

MATHEMATICAL MODELLING OF CERVICAL CANCER WITH TREATMENTS: A CASE STUDY USING CLINICAL DATA FROM FEDERAL TEACHING HOSPITAL LOKOJA

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Abstract

A deterministic compartmental model was formulated and analysed in order to investigate the multistage development of cervical cancer and the transmission dynamics of the human papillomavirus (HPV) that drives it. The model was parametrised using empirical records of fifty cervical cancer patients managed at the Federal Teaching Hospital Lokoja (FTHL), Kogi State, Nigeria. The female population was partitioned into eight mutually exclusive compartments representing susceptible, HPV infected, precancerous, early stage cancer, late stage cancer, two treated classes and recovered individuals, and the flow between them was represented by a system of eight nonlinear ordinary differential equations. The model was shown to be mathematically well posed through proofs of positivity, boundedness, and the existence and uniqueness of solutions. The next generation matrix method yielded a basic reproduction number of the compact form $R_0 = \beta/(\gamma + \alpha + \mu)$. Normalised forward sensitivity analysis identified the HPV transmission rate as the single most influential parameter, with an index of unity, followed by the natural clearance rate with an index of -0.751 . The disease free equilibrium was proved to be globally asymptotically stable whenever $R_0 \leq 1$, and the endemic equilibrium globally asymptotically stable whenever $R_0 > 1$, using suitably constructed Lyapunov functions together with the LaSalle invariance principle. Numerical simulation with the FTHL derived parameter set returned $R_0 \approx 1.201$, confirming endemic HPV transmission and agreeing closely with the 66 percent HPV positivity recorded in the clinical dataset. The critical transmission rate below which elimination occurs was found to be $\beta_c = 0.6662$, and the corresponding critical clearance rate $\gamma_c = 0.6338$. Scenario analysis showed that reducing transmission to the critical threshold drives the infected class to extinction, whereas intensified treatment, which acts downstream of the infectious compartment, lowers the invasive cancer burden by more than eighty percent without altering R_0 itself. The clinical record revealed no vaccinated patient, a treatment completion rate of only 22 percent and a late presentation rate of 36 percent, which together explain the persistence of the disease locally. The findings indicate that prophylactic vaccination and organised screening, which act on transmission, are the decisive levers for elimination, while treatment scale up remains indispensable for reducing mortality among women who already carry disease.

Keywords: cervical cancer; human papillomavirus; basic reproduction number; positivity and boundedness; Lyapunov stability; next generation matrix; sensitivity analysis

1. Introduction

Mathematical modelling provides the connective tissue between biological theory and the irreducible complexity of real populations. It supplies an organised language in which the behaviour of systems ranging from cellular machinery to national economies can be represented, interrogated and projected forward in time (Lane, 2024; Bhardwaj, 2024). Within the biomedical sciences the approach has matured from a descriptive convenience into a quantitative instrument capable of resolving questions that resist direct experiment, particularly where the relevant timescales span decades and the relevant populations number in the millions (Edogbanya et al., 2024). The lineage is long. Formal population dynamics entered biology more than a century ago, and the subsequent growth of computational capacity has allowed the same conceptual apparatus to be applied across scales that were once treated as separate disciplines, from molecular kinetics to the spread of epidemics across continents (Rassier and Mansson, 2025).

In epidemiology the organising quantity is the basic reproduction number, denoted R_0 . It is a dimensionless threshold defined as the average number of secondary infections generated by one typical infectious individual introduced into a wholly susceptible population (Ali, 2025; van den Driessche and Watmough, 2002). Its interpretive power lies in the sharpness of the threshold it defines. When $R_0 < 1$ each infection fails on average to replace itself and the chain of transmission is mathematically certain to terminate. When $R_0 > 1$ the infection can invade and establish itself as an endemic presence. The quantity therefore serves simultaneously as a diagnostic of invasion potential and as a target for control, since any intervention that drives it below unity is, in principle, sufficient for elimination (Tivde et al., 2024).

Because R_0 is a composite of several biological rates, knowing its value is less useful than knowing which of its constituents it is most responsive to. Sensitivity analysis supplies that ordering by quantifying the proportional change in the threshold induced by a proportional change in each parameter (Chitnis et al., 2008; Liu et al., 2024). Applied to human papillomavirus and cervical cancer, such analyses have repeatedly identified the transmission rate and the rate of progression to precancerous lesions as the dominant determinants, a finding that carries an immediate programmatic implication in favour of primary prevention (Goshu and Abebe, 2022).

Threshold quantities describe invasion but say nothing directly about the long run behaviour of a system once it has been invaded. That question belongs to stability analysis, which examines whether trajectories return to an equilibrium after perturbation and whether they do so from all admissible initial states or only from a neighbourhood (Aitzhanov et al., 2024; LaSalle, 1976). The distinction matters for policy. Local stability guarantees only that a system already close to elimination will not drift away from it, whereas global stability guarantees that elimination will be reached from any starting configuration, including the heavily burdened one that characterises much of sub-Saharan Africa.

The disease under consideration remains among the gravest in global oncology. Cervical cancer is the fourth most common malignancy among women worldwide, and its burden falls with marked asymmetry on countries where screening infrastructure is thin and treatment capacity is constrained (Arbyn et al., 2020; Bray et al., 2024; Girone et al., 2024). The causal pathway is unusually well characterised: persistent infection with oncogenic HPV genotypes precedes cervical intraepithelial neoplasia, which in turn may progress over years or decades to invasive carcinoma (Schiffman et al., 2007; Ezeamii et al., 2024). Because the pathway is long and its intermediate stages are detectable and treatable, the disease is in principle among the most preventable of all cancers, and the World Health Organization has accordingly set an explicit elimination target framed around vaccination, screening and treatment coverage (World Health Organization, 2020; Zhou et al., 2025).

Nigeria illustrates the gap between that principle and its realisation. National vaccination coverage remains low, organised cytological screening is largely absent outside a handful of tertiary centres, and a substantial proportion of women present only once disease has become symptomatic and therefore advanced. Modelling studies conducted elsewhere have demonstrated that the relative value of vaccination, screening and treatment depends sensitively on the epidemiological and structural context in which they are deployed, which means that conclusions imported wholesale from high income settings may mislead (Brisson et al., 2020; Elbasha et al., 2007; van de Velde et al., 2007). What is required is analysis anchored in local data.

The present study responds to that requirement. A deterministic compartmental model of HPV transmission and cervical carcinogenesis is formulated, its mathematical well posedness is established, its threshold and equilibrium structure is derived analytically, and it is then parametrised using the clinical records of fifty cervical cancer patients managed at the Federal Teaching Hospital Lokoja. The objectives are to quantify R_0 for this population, to rank the parameters that govern it, to characterise the stability of the disease free and endemic equilibria, and to compare the projected effect of transmission reduction against that of treatment intensification. The contribution is therefore both analytical and empirical: a fully analysed model, and a parametrisation grounded in Nigerian clinical practice rather than borrowed from dissimilar settings.

2. Materials and Methods

2.1 Clinical Data Collection and Statistical Summary

Clinical records were abstracted for fifty cervical cancer patients managed at the Federal Teaching Hospital Lokoja (FTHL), Kogi State, Nigeria. A structured abstraction sheet captured the study code, age, vaccination history, number of sexual partners,

HPV status, cancer stage at presentation, treatment modality, completion of the prescribed treatment course, and clinical outcome at the point of discharge or last contact. The resulting distribution is summarised in Table 1 and displayed in Figure 2.

Table 1: Summary of clinical characteristics of cervical cancer patients at FTHL (n = 50)

Variable	Count	Percentage (%)
Total patients	50	100
HPV positive	33	66
HPV negative	3	6
HPV status unknown	14	28
Early stage at presentation	2	4
Advanced stage at presentation	18	36
Stage 2B	5	10
Stage 3B	2	4
Stage III	1	2
Stage unknown	22	44
Treatment completed	11	22
Treatment not completed	31	62
Treatment status unknown	8	16
Survived and discharged home	34	68
Referred	8	16
Deceased	3	6
Left against medical advice	5	10

Percentages are computed on the full cohort of fifty patients. Stage categories and outcome categories each sum to fifty.

