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MATHEMATICAL MODELING AND NUMERICAL SIMULATION OF THE DYNAMICS OF HUMAN PAPILLOMAVIRUS (HPV) AND CERVICAL CANCER IN BURKINA FASO

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Abstract. Cervical cancer, primarily caused by the human papillomavirus (HPV), remains a significant global health burden, especially in low-income countries. This study develops a mathematical model based on the SEIR framework to analyze the transmission dynamics of HPV and its progression to cervical cancer in Burkina Faso. A distinctive feature of this model is the inclusion of both same-sex transmission and vertical transmission mechanisms often overlooked in most existing models. We establish the existence of non-negative solutions and determine the global stability of both the disease-free and endemic equilibria. The basic reproduction number, $\mathcal{R}_0$, is derived, and sensitivity analysis is performed on key parameters. Numerical simulations, grounded in local epidemiological data, confirm the theoretical findings and provide actionable insights for prevention strategies in resource-limited settings like Burkina Faso. Our results suggest that targeting both same-sex and vertical transmission pathways is crucial for comprehensive HPV control.

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1. Introduction

Cervical cancer, predominantly caused by human papillomavirus (HPV), represents a substantial public health concern, especially in low-income countries. Globally, it is the fourth most common cancer among women, with the highest burden concentrated in regions like sub-Saharan Africa, where healthcare access is often limited. In 2020, around 90% of the 604,000 new cases and 342,000 deaths related to cervical cancer occurred globally [1,2]. In Burkina Faso, the disease's impact is alarming, with 1,132 new cases and a high mortality rate of 74.1% recorded in the same year [3]. HPV, a sexually transmitted infection, remains the primary cause of cervical cancer, making it vital to understand its transmission mechanisms.

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Despite significant research on HPV, most existing models overlook two transmission pathways: same-sex transmission and vertical transmission from mother to child. Our study seeks to address these gaps by developing a comprehensive SEIR-based model that integrates these transmission routes to better understand HPV dynamics in Burkina Faso.

This study is timely, given the high prevalence of HPV in Burkina Faso, exacerbated by factors such as poverty, internal displacement, and social stigma surrounding sexually transmitted infections. By capturing the full spectrum of transmission pathways, the model offers insights into effective prevention strategies, including vaccination, screening, and targeted education efforts.

The manuscript is organised as follows: Section 2 presents the formulation of the mathematical model, highlighting the integration of both same-sex and vertical transmission in HPV dynamics. In Section 3, we analyze the mathematical properties of the model, including the existence of solutions, positivity, boundedness, and stability of both disease-free and endemic equilibria.

A sensitivity analysis of key parameters is also conducted at the end of Section 3. Section 4 focuses on the numerical methods and simulations, where local epidemiological data are used to validate the model. Finally, Section 5 concludes with a discussion of the findings and potential public health implications for HPV control in Burkina Faso.

2. Model Formulation

A SEIR model (Susceptible S, Exposed E, Infected I, Recovered R) has been formulated and includes vertical transmission. The total birth population is denoted as Λ_f for females and Λ_m for males. Accounting for vertical transmission reduces the birth rate of females by an amount of $\rho_1 I_2 + \rho_2 P + \rho_3 Q$ because babies from this group are infected. Thus, $\rho_1 I_2 + \rho_2 P + \rho_3 Q$ appears in compartment E_1. HPV is vertically transmitted by some females aged 9-15 years and over 15 years who are exposed. It is also transmitted by infected females aged 9-15 years and over 15 years, as well as by chronically infected females and those in the pre-cancerous phase. The description of variables is provided in Table 1 and that of parameters in Table 2.

Throughout this document, the following assumptions are made:

(H1) The choice of the vertical transmission model is used to account for vertical transmission.

(H2) The total population at time t, denoted by $N(t)$, is divided into two classes: the total female population ($N_f(t)$) and the total male population ($N_m(t)$).

(H3) The total female population is subdivided into 13 compartments: S_1 (9 years - 15 years), S_2 (>15 years), E_1, E_2, I_1, I_2, P, Q, T, C, R_1, R_2, R_c.

(H4) The total male population is subdivided into 4 compartments: S_m, E_m, I_m, R_m.

(H5) Recruitment is $\Lambda_f - \rho_1 I_2 - \rho_2 P - \rho_3 Q$.

(H6) General recruitment for males is Λ_m.

(H7) Susceptibles S_1 and S_2 contract the virus through sexual intercourses with an infected man at a rate λ_m.

(H8) Susceptibles S_2 contract the virus through sexual intercourses with an infected woman at a rate B_f.

(H9) Susceptibles S_m contract the virus through sexual intercourses with an infected man at a rate λ_f.

(H10) Susceptibles S_m contract the virus through sexual intercourses with an infected woman at a rate B_m.

(H11) Classes R_1, R_2, and R_m can reacquire the virus.

(H12) Only individuals presenting pre-cancerous lesions are treated.

(H13) The parameters $\beta_f,\ \beta_m,\ c_f,\ c_m,\ p_1,\ \mu_f,\ \mu_q,\ \mu_t,\ \mu_m,\ \mu_c,\ \sigma_i,\ \sigma_1,\ \sigma_2,\ \sigma_3,\ \sigma_4,\ \sigma_5,\ \rho_1,\ \rho_2,\ \rho_3,\ \theta_1,\ \theta_2,\ \theta_3,\ \theta_4,\ \theta_5,\ \theta_6,\ \theta_n,\ \theta_m,\ k_f,\ r,\ r_1,\ r_2,\ r_3,\ r_4,\ r_5,\ m_1,\ m_2,\ r_m,\ k_m,\ f_1,\ f_2, f_3,\ f_4,\ f_5,\ f_6,\ k_1,\ k_2,\ \tau_1,\ \tau_2$ are assumed to be strictly positive.

(H14) All individuals in compartments $S_1, S_2, E_1, E_2, I_1, I_2, R_1, R_2, P$, and R_c have the same mortality rate μ_f.

Compartment S_1 represents susceptible females aged 9 to 15, who may become exposed (E_1) through sexual contacts with infectious males, with a strength of infection λ_m. As they grow older, a proportion p_1 of these women move into the susceptible compartment S_2. In S_2, susceptible women over the age of 15 may become exposed and enter E_2 with probability $\lambda_m + B_f$. Exposed individuals in E_1 progress to infectious state I_1 at rate $\tau_1\sigma_i$, while those in E_2 progress to I_2 at rate τ_2. Infectious women in I_1 heal and go into R_1 with probability $r_1\sigma_1$ or evolve into the infectious state I_2. Similarly, women in I_2 heal and go into R_2 at a rate $r_2\sigma_2$ or develop persistent infections to go into the P compartment. Persistent infections in P may resolve, leading to healing in R_2, or progress to precancerous lesions in Q at the rate $(1-r_3)\sigma_3$. Since Q, individuals can receive treatment (T) or develop cervical cancer (C). A proportion of women treated in T recover to go into R_2 and another proportion progress to cancer at the rate $(1-r_5)\sigma_5$. Patients with cancer in C may heal and progress to R_c at the rate r_4. The S_m compartment represents susceptible men, who may become exposed (E_m) through contacts with infectious women, with probability $\lambda_f + B_m$. Exposed men in E_m progress to the infectious state I_m at rate τ_m and either recover to go into R_m with probability r_m, or remain in E_m at rate $k_m\lambda_f$. This dynamic framework shows the essential interactions between individuals in the different compartments, taking into account the different states of infection, cure, treatment and disease progression, in both male and female populations.

