

Dynamics and Control of Human Papillomavirus (HPV) Infection Using an SVITR Compartmental Model

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ABSTRACT

Human papillomavirus (HPV) remains a significant public health concern due to its high transmissibility and associated health risks. This study underscores the pivotal role of vaccination in reducing HPV transmission, while also highlighting the limitations of relying solely on vaccination for infection control. In this study, we present a deterministic compartmental model to investigate the transmission dynamics of Human Papillomavirus (HPV). The model stratifies the population into five compartments: susceptible individuals $S(t)$, Vaccinated individuals $V(t)$, HPV Infected individuals $I(t)$, treated HPV-infected individuals $T(t)$ and recovered individuals $R(t)$. We establish the existence and uniqueness of the model solution and also examine the existence of disease-free and endemic equilibrium and analyze their stability properties. Numerical simulations were performed to explore the temporal evolution of the compartments, assess the sensitivity of key parameters, and investigated the behaviour of the basic reproduction number \mathcal{R}_0 . Our findings were that a comprehensive strategy, incorporating both preventive vaccination and therapeutic management, is essential for achieving sustainable control of HPV spread. Strengthening these measures, alongside reducing transmission through demographic interventions, offers the best way for long-term management of the infection. These results provide insights into the impact of vaccination and treatment strategies on HPV transmission and highlight critical factors for public health.



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I. INTRODUCTION

The mathematical modeling of infectious diseases and their spread within populations has its origins in the pioneering work of several scientists. In the 18th century, Daniel Bernoulli was among the first to formalize a mathematical model aimed at estimating the impact of variolation on smallpox-related mortality [5]. This early contribution laid the groundwork for epidemiological modeling by introducing a probabilistic approach to disease prevention [4].

In the early 20th century, Ronald Ross advanced the field by developing models describing the transmission of malaria.

His research highlighted the critical role of vectors in disease dynamics and emphasized the importance of public health interventions in controlling infections [22].

Later, Kermack and McKendrick [18] introduced compartmental models, notably the well-known SIR model, to study the spread of diseases such as measles. Their framework structured epidemic analysis around transitions between health states susceptible, infected, and recovered paving the way for more systematic and predictive approaches to modeling infectious phenomena.

Human papillomavirus (HPV) is one of the most widespread sexually transmitted infections worldwide and is implicated in the etiology of several cancers, notably cervical, anal, and oropharyngeal cancers. Epidemiological analyses reported in [13] highlight the high prevalence of HPV and marked regional disparities, particularly in low-resource settings where access to vaccination, screening, and healthcare remains insufficient. These observations underline the need for analytical tools capable of assessing the potential impact of prevention and management strategies in constrained healthcare contexts.

In this framework, mathematical modeling has emerged as a central tool for analyzing HPV transmission dynamics and informing public health policies. Numerous studies have shown that deterministic compartmental models provide a coherent representation of infection and prevention processes, particularly for evaluating vaccination strategies [8,19].

Moreover, some studies have explicitly incorporated the distinction between treated and untreated HPV-infected individuals and have analyzed the impact of treatment on HPV transmission by comparing scenarios with and without therapeutic intervention [23]. These studies highlight the potential role of treatment in reducing transmission, particularly through decreased infectivity. However, the analysis is mainly limited to the computation of the basic reproduction number, without a comprehensive qualitative analysis of the model, such as the study of solution positivity, invariance of the biologically feasible domain, or equilibrium stability. This limitation justifies the need to develop simpler frameworks that allow for a more exhaustive and rigorous mathematical analysis.

In addition, several studies have revealed nonlinear dynamic behaviors, such as the existence of backward bifurcations in the presence of imperfect vaccination and treatment, suggesting that the classical condition $\mathcal{R}_0 < 1$ may not be sufficient to guarantee disease elimination [3]. At the same time, the strong variability of biological and epidemiological parameters reported in the literature [2] emphasizes the need to develop flexible models capable of adapting to local realities and healthcare system constraints.

It is within this context that the present study is situated. We develop a simplified SVITR-type compartmental mathematical model to analyze HPV transmission dynamics within a female population. The model is structured around five compartments representing susceptible (S), vaccinated (V), infected (I), treated infected (T), and recovered (R) individuals. This deliberately reduced formulation preserves the essential mechanisms of HPV transmission dynamics while avoiding the excessive complexity of more detailed natural-history models.

The structure of the proposed model is grounded in biological and epidemiological assumptions consistent with the natural history of HPV infection. After infection, individuals enter the infected compartment I , which primarily represents asymptomatic and undetected infections. A proportion of these individuals is identified through screening

programs, medical consultations, or health surveillance initiatives and subsequently transitions to the treated infected compartment T at rate γ_1 . The remaining infected individuals may clear the virus naturally through an effective immune response without medical intervention, a process described by the natural recovery rate γ_2 . Individuals in compartment T may recover following successful treatment and move to the recovered compartment R at rate η . These assumptions capture the generally persistent yet often silent nature of HPV infection, while explicitly accounting for the role of treatment in disease control.

The main originality of this work lies in the explicit extension of the classical SIR and SVIR frameworks through the introduction of a specific compartment dedicated to infected individuals under treatment. Unlike many previous approaches in which treatment is incorporated implicitly or neglected, the T compartment allows for a direct quantification of the impact of treatment on HPV transmission, particularly through reduced infectivity. Grouping high-risk and low-risk HPV infections under treatment also reflects current clinical practice while maintaining a mathematically simple and analytically tractable structure.

The contribution of this study is therefore twofold. On the one hand, it is structural, proposing a minimalistic SVITR formulation that is more informative than classical SVIR models. On the other hand, it is mathematical, through a comprehensive qualitative and quantitative analysis of the model, including the existence and uniqueness of solutions, positivity of solutions, invariance of the biologically feasible domain, boundedness of trajectories, and stability analysis of equilibria, particularly via the computation and analysis of the basic reproduction number \mathcal{R}_0 .

Finally, beyond the theoretical analysis, the developed model constitutes a relevant decision-support tool for public health. It allows the evaluation of the combined impact of vaccination and treatment on HPV transmission dynamics and is fully aligned with current strategies aimed at controlling HPV-associated cancers [29]. This approach is particularly suitable for low-resource settings, where simple, transparent, and easily interpretable mathematical models are required to effectively guide infection control and prevention strategies.