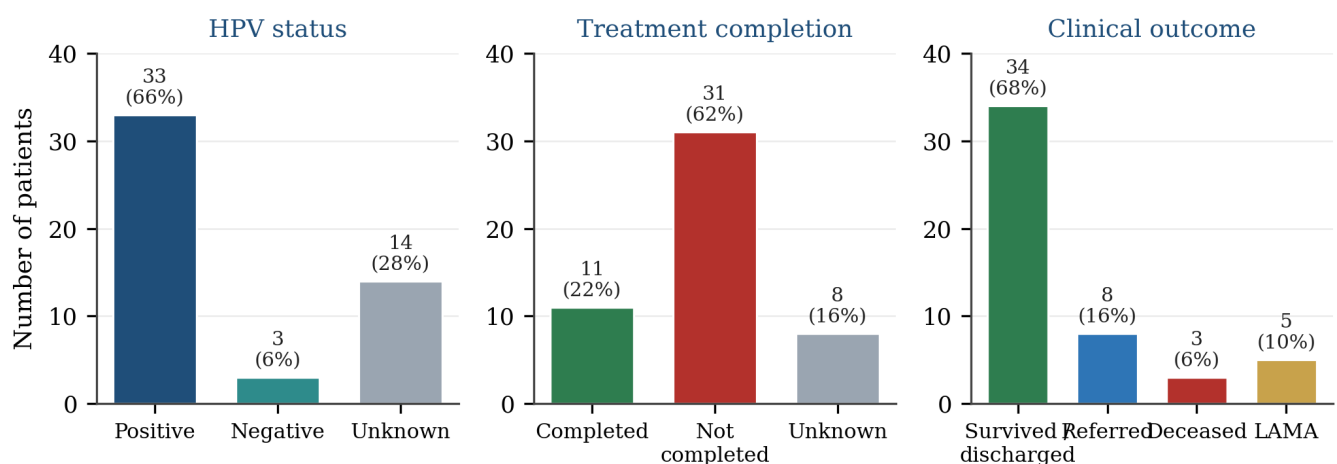


Figure 2: Clinical profile of the FTHL cohort. Panel (a) shows HPV status, with two thirds of the cohort testing positive and a further 28 percent untested. Panel (b) shows treatment completion, where fewer than one in four women completed the prescribed course. Panel (c) shows clinical outcome at discharge or last contact. Counts appear above each bar with the corresponding percentage of the cohort in parentheses.

2.2 Model Assumptions

The construction of the model rests on the following assumptions.

- The susceptible class comprises all sexually active females in the study population.
- Recruitment into the susceptible class, natural mortality and cervical cancer mortality are each represented explicitly.
- Only unvaccinated females are considered, consistent with the absence of any vaccinated patient in the FTHL record.
- Disease induced mortality occurs only in the late stage cancer class.
- Natural mortality occurs in every compartment at the constant per capita rate μ .
- Recovered individuals lose immunity at rate ϕ and return to the susceptible class.
- There is no vertical transmission, so that newborns enter the population uninfected.

2.3 Model Description and Flow Structure

The total female population at time t , denoted $N(t)$, is partitioned into eight mutually exclusive compartments: susceptible individuals $S(t)$, HPV infected individuals $I(t)$, individuals with persistent infection and precancerous lesions $P(t)$, early stage

cancer cases $C_1(t)$, late stage cancer cases $C_2(t)$, individuals under treatment for precancerous or early stage disease $T_1(t)$, individuals under treatment for late stage disease $T_2(t)$, and recovered individuals $R(t)$. Hence

$$N(t) = S(t) + I(t) + P(t) + C_1(t) + C_2(t) + T_1(t) + T_2(t) + R(t). \quad (1)$$

Susceptible individuals are recruited at the constant rate π and acquire HPV through contact with infected individuals at the standard incidence rate $\beta SI/N$. A proportion of infections clears naturally at rate γ , returning those individuals directly to the susceptible class, while a proportion persists and progresses to the precancerous class at rate α . Progression continues from precancerous lesion to early stage carcinoma at rate δ , and from early to late stage carcinoma at rate η . Treatment removes individuals from the precancerous and early stage classes at rates τ_1 and τ_2 respectively into the class T_1 , and from the late stage class at rate τ_3 into the class T_2 . Individuals in T_1 recover at rate ψ and subsequently lose immunity at rate ϕ . Late stage disease carries an additional mortality hazard σ . The complete flow structure is shown in Figure 1.

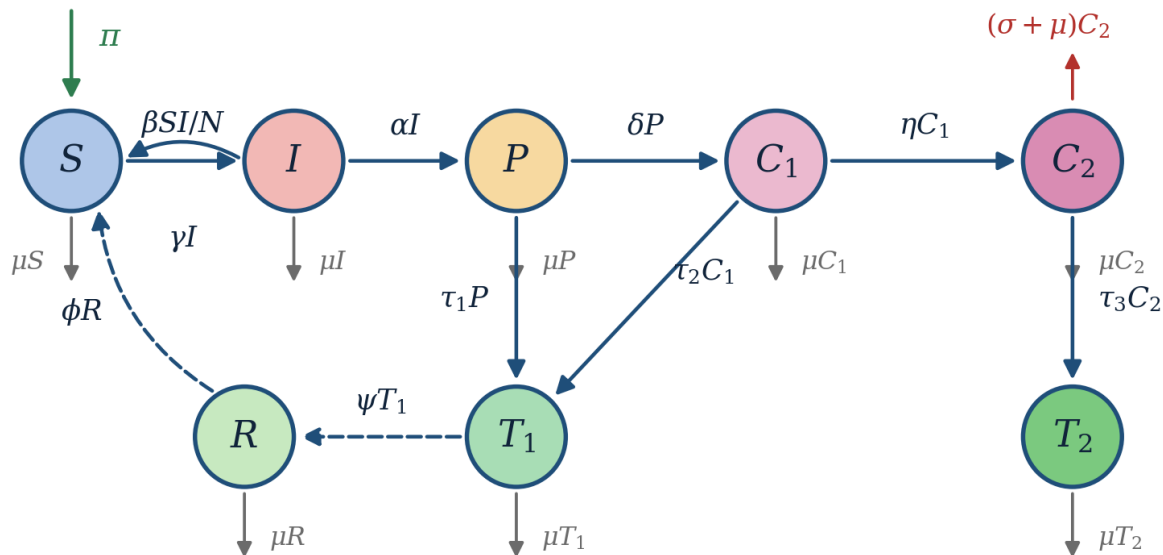


Figure 1: Flow diagram of the eight compartment cervical cancer model. Solid arrows denote infection, progression and treatment transitions; broken arrows denote recovery and the subsequent loss of immunity. Grey arrows represent natural mortality acting on every compartment, and the red arrow denotes the additional disease induced mortality borne by the late stage cancer class.

2.4 Model Equations

The transmission and progression dynamics described above are governed by the following system of eight nonlinear ordinary differential equations.

$$dS/dt = \pi + \phi R + \gamma I - (\beta I/N + \mu)S \quad (2)$$

$$dI/dt = \beta SI/N - (\gamma + \alpha + \mu)I \quad (3)$$

$$dP/dt = \alpha I - (\delta + \tau_1 + \mu)P \quad (4)$$

$$dC_1/dt = \delta P - (\eta + \tau_2 + \mu)C_1 \quad (5)$$

$$dC_2/dt = \eta C_1 - (\sigma + \tau_3 + \mu)C_2 \quad (6)$$

$$dT_1/dt = \tau_1 P + \tau_2 C_1 - (\psi + \mu)T_1 \quad (7)$$

$$dT_2/dt = \tau_3 C_2 - \mu T_2 \quad (8)$$

$$dR/dt = \psi T_1 - (\phi + \mu)R \quad (9)$$

subject to the nonnegative initial conditions $S(0) > 0$, $I(0) \geq 0$, $P(0) \geq 0$, $C_1(0) \geq 0$, $C_2(0) \geq 0$, $T_1(0) \geq 0$, $T_2(0) \geq 0$ and $R(0) \geq 0$, where $N = S + I + P + C_1 + C_2 + T_1 + T_2 + R$ denotes the total population.

2.5 Model Parameters

Table 2: Description of the model parameters

Parameter	Description
π	Recruitment rate into the susceptible population
μ	Natural death rate
β	HPV transmission rate
γ	Natural clearance rate of HPV infection
α	Progression rate from HPV infection to the persistent or precancerous stage
δ	Progression rate from persistent HPV infection to cervical intraepithelial neoplasia
η	Progression rate between cancer stages, early to late
σ	Death rate from metastatic cancer, stage III or IV
τ_1	Treatment rate for the precancerous stage
τ_2	Treatment rate for early stage cancer, stages 0, I and II
τ_3	Treatment rate for late stage cancer, stages III and IV
ψ	Recovery rate from treatment
ϕ	Rate of immunity loss and return to the susceptible class

3. Basic Analysis of the Model

Before a model is used to draw epidemiological inference it must be shown to be mathematically well posed. Three properties are required: solutions that begin in the nonnegative orthant must remain there, since negative population sizes carry no meaning; solutions must remain bounded, since unbounded growth is biologically impossible; and solutions must exist and be unique, since otherwise a single initial state could generate conflicting projections. Each is established in turn.

3.1 Positivity of Solutions

Theorem 1. *The solutions $S(t)$, $I(t)$, $P(t)$, $C_1(t)$, $C_2(t)$, $T_1(t)$, $T_2(t)$, $R(t)$ of the model system with nonnegative initial conditions remain nonnegative for all $t > 0$.*

Proof. Consider the first equation of the system. Since every term removing individuals from S is proportional to S itself, and the remaining terms are nonnegative,

$$dS/dt = \pi + \phi R + \gamma I - (\beta I + \mu)S \geq -(\mu + \beta I)S, \quad (10)$$

so that separation of variables gives

$$dS/S \geq -(\mu + \beta I) dt. \quad (11)$$

Integrating both sides from 0 to t yields

$$\int_0^t dS/S \geq -\int_0^t (\mu + \beta I) dt, \quad (12)$$

and therefore

$$S(t) \geq S(0) e^{-[\mu + \beta I]t} \geq 0. \quad (13)$$

An identical argument applied to each of the remaining seven equations, in which every outflow term is likewise proportional to the state variable being differentiated, shows that $I(t)$, $P(t)$, $C_1(t)$, $C_2(t)$, $T_1(t)$, $T_2(t)$ and $R(t)$ remain nonnegative for all $t > 0$. The nonnegative orthant is therefore positively invariant under the flow of the system.