TABLE 1. Description of variables used in the model

Variables	Biological description
$S_1(t)$	Population of susceptible females aged between 9 and 15
$S_2(t)$	Population of susceptible females above age 15
$E_1(t)$	Population of females aged between 9 and 15 exposed to HPV infection
$I_1(t)$	Population of HPV-infected females aged between 9 and 15
$R_1(t)$	Population of females aged between 9 and 15 who have recovered from HPV infection
$E_2(t)$	Population of females aged above 15 exposed to HPV infection
$I_2(t)$	Population of HPV-infected females above age 15
$R_2(t)$	Population of females above age 15 who have recovered from HPV infection
$P(t)$	Population of females with persistent HPV infection
$Q(t)$	Population of females with precancerous lesions
$T(t)$	Population of females with precancerous lesions treated
$C(t)$	Population of females with cervical cancer
$R_C(t)$	Population of females who recovered from cervical cancer
$S_m(t)$	Population of susceptible males
$E_m(t)$	Population of males exposed to HPV infection
$I_m(t)$	Population of HPV-infected males
$R_m(t)$	Population of males who have recovered from HPV infection

TABLE 2. Parameters used in the model

Parameter	Description	Values	References
Λ_f	Recruitment of new sexually-active females	424,553	[21]
Λ_m	Recruitment of new sexually-active males	396580	[21]
ρ_1	Rate of births infected by vertical transmission from I_2	0.09	Assumed
ρ_2	Rate of births infected by vertical transmission from P	0.09	Assumed
ρ_3	Rate of births infected by vertical transmission from Q	0.09	Assumed
p_1	Proportion of S_1 who grow healthy to enter in S_2	0.11	Assumed
σ_i	Departure rate of females (9-15 years) from class E_1	0.21	[23]
τ_m	Fraction of males that are infectious	0.47	Assumed
τ_1, τ_2	Fraction of females aged 9-15 years and over 15 years that are infectious, respectively	0.47	Assumed

Continued on next page

Table 2 – Continuation of previous table

Parameter	Description	Values	References
r	Fraction of females that are treated	0.9	[23]
r_1	Rate of females (9-15 years) leaving class I_1 that recover	0.82	[23]
r_2, r_3, r_4, r_5	Fraction of females over 15 leaving class I_2, P, C, and T, respectively recovering	0.9, 0.9, 0.5, 0.9	[23]
r_m	Male recovery rate	0.9	[23]
$\beta_m(\beta_f)$	Probability of transfer of infection from males to females (females to males) per contact	0.8 (0.7)	[23]
σ_1, σ_2	Departure rate of females S_1 from I_1 and I_2	0.61, 0.5	[23]
σ_3, σ_4, σ_5	Departure rate of females S_2 of age from class P, cervical neoplasia Q, and treated T, respectively	0.58	[23]
k_f	Proportion of females aged 9-15 or over 15 who will be reinfected through sexual contact	0.01	Assumed
k_m	Proportion of males reinfected through sexual contacts	0.01	Assumed
c_m	Average number of sexual contacts per male	2	[23]
c_f	Average number of sexual contacts per female	$\frac{c_m N_m}{N_f}$	[23]
μ_f, μ_m	Natural mortality rate of females and males respectively, i.e., mortality not caused by HPV	0.0085, 0.0099	[21]
μ_q	Mortality rate caused by cervical neoplasia	0.002	Assumed
μ_t	Mortality rate in treated precancerous women	0.004	Assumed
μ_c	Mortality rate caused by cervical cancer	0.001	Assumed
θ_m	Rate of latently infected men E_m who are infectious	0.95	Assumed
θ_n	Fraction of infected men I_m capable of transmitting the HPV virus	0.99	Assumed
θ_1, θ_2, θ_3, θ_4, θ_5, θ_6	Fraction of latently infected females E_1, infected females aged between 9 and 15 I_1, latently infected females E_2, infected females above age 15 I_2, females with persistent HPV infection P, and females with precancerous lesions Q respectively capable of transmitting the HPV virus	0.8, 0.95, 0.99, 0.94, 0.90, 0.97, 0.98, 0.99	Assumed
f_1, f_2, f_3, f_4, f_5, f_6	Fraction of latently infected females E_1, infected females aged between 9 and 15 I_1, latently infected females E_2, infected females above age 15 I_2, females with persistent HPV infection P, and females with precancerous lesions Q respectively capable of transmitting the HPV virus between women	0.1, 0.15, 0.1, 0.3, 0.35, 0.35	Assumed
m_1, m_2	Fraction of latently infected E_m males and HPV-infected males I_m respectively capable of transmitting the HPV virus between men	0.2, 0.3	Assumed