We conduct both quantitative and qualitative analyses of the model, including the study of positivity, invariance, boundedness, and stability of equilibria. We then perform detailed numerical simulations to illustrate the dynamical behaviour of the system, followed by a sensitivity analysis of the basic reproduction number \mathcal{R}_0 .

II. MATHEMATICAL FORMULATION

Our study uses a compartmental modelling framework, in which the population is divided into five compartments, each representing a different clinical stage of Human Papillomavirus (HPV). The Movement between these compartment reflects the natural progression of the disease,

enabling us to analyze the dynamics of HPV transmission over time.

The model incorporates the following assumptions :

A1. We consider a homogeneous mixture within the total individual population N at time t is divided into five (5) mutually exclusive subpopulation, each representing a specific clinical state in the progression from HPV infection. This compartmental structure enables the detailed analysis of the dynamics of individuals at different stages of infection with the disease:

$S(t)$, compartment of susceptible individuals, i.e but are not yet infected at time t ,

$V(t)$, compartment of individuals vaccinated at time t ,

$I(t)$, compartment of infected HPV individuals with no symptoms at time t ,

$T(t)$, compartment of infected HPV individuals receiving treatment at time t ,

$R(t)$, compartment of recovered HPV individuals at time t .

A2. Susceptible individuals are vaccinated with the vaccine efficacy rate range between $0 \leq \theta \leq 1$.

A3. Contact between susceptible and infectious is random.

A4. The standard incidence function $f(I, T)$ determining the rate of new infections is given by

$$\lambda = \frac{\beta(I + p T)}{N}$$

A5. All model parameters are shown in table 1 and are positive.

A6. High-risk and low-risk human papillomavirus (HPV) are classified in the compartments T .

A7. All compartments decrease due to natural mortality at the natural death rate μ .

A8. Compartment T , decrease according to the disease-specific mortality rates μ_1 .

A9. The total individual population is

$$N(t) = S(t) + V(t) + I(t) + T(t) + R(t)$$

The model resulting from these assumptions is given in figure 1 and system (1).

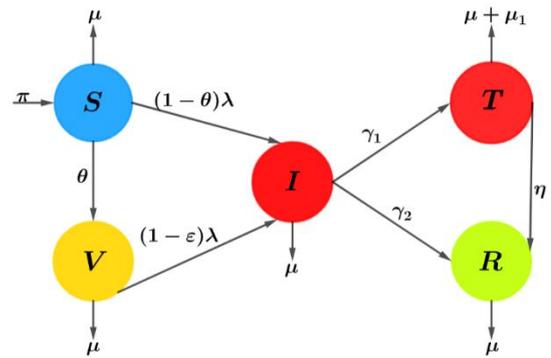


Figure 1. HPV transmission diagram

The transitions between compartments that make up the model's population are as follows:

The compartment of Susceptible increases by the natural recruitment of new sexually active individuals at a rate π . The population decreases due to several factors: a fraction θ of these individuals is vaccinated, while the remaining proportion $(1 - \theta)$ is not vaccinated; some individuals contract the infection after contact with infectious individuals, according to the force of infection λ , and the natural mortality rate μ .

The dynamics in the Susceptible (S) compartment is given by

$$S' = \pi - (1 - \theta)\lambda S - (\mu + \theta)S$$

The vaccinated compartment V increases at the vaccination rate θ but decreases because a proportion $(1 - \varepsilon)$ of vaccinated individuals can still become infected at a rate $(1 - \varepsilon)\lambda V$, and due to the natural mortality rate μ .

Based on the provided information, the dynamics in the Vaccinated (V) compartment is given by

$$V' = \theta S - (1 - \varepsilon)\lambda S - \mu V$$

The HPV-infected compartment I increases due to infections among susceptibles at the rate $(1 - \theta)\lambda S$ and among vaccinated individuals at the rate $(1 - \varepsilon)\lambda V$. It decreases due to the natural mortality rate μ , the HPV treatment rate γ_1 , and the HPV recovered rate γ_2 .

Thus, the dynamics in the HPV-infected compartment is expressed as follows:

$$I' = (1 - \theta)\lambda S + (1 - \varepsilon)\lambda V - (\mu + \gamma_1 + \gamma_2)I$$

The treated HPV-infected (T) compartment consists of individuals who are treated of HPV that we simplified the model by combining individuals with high-risk and low-risk HPV infections into the same compartment. The treated HPV-infected increases with the treatment of HPV rate γ_1 . It decreases due to the recovered rate of treated individuals η , the natural mortality rate μ , and the disease-induced mortality rate.

The dynamics in this compartment is given by:

$$T' = \gamma_1 I - (\eta + \mu + \mu_1)T$$

The Recovered R compartment increases due to the recovered of HPV-infected at the rate γ_2 and the recovered of

treated individuals at the rate η . It decreases due to the natural mortality rate μ .

The dynamics in this compartment is given by:

$$R' = \gamma_2 I + \eta T - \mu R$$

The overall dynamics of the epidemiological model is thus given by the following system of differential equations

$$\begin{cases} S' &= \Pi - (1 - \theta)\lambda S - \mu S - \theta S \\ V' &= \theta S - (1 - \epsilon)\lambda V - \mu V \\ I' &= (1 - \theta)\lambda S + (1 - \epsilon)\lambda V - (\mu + \gamma_1 + \gamma_2)I \\ T' &= \gamma_1 I - (\eta + \mu + \mu_1)T \\ R' &= \gamma_2 I + \eta T - \mu R \end{cases} \quad (1)$$

The system (1) is solved under the initial conditions:

$$(S(0), V(0), I(0), T(0), R(0)) \geq 0 \quad (2)$$

TABEL I

PARAMETERS AND THEIR DESCRIPTION

Parameter	Description
π	The recruitment of new sexually active individuals
β	Probability of transmission of HPV from male to female
ϵ	Vaccine efficacy rate
ρ	Reduction in infectivity of treated individuals
θ	Vaccination rate
μ	Natural death rate
μ_1	Disease-induced mortality rate due to HPV
γ_1	Rate of infectivity of HPV receiving treatment
γ_2	Recovered rate of HPV receiving no treatment
η	Recovered rate from HPV infected individuals

III. MATERIALS USING

The SVITR model (see Figure 1) is formulated as a system of ordinary differential equations (ODEs), presented in equation (1). The structural diagram of the model was produced using GeoGebra (version 6.0.848.0) on a computer equipped with an Intel(R) Celeron(R) N4500 @ 1.10 GHz processor, 20 GB of RAM, a 1 TB SSD, and running Windows 11.