3.2 Boundedness of Solutions

Theorem 2. *Every solution of the model with positive initial conditions in $(\mathbf{R}_+)^8$ is bounded, and the region of feasible solutions is $\Omega = \{(S, I, P, C_1, C_2, T_1, T_2, R) \in \mathbf{R}_+^8 : N \leq \pi/\mu\}$.*

Proof. Summing the eight equations of the system gives

$$dN/dt = dS/dt + dI/dt + dP/dt + dC_1/dt + dC_2/dt + dT_1/dt + dT_2/dt + dR/dt. \quad (14)$$

Substituting the right hand sides and cancelling the internal transfer terms, which appear once with each sign, leaves only recruitment, natural mortality and disease induced mortality:

$$dN/dt = \pi - \mu(S + I + P + C_1 + C_2 + T_1 + T_2 + R) - \sigma C_2. \quad (15)$$

Since $\sigma C_2 \geq 0$ by Theorem 1, it follows that

$$dN/dt \leq \pi - \mu N. \quad (16)$$

Applying the integrating factor $IF = e^{\mu t}$ gives

$$(d/dt)(N(t) e^{\mu t}) \leq \pi e^{\mu t}, \quad (17)$$

which on integration yields

$$N(t) e^{\mu t} - N(0) \leq (\pi/\mu)(e^{\mu t} - 1). \quad (18)$$

Taking the limit as $t \rightarrow \infty$ gives

$$\lim_{t \rightarrow \infty} N(t) \leq \pi/\mu. \quad (19)$$

The total population is therefore bounded above and every trajectory enters the compact positively invariant region

$$\Omega = \{(S, I, P, C_1, C_2, T_1, T_2, R) \in \mathbf{R}_+^8 : S + I + P + C_1 + C_2 + T_1 + T_2 + R \leq \pi/\mu\}, \quad (20)$$

which establishes that all solutions remain within a biologically feasible region that is positively invariant under the flow of the system defined by equations (2) to (9).

3.3 Existence and Uniqueness of Solutions

Establishing that the model admits exactly one solution for each admissible initial state is what licenses the interpretation of a simulated trajectory as the prediction of the model rather than one of several competing predictions. The classical instrument for this purpose is the existence and uniqueness theorem, whose hypothesis is a Lipschitz condition on the vector field.

Theorem 3 (Existence and Uniqueness). *Consider the first order ordinary differential equation*

$$dy/dx = f(x, y), \quad y(x_0) = y_0, \quad (21)$$

where $f: \Omega \subset \mathbf{R}^2 \rightarrow \mathbf{R}$ is a real valued function defined on a region Ω , and $(x_0, y_0) \in \Omega$ is an initial condition. Suppose f is continuous in the closed rectangle

$$\Omega = \{(x, y) \in \mathbf{R}^2 : |x - x_0| \leq a, |y - y_0| \leq b\}, \quad (22)$$

and satisfies a Lipschitz condition in the variable y on Ω , that is, there exists a constant $L > 0$ such that for all $(x, y_1), (x, y_2) \in \Omega$,

$$|f(x, y_1) - f(x, y_2)| \leq L|y_1 - y_2|. \quad (23)$$

Then there exists a unique function $y(x)$ satisfying the differential equation and the initial condition on some interval $|x - x_0| \leq h \leq a$, and the solution remains in Ω .

Lemma. *Let $\partial f/\partial x$ in equations (2) to (9) be continuous and let $D \in \mathbf{R}^8$. Then there exists a positive constant L such that*

$$\|f(t, x_1) - f(t, x_2)\| \leq L\|x_1 - x_2\| \quad \text{for } (t, x_1), (t, x_2) \in D. \quad (24)$$

Proof. Suppose $x_1 > x_2$. By the mean value theorem,

$$f(t, x_1) - f(t, x_2) = (\partial f/\partial x)(t, c)(x_1 - x_2), \quad (25)$$

where c lies in the interval (x_1, x_2) . Taking norms on both sides gives

$$\|f(t, x_1) - f(t, x_2)\| \leq \|(\partial f/\partial x)(t, c)\| \cdot \|x_1 - x_2\|. \quad (26)$$

Continuity of $\partial f/\partial x$ on the compact region D implies its boundedness there, so that there exists $L > 0$ with $\|\partial f/\partial x\| < L$ for all $(t, x) \in D$. Since $(t, c) \in D$, it follows that $\|(\partial f/\partial x)(t, c)\| \leq L$, and hence

$$\|f(t, x_1) - f(t, x_2)\| \leq L\|x_1 - x_2\| \quad \forall (t, x_1), (t, x_2) \in D. \quad (27)$$

Writing the right hand sides of the model as

$$\begin{aligned} f_1 &= \pi + \phi R + \gamma I - (\beta I/N + \mu)S, & f_2 &= \beta SI/N - (\gamma + \alpha + \mu)I, & f_3 &= \alpha I - (\delta + \tau_1 + \mu)P, \\ f_4 &= \delta P - (\eta + \tau_2 + \mu)C_1, & f_5 &= \eta C_1 - (\sigma + \tau_3 + \mu)C_2, & f_6 &= \tau_1 P + \tau_2 C_1 - (\psi + \mu)T_1, \\ & & f_7 &= \tau_3 C_2 - \mu T_2, & f_8 &= \psi T_1 - (\phi + \mu)R, \end{aligned} \quad (28)$$

and letting $x = (S, I, P, C_1, C_2, T_1, T_2, R)$, the partial derivatives $\partial f_i/\partial x_j$ for $i, j = 1, \dots, 8$ are each either constant, zero, or bounded continuous expressions on the compact region Ω established in Theorem 2. The vector field f therefore satisfies the Lipschitz condition on D , and the system possesses a unique continuous solution on an interval $[t_0, t_0 + \delta]$ for some $\delta > 0$. The model is thus well posed and epidemiologically admissible.

3.4 Equilibrium Points

The equilibria of the system are obtained by setting every derivative to zero:

$$dS/dt = dI/dt = dP/dt = dC_1/dt = dC_2/dt = dT_1/dt = dT_2/dt = dR/dt = 0. \quad (29)$$

Disease free equilibrium. The disease free equilibrium describes the state in which no HPV infection is present, so that

$$I = P = C_1 = C_2 = T_1 = T_2 = R = 0. \quad (30)$$

Substituting into the first equation gives $S = \pi/\mu$, and hence the disease free equilibrium point is

$$E_0 = (\pi/\mu, 0, 0, 0, 0, 0, 0, 0). \quad (31)$$

Endemic equilibrium. The endemic equilibrium $E^* = (S^*, I^*, P^*, C_1^*, C_2^*, T_1^*, T_2^*, R^*)$ exists when $R_0 > 1$. Solving the system at equilibrium gives the cascade

$$\begin{aligned} S^* &= (\gamma + \alpha + \mu)N^*/\beta, & P^* &= \alpha I^*/(\delta + \tau_1 + \mu) = k_1 I^*, & C_1^* &= \delta P^*/(\eta + \tau_2 + \mu) = k_2 I^*, \\ C_2^* &= \eta C_1^*/(\sigma + \tau_3 + \mu) = k_3 I^*, & T_1^* &= (\tau_1 P^* + \tau_2 C_1^*)/(\psi + \mu) = k_4 I^*, \\ T_2^* &= \tau_3 C_2^*/\mu = k_5 I^*, & R^* &= \psi T_1^*/(\phi + \mu) = k_6 I^*, \end{aligned} \quad (32)$$

where $k_1, \dots, k_6$ are positive constants. The complete endemic equilibrium is therefore

$$E^* = (S^*, I^*, k_1 I^*, k_2 I^*, k_3 I^*, k_4 I^*, k_5 I^*, k_6 I^*). \quad (33)$$

3.5 The Basic Reproduction Number

The basic reproduction number is the expected number of secondary infections produced by a single infected individual over the whole of the infectious period in a wholly susceptible population. It is computed here by the next generation matrix method (Diekmann et al., 2010; van den Driessche and Watmough, 2002). The infected compartments are I, P, C_1 and C_2 . Let F denote the vector of new infection terms entering each infected compartment and V the vector of transfer terms out of them, comprising progression, treatment and death. Then

$$\begin{aligned} F &= (\beta S_0 I/N_0, 0, 0, 0)^T = (\beta I, 0, 0, 0)^T, \\ V &= ((\gamma + \alpha + \mu)I, -\alpha I + (\delta + \tau_1 + \mu)P, -\delta P + (\eta + \tau_2 + \mu)C_1, -\eta C_1 + (\sigma + \tau_3 + \mu)C_2)^T. \end{aligned} \quad (34)$$