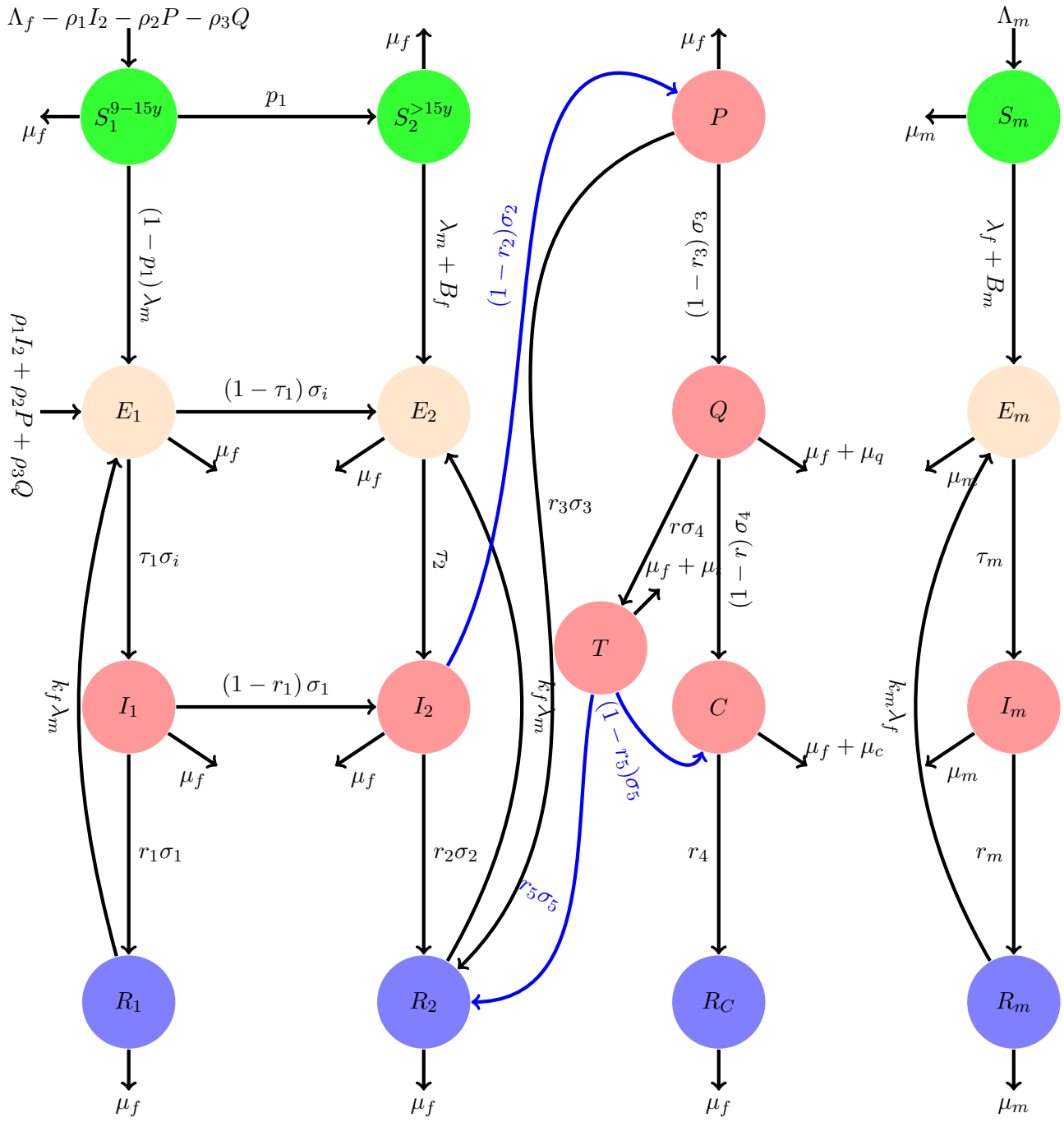

FIGURE 1. Schematic Diagram of the Dynamics of HPV

By doing a mass balance through the compartments, we obtain the system of differential equations:

$$\begin{cases}
\frac{dS_1}{dt} &= \Lambda_f - \rho_1 I_2 - \rho_2 P - \rho_3 Q - (1-p_1)\,\lambda_m S_1 - (p_1+\mu_f)\,S_1 \\
\frac{dS_2}{dt} &= p_1 S_1 - \lambda_m S_2 - B_f S_2 - \mu_f S_2 \\
\frac{dE_1}{dt} &= (1-p_1)\,\lambda_m S_1 + \lambda_m k_f R_1 + \rho_1 I_2 + \rho_2 P + \rho_3 Q - (\sigma_i+\mu_f)\,E_1 \\
\frac{dI_1}{dt} &= \tau_1 \sigma_i E_1 - (\sigma_1+\mu_f)\,I_1 \\
\frac{dR_1}{dt} &= r_1 \sigma_1 I_1 - \lambda_m k_f R_1 - \mu_f R_1 \\
\frac{dE_2}{dt} &= (1-\tau_1)\,\sigma_i E_1 + \lambda_m S_2 + B_f S_2 + \lambda_m k_f R_2 - (\tau_2+\mu_f)\,E_2 \\
\frac{dI_2}{dt} &= \tau_2 E_2 + (1-r_1)\,\sigma_1 I_1 - (\sigma_2+\mu_f)\,I_2 \\
\frac{dR_2}{dt} &= r_2 \sigma_2 I_2 + r_3 \sigma_3 P + r_5 \sigma_5 T - \lambda_m k_f R_2 - \mu_f R_2 \\
\frac{dP}{dt} &= (1-r_2)\,\sigma_2 I_2 - (\sigma_3+\mu_f)\,P \\
\frac{dQ}{dt} &= (1-r_3)\,\sigma_3 P - (\sigma_4+\mu_f+\mu_q) Q \\
\frac{dT}{dt} &= r \sigma_4 Q - (\sigma_5+\mu_f+\mu_t)\,T \\
\frac{dC}{dt} &= (1-r)\,\sigma_4 Q + (1-r_5)\,\sigma_5 T - (r_4+\mu_f+\mu_c)\,C \\
\frac{dR_C}{dt} &= r_4 C - \mu_f R_C \\
\frac{dS_m}{dt} &= \Lambda_m - \lambda_f S_m - B_m S_m \\
\frac{dE_m}{dt} &= \lambda_f k_m R_m + \lambda_f S_m + B_m S_m - (\tau_m+\mu_m)\,E_m \\
\frac{dI_m}{dt} &= \tau_m E_m - (r_m+\mu_m)\,I_m \\
\frac{dR_m}{dt} &= r_m I_m - \lambda_f k_m R_m - \mu_m R_m
\end{cases} \tag{1}$$

With

$$\begin{aligned}
\lambda_f &= \frac{\beta_f c_m\,(\theta_1 E_1 + \theta_2 I_1 + \theta_3 E_2 + \theta_4 I_2 + \theta_5 P + \theta_6 Q)}{N_f}, \\
B_f &= \frac{f_1\theta_1 E_1 + f_2\theta_2 I_1 + f_3\theta_3 E_2 + f_4\theta_4 I_2 + f_5\theta_5 P + f_6\theta_6 Q}{N_f}, \\
N_f &= S_1 + S_2 + E_1 + I_1 + R_1 + E_2 + I_2 + R_2 + P + Q + T + C + R_C,
\end{aligned} \tag{2}$$

$$\begin{aligned}
\lambda_m &= \frac{\beta_m c_f (\theta_m E_m + \theta_n I_m)}{N_m}, \\
B_m &= \frac{m_1 \theta_m E_m + m_2 \theta_n I_m}{N_m}, \\
N_m &= S_m + E_m + I_m + R_m, \\
c_m N_m(t) &= c_f N_f(t).
\end{aligned}$$

3. Mathematical Analysis

3.1. **Invariant Regions.** We begin the analysis of the two-sex HPV infection model(1) by considering its behavior in a biologically feasible region. First, we demonstrate that the system (1) is dissipative, meaning that all biologically relevant solutions remain uniformly bounded within a suitable subset $D \subset \mathbb{R}^{17}_+$. The model is structured into two subsystems: one for the female population, N_f, and the other for the male population, N_m. The feasible region is defined as $D = D_f \cup D_m \subset \mathbb{R}^{13}_+ \times \mathbb{R}^4_+$, with:

$$\begin{aligned}
D_f &= \left\{ (S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_c) \in \mathbb{R}^{13}_+ : N_f \leq \frac{\Lambda_f}{\mu_f} \right\}, \\
D_m &= \left\{ (S_m, E_m, I_m, R_m) \in \mathbb{R}^4_+ : N_m \leq \frac{\Lambda_m}{\mu_m} \right\}.
\end{aligned} \tag{3}$$