In the quantitative analysis, we will show that the system of differential equations admits a unique solution, ensuring that the model's evolution is well defined for any biologically admissible initial condition.

The qualitative analysis was carried out using SageMath (version 9.2).

The following tasks were performed:

- Computation of equilibrium points;
- determination of the basic reproduction number \mathcal{R}_0 ;
- assessment of the local and global stability of both the disease-free and endemic equilibrium;
- sensitivity analysis of the parameters influencing \mathcal{R}_0 .

All these procedures were executed using SageMath (version 9.2).

For the numerical simulations, the use of reliable empirical data is essential. However, in the context of the Democratic Republic of Congo (DRC), epidemiological parameters specific to human papillomavirus (HPV) are not available in national databases or have not yet been systematically collected. This lack of information represents a major obstacle to calibrating the model directly using local data.

We therefore use estimated parameter values, based on data from similar countries or from published studies. The simulations are intended for exploratory purposes: to investigate the model's dynamics, examine the effect of key parameters, and explore potential control strategies, while acknowledging that the results may not exactly represent the situation in the DRC.

IV. QUANTITATIVE AND QUALITATIVE ANALYSIS OF SYSTEM

A. Results

In this section, we investigate the fundamental mathematical characteristic of HPV model. Our analysis focuses on the positivity and boundedness of solutions, the invariance of the feasible region, the existence of equilibrium points, the computation of the basic reproduction number, and analyzes of the stability of the equilibria from both local and global.

1. Well-posedness of the model system and boundary

Consider the following compact invariant domain

$$\Omega = \left\{ (S, V, I, T, R) \in \mathbb{R}_+^5 : 0 < N \leq \frac{\pi}{\mu} \right\} \quad (3)$$

Theorem 1. The HPV model (1) is biologically and mathematically well-established.

Proof: Theorem 1, is structured into four main steps.

Step 1: The right-hand side of system (1) consists of C^∞ functions, and therefore they are also C^1 . Consequently, the function f is differentiable. By the standard results of dynamical systems theory [25], this ensures that f is locally Lipschitz continuous in some open ball containing the initial conditions $S(0), V(0), I(0), T(0), R(0)$. Hence, by applying the Cauchy–Lipschitz theorem, we conclude that system (1) admits a unique local solution for system (1) in Ω .

Step 2. We demonstrate that the solutions of system (1) are positive. Starting from system (1), we obtain:

$$\begin{aligned} \frac{dS}{dt} \Big|_{S=0} &= \pi \geq 0, & \frac{dV}{dt} \Big|_{V=0} &= \theta S \geq 0, \\ \frac{dI}{dt} \Big|_{I=0} &= (1 - \theta)\lambda S + (1 - \epsilon)\lambda V \geq 0, \\ \frac{dT}{dt} \Big|_{T=0} &= \gamma_1 I \geq 0, \\ \frac{dR}{dt} \Big|_{R=0} &= \gamma_2 I + \eta T \geq 0 \end{aligned}$$

Thus, all solutions of system (1) are positive.

Step 3. Adding all the equations of system (1), we obtain

$$\begin{aligned} N' &= \pi - \mu N - \mu_1 T \\ &\leq \pi - \mu N \end{aligned} \tag{4}$$

Solving (4) by applying integration factor and the Gronwall inequality $N(0) = N_0$ yields

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu} (1 - e^{-\mu t}) \tag{5}$$

By the constant variation formula, it follows that.

$$\limsup_{t \rightarrow +\infty} N(t) = \frac{\pi}{\mu} \tag{6}$$

From equation (6), $N(t) = \frac{\pi}{\mu}, \forall t \geq 0$. This establishes that the total population remains bounded, implying that the biologically feasible region Ω is positively invariant and attracting $\forall t \geq 0$. Consequently, all solutions of system (1) are bounded in the region Ω which attracts all solutions in \mathbb{R}_+^6 .

Given all this, we conclude that the initial condition $(S(0), V(0), I(0), T(0), R(0))$ contained in the positively invariant domain Ω , system (1) admits a unique, non-negative solution.

2. The system equilibria

The model admits two equilibrium states: a disease-free equilibrium (DFE), where no infection is present, and an endemic equilibrium (EE), where infection persists at a constant level in the population. The equilibrium points of system (1) obtained by the right hand side of system (1) equal zero and solving the rest by algebraic system.

We solve system (1) and we obtain the disease-free equilibrium points $Y^* \equiv (\lambda = 0, I = 0, T = 0)$, and the endemic equilibrium $Y^{**} \equiv (I \neq 0, T \neq 0)$. From the above, Disease-free equilibrium is given by

$$Y^* \equiv (S^*, V^*, I^*, T^*, R^*) = \left(\frac{\pi}{\mu + \theta}, \frac{\theta \pi}{\mu(\mu + \theta)}, 0, 0, 0 \right)$$

and the endemic equilibrium point is given by

$$Y^{**} \equiv (S^{**}, V^{**}, I^{**}, T^{**}, R^{**})$$

where

$$S^{**} = \frac{\pi}{\theta + (1 - \theta)\lambda^{**} + \mu}, T^{**} = \frac{\gamma_1 I^{**}}{\eta + \gamma_1 + \gamma_2},$$

$$R^{**} = \frac{[\gamma_2(\eta + \gamma_1 + \gamma_2) + \eta \gamma_1] I^{**}}{(\eta + \gamma_1 + \gamma_2)},$$

$$V^{**} = \frac{\theta[\theta + \mu + (1 - \theta)\lambda^{**}] + \pi}{[(1 - \varepsilon)\lambda^{**} + \mu][(\theta + \mu) + (1 - \theta)\lambda^{**}]}$$

where

$$\lambda^{**} = \beta \frac{I^{**} + p T^{**}}{N^{**}}$$

Thus, $Y^{**} \equiv (S^{**}, V^{**}, I^{**}, T^{**}, R^{**})$ is an uniqueness endemic equilibrium.

3. Basic Reproduction Number

The next generation matrix method is employed to determine the basic reproduction number, (\mathcal{R}_0) . This parameter (\mathcal{R}_0) quantifies the average number of new infections that a single infectious individual can generate when introduced into an entirely susceptible population [21]. From the system of equations (1), the infectious compartments (I, T) contributing to the transmission dynamics HPV.

Specifically, the infectious compartments considered are HPV-infected individuals (I) and HPV-infected individuals under treatment (T), whose infectivity is reduced by treatment.

