervical cancer or hysterectomy status
  $x = 1$ if $(h = 6$ or $s = 6)$, Cervical cancer, local, undiagnosed; else, no cancer or hysterectomy; $x = 2$ for Cervical cancer, regional, undiagnosed; $x = 3$ for Cervical cancer, distant, undiagnosed; $x = 4$ for Cervical cancer, local, diagnosed & untreated; $x = 5$ for Cervical cancer, regional, diagnosed & untreated; $x = 6$ for Cervical cancer, distant, diagnosed & untreated; $x = 7$ for Cervical cancer, local,

treated; $x = 8$ for Cervical cancer, regional, treated; $x = 9$ for Cervical cancer, distant, treated; $x = 10$ for Hysterectomy

- p refers to vaccination and screening history
  $p = 1$ for Non-vaccinated, non-screened; $p = 2$ for Vaccinated; $p = 3$ for Screened; $p = 4$ for Vaccinated and screened
- g refers to gender
  $g = 1$ for Men; $g = 2$ for Women
- a refers to age
  $a = 1$ for ages 0-4; $a = 2$ for ages 5-9; $a = 3$ for ages 10-14 ...; $a = 16$ for ages 75-79
- r refers to sexual risk group defined by number of sexual partnerships per year
  $r = 1$ for low risk; $r = 2$ for moderate risk; $r = 3$ for high risk

We calculate changes in HIV stages defined by CD4 count, viral load, and treatment status. The HIV-negative population can acquire HIV after sexual debut with a force of infection that is reduced by circumcision among men and condom use by either gender. We only track circumcision among HIV-negative men. Individuals with HIV infection experience HIV-associated mortality, CD4 and viral load stage progression, and ART initiation and discontinuation. CD4 and viral load stage are not tracked among persons on treatment. The ODEs for the eight HIV disease states are:

$$\frac{dX^{1,1,h,s,x,p}_{g,a,r}(t)}{dt} = -\left(\psi_{\text{HIV}g} \cdot \lambda_{\text{HIV}g,a,r}(t) + P_{g,a}(t)\right) X^{1,1,h,s,x,p}_{g,a,r}(t)$$

HIV-negative, circumcised

$$\frac{dX^{2,1,h,s,x,p}_{g,a,r}(t)}{dt} = P_{g,a}(t) \cdot X^{1,1,h,s,x,p}_{g,a,r}(t) - \left(\psi_{\text{HIV}g} \cdot \rho_{\text{HIV}g} \cdot \lambda_{\text{HIV}g,a,r}(t)\right) X^{2,1,h,s,x,p}_{g,a,r}(t)$$

HIV-positive, acute infection

$$\begin{aligned}\frac{dX^{3,1,h,s,x,p}_{g,a,r}(t)}{dt} = \; & \psi_{\text{HIV}g} \cdot \lambda_{\text{HIV}g,a,r}(t) \cdot X^{1,1,h,s,x,p}_{g,a,r}(t) + \psi_{\text{HIV}g} \cdot \rho_{\text{HIV}g} \cdot \lambda_{\text{HIV}g,a,r}(t) \\ & \cdot X^{2,1,h,s,x,p}_{g,a,r}(t) + \sigma^{3,1}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t) \\ & - \left(\mu^{3}_{\text{HIV}g,a} + \omega^{3} + A^{3}_{g,a}(t)\right) X^{3,1,h,s,x,p}_{g,a,r}(t)\end{aligned}$$

HIV-positive, CD4 > 500 cells/μL

$$\begin{aligned}\frac{dX^{4,v,h,s,x,p}_{g,a,r}(t)}{dt} = & \; \omega^{3} \cdot X^{3,v,h,s,x,p}_{g,a,r}(t) \\ & + l^{v-1} \cdot X^{4,v-1,h,s,x,p}_{g,a,r}(t) \\ & + \sigma^{4,v}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t) \\ & - \left(\mu^{4}_{\text{HIV}g,a} + \omega^{4} + l^{v} + A^{4}_{g,a}(t)\right) X^{4,v,h,s,x,p}_{g,a,r}(t)\end{aligned}$$

HIV-positive, CD4 350-500 cells/μL

$$\begin{aligned}\frac{dX^{5,v,h,s,x,p}_{g,a,r}(t)}{dt} = & \; \omega^{4} \cdot X^{4,v,h,s,x,p}_{g,a,r}(t) \\ & + l^{v-1} \cdot X^{5,v-1,h,s,x,p}_{g,a,r}(t) \\ & + \sigma^{5,v}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t) \\ & - \left(\mu^{5}_{\text{HIV},g,a} + \omega^{5} + l^{v} + A^{5}_{g,a}(t)\right) X^{5,v,h,s,x,p}_{g,a,r}(t)\end{aligned}$$

HIV-positive, CD4 200-350 cells/μL

$$\begin{aligned}\frac{dX^{6,v,h,s,x,p}_{g,a,r}(t)}{dt} &= \omega^5 \cdot X^{5,v,h,s,x,p}_{g,a,r}(t) \\ &+ l^{v-1} \cdot X^{6,v-1,h,s,x,p}_{g,a,r}(t) \\ &+ \sigma^{6,v}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t) \\ &- \left(\mu^6_{\text{HIV},g,a} + \omega^6 + l^v + A^6_{g,a}(t)\right) X^{6,v,h,s,x,p}_{g,a,r}(t)\end{aligned}$$

HIV-positive, CD4 ≤ 200 cells/μL

$$\begin{aligned}\frac{dX^{7,v,h,s,x,p}_{g,a,r}(t)}{dt} &= \omega^6 \cdot X^{6,v,h,s,x,p}_{g,a,r}(t) \\ &+ l^{v-1} \cdot X^{7,v-1,h,s,x,p}_{g,a,r}(t) \\ &+ \sigma^{7,v}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t) \\ &- \left(\mu^7_{\text{HIV},g,a} + \omega^7 + l^v + A^7_{g,a}(t)\right) X^{7,v,h,s,x,p}_{g,a,r}(t)\end{aligned}$$

HIV-positive, on ART

$$\frac{dX^{8,6,h,s,x,p}_{g,a,r}(t)}{dt} = \sum_{d=3}^{7}\sum_{v=1}^{5}\left(A^d_{g,a}(t) \cdot X^{d,v,h,s,x,p}_{g,a,r}(t) - \sigma^{d,v}_{g,a,r}(t) \cdot X^{8,6,h,s,x,p}_{g,a,r}(t)\right)$$

The equation variables are:

Variable	Description
$\mu\_HIV^d_{g,a}$	Annual HIV-associated mortality rate by gender g, age a, and HIV disease stage d for $3 \leq d \leq 8$.
$\lambda\_HIV_{g,a,r}(t)$	Force of HIV infection for HIV-negative persons by gender g, age a, and risk r.
ρ_HIV_g	Reduction in HIV acquisition due to circumcision by gender. Only men receive circumcision ($\rho\_HIV_2 = 1$).
ψ_HIV_g	Reduction in HIV acquisition due to population-level condom use by gender.
ω^d	The rate of progressing from HIV stage d to $d+1$, for $3 \leq d \leq 7$.
l^d	The rate of progressing from viral load stage v to $v+1$, for $1 \leq v \leq 5$.
$P_{g,a}(t)$	The proportion of HIV-negative persons of gender g and age a that are circumcised. Only men receive circumcision ($P_{2,a}(t) = 0$).
$A^d_{g,a}(t)$	The proportion of persons living with HIV of disease stage d, gender g, and age a that initiate ART.
$\sigma^{d,v}_{g,a,r}(t)$	The proportion of persons who discontinue ART based on the recent distribution of persons initiating ART by gender g, age a, risk r, disease d, and viral v.