3.2. **Positivity and Boundedness Properties.**

Theorem 3.1. *Let the initial conditions be:*

$$\begin{aligned}
&(S_1(0), S_2(0), E_1(0), I_1(0), R_1(0), E_2(0), I_2(0), R_2(0), \\
&P(0), Q(0), T(0), C(0), R_C(0), S_m(0), E_m(0), I_m(0), R_m(0)) \in \mathbb{R}^{17}_+
\end{aligned}$$

such that: $S_1(0) + S_2(0) + E_1(0) + I_1(0) + R_1(0) + E_2(0) + I_2(0) + R_2(0) + P(0) + Q(0) + T(0) + C(0) + R_C(0) + S_m(0) + E_m(0) + I_m(0) + R_m(0) = N(0)$ *where* $\Lambda_f > 0$, $\Lambda_m > 0$, *and* $N(0) \leq \frac{\Lambda}{\mu}$. *Additionally, the following parameter constraints hold:*

$$\begin{aligned}
&0 \leq \lambda_f, \lambda_m, c_f, c_m, p_1, \mu_f, \mu_Q, \mu_m, \mu_C, \sigma_i, \sigma_1, \sigma_2, \sigma_3, \sigma_4, \sigma_5, \rho_1, \rho_2, \rho_3, \theta_1, \theta_2, \theta_3, \theta_4, \theta_5, \theta_6, \theta_m, \theta_n, k_f, r, r_1, \\
&\quad r_2, r_3, r_4, r_5, m_1, m_2, r_m, k_m, f_1, f_2, f_3, f_4, f_5, f_6, k_1, k_2 \leq 1.
\end{aligned}$$

Then, the system (1) *has a unique global solution* [8] *that remains non-negative and bounded for all* $t > 0$. *Moreover, the following conditions hold:*

(1) $0 < N(t) < \frac{\Lambda}{\mu}$, *where* $\Lambda = \Lambda_f + \Lambda_m$, $\mu = \min(\mu_f, \mu_m)$, *and* $N(t) = N_f(t) + N_m(t)$.
(2) *If* $S_1(0) \leq \frac{\Lambda_f}{p_1 + \mu_f}$, $S_2(0) \leq \frac{p_1 \Lambda_f}{\mu_f (p_1 + \mu_f)}$, *and* $S_m(0) \leq \frac{\Lambda_m}{\mu_m}$, *then for all* $t > 0$, $S_1(t) \leq \frac{\Lambda_f}{p_1 + \mu_f}$, $S_2(t) \leq \frac{p_1 \Lambda_f}{\mu_f (p_1 + \mu_f)}$, *and* $S_m(t) \leq \frac{\Lambda_m}{\mu_m}$.

Proof. For local existence, all functions in system (1) are locally Lipschitz continuous. Hence, there exists a unique local solution on $t \in [0, T_{max}[$, where T_{max} is the explosion time. The analysis of such

systems is based on elementary methods of ordinary differential equations. The existence of unique solutions is guaranteed by various fixed-point theorems on a maximal interval $[0, T_{max}[$. By proving that the components of the solution vector are uniformly bounded over any bounded interval $[0, T_{max}[$, we ensure $T_{max} = \infty$. We notice that the components of the vector M,

$$M = \begin{pmatrix} M_1(S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_C, S_m, E_m, I_m, R_m) \\ M_2(S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_C, S_m, E_m, I_m, R_m) \\ \dots \\ M_{17}(S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_C, S_m, E_m, I_m, R_m) \end{pmatrix} \tag{4}$$

are quasi-positive. Therefore, as the initial conditions are non-negative, this implies that the components of the solution are non-negative for all $t \in [0, T_{max}[$. Let's define the function N as follows:

$N(t) = S_1(t) + S_2(t) + E_1(t) + I_1(t) + R_1(t) + E_2(t) + I_2(t) + R_2(t) + P(t) + Q(t) + T(t) + C(t) + R_C(t) + S_m(t) + E_m(t) + I_m(t) + R_m(t)$ One obtains:

$$\begin{aligned} \frac{dN}{dt} &= \Lambda_f + \Lambda_m - \mu_f N_f - \mu_m N_m - \mu_q Q - \mu_t T - \mu_c C \\ \frac{dN}{dt} &\leq \Lambda - \mu(N_f + N_m) \\ \frac{dN}{dt} &\leq \Lambda - \mu N(t) \\ \frac{dN}{dt} + \mu N(t) &\leq \Lambda \end{aligned} \tag{5}$$

$$\begin{cases} \frac{dN}{dt} \leq \Lambda - \mu N(t) \\ N(0) = N_0 \end{cases} \tag{6}$$

Solving the inequality:

$$\begin{aligned} \frac{dN}{dt} + \mu N(t) &\leq 0 \\ N(t) &\leq k(t) \exp^{-\mu t} \\ N'(t) &\leq k'(t) \exp^{-\mu t} - \mu \exp^{-\mu t} k(t) \end{aligned} \tag{7}$$

Substituting $N'(t)$ and $N(t)$ in (5):

$$\begin{aligned} k'(t) \exp^{-\mu t} - \mu \exp^{-\mu t} k(t) + \mu k(t) \exp^{-\mu t} &\leq \Lambda \\ k'(t) \exp^{-\mu t} &\leq \Lambda \\ k'(t) &\leq \Lambda \exp^{\mu t} \\ k(t) &= \frac{\Lambda}{\mu} \exp^{\mu t} + c \end{aligned} \tag{8}$$

Substituting $k(t)$in (7):

$$\begin{aligned} N(t) &\leq \left(\frac{\Lambda}{\mu}\exp^{\mu t} + c\right)\exp^{-\mu t} \\ N(0) &\leq \left(\frac{\Lambda}{\mu} + c\right) \\ c &\geq N(0) - \frac{\Lambda}{\mu} \\ N(t) &\leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)\exp^{-\mu t} \end{aligned} \tag{9}$$

Since $N(0) \leq \dfrac{\Lambda}{\mu}$, then $N(t) \leq \dfrac{\Lambda}{\mu}$.

Conclusion: $0 < N(t) < \dfrac{\Lambda}{\mu}$, hence $T_{max} = \infty$ and the existence of a unique global non-negative solution is proven. Similarly, solving for S_1:

$$\begin{aligned} \frac{dS_1}{dt} + (p_1 + \mu_f)S_1(t) &\leq 0 \\ S_1(t) &\leq k(t)\exp^{-(p_1+\mu_f)t} \end{aligned} \tag{10}$$

The same reasoning applies to S_2 and S_3, concluding the proof of the theorem. □

3.3. **Disease-free equilibrium point ε^0 and basic reproduction number $\mathcal{R}_0$.** This section is dedicated to calculating the reproduction number $\mathcal{R}_0$ of the proposed model. To do this, we will use the method of the generation matrix that was developed and then adopted for finite-dimensional systems.

Theorem 3.2. *Consider the system* (1) *with the given parameters*

$$\Lambda_f > 0, \quad \Lambda_m > 0.$$

$$0 \leq \lambda_f, \lambda_m, c_f, c_m, p_1, \mu_f, \mu_Q, \mu_m, \mu_C, \sigma_i, \sigma_1, \sigma_2, \sigma_3, \sigma_4, \sigma_5, \rho_1, \rho_2, \rho_3, \theta_1, \theta_2, \theta_3, \theta_4, \theta_5, \theta_6, \theta_m, \theta_n, k_f, r, r_1,$$
$$r_2, r_3, r_4, r_5, m_1, m_2, r_m, k_m, f_1, f_2, f_3, f_4, f_5, f_6, k_1, k_2 \leq 1.$$

Then,

(*i*) *The disease-free equilibrium point* (*DFE*) *is*

$$\varepsilon^0 = \left(S_1^0,\ S_2^0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ S_m^0,\ 0,\ 0,\ 0\right) \tag{11}$$

where

$$S_1^0 = \frac{\Lambda_f}{p_1 + \mu_f}, \quad S_2^0 = \frac{p_1\Lambda_f}{\mu_f\left(p_1 + \mu_f\right)}, \quad S_m^0 = \frac{\Lambda_m}{\mu_m} \tag{12}$$