Therefore, we write the system (1) into the reduced form as

$$\begin{cases} I' = (1 - \theta) \lambda S + (1 - \varepsilon) \lambda V - (\mu + \gamma_1 + \gamma_2) I \\ T' = \gamma_1 I - (\eta + \mu + \mu_1) T \end{cases}$$

It is calculated using the algorithm of van den Driessche [7]. After calculation in SageMath, we obtain the new infection production matrix F , the transition matrix V , and the inverse of the matrix V^{-1} respectively.

$$F = \begin{pmatrix} \frac{\beta(\mu(1 - \theta) + \theta(1 - \varepsilon))}{\mu + \theta} & \frac{p\beta(\mu(1 - \theta) + \theta(1 - \varepsilon))}{\mu + \theta} \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \mu + \gamma_1 + \gamma_2 & 0 \\ -\gamma_1 & \eta + \mu + \mu_1 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu + \gamma_1 + \gamma_2} & 0 \\ \frac{\gamma_1}{(\eta + \mu + \mu_1)(\mu + \gamma_1 + \gamma_2)} & \frac{1}{\eta + \mu + \mu_1} \end{pmatrix}$$

Thus, we obtained the basic reproduction number by the spectral radius of the new generation matrix (FV^{-1}) :

$$\mathcal{R}_0 = \beta \frac{[(1 - \theta)\mu + \theta(1 - \varepsilon)](\mu + \mu_1 + \eta + p\gamma_1)}{(\mu + \theta)(\mu + \mu_1 + \eta)(\mu + \gamma_1 + \gamma_2)}$$

TABEL II
PARAMETERS AND THEIR VALUE

Parameter	Value	Reference
β	0.536	assume
ε	0.5	[12]
μ	0.0002	assume
μ_1	0.05	assume
θ	0.4	[6,17]
γ_1	0.3	assume
γ_2	0.2	assume
p	0.3	[6]
η	0.15	assume

$$\mathcal{R}_0 = 0.776726007631547$$

Parameter	Value	Reference
β	0.8	[20]
ε	0.9	[10]
μ	0.00097	[6]
μ_1	0.0005	assume
θ	0.6	[12]
γ_1	0.003	assume
γ_2	0.002	assume
p	0.6	assume
η	0.4	assume