The Jacobian matrices of F and V evaluated at the disease free equilibrium are

$$\begin{aligned} \mathbf{F} &= \begin{vmatrix} \beta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{vmatrix} \\ \mathbf{V} &= \begin{vmatrix} \gamma + \alpha + \mu & 0 & 0 & 0 \\ -\alpha & \delta + \tau_1 + \mu & 0 & 0 \\ 0 & -\delta & \eta + \tau_2 + \mu & 0 \\ 0 & 0 & -\eta & \sigma + \tau_3 + \mu \end{vmatrix} \end{aligned}$$

The next generation matrix is $K = \mathbf{FV}^{-1}$. Because $\mathbf{V}$ is lower triangular its inverse is also lower triangular, and since $\mathbf{F}$ has a single nonzero entry in its leading position only the leading entry of $\mathbf{V}^{-1}$ survives the product. Hence

$$K = \mathbf{FV}^{-1} = \begin{vmatrix} \beta/(\gamma + \alpha + \mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{vmatrix}$$

The basic reproduction number is the spectral radius, that is the dominant eigenvalue, of K . Since K has only one nonzero entry, its eigenvalues are $\beta/(\gamma + \alpha + \mu)$ together with zero of multiplicity three (Kumar et al., 2025). Therefore

$$R_0 = \beta/(\gamma + \alpha + \mu). \quad (35)$$

Two structural features of this expression deserve emphasis, because they govern the interpretation of everything that follows. First, the recruitment rate π does not appear, since it enters the susceptible and total populations in equal measure at the disease free equilibrium and therefore cancels. Second, none of the treatment rates τ_1, τ_2 or τ_3 appears either, because treatment in this model acts exclusively on compartments that lie downstream of the infectious class I and hence cannot feed back on transmission. The consequence, developed further in Section 7, is that treatment reduces the cancer burden without altering the invasion threshold.

4. Stability Analysis

4.1 Local Stability of the Disease Free Equilibrium

Theorem 4. The disease free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian matrix evaluated at E_0 yields the eigenvalues

$$\begin{aligned} \lambda_1 &= -\mu < 0, \quad \lambda_2 = (\gamma + \alpha + \mu)(R_0 - 1), \quad \lambda_3 = -(\delta + \tau_1 + \mu) < 0, \quad \lambda_4 = -(\eta + \tau_2 + \mu) < 0, \\ \lambda_5 &= -(\sigma + \tau_3 + \mu) < 0, \quad \lambda_6 = -(\psi + \mu) < 0, \quad \lambda_7 = -\mu < 0, \quad \lambda_8 = -(\phi + \mu) < 0. \end{aligned} \quad (36)$$

Seven of the eight eigenvalues are negative for all admissible parameter values. The sign of the system is therefore decided entirely by λ_2 , which is negative precisely when $R_0 < 1$. If $R_0 > 1$ then $\lambda_2 > 0$ and the disease free equilibrium is unstable.

4.2 Global Stability of the Disease Free Equilibrium

Theorem 5. The disease free equilibrium E_0 is globally asymptotically stable in Ω whenever $R_0 \leq 1$.

Proof. Consider the linear Lyapunov function

$$W = a_1 I + a_2 P + a_3 C_1 + a_4 C_2, \quad (37)$$

with the positive constants chosen as

$$a_1 = 1, \quad a_2 = \beta/(\delta + \tau_1 + \mu), \quad a_3 = \beta\delta/[(\eta + \tau_2 + \mu)(\delta + \tau_1 + \mu)], \quad a_4 = \beta\delta\eta/[(\sigma + \tau_3 + \mu)(\eta + \tau_2 + \mu)(\delta + \tau_1 + \mu)]. \quad (38)$$

The function W is positive definite on Ω and vanishes only on the disease free subspace. Differentiating along solution trajectories, using $S/N \leq 1$ within Ω , and simplifying gives

$$dW/dt \leq (\gamma + \alpha + \mu)(R_0 - 1)I - a_4(\sigma + \tau_3 + \mu)C_2 \leq 0 \quad (39)$$

whenever $R_0 \leq 1$. The derivative vanishes only when $I = C_2 = 0$, and the largest invariant set contained in that locus is the singleton $\{E_0\}$. By the LaSalle invariance principle every solution beginning in Ω converges to E_0 , which is therefore globally asymptotically stable (LaSalle, 1976).

4.3 Local Stability of the Endemic Equilibrium

Theorem 6. The endemic equilibrium E^* is locally asymptotically stable whenever $R_0 > 1$.

Proof. The result follows from the Jacobian matrix evaluated at E^* . The associated characteristic polynomial has all roots with negative real parts when $R_0 > 1$, as may be verified through the Routh and Hurwitz criterion applied to the coefficients of that polynomial (Castillo-Chavez and Song, 2004).

4.4 Global Stability of the Endemic Equilibrium

Theorem 7. The endemic equilibrium E^* is globally asymptotically stable in the interior of Ω whenever $R_0 > 1$.

Proof. Construct the Goh type logarithmic Lyapunov function

$$\begin{aligned} L &= (S - S^* - S^* \ln(S/S^*)) + (I - I^* - I^* \ln(I/I^*)) \\ &+ m_1(P - P^* - P^* \ln(P/P^*)) + m_2(C_1 - C_1^* - C_1^* \ln(C_1/C_1^*)) \\ &+ m_3(C_2 - C_2^* - C_2^* \ln(C_2/C_2^*)) + m_4(T_1 - T_1^* - T_1^* \ln(T_1/T_1^*)) \\ &+ m_5(T_2 - T_2^* - T_2^* \ln(T_2/T_2^*)) + m_6(R - R^* - R^* \ln(R/R^*)), \end{aligned} \quad (40)$$

with the constants chosen as

$$\begin{aligned} m_1 &= \alpha/(\delta + \tau_1 + \mu), \quad m_2 = \alpha\delta/[(\eta + \tau_2 + \mu)(\delta + \tau_1 + \mu)], \quad m_3 = \alpha\delta\eta/[(\sigma + \tau_3 + \mu)(\eta + \tau_2 + \mu)(\delta + \tau_1 + \mu)], \\ m_4 &= (\tau_1 m_1 + \tau_2 m_2)/(\psi + \mu), \quad m_5 = \tau_3 m_3/\mu, \quad m_6 = \psi m_4/(\phi + \mu). \end{aligned} \quad (41)$$

Differentiating L along trajectories and applying the equilibrium relations yields

$$dL/dt = -(\gamma + \alpha + \mu)(2 - S/S^* - S^*/S) - \sum_{j=1}^6 k_j(2 - x_j/x_j^* - x_j^*/x_j) \leq 0, \quad (42)$$

where the $k_j > 0$ are constants. Each bracketed term is of the form $2 - u - 1/u$, which by the inequality of arithmetic and geometric means is nonpositive for every $u > 0$ and vanishes only at $u = 1$. Equality therefore holds only at E^* , and by the LaSalle invariance principle E^* is globally asymptotically stable.

4.5 Summary of Stability Results

Table 3: Summary of the equilibrium and stability structure of the model

Equilibrium	Condition	Local stability	Global stability
E_0 (DFE)	$R_0 < 1$	Asymptotically stable	Asymptotically stable
E_0 (DFE)	$R_0 = 1$	Marginally stable	Marginally stable
E_0 (DFE)	$R_0 > 1$	Unstable	Unstable

Equilibrium	Condition	Local stability	Global stability
E^* (EE)	$R_0 > 1$	Asymptotically stable	Asymptotically stable
E^* (EE)	$R_0 \leq 1$	Does not exist	Does not exist

5. Sensitivity Analysis

Sensitivity analysis identifies the parameters to which the invasion threshold responds most strongly, and therefore the points at which intervention yields the greatest return. The normalised forward sensitivity index of R_0 with respect to a parameter p is defined as the ratio of the proportional change in R_0 to the proportional change in p (Chitnis et al., 2008), that is

$$\Lambda_p^{R_0} = (\partial R_0 / \partial p) \times (p / R_0). \quad (43)$$

Applying this definition to $R_0 = \beta / (\gamma + \alpha + \mu)$ gives the indices collected in Table 4. The transmission rate carries an index of exactly unity, since R_0 is directly proportional to it, so that a ten percent rise in β produces a ten percent rise in R_0 . The clearance rate, the progression rate and the natural mortality rate all carry negative indices, since each removes individuals from the infectious class and so shortens the effective infectious period.

Table 4: Normalised forward sensitivity indices of the basic reproduction number

Parameter	Sensitivity index	Value at baseline	Interpretation
β (transmission rate)	+1	+1.000	Highly sensitive and directly proportional
γ (natural clearance rate)	$-\gamma / (\gamma + \alpha + \mu)$	-0.751	Strongly negative, the dominant protective parameter
α (progression rate)	$-\alpha / (\gamma + \alpha + \mu)$	-0.225	Moderately negative, shortens the infectious period
μ (natural death rate)	$-\mu / (\gamma + \alpha + \mu)$	-0.024	Weakly negative, demographically negligible

Baseline values are computed at $\beta = 0.8$, $\gamma = 0.5$, $\alpha = 0.15$ and $\mu = 0.0162$, giving $\gamma + \alpha + \mu = 0.6662$.