(*ii*) *The basic reproductive number is*

$$\mathcal{R}_0 = \sqrt{\frac{\left(m_1\mu_m\theta_m + m_1 r_m\theta_m + m_2\theta_n r_m\right)\left(\dfrac{p_{11}}{2} + \dfrac{p_{19}}{2} + \dfrac{\sqrt{p_{11}^2 - 2p_{11}p_{19} + 4p_{13}p_{17} + p_{19}^2}}{2}\right)}{\mu_m^2 + \mu_m r_m + \mu_m\tau_m + r_m\tau_m}} \tag{13}$$

where

$$p_{11}, \quad p_{13}, \quad p_{17}, \quad p_{19}, \quad are \quad given \quad by \quad (32), \quad (34) \quad and \quad (35) \tag{14}$$

Proof. Here, we consider the proposed mathematical model(1) with seventeen homogeneous compartments. This model can be written as:

$\frac{d}{dt}(S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_C, S_m, E_m, I_m, R_m)^T =$

$F(S_1, S_2, E_1, I_1, R_1, E_2, I_2, R_2, P, Q, T, C, R_C, S_m, E_m, I_m, R_m)^T$

Where F is defined by(4) . The point ε^0 defined by (11) satisfies $F(\varepsilon^0) = 0$.

The T and C compartments already have precancerous lesions treated and cancerous cells, so will no longer be part of the virus propagation process.

To compute the reproduction rate $\mathcal{R}_0$, using the next generation operator method [9–11] on the system model(1). Only the infected and infectious non-cancerous compartments are to be considered, satisfying the following seventh-order system:

$$\frac{d}{dt}\begin{pmatrix} E_1 \\ I_1 \\ E_2 \\ I_2 \\ P \\ Q \\ E_m \\ I_m \end{pmatrix} = \begin{pmatrix} (1-p_1)\,\lambda_m S_1 + \lambda_m k_f R_1 + \rho_1 I_2 + \rho_2 P + \rho_3 Q - (\sigma_i + \mu_f)\,E_1 \\ \tau_1 \sigma_i E_1 - (\sigma_1 + \mu_f)\,I_1 \\ (1-\tau_1)\,\sigma_i E_1 + \lambda_m S_2 + B_f S_2 + \lambda_m k_f R_2 - (\tau_2 + \mu_f)\,E_2 \\ \tau_2 E_2 + (1-r_1)\,\sigma_1 I_1 - (\sigma_2 + \mu_f)\,I_2 \\ (1-r_2)\,\sigma_2 I_2 - (\sigma_3 + \mu_f)\,P \\ (1-r_3)\,\sigma_3 P - (\sigma_4 + \mu_f + \mu_q)Q \\ \lambda_f k_m R_m + \lambda_f S_m + B_m S_m - (\tau_m + \mu_m)\,E_m \\ \tau_m E_m - (r_m + \mu_m)\,I_m \end{pmatrix} \tag{15}$$

$$\frac{d}{dt}\begin{pmatrix} E_1 \\ I_1 \\ E_2 \\ I_2 \\ P \\ Q \end{pmatrix} = \begin{pmatrix} (1-p_1)\,\lambda_m S_1 + \lambda_m k_f R_1 + \rho_1 I_2 + \rho_2 P + \rho_3 Q - (\sigma_i + \mu_f)\,E_1 \\ \tau_1 \sigma_i E_1 - (\sigma_1 + \mu_f)\,I_1 \\ (1-\tau_1)\,\sigma_i E_1 + \lambda_m S_2 + B_f S_2 + \lambda_m k_f R_2 - (\tau_2 + \mu_f)\,E_2 \\ \tau_2 E_2 + (1-r_1)\,\sigma_1 I_1 - (\sigma_2 + \mu_f)\,I_2 \\ (1-r_2)\,\sigma_2 I_2 - (\sigma_3 + \mu_f)\,P \\ (1-r_3)\,\sigma_3 P - (\sigma_4 + \mu_f + \mu_q)Q \end{pmatrix} \tag{16}$$

$$\frac{d}{dt}\begin{pmatrix} E_m \\ I_m \end{pmatrix} = \begin{pmatrix} \lambda_f k_m R_m + \lambda_f S_m + B_m S_m - (\tau_m + \mu_m)\,E_m \\ \tau_m E_m - (r_m + \mu_m)\,I_m \end{pmatrix} \tag{17}$$

The rate of occurrence of new infection in the seven compartments $(E_1, I_1, E_2, I_2, P, Q, E_m, I_m,)$ is represented by the vector $\mathcal{F}$ as follows :

$$\mathcal{F} = \begin{pmatrix} (1-p_1)\dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} S_1 + \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} k_f R_1 + \rho_1 I_2 + \rho_2 P + \rho_3 Q \\ 0 \\ \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} S_2 + B_f S_2 + \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} k_f R_2 \\ 0 \\ 0 \\ 0 \\ \dfrac{\beta_f c_m(\theta_1 E_1 + \theta_2 I_1 + \theta_3 E_2 + \theta_4 I_2 + \theta_5 P + \theta_6 Q)}{N_f}(k_m R_m + S_m) + B_m S_m \\ 0 \end{pmatrix} \tag{18}$$

$$\mathcal{F}_f = \begin{pmatrix} (1-p_1)\dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} S_1 + \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} k_f R_1 + \rho_1 I_2 + \rho_2 P + \rho_3 Q \\ 0 \\ \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} S_2 + B_f S_2 + \dfrac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m} k_f R_2 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{19}$$

$$\mathcal{F}_m = \begin{pmatrix} \dfrac{\beta_f c_m(\theta_1 E_1 + \theta_2 I_1 + \theta_3 E_2 + \theta_4 I_2 + \theta_5 P + \theta_6 Q)}{N_f}(k_m R_m + S_m) + B_m S_m \\ 0 \end{pmatrix} \tag{20}$$

The transfer rate of individuals into and out of the infected compartments is given by the vector:

$$\mathcal{V} = \begin{pmatrix} (\sigma_i + \mu_f) E_1 \\ -\tau_1 \sigma_i E_1 + (\sigma_1 + \mu_f) I_1 \\ -(1-\tau_1)\sigma_i E_1 + (\tau_2 + \mu_f) E_2 \\ -\tau_2 E_2 - (1-r_1)\sigma_1 I_1 + (\sigma_2 + \mu_f) I_2 \\ -(1-r_2)\sigma_2 I_2 + (\sigma_3 + \mu_f) P \\ -(1-r_3)\sigma_3 P + (\sigma_4 + \mu_f + \mu_q) Q \\ (\tau_m + \mu_m) E_m \\ -\tau_m E_m + (r_m + \mu_m) I_m \end{pmatrix} \tag{21}$$

$$\mathcal{V}_f = \begin{pmatrix} (\sigma_i + \mu_f)\, E_1 \\ -\tau_1\sigma_i E_1 + (\sigma_1 + \mu_f)\, I_1 \\ -(1-\tau_1)\,\sigma_i E_1 + (\tau_2 + \mu_f)\, E_2 \\ -\tau_2 E_2 - (1-r_1)\,\sigma_1 I_1 + (\sigma_2 + \mu_f)\, I_2 \\ -(1-r_2)\,\sigma_2 I_2 + (\sigma_3 + \mu_f)\, P \\ -(1-r_3)\,\sigma_3 P + (\sigma_4 + \mu_f + \mu_q) Q \end{pmatrix} \tag{22}$$

$$\mathcal{V}_m = \begin{pmatrix} (\tau_m + \mu_m)\, E_m \\ -\tau_m E_m + (r_m + \mu_m)\, I_m \end{pmatrix} \tag{23}$$