$$\mathcal{R}_0 = 13.5255933650116$$

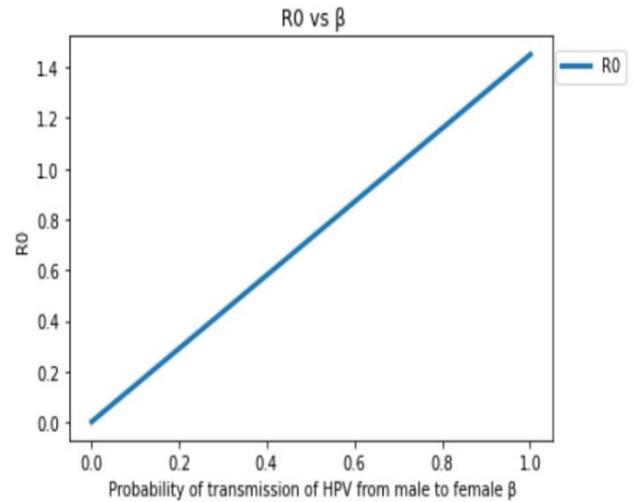


Figure 2. \mathcal{R}_0 profil as a function of β

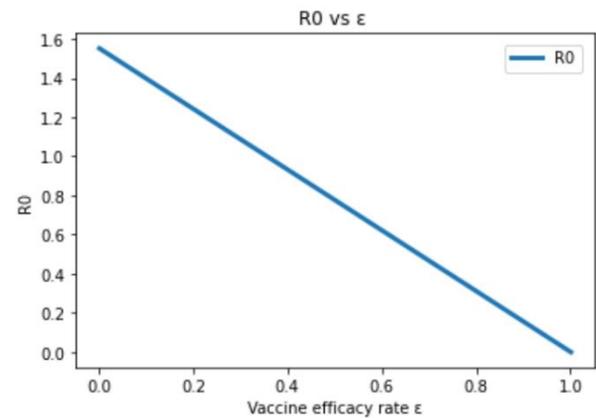


Figure 3. \mathcal{R}_0 profil as a function of ε

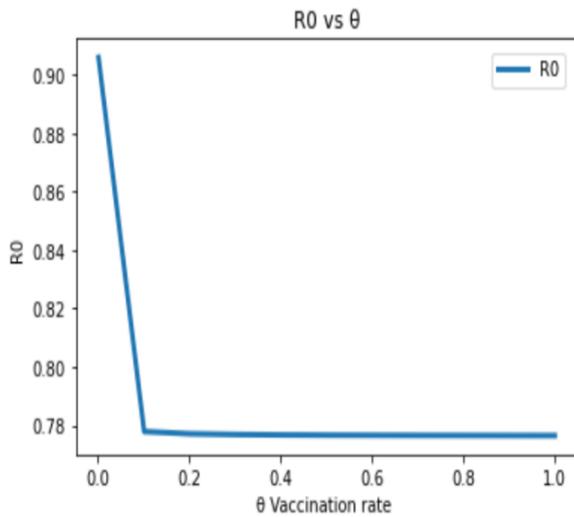


Figure 1. \mathcal{R}_0 profil as a function of θ

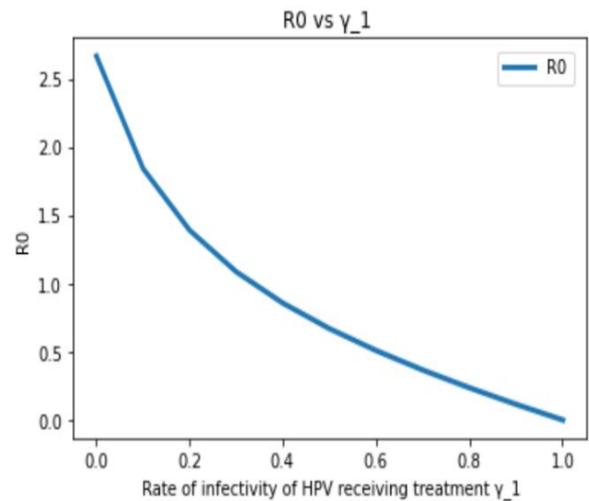


Figure 4. \mathcal{R}_0 profil as a function of γ_1

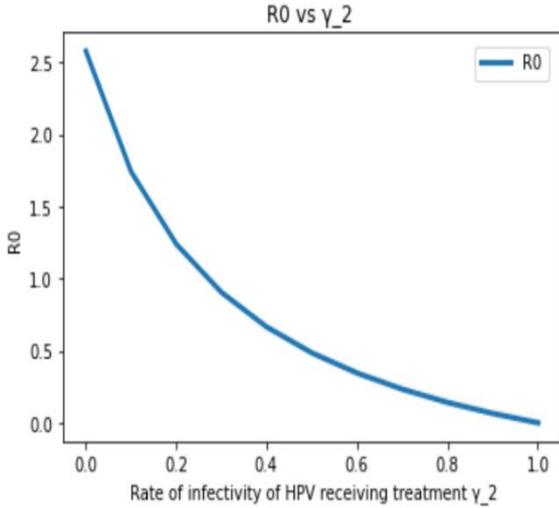


Figure 5. \mathcal{R}_0 profil as a function of γ_2

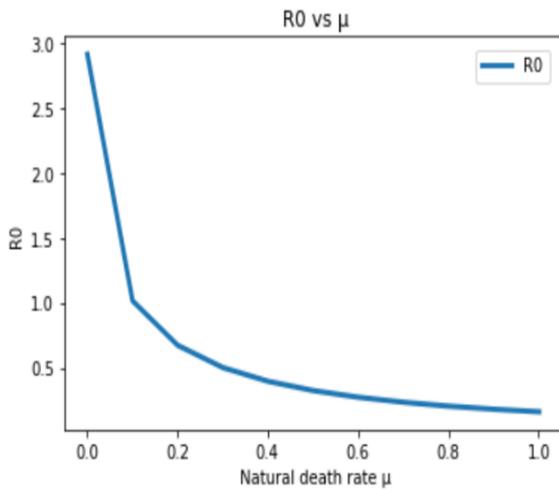


Figure 6. \mathcal{R}_0 profil as a function of μ

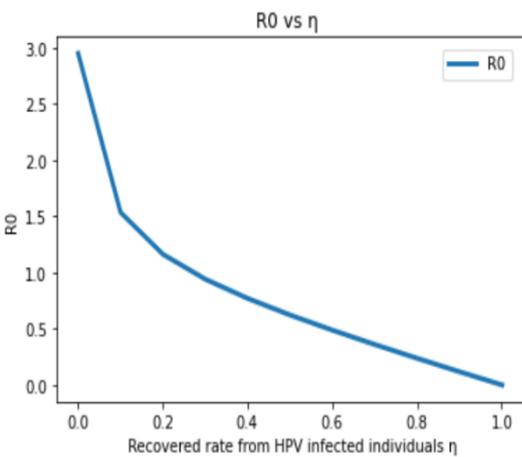


Figure 7. \mathcal{R}_0 profil as a function of η

Global Stability of DFE

Theorem 2. The disease free equilibrium

$Y^* = \left(\frac{\pi}{\mu+\theta}, \frac{\theta\pi}{\mu(\mu+\theta)}, 0, 0, 0 \right)$ of system (1) is globally asymptotically stable in the positively invariant compact set Ω if $\mathcal{R}_0 \leq 1$. If $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable and the system is uniformly persistent and there is at least one equilibrium in $\text{int } \Omega$.

Proof. To construct the Lyapunov function, we apply the matrix-theoretical method as described in [24]. Let $x = (I, T)^T$ and $y = (S, V, R)^T$. We define $f(x, y) = (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y)$ such as

$$f(x, y) = \begin{pmatrix} [(1-\theta)(S^* - S) + (1-\varepsilon)(V^* - V)](I + pT)\frac{\beta}{N} \\ 0 \end{pmatrix}$$

and

$$V^{-1}F = \begin{pmatrix} k_1 & pk_1 \\ k_2 & pk_2 \end{pmatrix}$$

where

$$k_1 = \frac{\beta(\mu(1-\theta) + (1-\varepsilon)\theta)}{(\mu+\theta)(\mu+\gamma_1+\gamma_2)}, k_2 = \frac{\beta\gamma_1(\mu(1-\theta) + (1-\varepsilon)\theta)}{(\mu+\theta)(\eta+\mu+\mu_1)(\mu+\gamma_1+\gamma_2)}$$

Since $S \leq N$, then $f(x, y) \geq 0$. We also observe that $F \geq 0, V^{-1} \geq 0$. Hence the matrix $V^{-1}F$ is reducible, we can use Theorem 2.1 of [24] to construction the Lyapounov function of system (1). Let $w^T = (w_1, w_2) \geq 0$ the lefteigen vector of the non negative matrix $V^{-1}F$ corresponding to the eigenvalue \mathcal{R}_0 . Then

$$(w_1, w_2)V^{-1}F = \mathcal{R}_0(w_1, w_2) \tag{7}$$

From the left-hand side of (7), we have:

$$w^T V^{-1}F = (w_1, w_2) \begin{pmatrix} k_1 & pk_1 \\ k_2 & pk_2 \end{pmatrix} = (a, b) \tag{8}$$

Where

$$a = k_1w_1 + k_2w_2, b = pk_1w_1 + pk_2w_2$$

From equation (7) and (8) we obtain:

$$w_1 = \frac{\mathcal{R}_0 - pk_2}{pk_1} w_2, \text{ where } w_2 \in \mathbb{R}^+.$$

$$\text{Hence, } w^T = \left(\frac{\mathcal{R}_0 - pk_2}{pk_1}, 1 \right).$$

The function below is as a candidate Lyapounov function for the system (1) defined such as

$$L = w^T V^{-1}Fx = \left(\frac{\mathcal{R}_0 - pk_2}{pk_1}, 1 \right) \begin{pmatrix} k_1 & pk_1 \\ k_2 & pk_2 \end{pmatrix} \begin{pmatrix} I \\ T \end{pmatrix}$$