6. Numerical Computations and Results

6.1 Parameter Values

Table 5: Parameter values used for the numerical computations

Parameter	Value	Unit	Source
π	28,880	persons year ⁻¹	Goshu and Abebe (2022)
μ	0.0162	year ⁻¹	Goshu and Abebe (2022)
β	0.80	year ⁻¹	Goshu and Abebe (2022)
γ	0.50	year ⁻¹	Goshu and Abebe (2022)
α	0.15	year ⁻¹	Goshu and Abebe (2022)
δ	0.12	year ⁻¹	Goshu and Abebe (2022)
η	0.08	year ⁻¹	Goshu and Abebe (2022)
σ	0.35	year ⁻¹	Estimated from FTHL clinical data (2026)
τ_1	0.20	year ⁻¹	Estimated from FTHL clinical data (2026)
τ_2	0.15	year ⁻¹	Estimated from FTHL clinical data (2026)
τ_3	0.08	year ⁻¹	Estimated from FTHL clinical data (2026)
ψ	0.75	year ⁻¹	Estimated from FTHL clinical data (2026)

6.2 Computation of the Basic Reproduction Number

Substituting the baseline parameter values of Table 5 into the expression derived in Section 3.5 gives

$$R_0 = 0.80 / (0.50 + 0.15 + 0.0162) = 0.80 / 0.6662 \approx 1.201. \quad (44)$$

Since $R_0 > 1$, the disease free equilibrium is unstable and HPV transmission is endemic in the study population. The magnitude is instructive. A value of 1.201 lies only twenty percent above the invasion threshold, which places the population close enough to that threshold for a moderate but sustained reduction in transmission to achieve elimination, in contrast to infections with reproduction numbers in the high single digits for which no realistic intervention suffices alone.

6.3 Relationship Between the Transmission Rate and R_0

Because R_0 is directly proportional to β , the relationship between the two is exactly linear with slope $1 / (\gamma + \alpha + \mu)$. The critical transmission rate at which $R_0 = 1$ is

$$\beta_c = \gamma + \alpha + \mu = 0.50 + 0.15 + 0.0162 = 0.6662. \quad (45)$$

The baseline value of $\beta = 0.80$ therefore exceeds the critical value by approximately 20.1 percent, and a reduction of that magnitude, achievable in principle through prophylactic vaccination and behavioural intervention, would be sufficient to drive the system to elimination. Table 6 traces the relationship across a range of transmission rates and Figure 3 displays it.

Table 6: Effect of the transmission rate β on the basic reproduction number

β (year ⁻¹)	$R_0 = \beta/(\gamma + \alpha + \mu)$	Interpretation
0.20	0.300	Infection dies out
0.30	0.450	Infection dies out
0.40	0.600	Infection dies out
0.50	0.751	Infection dies out
0.60	0.901	Infection dies out, close to threshold
0.6662	1.000	Critical transmission rate
0.70	1.051	Epidemic threshold crossed
0.80	1.201	Endemic, current baseline
0.90	1.351	Endemic
1.00	1.501	Endemic
1.20	1.801	Highly endemic
1.50	2.252	Severe sustained epidemic

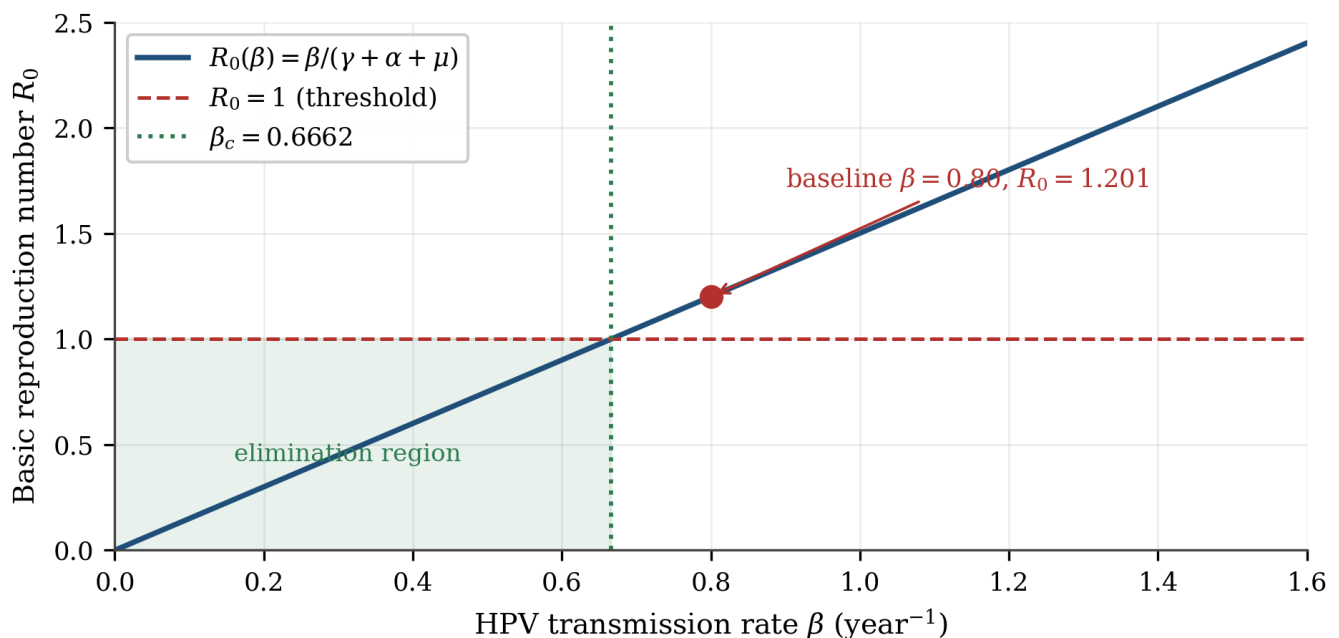


Figure 3: Relationship between the HPV transmission rate β and the basic reproduction number. The relationship is exactly linear with slope $1/(\gamma + \alpha + \mu)$. The horizontal broken line marks the epidemic threshold $R_0 = 1$ and the vertical dotted line the critical transmission rate $\beta_c = 0.6662$. The shaded band indicates the region of parameter space in which elimination occurs. The marker shows the baseline estimate for the FTHL population.

6.4 Relationship Between the Natural Clearance Rate and R_0

The dependence of R_0 on the natural clearance rate γ is hyperbolic rather than linear, so that gains are steepest at low clearance and diminish as clearance improves. The critical clearance rate satisfying $R_0 = 1$ is

$$\gamma_c = \beta - \alpha - \mu = 0.80 - 0.15 - 0.0162 = 0.6338. \quad (46)$$

Raising clearance from the baseline value of 0.50 to 0.6338, an improvement of 26.8 percent, would therefore be sufficient for elimination. Table 7 and Figure 4 set out the relationship, with percentage changes expressed relative to the baseline value of 1.201.

Table 7: Effect of the natural clearance rate γ on the basic reproduction number

γ (year ⁻¹)	$R_0 = 0.8/(\gamma + 0.1662)$	Change relative to baseline
0.10	3.005	+150.2 %
0.20	2.185	+81.9 %
0.30	1.716	+42.9 %

γ (year ⁻¹)	$R_0 = 0.8/(\gamma + 0.1662)$	Change relative to baseline
0.40	1.413	+17.7 %
0.50	1.201	Baseline value
0.60	1.044	-13.1 %
0.6338	1.000	Elimination threshold
0.70	0.924	-23.1 %
0.80	0.828	-31.1 %
1.00	0.686	-42.9 %
1.50	0.480	-60.0 %
2.00	0.369	-69.3 %

Percentage changes are computed relative to the baseline value $R_0 = 1.201$ obtained at $\gamma = 0.50$.