HPV Natural History

We calculate changes in HPV status and precancer or disease stage for women without hysterectomy ($1 \leq x \leq 9$). We track vaccine-type and non-vaccine type HPV independently, but only count cancer incidence for the first infection to progress to local cervical cancer. CIN1,2,3 can regress, and HPV infection can clear naturally. Women who clear HPV temporarily develop partial natural immunity against reinfection with the same HPV type group while men who clear HPV do not develop natural immunity. Persons susceptible to type-specific HPV infection or with temporary natural immunity can acquire HPV after sexual debut. Individuals with HIV experience higher rates of HPV acquisition and disease progression, and lower rates of HPV clearance, immunity waning, and disease regression. Cervical cancer mortality varies by cancer stage, whether it is treated, and HIV status, and affects individuals regardless of type-specific etiology. We assume that the nonavalent HPV vaccine provides lifelong protection against vaccine-type HPV and no protection against non-vaccine-type HPV. We assume the vaccine is ineffective for persons with current vaccine-type HPV infection, and the equations therefore only reflect vaccination of persons susceptible or immune to vaccine-type HPV. Vaccination does not depend on non-vaccine-type HPV infection status. Although not shown in the equations below, persons are screened according to their age a and lose their screened status upon aging out of the screened age group.

The ODEs for vaccine-type HPV and precancer equations are:

Men, susceptible

$$\frac{dX_{1,a,r}^{d,v,1,s,1,1}(t)}{dt} = l_1 \cdot \zeta_v^{d,2,1} \cdot k_{v,1,a}^{2,1} \cdot X_{1,a,r}^{d,v,2,s,1,1}(t) - \left(\xi_d \cdot \psi_{\text{HPV},1} \cdot \lambda_{v\text{HPV},1,a,r}(t) + V_{1,a}^d\right) X_{1,a,r}^{d,v,1,s,1,1}(t)$$

Men, HPV-infected

$$\frac{dX_{1,a,r}^{d,v,2,s,1,1}(t)}{dt} = \xi_d \cdot \psi_{\text{HPV},1} \cdot \lambda_{v\text{HPV},1,a,r}(t) \cdot X_{1,a,r}^{d,v,1,s,1,1}(t) - l_1 \cdot ж_v^{d,2,1} \cdot k_{v,1,a}^{2,1} \cdot X_{1,a,r}^{d,v,2,s,1,1}(t)$$

Men, susceptible, vaccinated

$$\frac{dX_{1,a,r}^{d,v,1,s,1,2}(t)}{dt} = V_{1,a}^d \cdot X_{1,a,r}^{d,v,1,s,1,1}(t) + l_1 \cdot \zeta_v^{d,2,1} \cdot k_{v,1,a}^{2,1} \cdot X_{1,a,r}^{d,v,2,s,1,2}(t) - \phi_a \cdot ж_d \cdot \psi_{HPV1} \cdot \lambda_{v\text{HPV},1,a,r}(t) \cdot X_{1,a,r}^{d,v,1,s,1,2}(t)$$

Men, HPV-infected, vaccinated

$$\frac{dX_{1,a,r}^{d,v,2,s,1,2}(t)}{dt} = \phi_a \cdot ж_d \cdot \psi_{\text{HPV},1} \cdot \lambda_{v\text{HPV1},a,r}(t) \cdot X_{1,a,r}^{d,v,1,s,1,2}(t) - l_1 \cdot \zeta_v^{d,2,1} \cdot k_{v,1,a}^{2,1} \cdot X_{1,a,r}^{d,v,2,s,1,2}(t)$$

Women, susceptible

Untreated cervical cancer health states

$$\frac{dX_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[1,3]}(t)}{dt} = \zeta_v^{d,7,1} \cdot r_2 \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t) - \left(ж_d \cdot \psi_{\text{HPV}2} \cdot \lambda_{v\text{HPV}2,a,r}(t) + V_{2,a}^d + \mu_{\text{utHPV}2}^{d,1,s,[1,2,3,4,5,6]}\right) X_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[1,3]}(t)$$

Treated cervical cancer health states

$$\frac{dX_{2,a,r}^{d,v,1,s,[7,8,9],[1,3]}(t)}{dt} = \zeta_v^{d,7,1} \cdot r_2 \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t) - \left(\xi_d \cdot ж_{\text{HPV}2} \cdot \lambda_{v\text{HPV},2,a,r}(t) + V_{2,a}^d + \mu_{\text{tHPV},2}^{d,1,s,[7,8,9]}\right) X_{2,a,r}^{d,v,1,s,[7,8,9],[1,3]}(t)$$

Women, immune

Untreated cervical cancer health states

$$\frac{dX_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t)}{dt} = V_{2,a}^d \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t) + \zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[1,3]}(t) - \left(\zeta_v^{d,7,1} \cdot k_{v,2,a}^{7,1} + \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV},2} \cdot \lambda_{v\text{HPV},2,a,r}(t) + V_{2,a}^d + \mu_{\text{utHPV},2}^{d,7,s,[1,2,3,4,5,6]}\right) X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t)$$

Treated cervical cancer health states

$$\frac{dX_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t)}{dt} = V_{2,a}^d \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t) + \zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],[1,3]}(t) - \left(\zeta_v^{d,7,1} \cdot k_{v,2,a}^{7,1} + \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV}2} \cdot \lambda_{v\text{HPV},2,a,r}(t) + V_{2,a}^d + \mu_{\text{tHPV},2}^{d,7,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t)$$