Differentiation of Lyapounov function is

$$L' = (\mathcal{R}_0 - 1)w^T x - w^T V^{-1} f(x, y) \quad (10)$$

$$L' = (\mathcal{R}_0 - 1) \left(\frac{\mathcal{R}_0 - pk_2}{pk_1} I + T \right) - \beta \xi_1 \xi_2$$

where

$$\xi_1 = \left[(1 - \theta) \left(\frac{\mu}{\mu + \theta} - \frac{S}{N} \right) + (1 - \varepsilon) \left(\frac{\theta}{\mu + \theta} - \frac{V}{N} \right) \right] (I + pT)$$

$$\xi_2 = \left(\frac{\mathcal{R}_0 - pk_2}{pk_1} + \frac{\gamma_1}{(\eta + \mu + \mu_i)(\mu + \gamma_1 + \gamma_2)} \right)$$

Since $V \leq S$ and $S \leq N$ we also deduce if $\mathcal{R}_0 < 1$ then $L' < 0$ this implies that is indeed a Lyapounov function for the system (1). Thus, the largest invariant set of the model when $L' = 0$ within Ω is the singleton Y^* . Therefore, by LaSalle's invariance principle [28], the disease free equilibrium Y^* is GAS if $\mathcal{R}_0 < 1$. On the other hand, if, $\mathcal{R}_0 > 1$ then $L' > 0$ there is an endemic equilibrium Y^{**} .

4. Global Stability of Endemic Equilibrium

Theorem 3. *The Y^{**} endemic equilibrium of model (1) is GAS when $\mathcal{R}_0 > 1$.*

Proof. Suppose that the function below is defined positive and continuously differentiable [9,17]:

$$L = \frac{1}{2} \left[(S - S') + (V - V') + (I - I') + (T - T') + (R - R') \right]^2 \quad (11)$$

For the computing time derivative of L, we obtain

$$\begin{aligned} \frac{dL}{dt} &= \frac{1}{2} [(S - S') + (V - V') + (I - I') + (T - T') + (R - R')] \\ &\quad \times \frac{d(S + V + I + T + R)}{dt} \\ &= \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &\quad \times ((\pi - \mu(S + V + I + T + R) - \mu_1)) \\ &\leq \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &\quad \times (\pi - \mu(S + V + I + T + R)) \\ &= \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &\quad \times (\mu N' - \mu(S + V + I + T + R)) \quad \text{we know that } N' = \frac{\pi}{\mu} \\ &= \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &\quad \times \left(\mu(S' + V' + I' + T' + R') - \mu(S + V + I + T + R) \right) \\ &= \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &\quad \times -\mu \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right] \\ &= -\mu \left[(S + V + I + T + R) - (S' + V' + I' + T' + R') \right]^2 \end{aligned}$$

It is obvious $\frac{dL}{dt}$ is negative definite. We can say that the function chosen above satisfies a Lyapunov function. $\frac{dL}{dt} = 0$ if and only when $S = S^*, V = V^*, I = I^*, T = T^*, R = R^*$. According to LaSalle's invariance principle [28], the largest invariant set in

$$\Omega = \{(S, V, I, T, R) \in \mathbb{R}_+^5 : L' = 0\}$$

is the singleton $\{Y^{**}\}$. Therefore, the endemic equilibrium is GAS.

5. Sensitivity analysis

To evaluate the impact of the model parameters on the transmission dynamics of HPV, a sensitivity analysis of model (1) is carried out. This analysis aims to quantify how variations in each parameter influence the basic reproduction number \mathcal{R}_0 .

The equation is given by :

$$\frac{\partial \mathcal{R}_0}{\partial \delta} \times \frac{\delta}{\mathcal{R}_0} \quad (13)$$

where δ is the parameter of \mathcal{R}_0 .

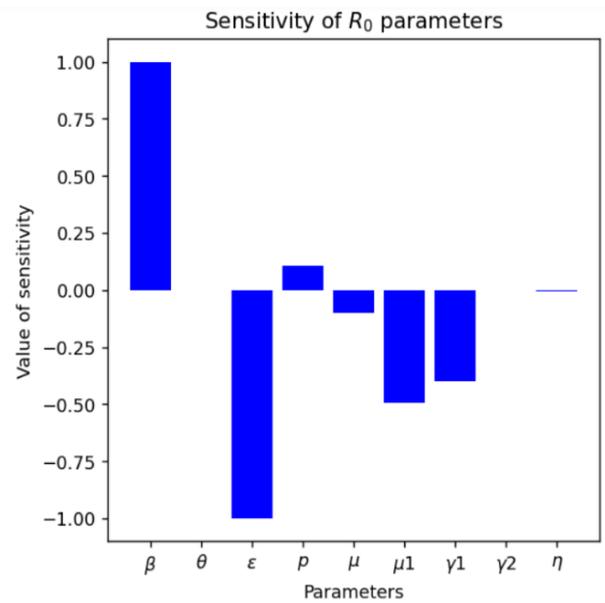


Figure 8. Sensitivity Analysis

V. NUMERICAL SIMULATIONS

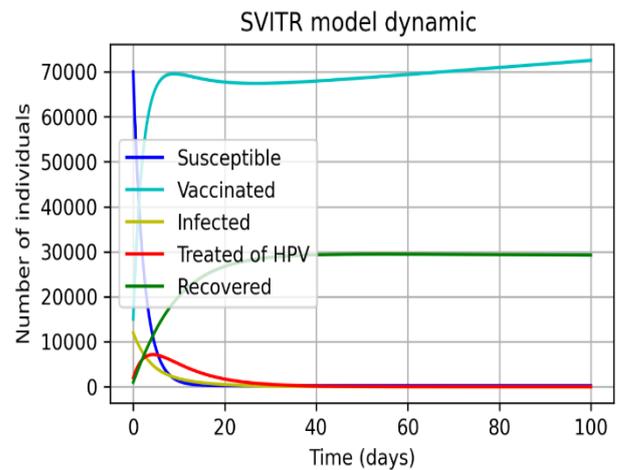


Figure 9. Evolution of HPV- Infected over time. ($\mathcal{R}_0 < 1$)

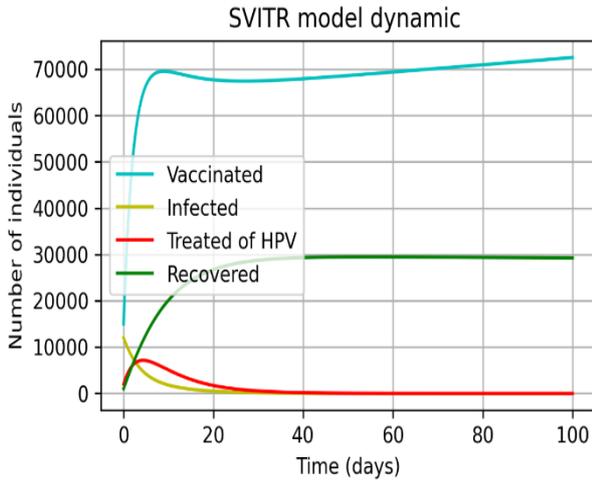


Figure 10. Evolution of HPV- Infected over time. ($\mathcal{R}_0 < 1$)