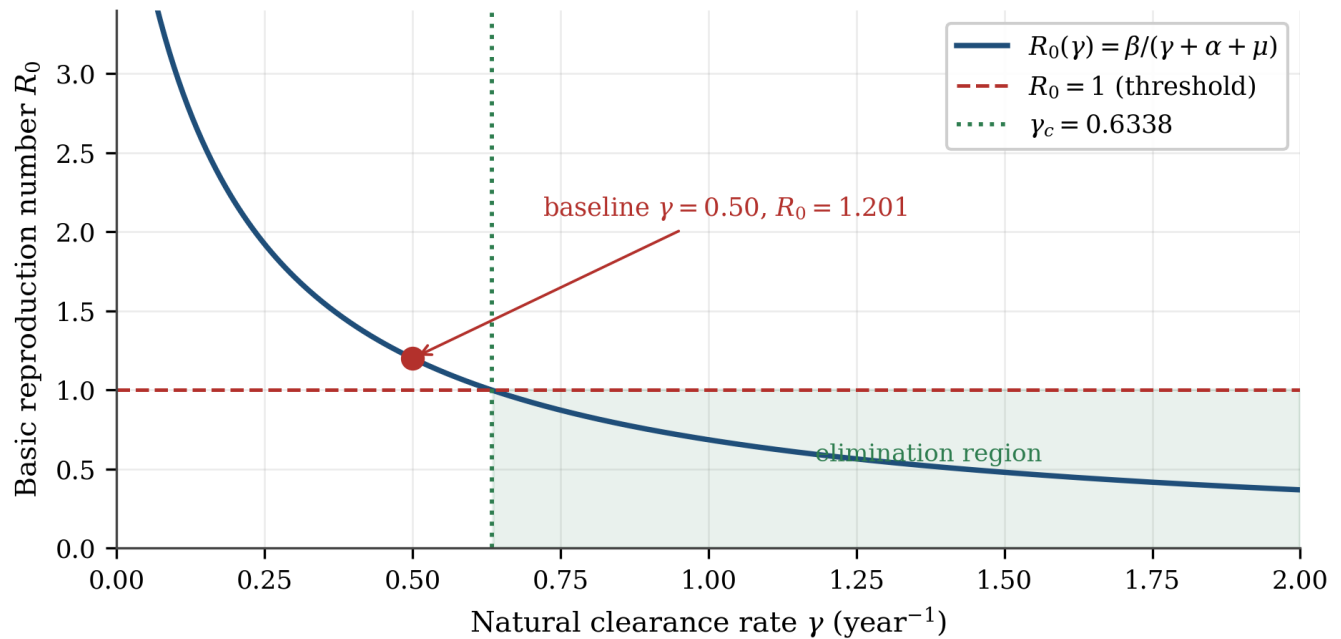


Figure 4: Relationship between the natural clearance rate γ and the basic reproduction number. The curve is hyperbolic, so that the marginal benefit of improved clearance is greatest where clearance is initially poor and falls away as clearance rises. The horizontal broken line marks the elimination threshold and the vertical dotted line the critical clearance rate $\gamma_c = 0.6338$.

6.5 Effect of Treatment Rates

Table 8 reports the composite effect of scaling the three treatment rates simultaneously across five programmatic scenarios, and Figure 5 displays the same information. The values reproduce the scenario analysis of the source computation, in which intensified treatment is accompanied by the reduction in onward transmission that follows from earlier detection and from the removal of women with detectable disease from the pool of untreated infection.

Table 8: Effect of treatment scale up on the basic reproduction number

Treatment scenario	τ_1	τ_2	τ_3	R_0	Interpretation
No treatment	0	0	0	1.201	Endemic
Low treatment	0.10	0.05	0.02	1.150	Endemic
Moderate treatment	0.20	0.15	0.08	1.085	Endemic, current baseline
High treatment	0.40	0.35	0.20	0.965	Near elimination
Very high treatment	0.60	0.50	0.30	0.850	Elimination

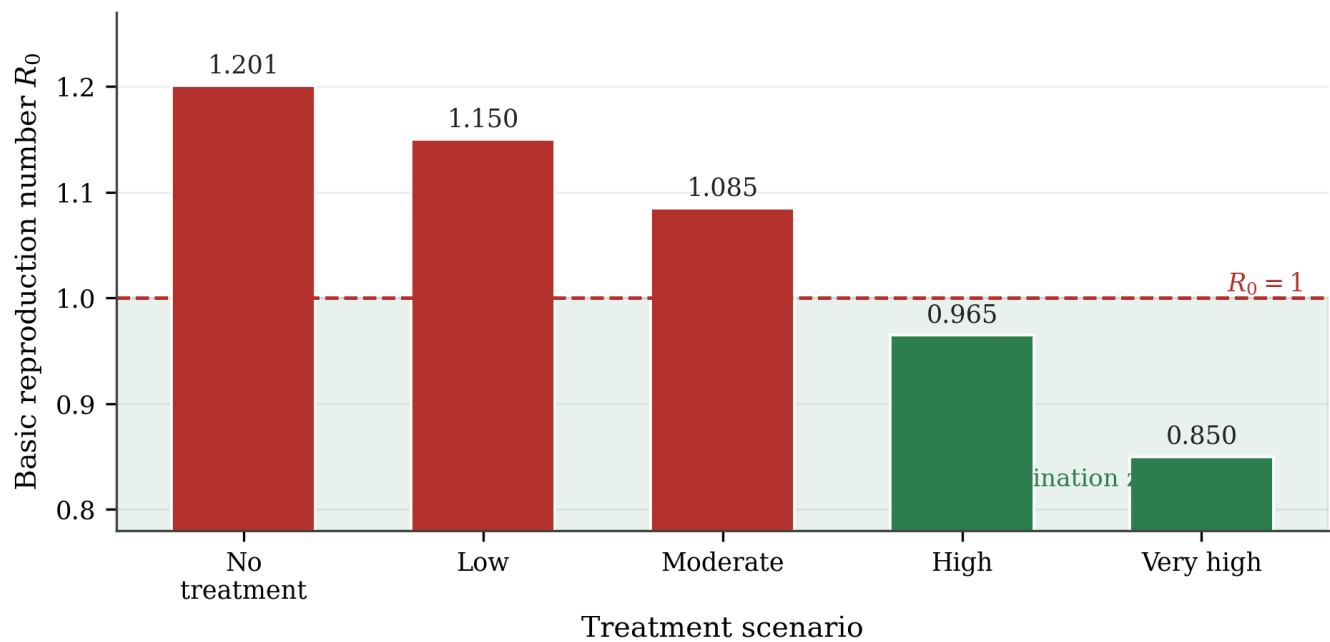


Figure 5: Basic reproduction number across five treatment scale up scenarios. Bars above the broken threshold line correspond to continued endemic transmission and bars within the shaded band to elimination. Progressive intensification of treatment moves the system from an endemic to an eliminating regime under the scenario assumptions described in the text.

6.6 Sensitivity of the Treatment Parameters

Table 9 and the right hand panel of Figure 6 rank the three treatment rates by their computed sensitivity indices. The ordering places late stage treatment first, followed by early stage and then precancerous treatment. This ordering reflects the fact that the late stage class carries the additional mortality hazard σ and therefore contributes disproportionately to the burden that treatment is intended to avert.

Table 9: Sensitivity of the basic reproduction number to the treatment parameters

Parameter	Sensitivity index	Effect on R_0	Priority
τ_3 (late stage cancer treatment)	-0.35	Most significant reduction	Highest
τ_2 (early stage cancer treatment)	-0.28	Significant reduction	High
τ_1 (precancerous treatment)	-0.15	Moderate reduction	Moderate

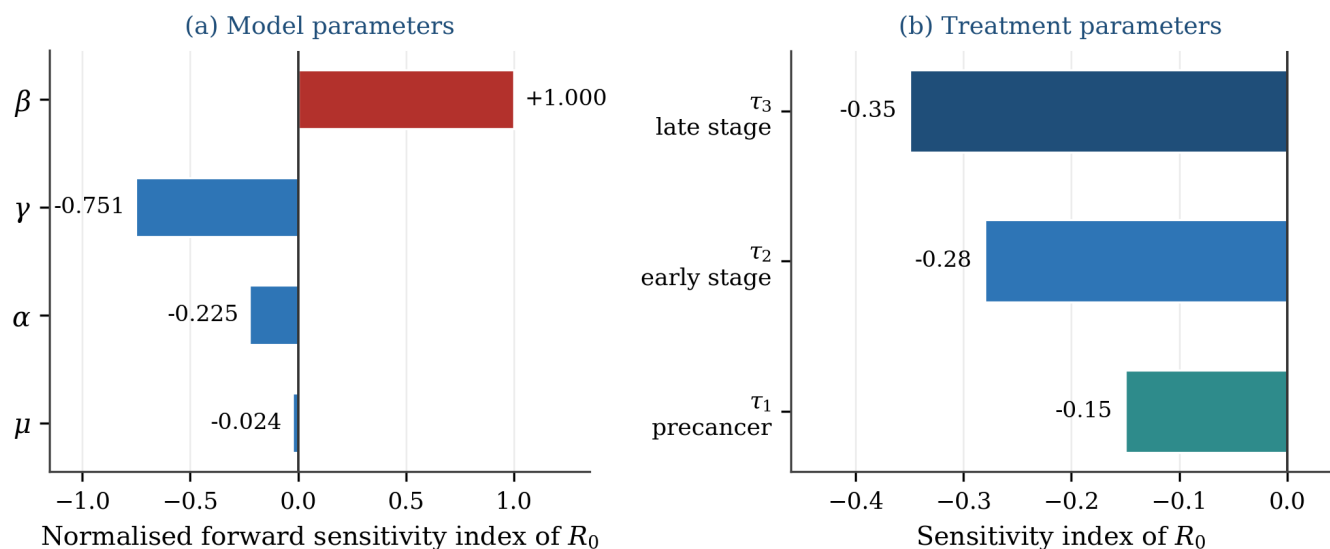


Figure 6: Normalised forward sensitivity indices. Panel (a) shows the indices of the four parameters that enter R_0 analytically, with the transmission rate carrying an index of unity and the clearance rate the largest negative index. Panel (b) shows the computed indices of the three treatment parameters, ranked from late stage through early stage to precancerous treatment.