By linearizing $\mathcal{F}$ and $\mathcal{V}$ in ε^0 and using (6), we obtain :

$$F = \begin{pmatrix} 0 & 0 & 0 & \rho_1 & \rho_2 & \rho_3 & \theta_m\Phi_1 & \theta_n\Phi_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{f_1\theta 1 S_2^0}{S_1^0+S_2^0} & \frac{f_2\theta 2 S_2^0}{S_1^0+S_2^0} & \frac{f_3\theta 3 S_2^0}{S_1^0+S_2^0} & \frac{f_4\theta 4 S_2^0}{S_1^0+S_2^0} & \frac{f_5\theta 5 S_2^0}{S_1^0+S_2^0} & \frac{f_6\theta 6 S_2^0}{S_1^0+S_2^0} & \theta_m\Phi_2 & \theta_n\Phi_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \theta_1\Phi_3 & \theta_3\Phi_3 & \theta_2\Phi_3 & \theta_4\Phi_3 & \theta_5\Phi_3 & \theta_6\Phi_3 & m_1\theta_m & m_2\theta_n \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{24}$$

With:

$$F_1 = \begin{pmatrix} 0 & 0 & 0 & \rho_1 & \rho_2 & \rho_3 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{f_1\theta 1 S_2^0}{S_1^0+S_2^0} & \frac{f_2\theta 2 S_2^0}{S_1^0+S_2^0} & \frac{f_3\theta 3 S_2^0}{S_1^0+S_2^0} & \frac{f_4\theta 4 S_2^0}{S_1^0+S_2^0} & \frac{f_5\theta 5 S_2^0}{S_1^0+S_2^0} & \frac{f_6\theta 6 S_2^0}{S_1^0+S_2^0} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{25}$$

and

$$F_2 = \begin{pmatrix} m_1\theta_m & m_2\theta_n \\ 0 & 0 \end{pmatrix} \tag{26}$$

$$V = \begin{pmatrix} d_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\tau_1\sigma_i & d_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ -(1-\tau_1)\sigma_i & 0 & d_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(1-r_1)\sigma_1 & -\tau_2 & d_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(1-r_2)\sigma_2 & d_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(1-r_3)\sigma_3 & d_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & d_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\tau_m & d_8 \end{pmatrix} \tag{27}$$

With:

$$V_1 = \begin{pmatrix} d_1 & 0 & 0 & 0 & 0 & 0 \\ -\tau_1\sigma_i & d_2 & 0 & 0 & 0 & 0 \\ -(1-\tau_1)\sigma_i & 0 & d_3 & 0 & 0 & 0 \\ 0 & -(1-r_1)\sigma_1 & -\tau_2 & d_4 & 0 & 0 \\ 0 & 0 & 0 & -(1-r_2)\sigma_2 & d_5 & 0 \\ 0 & 0 & 0 & 0 & -(1-r_3)\sigma_3 & d_6 \end{pmatrix} \tag{28}$$

and

$$V_1^{-1} = \begin{pmatrix} \frac{1}{d_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\sigma_i\tau_1}{d_2d_1} & \frac{1}{d_2} & 0 & 0 & 0 & 0 \\ \frac{\pi_i}{d_3d_1} & 0 & \frac{1}{d_3} & 0 & 0 & 0 \\ \frac{\pi_1d_3\sigma_i\tau_1+\pi_i\tau_2d_2}{d_4d_3d_2d_1} & \frac{\pi_1}{d_4d_2} & \frac{\tau_2}{d_4d_3} & \frac{1}{d_4} & 0 & 0 \\ \frac{\pi_2(\pi_1d_3\sigma_i\tau_1+\pi_i\tau_2d_2)}{d_4d_5d_3d_2d_1} & \frac{\pi_1\pi_2}{d_4d_5d_2} & \frac{\pi_2\tau_2}{d_4d_5d_3} & \frac{\pi_2}{d_4d_5} & \frac{1}{d_5} & 0 \\ \frac{\pi_2\pi_3(\pi_1d_3\sigma_i\tau_1+\pi_i\tau_2d_2)}{d_4d_5d_3d_2d_6d_1} & \frac{\pi_1\pi_2\pi_3}{d_4d_5d_6d_2} & \frac{\pi_2\pi_3\tau_2}{d_4d_5d_6d_3} & \frac{\pi_2\pi_3}{d_4d_6d_5} & \frac{\pi_3}{d_6d_5} & \frac{1}{d_6} \end{pmatrix} \tag{29}$$

$$V_2 = \begin{pmatrix} \tau_m+\mu_m & 0 \\ -\tau_m & r_m+\mu_m \end{pmatrix} \tag{30}$$

and

$$V_2^{-1} = \begin{pmatrix} \frac{1}{\tau_m+\mu_m} & 0 \\ \frac{r_m}{(\tau_m+\mu_m)(r_m+\mu_m)} & \frac{1}{r_m+\mu_m} \end{pmatrix} \tag{31}$$

$\Phi_1 = \frac{\beta_m c_f \mu_m \Lambda_f (1-p_1)}{\Lambda_m (p_1+\mu_f)}$; $\Phi_2 = \frac{\beta_m c_f \mu_m p_1 \Lambda_f}{\Lambda_m \mu_f (p_1+\mu_f)}$; $\Phi_3 = \frac{\beta_f c_m \Lambda_m \mu_f}{\Lambda_f \mu_m}$;

$S_2^0 = \frac{p_1\Lambda_f}{\mu_f(p_1+\mu_f)}$; $S_1^0 = \frac{\Lambda_f}{p_1+\mu_f}$

Where $d_1 = \sigma_i + \mu_f$, $d_2 = \sigma_1 + \mu_f$, $d_3 = \tau_2 + \mu_f$, $d_4 = \sigma_2 + \mu_f$, $d_5 = \sigma_3 + \mu_f$,