Women, HPV-infected

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[1,3]}(t)}{dt} = \; & \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} \cdot X_{2,a,r}^{d,v,3,s,[1,2,3,4,5,6],[1,3]}(t) \\ & + ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[1,3]}(t) \\ & + \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t) \\ & - \left(\zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} + \mu_{\text{utHPV2}}^{d,2,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[1,3]}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,2,s,[7,8,9],[1,3]}(t)}{dt} = \; & \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} \cdot X_{2,a,r}^{d,v,3,s,[7,8,9],[1,3]}(t) \\ & + ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,1,s,[7,8,9],[1,3]}(t) \\ & + \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t) \\ & - \left(\zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} + \mu_{t\text{HPV2}}^{d,2,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],[1,3]}(t)\end{aligned}$$

Women, susceptible, vaccinated

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[2,4]}(t)}{dt} = \; & \zeta_v^{d,7,1} \cdot r_2 \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[2,4]}(t) \\ & + V_{2,a}^{d} \cdot X_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[1,3]}(t) \\ & - \left(\phi_a \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) + \mu_{\text{utHPV2}}^{d,1,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,1,s,[1,2,3,4,5,6],[2,4]}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,1,s,[7,8,9],[2,4]}(t)}{dt} = \; & \zeta_v^{d,7,1} \cdot r_2 \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[2,4]}(t) \\ & + V_{2,a}^{d} \cdot X_{2,a,r}^{d,v,1,s,[7,8,9],[1,3]}(t) \\ & - \left(\phi_a \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) + \mu_{t\text{HPV2}}^{d,1,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,1,s,[7,8,9],[2,4]}(t)\end{aligned}$$

Women, immune, vaccinated

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[2,4]}(t)}{dt} = \; & \zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[2,4]}(t) \\ & + V_{2,a}^{d} \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[1,3]}(t) \\ & - \left(\zeta_v^{d,7,1} \cdot k_{v,2,a}^{7,1} + \phi_a \cdot \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \right. \\ & \left. + \mu_{\text{utHPV2}}^{d,7,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[2,4]}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,7,s,[7,8,9],[2,4]}(t)}{dt} = \; & \zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],[2,4]}(t) \\ & + V_{2,a}^{d} \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[1,3]}(t) \\ & - \left(\zeta_v^{d,7,1} \cdot k_{v,2,a}^{7,1} + \phi_a \cdot \xi_{2,a} \cdot ж_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \right. \\ & \left. + \mu_{t\text{HPV2}}^{d,7,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[2,4]}(t)\end{aligned}$$

Women, HPV-infected, vaccinated

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[2,4]}(t)}{dt} = & \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} \cdot X_{2,a,r}^{d,v,3,s,[1,2,3,4,5,6],[2,4]}(t) \\ & + \phi_a \cdot \text{ж}_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[2,4]}(t) \\ & + \phi_a \cdot \xi_{2,a} \cdot \text{ж}_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,7,s,[1,2,3,4,5,6],[2,4]}(t) \\ & - \left(\zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} + \mu_{\text{utHPV2}}^{d,2,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],[2,4]}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,2,s,[7,8,9],[2,4]}(t)}{dt} = & \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} \cdot X_{2,a,r}^{d,v,3,s,[7,8,9],[2,4]}(t) \\ & + \phi_a \cdot \text{ж}_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],[2,4]}(t) \\ & + \phi_a \cdot \xi_{2,a} \cdot \text{ж}_d \cdot \psi_{\text{HPV2}} \cdot \lambda_{v\text{HPV},2,a,r}(t) \cdot X_{2,a,r}^{d,v,7,s,[7,8,9],[2,4]}(t) \\ & - \left(\zeta_v^{d,2,7} \cdot k_{v,2,a}^{2,7} + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} + \mu_{t\text{HPV2}}^{d,2,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],[2,4]}(t)\end{aligned}$$

Women, CIN1

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,3,s,[1,2,3,4,5,6],p}(t)}{dt} = & \zeta_v^{d,4,3} \cdot k_{v,2,a}^{4,3} \cdot X_{2,a,r}^{d,v,4,s,[1,2,3,4,5,6],p}(t) \\ & + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} \cdot X_{2,a,r}^{d,v,2,s,[1,2,3,4,5,6],p}(t) \\ & - \left(\zeta_v^{d,3,4} \cdot k_{v,2,a}^{3,4} + \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} + \mu_{\text{utHPV2}}^{d,3,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,3,s,[1,2,3,4,5,6],p}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,3,s,[7,8,9],p}(t)}{dt} = & \zeta_v^{d,4,3} \cdot k_{v,2,a}^{4,3} \cdot X_{2,a,r}^{d,v,4,s,[7,8,9],p}(t) \\ & + \zeta_v^{d,2,3} \cdot k_{v,2,a}^{2,3} \cdot X_{2,a,r}^{d,v,2,s,[7,8,9],p}(t) \\ & - \left(\zeta_v^{d,3,4} \cdot k_{v,2,a}^{3,4} + \zeta_v^{d,3,2} \cdot k_{v,2,a}^{3,2} + \mu_{t\text{HPV2}}^{d,3,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,3,s,[7,8,9],p}(t)\end{aligned}$$

Women, CIN2

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,4,s,[1,2,3,4,5,6],p}(t)}{dt} = & \zeta_v^{d,5,4} \cdot k_{v,2,a}^{5,4} \cdot X_{2,a,r}^{d,v,5,s,[1,2,3,4,5,6],p}(t) \\ & + \zeta_v^{d,3,4} \cdot k_{v,2,a}^{3,4} \cdot X_{2,a,r}^{d,v,3,s,[1,2,3,4,5,6],p}(t) \\ & - \left(\zeta_v^{d,4,5} \cdot k_{v,2,a}^{4,5} + \zeta_v^{d,4,3} \cdot k_{v,2,a}^{4,3} + \mu_{\text{utHPV2}}^{d,4,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,4,s,[1,2,3,4,5,6],p}(t)\end{aligned}$$

Treated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,4,s,[7,8,9],p}(t)}{dt} = & \zeta_v^{d,5,4} \cdot k_{v,2,a}^{5,4} \cdot X_{2,a,r}^{d,v,5,s,[7,8,9],p}(t) \\ & + \zeta_v^{d,3,4} \cdot k_{v,2,a}^{3,4} \cdot X_{2,a,r}^{d,v,3,s,[7,8,9],p}(t) \\ & - \left(\zeta_v^{d,4,5} \cdot k_{v,2,a}^{4,5} + \zeta_v^{d,4,3} \cdot k_{v,2,a}^{4,3} + \mu_{t\text{HPV2}}^{d,4,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,4,s,[7,8,9],p}(t)\end{aligned}$$