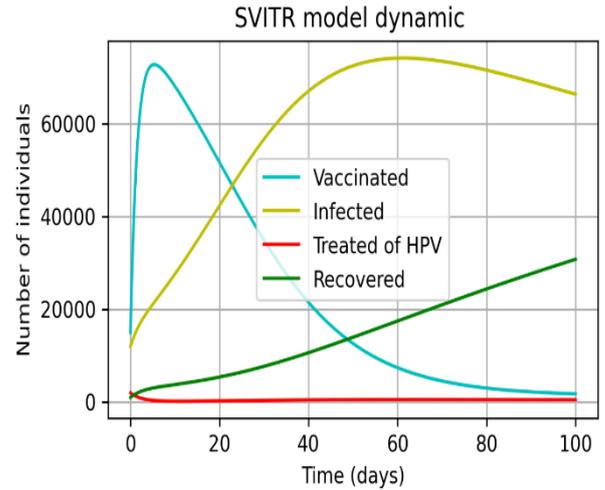


Figure 13. Evolution of HPV- Infected over time. ($\mathcal{R}_0 > 1$)

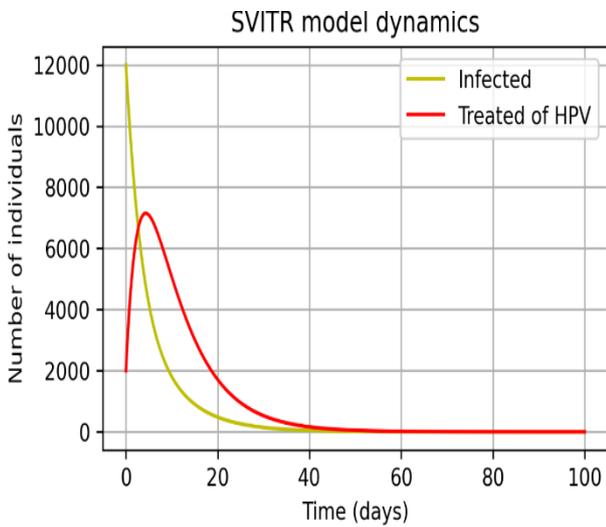


Figure 11. Evolution of HPV- Infected over time. ($\mathcal{R}_0 < 1$)

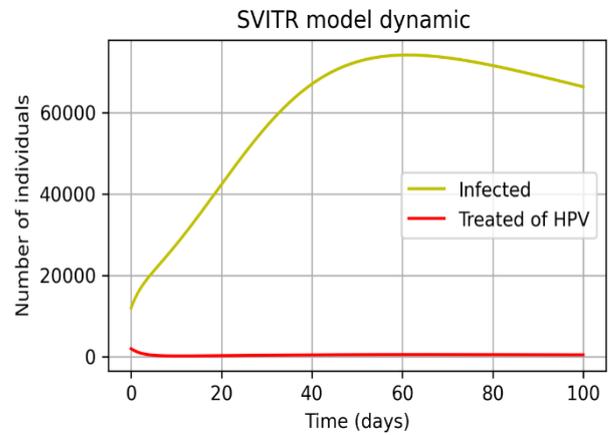


Figure 13. Evolution of HPV- Infected over time ($\mathcal{R}_0 > 1$)

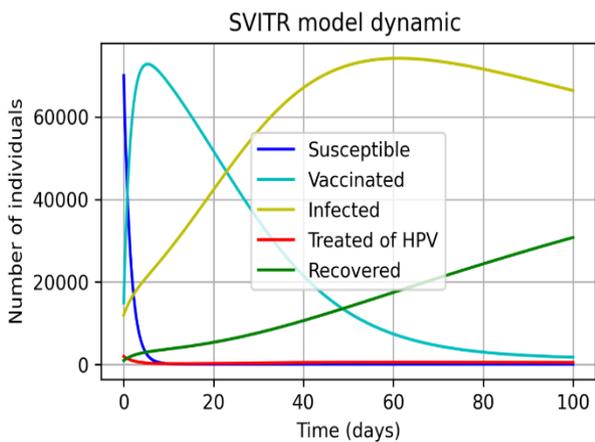


Figure 12. Evolution of HPV- Infected over time ($\mathcal{R}_0 > 1$)

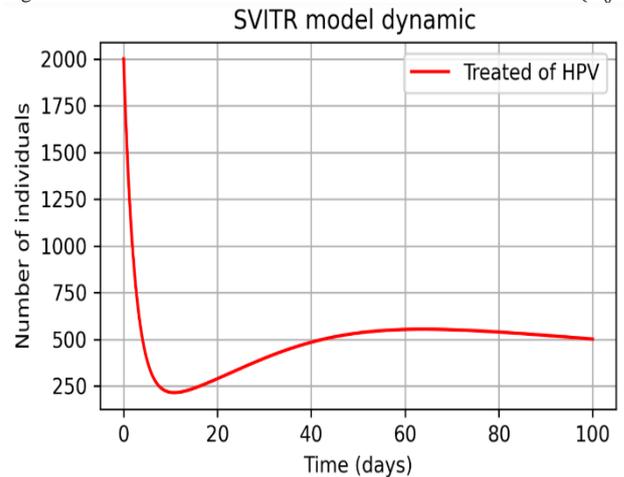


Figure 14. Evolution of HPV- Infected over time ($\mathcal{R}_0 > 1$)

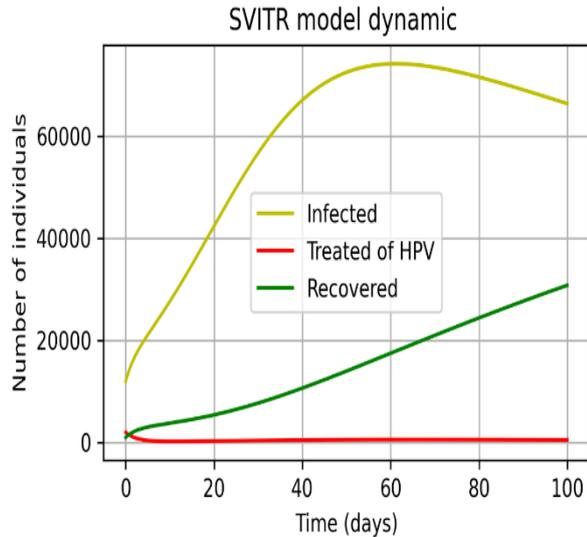


Figure 15. Evolution of HPV- Infected over time ($\mathcal{R}_0 > 1$)