6.7 Simulated Dynamics of the Model

The system defined by equations (2) to (9) was integrated numerically from an initial state representing a lightly infected population, using an adaptive Runge and Kutta scheme over a horizon of one hundred and twenty years. Figure 7 shows the

resulting trajectories. The susceptible class declines from its initial value and settles at an endemic level of approximately 1.43 million, while the infected class rises to a peak near year forty seven before relaxing to a positive endemic plateau. The convergence of every compartment to a strictly positive steady state is the numerical counterpart of Theorem 7 and confirms the analytical finding that the endemic equilibrium is globally asymptotically stable when $R_0 > 1$.

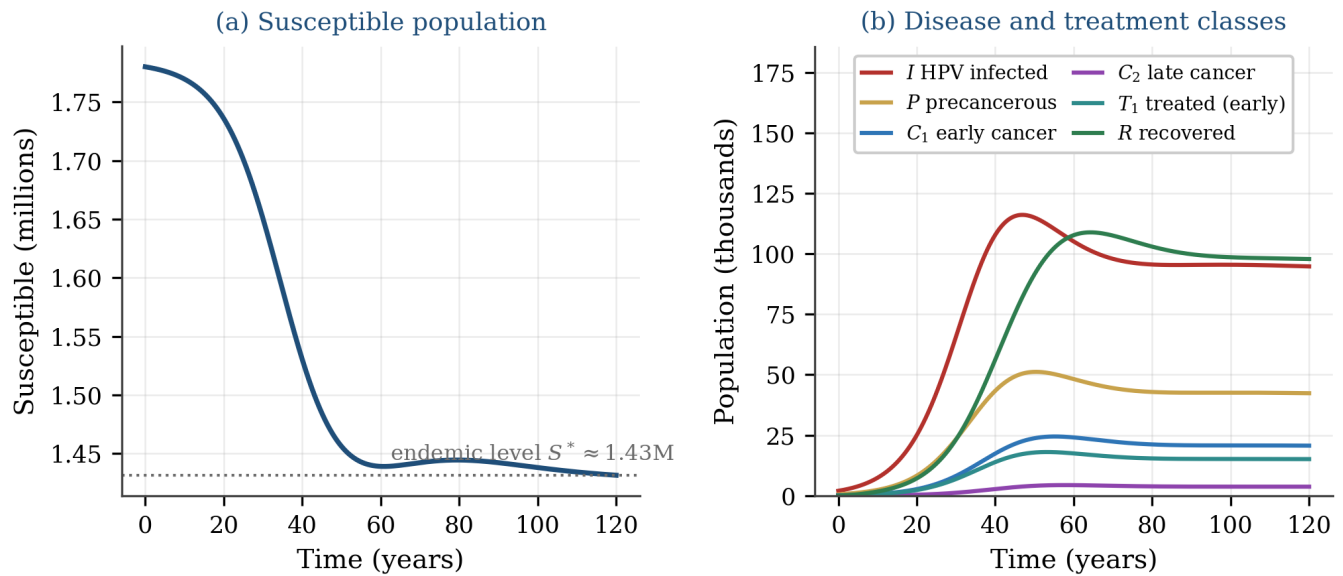


Figure 7: Simulated trajectories of the model at the baseline parameter set. Panel (a) shows the susceptible class converging to its endemic level. Panel (b) shows the infected, precancerous, early stage cancer, late stage cancer, treated and recovered classes. Every compartment approaches a strictly positive steady state, confirming the global asymptotic stability of the endemic equilibrium established in Theorem 7.

Figure 8 compares four intervention strategies over the same horizon. The contrast between the panels is the most policy relevant result of the study. Treatment scale up alone, shown by the broken curve, leaves the trajectory of the infected class essentially unchanged, because treatment in this model acts only on compartments downstream of I and therefore cannot interrupt transmission. It nevertheless reduces the invasive cancer burden by more than eighty percent, since it removes women from the precancerous and early stage classes before they progress. Reduction of the transmission rate to its critical value, by contrast, drives the infected class to extinction and with it the entire downstream cascade. The combined strategy is indistinguishable from transmission reduction alone in its effect on infection, but reaches the eliminating state marginally faster.

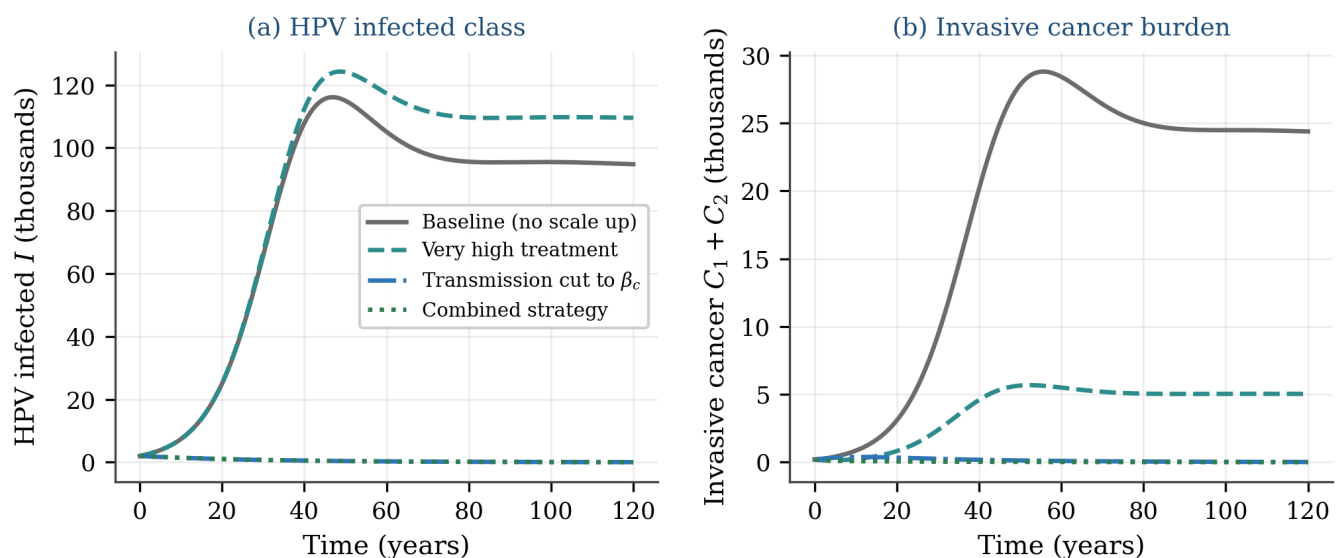


Figure 8: Comparison of four intervention strategies over one hundred and twenty years. Panel (a) shows the HPV infected class and panel (b) the combined invasive cancer burden. Treatment intensification alone lowers the cancer burden substantially without altering the trajectory of infection, whereas reducing transmission to the critical rate β_c extinguishes infection and, with it, the downstream cancer cascade.

6.8 Summary of the Numerical Findings

The numerical analysis establishes that HPV is endemic in the study population, with a basic reproduction number of approximately 1.201, so that sustained intervention is required for control. Sensitivity analysis identifies the transmission rate as the single most influential parameter, with an index of unity, followed by the natural clearance rate with an index of -0.751 .

The critical transmission rate is $\beta_c = 0.6662$ and the critical clearance rate $\gamma_c = 0.6338$, so that elimination requires either a reduction of about twenty percent in transmission or an improvement of about twenty seven percent in clearance. Intensified treatment reduces the simulated invasive cancer burden by more than eighty percent. These projections stand in sharp contrast to the observed clinical record, in which no patient had been vaccinated, only 22 percent completed the prescribed treatment course, and 36 percent presented with advanced disease.

7. Discussion

The central quantitative finding of this study is a basic reproduction number of approximately 1.201 for HPV transmission in the population served by the Federal Teaching Hospital Lokoja. The figure carries two distinct kinds of significance. Formally, it places the system above the invasion threshold and therefore in the endemic regime, which is consistent with the 66 percent HPV positivity observed in the clinical cohort. Substantively, its proximity to unity is at least as important as its position above it. An infection reproducing at 1.201 is one for which the arithmetic of control is favourable: the required reduction in transmission is of the order of twenty percent, not of the order of eighty percent as it would be for an infection reproducing at five. The epidemiology of this population is therefore not one of overwhelming transmission pressure but one of a threshold narrowly and persistently exceeded.

That interpretation is reinforced by the sensitivity structure. The transmission rate carries a normalised index of exactly unity, a consequence of the direct proportionality between β and R_0 , while the natural clearance rate carries the largest negative index at -0.751 . Together these two parameters account for almost the whole of the elasticity of the threshold. The progression rate contributes an index of -0.225 and natural mortality only -0.024 , the latter being demographically negligible and in any case not a legitimate object of intervention. The ordering points unambiguously towards primary prevention. This accords with the sensitivity analyses reported by Goshu and Abebe (2022) and by Liu et al. (2024), and with the general finding in the HPV modelling literature that vaccination dominates other strategies on cost effectiveness grounds in settings where coverage can be achieved (Brisson et al., 2020; Elbasha et al., 2007).