Women, CIN3

Untreated cervical cancer health states

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,5,s,[1,2,3,4,5,6],p}(t)}{dt} = & \zeta_v^{d,4,5} \cdot k_{v,2,a}^{4,5} \cdot X_{2,a,r}^{d,v,4,s,[1,2,3,4,5,6],p}(t) \\ & - \left(\zeta_v^{d,5,6} \cdot k_{v,2,a}^{5,6} + \zeta_v^{d,5,4} \cdot k_{v,2,a}^{5,4} + \mu_{\text{utHPV2}}^{d,5,s,[1,2,3,4,5,6]}\right) \cdot X_{2,a,r}^{d,v,5,s,[1,2,3,4,5,6],p}(t)\end{aligned}$$

Treated cervical cancer health states

$$\frac{dX_{2,a,r}^{d,v,5,s,[7,8,9],p}(t)}{dt} = \zeta_v^{d,4,5} \cdot k_{v,2,a}^{4,5} \cdot X_{2,a,r}^{d,v,4,s,[7,8,9],p}(t) - \left(\zeta_v^{d,5,6} \cdot k_{v,2,a}^{5,6} + \zeta_v^{d,5,4} \cdot k_{v,2,a}^{5,4} + \mu_{t\text{HPV2}}^{d,5,s,[7,8,9]}\right) \cdot X_{2,a,r}^{d,v,5,s,[7,8,9],p}(t)$$

Non-vaccine-type HPV and precancer equations

The non-vaccine-type HPV and precancer equations follow the same pattern as the vaccine-type HPV equations with a few updates. All values of s equal the values of h in the vaccine-type equations, and h equals any value. The appropriate force of infection, transition rates, and transition rate multipliers for individuals living with HIV should be used. Vaccination does not depend on non-vaccine-type HPV infection status.

Cervical cancer equations

Female cervical cancer, local

(where $h = 6$)

$$\frac{dX_{2,a,r}^{d,v,6,s,x,p}(t)}{dt} = \zeta_v^{d,5,6} \cdot k_{v,2,a}^{5,6} \cdot X_{2,a,r}^{d,v,5,s,x,p}(t)$$

(where $s = 6$)

$$\frac{dX_{2,a,r}^{d,v,h,6,x,p}(t)}{dt} = \zeta_v^{d,5,6} \cdot k_{v,2,a}^{5,6} \cdot X_{2,a,r}^{d,v,h,6,x,p}(t)$$

(where $h = 6$ or $s = 6$, and $x = 1$ or $x = 4$)

$$\frac{dX_{2,a,r}^{d,v,h,s,[1,4],p}(t)}{dt} = -\left(\phi_2^{h,s,[1,4],[2,5]} + \mu_{\text{utHPV2}}^{d,h,s,[1,4]}\right) \cdot X_{2,a,r}^{d,v,h,s,[1,4],p}(t)$$

(where $h = 6$ or $s = 6$, and $x = 7$)

$$\frac{dX_{2,a,r}^{d,v,h,s,7,p}(t)}{dt} = -\mu_{t\text{HPV2}}^{d,h,s,7} \cdot X_{2,a,r}^{d,v,h,s,7,p}(t)$$

Female cervical cancer, regional

(where $h = 6$ or $s = 6$, and $x = 2$ or $x = 5$)

$$\frac{dX_{2,a,r}^{d,v,h,s,[2,5],p}(t)}{dt} = \phi_2^{h,s,[1,4],[2,5]} \cdot X_{2,a,r}^{d,v,h,s,[1,4],p}(t) - \left(\phi_2^{h,s,[2,5],[3,6]} + \mu_{\text{utHPV2}}^{d,h,s,[2,5]}\right) \cdot X_{2,a,r}^{d,v,h,s,[2,5],p}(t)$$

(where $h = 6$ or $s = 6$, and $x = 8$)

$$\frac{dX_{2,a,r}^{d,v,h,s,8,p}(t)}{dt} = -\mu_{t\text{HPV2}}^{d,h,s,8} \cdot X_{2,a,r}^{d,v,h,s,8,p}(t)$$

Female cervical cancer, distant

(where h=6 or s=6, and x=3 or x=6)

$$\frac{dX_{2,a,r}^{d,v,h,s,[3,6],p}(t)}{dt} = \phi_2^{h,s,[2,5],[3,6]} \cdot X_{2,a,r}^{d,v,h,s,[2,5],p}(t) - \mu_{\text{utHPV2}}^{d,h,s,[3,6]} \cdot X_{2,a,r}^{d,v,h,s,[3,6],p}(t)$$

(where h=6 or s=6, and x=9)

$$\frac{dX_{2,a,r}^{d,v,h,s,9,p}(t)}{dt} = -\mu_{t\text{HPV2}}^{d,h,s,9} \cdot X_{2,a,r}^{d,v,h,s,9,p}(t)$$

Rate of Symptomatic Detection of Cervical Cancer

Female cervical cancer, local, untreated

$$\frac{dX_{2,a,r}^{d,v,h,s,4,p}(t)}{dt} = (1-\rho_{tx,2}) \cdot k_{sy}^{1} \cdot X_{2,a,r}^{d,v,h,s,1,p}(t)$$

Female cervical cancer, local, treated by other modalities

$$\frac{dX_{2,a,r}^{d,v,h,s,7,p}(t)}{dt} = \left[\rho_{tx,2} \cdot \left(1-\rho_{hy,2}^{1}\right)\right] \cdot k_{sy}^{1} \cdot X_{2,a,r}^{d,v,h,s,1,p}(t)$$

Female cervical cancer, regional, untreated

$$\frac{dX_{2,a,r}^{d,v,h,s,5,p}(t)}{dt} = (1-\rho_{tx,2}) \cdot k_{sy}^{2} \cdot X_{2,a,r}^{d,v,h,s,2,p}(t)$$

Female cervical cancer, regional, treated by other modalities

$$\frac{dX_{2,a,r}^{d,v,h,s,8,p}(t)}{dt} = \left[\rho_{tx,2} \cdot \left(1-\rho_{hy,2}^{2}\right)\right] \cdot k_{sy}^{2} \cdot X_{2,a,r}^{d,v,h,s,2,p}(t)$$

Female cervical cancer, distant, untreated

$$\frac{dX_{2,a,r}^{d,v,h,s,6,p}(t)}{dt} = (1-\rho_{tx,2}) \cdot k_{sy}^{3} \cdot X_{2,a,r}^{d,v,h,s,3,p}(t)$$

Female cervical cancer, distant, treated by other modalities

$$\frac{dX_{2,a,r}^{d,v,h,s,9,p}(t)}{dt} = \left[\rho_{tx,2} \cdot \left(1-\rho_{hy,2}^{3}\right)\right] \cdot k_{sy}^{3} \cdot X_{2,a,r}^{d,v,h,s,3,p}(t)$$