VI. DISCUSSION

Figures 1–7 illustrate the profile of \mathcal{R}_0 to various model parameters. The basic reproduction number \mathcal{R}_0 rises linearly with the transmission probability β , indicating that more efficient viral transmission accelerates the spread of HPV. In contrast, \mathcal{R}_0 declines with increasing vaccination efficacy ε and decreases exponential distribution with control and loss, related parameters, including natural death, disease induced death, recovered rate of HPV. These findings emphasize that HPV transmission is enhanced by higher transmissibility but can be effectively reduced through vaccination, therapeutic interventions, and demographic losses. Overall, the results highlight that controlling \mathcal{R}_0 requires both limiting transmission and implementing protective and treatment measures, underscoring the need for an integrated strategy to curb the epidemic (see also Figure 8 of sensitivity).

The sensitivity analysis of the basic reproduction number \mathcal{R}_0 , presented in the corresponding figure 8, allows the analytical stability results to be interpreted in light of their biological significance and epidemiological implications. In particular, it identifies the parameters whose variation directly affects the stability of the disease-free equilibrium ($\mathcal{R}_0 < 1$) and the emergence of an endemic equilibrium ($\mathcal{R}_0 > 1$) in the SVITR model.

The transmission rate β exhibits the highest positive sensitivity. An increase in β leads to a significant increase in \mathcal{R}_0 , destabilizing the disease-free equilibrium and promoting infection persistence. This result is epidemiologically consistent, as higher transmissibility increases the number of new infections and complicates disease control, highlighting the importance of strategies aimed at reducing transmission, such as behavioral prevention and early screening.

Vaccine efficacy ε appears as the parameter with the strongest negative effect on \mathcal{R}_0 . Its increase substantially contributes to reducing \mathcal{R}_0 and reinforcing the stability of the disease-

free equilibrium. This confirms that improving vaccine performance is a key lever for interrupting HPV transmission and preventing the establishment of endemicity.

The vaccination rate θ also shows a notable negative sensitivity. Higher vaccination coverage reduces the pool of susceptible individuals and helps maintain \mathcal{R}_0 below the critical threshold. The combined effect of ε and θ highlights that vaccination acts both through the quality of protection and its large-scale deployment within the population.

Treatment- and recovery-related parameters also play an important role. The recovery rate of treated infected individuals γ_1 exhibits a significant negative sensitivity, indicating that more effective therapeutic management reduces the duration of infectivity and limits secondary transmission. Similarly, the parameter η , representing recovery after treatment, contributes to reducing \mathcal{R}_0 by accelerating the transition to the recovered class. These results emphasize the importance of timely and effective access to treatment in HPV control strategies.

The parameter p , which measures the reduction in infectivity of treated individuals and appears directly in the force of infection $\lambda = \beta(I + pT)/N$, shows a slightly positive sensitivity. This indicates that when infectivity reduction is insufficient (i.e., when p remains relatively high), treated individuals may still contribute to virus transmission. Thus, treatment effectiveness should be evaluated not only in terms of recovery but also in terms of reducing transmission potential.

Natural mortality μ and disease-induced mortality μ_1 exhibit weak negative sensitivities. Although they may marginally influence the value of \mathcal{R}_0 , these parameters do not directly affect transmission mechanisms and therefore do not constitute effective public health control levers.

Overall, this analysis demonstrates that the stability of the disease-free equilibrium primarily relies on reducing the transmission rate, improving vaccine efficacy and coverage, and enhancing treatment effectiveness, both in accelerating recovery and reducing infectivity among treated individuals. These results provide a clear biological interpretation of the analytical findings and highlight the strategic priorities required to sustainably achieve $\mathcal{R}_0 < 1$.

The simulated dynamics of the five compartments of the SVITR model, illustrated in Figures 9–11 for the case where $\mathcal{R}_0 < 1$, clearly demonstrate a progressive extinction of the infection within the population. The susceptible compartment declines sharply at the beginning of the simulation and then stabilizes near zero, reflecting a rapid transfer of individuals into the vaccinated, infected, or recovered compartments, along with a relatively low influx of new susceptibles. In parallel, the number of vaccinated individuals rises quickly and reaches a high plateau, indicating sustained vaccine coverage that plays a central role in maintaining the condition $\mathcal{R}_0 < 1$.

The infected compartment exhibits an early, modest peak followed by a rapid decline to zero, illustrating a short-lived epidemic surge before the complete extinction of transmission. Similarly, the treated compartment displays a slightly delayed peak corresponding to the clinical management of initial cases, after which it gradually vanishes as the infection dies out. Finally, the recovered population increases markedly at the beginning before stabilizing at an intermediate level, indicating the development of natural immunity in a substantial fraction of the population. This immunity, together with vaccination, contributes decisively to the long-term control of the infection.

Figures 12–15 show the evolution of the compartments in the SVITR model for $\mathcal{R}_0 > 1$. Infected individuals (yellow) increase rapidly before stabilizing, indicating an endemic equilibrium. Vaccinated individuals (cyan) rise initially but then decline, suggesting that vaccination alone is insufficient to control the infection. Treated individuals (red) remain pic (see figure15) but stable, while recovered individuals (green) increase steadily, reflecting a continuous healing process. Overall, the dynamics highlight the need to strengthen vaccination, treatment, and transmission reduction measures to bring $\mathcal{R}_0 < 1$.

VII. CONCLUSION

The SVITR model highlights the complementary roles of vaccination and treatment in controlling HPV transmission. Vaccination progressively reduces the pool of susceptible individuals, thereby limiting the long-term persistence of the virus in the population. Treatment, in contrast, acts more rapidly by decreasing the infectiousness of infected individuals, leading to a short-term reduction in transmission. When implemented jointly, these interventions significantly reduce the basic reproduction number \mathcal{R}_0 , potentially bringing it below the epidemic threshold. This result indicates that the combined implementation of preventive vaccination and therapeutic management constitutes an effective strategy for the long-term control and possible elimination of HPV.

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