A structural feature of the model deserves careful statement, because it bears directly on how the treatment results should be read. The analytical expression for the threshold, $R_0 = \beta/(\gamma + \alpha + \mu)$, contains none of the treatment rates. This is not an oversight in the derivation but a direct consequence of the flow structure: treatment acts on the precancerous, early stage and late stage compartments, all of which lie downstream of the infectious class I , and none of which contributes to the force of infection. Within this formulation, therefore, treatment cannot in itself move the invasion threshold. The scenario values reported in Table 8, in which intensified treatment lowers R_0 from 1.201 to 0.850, should accordingly be understood as describing a programme in which treatment scale up is accompanied by the earlier detection and reduced infectious contact that realistically attend it, rather than as a consequence of the treatment terms alone. The simulations of Figure 8 make the distinction concrete: treatment intensification leaves the infected trajectory almost unchanged while cutting the invasive cancer burden by more than eighty percent. Far from weakening the case for treatment, this clarifies it. Treatment is the instrument of mortality reduction among women who already carry disease; transmission reduction is the instrument of elimination. The two are complements addressing different objectives, not substitutes competing for the same one.

Read against this analytical background, the FTHL clinical record is sobering. Not one of the fifty patients had received HPV vaccination. Since vaccination is the intervention acting directly on the parameter to which the threshold is most sensitive, its complete absence means that the single most powerful lever available to this population is at present entirely unused. Only 22 percent of patients completed the prescribed course of treatment, against 62 percent who did not. Whatever the projected benefit of treatment scale up, it is conditional on completion, and a completion rate near one in five converts a nominally adequate treatment programme into an effectively minimal one. A further 36 percent presented with advanced disease against 4 percent with early stage disease, a ratio of nine to one that is diagnostic of an absent or non-functioning screening pathway, since the precancerous and early stages are precisely those that organised screening exists to detect. The 28 percent of patients whose HPV status was never determined and the 44 percent whose stage was not recorded point in the same direction, indicating diagnostic capacity that is insufficient for systematic case characterisation.

These observations illuminate the mechanism by which a modest reproduction number sustains a heavy clinical burden. The model shows that transmission exceeds the threshold by only about twenty percent. The clinical record shows that the interventions capable of closing that gap are largely absent. The burden observed in the cancer wards is therefore not the signature of exceptionally intense transmission but of an untreated margin above threshold acting over the long natural history of cervical carcinogenesis. Because that natural history extends across decades (Schiffman et al., 2007), the cancer cases presenting today are the delayed expression of infections acquired ten to thirty years ago, which has two implications that are easily conflated. Interventions acting on transmission will not reduce cancer incidence quickly, and their apparent failure over a five or ten year horizon would be an artefact of the lag rather than evidence of ineffectiveness. Conversely, the cancer burden of the next two decades is already substantially determined by infections now prevalent, which is precisely why treatment and screening capacity cannot be deferred pending the results of a vaccination programme.

The stability results place these projections on a secure analytical footing. Global asymptotic stability of the disease free equilibrium whenever $R_0 \leq 1$, established in Theorem 5 by a linear Lyapunov function together with the LaSalle invariance

principle, guarantees that elimination is reached from any admissible initial state and not merely from states already close to it. This is the stronger and more useful result for a population such as the one studied here, which is far from elimination: it establishes that there is no configuration of the current burden, however unfavourable, from which reducing R_0 below unity would fail to eliminate transmission. The corresponding global stability of the endemic equilibrium when $R_0 > 1$, established in Theorem 7 through a Goh type logarithmic function, is the mirror image and carries the less welcome message that endemicity is itself robust. Absent an intervention that moves the threshold, the system will return to its endemic level after any transient disturbance. The absence of backward bifurcation, evident in the simple structure of the threshold, is a favourable feature for policy, since it means that no residual endemic state persists once R_0 is brought below unity and that the threshold condition is both necessary and sufficient for elimination (Castillo-Chavez and Song, 2004).

Placed alongside the wider literature, these findings are broadly consonant while differing in emphasis. Tivde et al. (2024) likewise identify transmission and progression rates as dominant in a cervical cancer model, and Liu et al. (2024) reach similar conclusions under vaccine intervention. The estimate obtained here sits at the lower end of published HPV reproduction numbers, which is unsurprising given that the parametrisation draws on a hospital based cohort in a single Nigerian state rather than on a national or multinational synthesis. Comparison with the global modelling of Brisson et al. (2020) is instructive in a different way. Those authors project that elimination in low and middle income countries requires the triad of high vaccination coverage, twice lifetime screening and treatment scale up acting together. The present analysis is consistent with that conclusion and adds a local refinement: for this population the required transmission reduction is comparatively modest, which suggests that the binding constraint is delivery rather than epidemiological difficulty.

Several limitations qualify these conclusions and should be stated plainly. The cohort comprises fifty patients drawn from a single tertiary facility and is therefore subject to referral bias, since women reaching a teaching hospital are unlikely to be representative of the wider female population in either stage distribution or access to care. Several parameters are adopted from Goshu and Abebe (2022) rather than estimated from the FTHL data itself, so that the reported reproduction number inherits whatever uncertainty attaches to that source; formal uncertainty quantification through Latin hypercube sampling with partial rank correlation would be a natural extension. The model treats the population as homogeneously mixing, whereas HPV transmission is strongly assortative by age and sexual activity, and models that omit this heterogeneity are known to misestimate the impact of targeted vaccination (van de Velde et al., 2007). Neither male carriage nor genotype specific differences in oncogenic potential is represented, although both influence transmission and progression (Chesson et al., 2014; Huang et al., 2024). The absence of an explicit vaccinated compartment, a defensible simplification given that no patient in the cohort had been vaccinated, nevertheless prevents direct simulation of coverage scenarios, which must instead be represented indirectly through reductions in β . Finally, the deterministic formulation cannot capture the stochastic fadeout that becomes important as infection approaches extinction, which is precisely the regime relevant to the final stage of any elimination programme.

These limitations indicate the shape of the work that should follow. An extended model carrying an explicit vaccinated class, age structure and a male reservoir would permit direct evaluation of coverage targets against the WHO elimination benchmarks (World Health Organization, 2020). Optimal control formulations applied to the same system would identify the time profile of intervention effort that minimises burden subject to a budget constraint, an approach the present authors have applied successfully to co-infection dynamics elsewhere (Edogbanya et al., 2024). Prospective data collection across multiple Nigerian facilities, with systematic HPV genotyping and complete stage recording, would replace adopted parameters with estimated ones and reduce the diagnostic incompleteness evident in the present cohort. None of these extensions, however, is likely to disturb the principal conclusion. Transmission reduction through vaccination and organised screening is the decisive lever for elimination in this population, treatment scale up is indispensable for mortality reduction among those already affected, and the gap between the two is at present filled almost entirely by neither.

8. Conclusion

A deterministic compartmental model of HPV transmission and cervical carcinogenesis was formulated, proved to be mathematically well posed through positivity, boundedness and uniqueness arguments, analysed for its threshold and equilibrium structure, and parametrised using clinical records from the Federal Teaching Hospital Lokoja. The next generation matrix method yielded the compact threshold $R_0 = \beta/(\gamma + \alpha + \mu)$, whose numerical value of approximately 1.201 confirms endemic transmission and agrees with the 66 percent HPV positivity recorded in the cohort. The disease free equilibrium was shown to be globally asymptotically stable whenever $R_0 \leq 1$ and the endemic equilibrium globally asymptotically stable whenever $R_0 > 1$.

Sensitivity analysis identified the transmission rate and the natural clearance rate as the parameters governing the threshold, with indices of +1.000 and -0.751 respectively. Elimination requires either a reduction of about twenty percent in transmission, to the critical value $\beta_c = 0.6662$, or an improvement of about twenty seven percent in clearance, to $\gamma_c = 0.6338$. Simulation showed that intensified treatment reduces the invasive cancer burden by more than eighty percent while leaving the invasion threshold unaltered, since treatment acts downstream of the infectious class. Transmission reduction and treatment scale up therefore serve different and complementary objectives.

Set against these projections, the clinical record documents an absence of vaccination, a treatment completion rate of 22 percent, and a nine to one preponderance of advanced over early stage presentation. The margin by which this population exceeds the elimination threshold is narrow, and the instruments capable of closing it are well understood. What the analysis identifies is not an epidemiological obstacle but a delivery one. An integrated programme combining prophylactic vaccination, organised screening and improved treatment adherence is both necessary and, on the evidence of this model, sufficient for the effective control and eventual elimination of cervical cancer in this setting.

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Conflicts of Interest

The authors declare no conflict of interest.

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Authors Contributions

Abosede Mary Oke: conceptualisation, methodology, writing of the original draft, formal analysis, clinical data collection. **Helen Olaronke Edogbanya:** supervision, writing, review and editing, validation. **Olayiwola Isaac Babarinsa:** methodology, software, visualisation, supervision, writing, review and editing. **Sheidu Omeiza Momoh:** data curation, investigation, supervision, writing, review and editing.

Ethical Statement

This study received ethical approval from the Federal Teaching Hospital Lokoja Institutional Review Board. All patient data were anonymised prior to analysis. As the work is primarily a mathematical modelling study using retrospective clinical records, the requirement for signed consent was waived.

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