Female cervical cancer, treated by hysterectomy

$$\begin{aligned}\frac{dX_{2,a,r}^{d,v,h,s,10,p}(t)}{dt} = & \left[\rho_{tx,2} \cdot \rho_{hy,2}^{1}\right] \cdot k_{sy}^{1} \cdot X_{2,a,r}^{d,v,h,s,1,p}(t) \\ & + \left[\rho_{tx,2} \cdot \rho_{hy,2}^{2}\right] \cdot k_{sy}^{2} \cdot X_{2,a,r}^{d,v,h,s,2,p}(t) \\ & + \left[\rho_{tx,2} \cdot \rho_{hy,2}^{3}\right] \cdot k_{sy}^{3} \cdot X_{2,a,r}^{d,v,h,s,3,p}(t)\end{aligned}$$

The equation variables are:

Variable	Description
$\mu_{t}HPVg^{d,h,s,x}$	Annual untreated cervical cancer-associated mortality rate by gender g, HIV disease stage d, vaccine-type HPV stage h, non-vaccine-type HPV stage s, and cervical cancer stage x for $1 \leq x \leq 6$. Only women have cervical cancer-associated mortality ($\mu_{H}PV1^{d,h,s,x} = 0$) and only when $h = 6$ or $s = 6$.
$\mu_{t}HPVg^{d,h,s,x}$	Annual treated cervical cancer-associated mortality rate by gender g, HIV disease stage d, vaccine-type HPV stage h, non-vaccine-type HPV stage s, and cervical cancer stage x for $7 \leq x \leq 9$. Only females have treated cervical cancer-associated mortality ($\mu_{t}HPV1^{d,h,s,x} = 0$) and only when $h = 6$ or $s = 6$.
$\lambda_{vHPVg,a,r(t)}$	Force of vaccine-type HPV infection for susceptible persons of gender g, age a, and risk r.
$\lambda_{nvHPVg,a,r}(t)$	Force of non-vaccine-type HPV infection for susceptible persons of gender g, age a, and risk r.
$\varkappa_{d}$	HPV acquisition risk multiplier for HIV-positive individuals with CD4 count $4 \leq d \leq 7$.
$\xi g, a$	HPV acquisition reduction multiplier by gender and age for individuals with type-specific natural immunity. Only women temporarily develop partial natural immunity ($\xi 1, a = 0$). Older women develop stronger natural immunity than young girls.
$\psi_{\text{HPV}g}$	HPV acquisition reduction multiplier due to population-level condom use by gender.
ϕa	Vaccine-type HPV acquisition reduction multiplier by age.
$k_{vg,a}^{h,h'}$	Transition rate of progressing or regressing from vaccine-type HPV precancer or disease stage h to stage h'. Only women develop precancerous lesions and cervical cancer ($k_{v1,a}^{h,h'} = 0$ except for HPV clearance when $h = 2$ and $h' = 1$).
$k_{nvg,a}^{s,s'}$	Transition rate of progressing or regressing from non-vaccine-type HPV precancer or disease stage s to stage s'. Only women develop precancerous lesions and cervical cancer ($k_{v}1, a^{s,s'} = 0$ except for HPV clearance when $s = 2$ and $s' = 1$).

Variable	Description
$\varphi_g^{h,s,x,x'}$	Progression rate of cervical cancer from stage x to stage x'. Only women develop cervical cancer ($\varphi_1^{h,s,x,x'} = 0$) and ($\varphi_2^{h,s,x'} > 0$ only when h or $s = 6$). Only untreated cervical cancers progress from stage x to stage x'.
r_g	Rate of waning natural immunity. Only women temporarily develop partial natural immunity ($r_1 = 0$).
$\zeta_v^{d,h,h'}$	Transition rate multiplier for individuals with HIV progressing or regressing from vaccine-type precancer or disease stage h to stage h' with CD4 count d. Transition rate multipliers for individuals with HIV are the same for vaccine-type and non-vaccine-type HPV.
$\zeta_{nv}^{d,s,s'}$	Transition rate multiplier for individuals with HIV progressing or regressing from non-vaccine-type precancer or disease stage s to stage s' with gender g and CD4 count d. Transition rate multipliers for individuals with HIV are the same for vaccine-type and non-vaccine-type HPV ($\zeta_v^{d,h,h'} = \zeta_n v^{d,s,s'}$ when $h = s$ and $h' = s'$).
l_g	Additional multiplier for clearance of vaccine or non-vaccine-type HPV infection. Only applied to men ($l_2 = 1$).
$V_{g,a}^d$	The proportion of persons with HIV disease status d, gender g, and age a vaccinated.
$k_{sy_g^x}$	Annual probability of being diagnosed with cervical cancer due to symptoms by cervical cancer stage x $(1 \leq x \leq 3)$.
$\rho_t x g$	The proportion of women who are diagnosed with cervical cancer and continue to treatment.
$\rho_h y g^x$	The proportion of women who are diagnosed and treated with cervical cancer who are treated with hysterectomy, by cancer stage x $(1 \leq x \leq 3)$.

Demography

At each iteration, the force of infection and the number of births are calculated and then used to evaluate the ODEs along with mortality and disease progression. The numbers of incident infections, HIV-related deaths, and individuals entering CD4≤200 cells/μL are also calculated to determine QALYs.

Births

The number of infant births of HIV disease stage d and gender g, $b_{g,a,r}^{d,1,h,s,x,p}(t)$ determines how many newborns enter the population. We assume that all newborns are born as low risk, no vertical transmission of HPV, and that if HIV is vertically transmitted, that infected newborns are born into the acute stage of HIV and that women age 15–49 give birth. Fertility rates are stratified by age and stage of disease. Births from uninfected mothers and women on ART, $b_s(t)$, and from HIV-positive mothers, $b_i(t)$, are:

$$b_s(t) = \sum_{h=1}^{7}\sum_{s=1}^{7}\sum_{x=1}^{3}\sum_{p=1}^{4}\sum_{a=4}^{10}\sum_{r=1}^{3}\left[\gamma_a^1(t)\cdot X_{g,a,r}^{1,1,h,s,x,p}(t) + \gamma_a^8(t)\cdot X_{g,a,r}^{8,6,h,s,x,p}(t)\right]$$

$$b_i(t) = \sum_{d=3}^{7}\sum_{v=1}^{5}\sum_{h=1}^{7}\sum_{s=1}^{7}\sum_{x=1}^{3}\sum_{p=1}^{4}\sum_{a=4}^{10}\sum_{r=1}^{3}\left[\gamma_a^d(t)\cdot X_{g,a,r}^{d,v,h,s,x,p}(t)\right]$$

HIV-negative, uncircumcised births

For $h = s = x = p = a = r = 1$,

$$b_{g,a,r}^{1,1,h,s,x,p}(t) = 0.5\cdot(b_s(t) + (1-\eta(t))\cdot b_i(t))$$

else,

$$b_{g,a,r}^{1,1,h,s,x,p}(t) = 0$$

HIV-positive births

For $h = s = x = p = a = r = 1$,

$$b_{g,a,r}^{3,1,h,s,x,p}(t) = 0.5\cdot\eta(t)\cdot b_i(t)$$

else,

$$b_{g,a,r}^{3,1,h,s,x,p}(t) = 0$$

The equation variables are:

Variable	Description
$\gamma_a^d(t)$	The annual fertility rate for women by age a and HIV disease stage d. Women aged 15-49 bear children.
$\eta(t)$	The proportion of births from women living with HIV that result in vertical transmission. Each birth is multiplied by 0.5 given an assumed gender ratio at birth of 1:1. The proportion of births from HIV-positive mothers that result in infection, $\eta(t)$, decreases linearly from 34% in 2004 to 29.2% in 2005, then to 7.1% in 2008[5-7].