$d_6 = \sigma_4 + \mu_f + \mu_q, \quad d_7 = \tau_m + \mu_m, \quad d_8 = r_m + \mu_m, \quad \pi_i = (1-\tau_1)\sigma_i, \quad \pi_1 = (1-r_1)\sigma_1,$
$\pi_2 = (1-r_2)\sigma_2, \quad \pi_3 = (1-r_3)\sigma_3$

$$p_{11} = \frac{\rho_1\left(\pi_1 d_3\sigma_i\tau_1 + \pi_i\tau_2 d_2\right)}{d_4 d_3 d_2 d_1} + \frac{\rho_2\pi_2\left(\pi_1 d_3\sigma_i\tau_1 + \pi_i\tau_2 d_2\right)}{d_4 d_5 d_3 d_2 d_1} + \frac{\rho_3\pi_2\pi_3\left(\pi_1 d_3\sigma_i\tau_1 + \pi_i\tau_2 d_2\right)}{d_4 d_5 d_6 d_3 d_2 d_1}, \tag{32}$$

$$p_{13} = \frac{\rho_1\tau_2}{d_4 d_3} + \frac{\rho_2\pi_2\tau_2}{d_4 d_5 d_3} + \frac{\rho_3\pi_2\pi_3\tau_2}{d_4 d_5 d_6 d_3} \tag{33}$$

$$p_{17} = \frac{S_2}{S_1+S_2}\left[\frac{f_1\theta_1}{d_1} + \frac{f_2\theta_2\sigma_i\tau_1}{d_2 d_1} + \frac{f_3\theta_3\pi_i}{d_3 d_1} + \frac{\left(\pi_1 d_3\sigma_i\tau_1 + \pi_i\tau_2 d_2\right)}{d_4 d_3 d_2 d_1}\left(f_4\theta_4 + \frac{f_5\theta_5\pi_2}{d_5} + \frac{f_6\theta_6\pi_2\pi_3}{d_6 d_5}\right)\right] \tag{34}$$

$$p_{19} = \frac{S_2}{S_1+S_2}\left[\frac{f_3\theta_3}{d_3} + \frac{f_4\theta_4\tau_2}{d_4 d_3} + \frac{f_5\theta_5\pi_2\tau_2}{d_4 d_5 d_3} + \frac{f_6\theta_6\pi_2\pi_3\tau_2}{d_6 d_4 d_5 d_3}\right] \tag{35}$$

Thus, the effective number of reproducers $\mathcal{R}_0$ for model 1 is given by :

$\mathcal{R}_0 = \rho(FV^{-1})$ where ρ represents the spectral radius

$\mathcal{R}_0 = \sqrt{\mathcal{R}_m\mathcal{R}_f}$ With :

$\mathcal{R}_f = \rho(F_1 V_1^{-1})$

$\mathcal{R}_f = \frac{p_{11}}{2} + \frac{p_{19}}{2} + \frac{\sqrt{p_{11}^2 - 2p_{11}p_{19} + 4p_{13}p_{17} + p_{19}^2}}{2}$

$\mathcal{R}_m = \rho(F_2 V_2^{-1})$

$\mathcal{R}_m = \frac{m_1\mu_m\theta_m + m_1 r_m\theta_m + m_2\theta_n r_m}{\mu_m^2 + \mu_m r_m + \mu_m\tau_m + r_m\tau_m}$

$$\mathcal{R}_0 = \sqrt{\frac{\left(m_1\mu_m\theta_m + m_1 r_m\theta_m + m_2\theta_n r_m\right)\left(\frac{p_{11}}{2} + \frac{p_{19}}{2} + \frac{\sqrt{p_{11}^2 - 2p_{11}p_{19} + 4p_{13}p_{17} + p_{19}^2}}{2}\right)}{\mu_m^2 + \mu_m r_m + \mu_m\tau_m + r_m\tau_m}} \tag{36}$$

$\mathcal{R}_0$ measures the average number of new HPV infections generated by a single infected individual introduced into a fully susceptible population [5–7].

$\mathcal{R}_f$ measures the average number of new HPV infections in the female population generated by a single infected male introduced into a fully susceptible female population.

$\mathcal{R}_m$ measures the average number of new HPV infections in the male population generated by a single infected female introduced into a fully susceptible male population. □

3.4. **Global stability of disease-free equilibrium point.** In this section, we will present the global stability of the disease-free equilibrium of the system(1) using the CASTILLO CHAVEZ technique [12,13]. Thus, the system (1) must be written in the form:

$$\begin{cases} \dfrac{dX}{dt} = F\left(X, I\right) \\ \dfrac{dI}{dt} = G\left(X, I\right), \\ G\left(X, 0\right) = 0 \end{cases} \tag{37}$$

where $X = (S_1, S_2, R_1, R_2, R_C, S_m, R_m) \in \mathbf{R}^7$ and $I = (E_1, I_1, E_2, I_2, P, Q, E_m, I_m) \in \mathbf{R}^8$ respectively denote the population of non-infected individuals and that of infected individuals (latent and infectious).

The disease-free equilibrium point calculated in Section 3 is:

$$\varepsilon^0 = \left(S_1^0,\ S_2^0,\ \ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ S_m^0,\ 0,\ 0,\ 0\right) \tag{38}$$

$$\varepsilon^0 = \left(S_1^0,\ S_2^0,\ S_m^0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\right) \tag{39}$$

$$\varepsilon^0 = \left(\frac{\Lambda_f}{p_1+\mu_f},\ \frac{p_1\Lambda_f}{\mu_f\left(p_1+\mu_f\right)},\ \frac{\Lambda_m}{\mu_m},\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\right) \tag{40}$$

ε^0 can be written as:

$$\varepsilon^0 = \left(S^0,\ 0\right) \tag{41}$$

The following conditions (H_1) and (H_2) below must be satisfied to ensure local asymptotic stability.

(H_1): if $\dfrac{dX}{dt} = F\left(X, 0\right)$, then ε^0 is globally asymptotically stable.

$(H_2) : G\left(X, I\right) = AI - \widehat{G}\left(X, I\right) \quad$ ou $\widehat{G}\left(X, I\right) \geq 0 \quad$ pour $(X, I) \in \Omega$

Here , $A = D_I G\left(S^0, 0\right)$ is an M matrix (off-diagonal elements of A are non-negative).

If the system satisfies these two conditions, the following theorem applies:

Theorem 3.3. *The fixed point $\varepsilon^0 = \left(S^0, 0\right)$ is a globally asymptotically stable equilibrium of the system provided $\mathcal{R}_0 < 1$ and both hypotheses $(H1)$ and (H_2) are satisfied.*

Proof. It is assumed that compartments T and C are non-infectious and therefore cannot transmit the virus.

$$F\left(X, I\right) = \begin{pmatrix} \Lambda_f - \rho_1 I_2 - \rho_2 P - \rho_3 Q - \left(1 - p_1\right)\dfrac{\beta_m c_f\left(\theta_m E_m + \theta_n I_m\right)}{N_m} S_1 - \left(p_1 + \mu_f\right) S_1 \\ p_1 S_1 - \dfrac{\beta_m c_f\left(\theta_m E_m + \theta_n I_m\right)}{N_m} S_2 - B_f S_2 - \mu_f S_2 \\ r_1\sigma_1 I_1 - \dfrac{\beta_m c_f\left(\theta_m E_m + \theta_n I_m\right)}{N_m} k_f R_1 - \mu_f R_1 \\ r_2\sigma_2 I_2 + r_3\sigma_3 P + r_5\sigma_5 T - \dfrac{\beta_m c_f\left(\theta_m E_m + \theta_n I_m\right)}{N_m} k_f R_2 - \mu_f R_2 \\ r_4 C - \mu_f R_C \\ \Lambda_m - \dfrac{\beta_f c_m\left(\theta_1 E_1 + \theta_2 I_1 + \theta_3 E_2 + \theta_4 I_2 + \theta_5 P + \theta_6 Q\right)}{N_f} S_m - B_m S_m - \mu_m S_m \\ r_m I_m - \dfrac{\beta_f c_m\left(\theta_1 E_1 + \theta_2 I_1 + \theta_3 E_2 + \theta_4 I_2 + \theta_5 P + \theta_6 Q\right)}{N_f} k_m R_m - \mu_m R_m \end{pmatrix} \tag{42}$$