Mortality

People leave the population due to death or aging past age 79. Annual background mortality rate by gender g and age a is represented by $\mu_{bkrdg,a}(t)$.

$$\frac{dX_{g,a,r}^{d,v,h,s,x,p}(t)}{dt} = -\mu_{bkrd,g,a}(t) \cdot X_{g,a,r}^{d,v,h,s,x,p}(t)$$

Force of Infection

The force of infection represents the cumulative risk of acquiring HIV or HPV from all possible partners, and depends on the adjusted contact rate, the per-partnership probability of transmission, and the proportion of sexually active persons who are HIV- or HPV-infected.

For HIV:

$$\lambda_{\text{HIV},g,a,r}(t) = \sum_{a'=1}^{16}\sum_{r'=1}^{3}\left(c_{g,a,a',r,r'}^{*}(t) \cdot \left[\frac{-\sum_{v'=1}^{6}\sum_{x'=1}^{4}\ln\left(1-\beta_{\text{HIV},g,a,r}^{v',x'}\right)\cdot\sum_{d'=3}^{8}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}{\sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}\right]\right)$$

Similarly, the force of infection $\lambda_{vHPV\,g,a,r}(t)$ determines vaccine-type HPV transmission:

$$\lambda_{v\text{HPV},g,a,r}(t) = \sum_{a'=1}^{16}\sum_{r'=1}^{3}\left(c_{g,a,a',r,r'}^{*}(t) \cdot \left[\frac{-\sum_{v'=1}^{6}\sum_{x'=1}^{4}\ln\left(1-\beta_{\text{HPV},g,a,r}^{v',x'}\right)\cdot\sum_{d'=1}^{8}\sum_{h'=2}^{6}\sum_{s'=1}^{7}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}{\sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}\right]\right)$$

and $\lambda_{nvHPV\,g,a,r}(t)$ defines non-vaccine-type HPV transmission:

$$\lambda_{\text{nvHPV},g,a,r}(t) = \sum_{a'=1}^{16}\sum_{r'=1}^{3}\left(c_{g,a,a',r,r'}^{*}(t) \cdot \left[\frac{-\sum_{v'=1}^{6}\sum_{x'=1}^{4}\ln\left(1-\beta_{\text{HPV},g,a,r}^{v',x'}\right)\cdot\sum_{d'=1}^{8}\sum_{h'=1}^{7}\sum_{s'=2}^{6}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}{\sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4}X_{g',a',r'}^{d',v',h',s',x',p'}(t)}\right]\right)$$

The equation variables are:

Variable	Description
$c_{g,a,a',r,r'}^{*}(t)$	Adjusted yearly contact rate for persons of gender g, age a, and risk group r, with persons of the opposite gender, age a', and risk group r'.
$\beta_{HIV\,g,a,r}^{v',x'}$	Annual per-partnership probability of HIV transmission from a person with HIV with viral load v' and cervical cancer stage x' to an HIV-susceptible partner with gender g, age a, and risk group r.
$\beta_{HPV\,g,a,r}^{v',x'}$	Annual per-partnership probability of HPV transmission from an HPV-infected person with viral load v' and cervical cancer stage x' to an HPV-susceptible partner with gender g, age a, and risk group r.

Mixing Matrix

Using methods similar to other models, the mixing matrix, $\rho_{g,a,a',r,r'}(t)$ describes patterns of sexual contact by calculating the proportion of one's sexual partners that come from a specific age and sexual-risk group[5].

$$\rho_{g,a,a',r,r'}(t) = \left(\epsilon_a \cdot \frac{\sum_{r'=1}^{3}\left(c_{g',a',r'} \cdot \sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X_{g',a',r'}^{d',v',h',s',x',p'}(t)\right)}{\sum_{a'=1}^{16}\sum_{r'=1}^{3}\left(c_{g',a',r'} \cdot \sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X_{g',a',r'}^{d',v',h',s',x',p'}(t)\right)} + (1-\epsilon_a)\cdot\delta_{g,a,a'}\right)$$
$$\cdot\left(\epsilon_r \cdot \frac{c_{g',a',r'} \cdot \sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X_{g',a',r'}^{d',v',h',s',x',p'}(t)}{\sum_{r'=1}^{3}\left(c_{g',a',r'} \cdot \sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X_{g',a',r'}^{d',v',h',s',x',p'}(t)\right)} + (1-\epsilon_r)\cdot\delta_{g,r,r'}\right)$$

The equation variables are

Variable	Description
$c_{g,a,r}$	Number of partners a person has per year of gender g, age a, and sexual-risk group r (i.e., the partner exchange rate or contact rate).
ϵ_a	Mixing parameter by age a. We assume a mixing pattern that is partially random and partially off-diagonal ($0 < \epsilon_a < 1$), where $\epsilon_a = 0$ indicates completely off-diagonal mixing, and $\epsilon_a = 1$ indicates completely random mixing.
ϵ_r	Mixing parameter by sexual-risk group r. We assume a mixing pattern that is partially random and partially on-diagonal ($0 < \epsilon_r < 1$), where $\epsilon_r = 0$ indicates completely on-diagonal mixing, and $\epsilon_r = 1$ indicates completely random mixing.
$\delta_{g,a,a'}$	Mixing pattern by age. In completely non-random mixing by age, women are most likely to form partnerships with men of the next oldest age group. We represent this pattern using an off-diagonal matrix. For men ($g = 1$) of age a mixing with women of age a': - $= 0.3$ if $(a = a')$ - $= 0.7$ if $(a = a' + 1)$ - $= 0.0$ if $(a = a' = 1)$ - $= 0.0$ if $(a = 2)$ and $(a' = 1)$ - $= 0.0$ if $(a = 2)$ and $(a' = 2)$ - $= 0.0$ if $(a = 3)$ and $(a' = 2)$ For women ($g = 2$) of age a mixing with men of age a': - $= 0.3$ if $(a = a')$ - $= 0.7$ if $(a = a' - 1)$ - $= 0.0$ if $(a = a' = 1)$ - $= 0.0$ if $(a = 1)$ and $(a' = 2)$ - $= 0.0$ if $(a = a' = 2)$ - $= 0.0$ if $(a = 2)$ and $(a' = 3)$
$\delta_{r,r'}$	Mixing pattern by risk. Completely non-random mixing by risk confines sexual encounters to individuals within the same risk group. We represent this pattern using an identity matrix: - $= 1.0$ if $(r = r')$ - $= 0.0$ if $(r \neq r')$

We assume that mixing is partially random and partially designated by a mixing pattern $\delta_{g,a,a'}$ or $\delta_{r,r'}$. The overall mixing matrix is therefore a weighted average of random mixing proportional to the number of available partnerships of each group and mixing among groups with similar characteristics. The off-diagonal pattern results in females of age a being more likely to form partnerships with males of age $a = a' - 1$, which is consistent with reports of such age discrepancies in KZN[6,7]. Although an off-diagonal mixing pattern results in the first and last ages groups (ages 10-14 and 75-79) having fewer than 100% of their partnerships, these age groups have relatively few partnerships and contribute marginally to overall infection transmission.

Per-Partnership Probability of Transmission

The per-partnership probability of transmission, $\beta_{\text{HIV}}{}_{g,a,r}^{v',x'}$, depends on the sexual risk group of the HIV-negative partner and the disease state of the HIV-positive partner. $\chi_{\text{HIV}}{}_{g}^{v',x'}$ is the per-act probability of HIV transmission to a person of gender based on the viral load of the partner living with HIV. We assume the probability of female-to-male HIV transmission is equal to the probability of male-to-female transmission across all viral load stages ($\chi_{\text{HIV}}{}_{1}^{v'} = \chi_{\text{HIV}}{}_{2}^{v'}$). We reduce HIV per-act transmission as a proxy for decreased sexual activity during late-stage HIV ($v' = 5$), regional or distant cervical cancer ($x' = 2$ or $x' = 3$), or hysterectomy ($x' = 4$). $A_{g,a,r}$ is the number of acts a person has per partnership of gender g, age a, and sexual-risk group r. We assume zero acts for individuals below the age of sexual debut (age 10). The probabilities of transmission per partnership are:

Per-partnership probability of HIV transmission to a male partner

$$\beta_{\text{HIV},1,a,r}^{v',x'} = 1 - \left(1 - \chi_{\text{HIV},1}^{v',x'}\right)^{A_{1,a,r}}$$

Per-partnership probability of HIV transmission to a female partner

$$\beta^{v',x'}_{\text{HIV},2,a,r} = 1 - \left(1 - \chi^{v',x'}_{\text{HIV},2}\right)^{A_{2,a,r}}$$

The per partnership probability of transmission, $\beta_{\text{HPV}}{}^{v',x'}_{g,a,r}$ is calculated in a similar manner for HPV. $\chi_{\text{HPV}}{}^{v',x'}_{g}$ is the per-act probability of HPV transmission to a person of gender by an an HPV-positive partner:

We calculate the per-partnership probability of HPV transmission to a male partner:

$$\beta_{\text{HPV}}{}^{v',x'}_{1,a,r} = 1 - \left(1 - \chi_{\text{HPV}}{}^{v',x'}_{1}\right)^{A_{1,a,r}}$$

Similarly, the per-partnership probability of HPV transmission to a female partner:

$$\beta_{\text{HPV}}{}^{v',x'}_{2,a,r} = 1 - \left(1 - \chi_{\text{HPV}}{}^{v',x'}_{2}\right)^{A_{2,a,r}}$$

Rate of Partner Change

Data on sexual behavior and specifically, sexual contact rates, $c_{g,a,r}$ are often subject to biases leading to contact rate data that, when assuming solely heterosexual contact, are inconsistent between males and females[8]. We account for this variability by using an adjusted contact rate, $c^*_{g,a,a',r,r'}(t)$ which equilibrates the reported number of sexual partners by males and females[5]. The adjusted contact rate can be male- or female-driven, as determined by the parameter θ, where $\theta = 1$ for male-driven, $\theta = 0$ for female-driven, and $\theta = 0.5$ when compromised equally. We assume $\theta = 0.5$ given the lack of data to assume otherwise. The adjusted contact rate for females is:

$$c^*_{2,a,a',r,r'}(t) = c_{2,a,r} \cdot \rho_{2,a,a',r,r'}(t) \cdot B_{a,a',r,r'}(t)^{\theta} \cdot \left(\frac{\sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X^{d',v',h',s',x',p'}_{1,a',r'}(t)}{\sum_{d=1}^{8}\sum_{v=1}^{6}\sum_{h=1}^{7}\sum_{s=1}^{7}\sum_{x=1}^{4}\sum_{p=1}^{4} X^{d,v,h,s,x,p}_{2,a,r}(t)} \right)^{-(1-\theta)}$$

For males, the adjusted contact rate is:

$$c^*_{1,a,a',r,r'}(t) = c_{1,a,r} \cdot \rho_{1,a,a',r,r'}(t) \cdot B_{a,a',r,r'}(t)^{-(1-\theta)} \cdot \left(\frac{\sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X^{d',v',h',s',x',p'}_{1,a',r'}(t)}{\sum_{d=1}^{8}\sum_{v=1}^{6}\sum_{h=1}^{7}\sum_{s=1}^{7}\sum_{x=1}^{4}\sum_{p=1}^{4} X^{d,v,h,s,x,p}_{2,a,r}(t)} \right)^{\theta}$$

The discrepancy between the two populations, $B^{a',r'}_{a,r}(t)$, is defined as:

$$B_{a,a',r,r'}(t) = \frac{c_{1,a,r} \cdot \rho_{1,a,a',r,r'}(t) \cdot \sum_{d'=1}^{8}\sum_{v'=1}^{6}\sum_{h'=1}^{7}\sum_{s'=1}^{7}\sum_{x'=1}^{4}\sum_{p'=1}^{4} X^{d',v',h',s',x',p'}_{1,a',r'}(t)}{c_{2,a,r} \cdot \rho_{2,a,a',r,r'}(t) \cdot \sum_{d=1}^{8}\sum_{v=1}^{6}\sum_{h=1}^{7}\sum_{s=1}^{7}\sum_{x=1}^{4}\sum_{p=1}^{4} X^{d,v,h,s,x,p}_{2,a,r}(t)}$$

Model Calibration

The model was calibrated through a phased approach to fit HIV, HPV, cervical cancer and population dynamics from 1996 to 2019. We used hand calibration to explore the sensitivity of model outcomes to individual parameters (Phase 0), informing our decision to divide the Bayesian calibration into three phases. In Phase 1, a Bayesian algorithm was used to fit sexual behavior and HIV natural history parameters to observed demographic and HIV prevalence data. Randomly resampling from the 50 best-fitting parameter sets of Phase 1, we then used the same Bayesian algorithm to fit HPV natural history parameters to observed data on HPV prevalence, CIN prevalence, cervical cancer incidence, and type distribution for Phase 2. Phase 3 was added to the calibration to fit parameters of probabilities of cervical cancer symptomatic detection by stage. Phase 3 involved randomly resampling from the 50 best-fitting parameter sets of Phase 2 and the corresponding parameter sets of Phase 1 and fitting to observed data on cervical cancer stage distribution before widespread cervical screening in South Africa. All our outputs followed independent normal distributions. For prevalence data, we assumed a normal approximation of the binomial distribution, and for incidence data, we assumed a normal approximation of a Poisson distribution. We assumed a normal distribution for the total population size of KwaZulu-Natal and that the 2019 estimate had the same variance as the 2011 estimate.

Population Aging

To age the population, one-fifth of each compartment enters the next age group while maintaining the same gender,disease state and sexual risk $\phi_{g,a,r}$. When individuals age to the next five-year group, they are redistributed into the closest unfilled risk group to match observed data on the age distribution of low, moderate and high-risk individuals. All compartments, except for the youngest and oldest age-groups, experience influx from the prior age and efflux into the next age. The 0 to 4 age-group only receives influx through births while the 75 to 79 age-group exits the population rather than entering the next age. Therefore, each state has a second ODE that occurs at each time step:

$$\frac{dX_{g,1,r}^{d,v,h,s,x,p}(t)}{dt} = -\frac{1}{5}\sum_{r=1}^{3}\left(X_{g,1,r}^{d,v,h,s,x,p}(t)\cdot\phi_{g,a,r}\right)$$

(for $a = 1$)

$$\begin{aligned}\frac{dX_{g,a,r}^{d,v,h,s,x,p}(t)}{dt} = & -\frac{1}{5}\sum_{r=1}^{3}\left(X_{g,a,r}^{d,v,h,s,x,p}(t)\cdot\phi_{g,a,r}\right) \\ & +\frac{1}{5}\sum_{r=1}^{3}\left(X_{g,a-1,r}^{d,v,h,s,x,p}(t)\cdot\phi_{g,a-1,r}\right)\end{aligned}$$

(for $a \neq 1$)

Interventions

ART Treatment:

We define ART coverage as the percentage of all persons living with HIV and age-eligible for ART who are on treatment and virally suppressed. Coverage of ART treatment for HIV-positive persons increased from 0% in 2004 to 44% for men and 60% for women in 2017[9]. The proportion of persons living with HIV who are virally suppressed remains at the estimated levels for 2017 for the simulation. Individuals on treatment with viral suppression are assumed to have zero probability of transmitting HIV and have reduced HIV-associated mortality. The probability of ART initiation is uniform by age or risk group. However, we do not model the discontinuation process of ART and the resulting loss of viral suppression, such that the cumulative probability of being on ART increases with age. Therefore, we apply age-specific minimum and maximum limits for viral suppression, ensuring that it is distributed appropriately across all age groups while also matching population-level levels of viral suppression by gender in the observed data. HIV-associated mortality among treated persons living with HIV decreases over time, reflecting higher baseline health among individuals initiating treatment. From 2004 to 2011, HIV-associated excess mortality among virally suppressed persons living with HIV was 0.5x the background rate. This multiplier decreases to 0.4x, 0.25x, and 0.15x the background mortality rate in 2011, 2015, and 2016, respectively.

Circumcision:

We model medical circumcision beginning in 1960 for age groups 15-19 and 20-24. Data suggest that circumcised males have a 60% ($\psi_0 = 0.6$) lower risk of acquiring HIV but are not at a reduced risk of transmitting HIV[10-12]. Therefore, the model does not track the circumcision status of HIV-positive persons. Before the initiation of the South Africa National VMMC program in 2010, circumcision was primarily targeted to young adult men as a rite of passage[13]. Starting circumcision in 1960 among youth resulted in circumcision prevalence among men aged 50 and older in 2012 corresponding to observed estimates[14]. We assume coverage increases linearly from 1960 to 2000 and between 2000 and 2008 to match coverage level estimates[13,15]. Following the initiation of the national VMMC program in 2010, we model scale-up of circumcision for all men aged 15 or older at levels extrapolated backward from 2012 to 2017[9,14,16]. In our future scenarios, the proportion of men circumcised remains at 2017 levels for the duration of the simulations.

HPV vaccination:

HPV vaccination begins in 2014 for 57% of nine-year-old girls . Our model was designed to evaluate the impact of the 9vHPV vaccine. However, the current vaccination program in South Africa uses the bivalent vaccine, which targets only two of the seven oncogenic types in the 9vHPV vaccine. To account for this, in the years 2014-2023 (when we assume bivalent vaccination is conducted), we adjust the 57% coverage by a factor

of (0.7/0.9), based on evidence that HPV types 16 and 18 contribute to approximately 70% of cervical cancer cases relative to the 90% attributable to one or more of the types included in the 9vHPV[17]. We assume that vaccination provides complete protection against the seven oncogenic types included in the 9vHPV vaccine and 0% protection against other hrHPV types. We assume lifelong protection from vaccination. Vaccine efficacy and uptake are assumed not to vary by HIV status. We model vaccine efficacy using a beta probability distribution that aligns with the results from a licensure trial of the bivalent vaccine, with an efficacy of 100% (95% CI 74.4, 100)[18,19]. The parameters of the beta distribution are $\alpha = 2.0$ and $\beta = 0.04$.

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Output Overview

Summary

This section describes outputs from the model.

Overview

The model tracks the prevalence and incidence of HPV, HIV, Cervical Cancer and their associated outcomes in each compartment for the entire simulated period. This allows for comparisons of population-level health outcomes in the absence and presence of various prevention and intervention strategies. Ultimately, this facilitates the study of the efficacy and cost-effectiveness of said strategies.

Output Listing

The results that are currently being produced are:

- Overall HIV prevalence
- Proportion of HIV-positive population on ART
- Population-level distribution of CD4 count and viral load among HIV-positive individuals
- HIV prevalence by age and gender
- HPV prevalence
- HPV health states
- CIN 2/3 prevalence by HIV status
- Cervical cancer health states
- Cervical cancer incidence
- New cervical cancer cases
- Cervical cancer mortality
- Deaths
- Screening and Treatment
- Vaccinations

MGH
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Summary

A guide to the results obtained from the model.

Overview

The following is a list of publications which showcase results from DRIVE.

Results List

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