$$G(X,I)=\begin{pmatrix}\frac{\beta_m c_f(\theta_m E_m+\theta_n I_m)}{N_m}((1-p_1)S_1+k_f R_1)+\rho_1 I_2+\rho_2 P+\rho_3 Q-(\sigma_i+\mu_f)E_1\\ \tau_1\sigma_i E_1-(\sigma_1+\mu_f)I_1\\ (1-\tau_1)\sigma_i E_1+\frac{\beta_m c_f(\theta_m E_m+\theta_n I_m)}{N_m}(S_2+k_f R_2)+B_f S_2-d_3 E_2\\ \tau_2 E_2+(1-r_1)\sigma_1 I_1-(\sigma_2+\mu_f)I_2\\ (1-r_2)\sigma_2 I_2-(\sigma_3+\mu_f)P\\ (1-r_3)\sigma_3 P-(\sigma_4+\mu_f+\mu_q)Q\\ \frac{\beta_f c_m(\theta_1 E_1+\theta_2 I_1+\theta_3 E_2+\theta_4 I_2+\theta_5 P+\theta_6 Q)}{N_f}(k_m R_m+S_m)+B_m S_m-(\tau_m+\mu_m)E_m\\ \tau_m E_m-(r_m+\mu_m)I_m\end{pmatrix} \tag{43}$$

$$G(X,I)=\begin{pmatrix}G_1(X,I)\\ G_2(X,I)\\ G_3(X,I)\\ G_4(X,I)\\ G_5(X,I)\\ G_6(X,I)\\ G_7(X,I)\\ G_8(X,I)\end{pmatrix} \tag{44}$$

It is clear that at the point DFE, $G(X,0)=0$.

Next, we will verify that if $\frac{dX}{dt}=F(X,0)$, then ε^0 is globally asymptotically stable. To do this, let's compute the eigenvalues of the Jacobian matrix $\left(D_X F\left(\varepsilon^0\right)\right)$ associated with F at the DFE.

$$D_X F\left(\varepsilon^0\right)=\begin{pmatrix}-(P_1+\mu_f) & 0 & 0 & 0 & 0 & 0 & 0\\ P_1 & -\mu_f & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & -\mu_f & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & -\mu_f & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & -\mu_f & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & -\mu_m & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m\end{pmatrix} \tag{45}$$

Given that the eigenvalues $\vartheta_1=-(P_1+\mu_f),\quad \vartheta_2=-\mu_f,\quad \vartheta_3=-\mu_f,\quad \vartheta_4=-\mu_f,\quad \vartheta_5=-\mu_f,\quad \vartheta_6=-\mu_f,\quad \vartheta_7=-\mu_f$ of the Jacobian matrix are all negative, the DFE point is globally asymptotically stable. To verify that $G(X,I)=A\times I-\widehat{G}(X,I)$ or $\widehat{G}(X,I)\geq 0$, we will first compute the matrix

$A = D_I G\left(S^0, 0\right)$

$A = F - V$

$$A = \begin{pmatrix} -d_1 & 0 & 0 & \rho_1 & \rho_2 & \rho_3 & \theta_m\Phi_1 & \theta_n\Phi_1 \\ \tau_1\sigma_i & -d_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\eta_1 S_2^0}{N_f^0} + \pi_i & \frac{\eta_2 S_2^0}{N_f^0} & \frac{\eta_3 S_2^0}{N_f^0} - d_3 & \frac{\eta_4 S_2^0}{N_f^0} & \frac{\eta_5 S_2^0}{N_f^0} & \frac{\eta_6 S_2^0}{N_f^0}\theta_m\Phi_2 & \theta_n\Phi_2 & \\ 0 & \pi_1 & \tau_2 & -d_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \pi_2 & -d_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \pi_3 & -d_6 & 0 & 0 \\ \theta_1\Phi_3 & \theta_3\Phi_3 & \theta_2\Phi_3 & \theta_4\Phi_3 & \theta_5\Phi_3 & \theta_6\Phi_3 & m_1\theta_m - d_7 & m_2\theta_n \\ 0 & 0 & 0 & 0 & 0 & 0 & \tau_m & -d_8 \end{pmatrix} \tag{46}$$

Where: $\eta_1 = f_1\theta_1$, $\eta_2 = f_2\theta_2$, $\eta_3 = f_3\theta_3$,$\eta_4 = f_4\theta_4$, $\eta_5 = f_5\theta_5$ and $\eta_6 = f_6\theta_6$

It is clear that matrix A is an M-matrix (the off-diagonal elements of A are non-negative).

We can now compute the function $\widehat{G}\left(X, I\right)$:

$$AI = \begin{pmatrix} -d_1E_1 + \rho_1I_2 + \rho_2P + \rho_3Q + \theta_m\Phi_1E_m + \theta_n\Phi_1I_m \\ \tau_1\sigma_iE_1 - d_2I_1 \\ \left(\frac{\eta_1 S_2^0}{N_f^0} + \pi_i\right)E_1 + \frac{\eta_2 S_2^0}{N_f^0}I_1 + \left(\frac{\eta_3 S_2^0}{N_f^0} - d_3\right)E_2 + \frac{\eta_4 S_2^0}{N_f^0}I_2 + \frac{\eta_5 S_2^0}{N_f^0}P + \frac{\eta_6 S_2^0}{N_f^0}Q \\ + \theta_m\Phi_2E_m + \theta_n\Phi_2I_m \\ \pi_1I_1 + \tau_2E_2 - d_4I_2 \\ \pi_2I_2 - d_5P \\ \pi_3P - d_6Q \\ \theta_1\Phi_3E_1 + \theta_3\Phi_3I_1 + \theta_2\Phi_3E_2 + \theta_4\Phi_3I_2 + \theta_5\Phi_3P + \theta_6\Phi_3Q + \left(m_1\theta_m - d_7\right)E_m + m_2\theta_nI_m \\ \tau_mE_m - d_8I_m \end{pmatrix} \tag{47}$$

$G\left(X, I\right) = AI - \widehat{G}\left(X, I\right) \Rightarrow \widehat{G}\left(X, I\right) = AI - G\left(X, I\right)$

$$\widehat{G}\left(X, I\right) = \begin{pmatrix} \widehat{G}_1\left(X, I\right) \\ 0 \\ \widehat{G}_3\left(X, I\right) \\ 0 \\ 0 \\ 0 \\ \widehat{G}_7\left(X, I\right) \\ 0 \end{pmatrix} \tag{48}$$

With

$$\widehat{G}_1(X,I) = \theta_m\Phi_1 E_m + \Phi_1\theta_n I_m - (1-p_1)\frac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m}S_1 - \frac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m}k_f R_1$$

$$\widehat{G}_1(X,I) = \Phi_1(\theta_m E_m + \theta_n I_m) - (\theta_m E_m + \theta_n I_m)\left((1-p_1)\frac{\beta_m c_f}{N_m}S_1 + \frac{\beta_m c_f}{N_m}k_f R_1\right)$$

$$\Phi_1 = \frac{\beta_m c_f \mu_m \Lambda_f (1-p_1)}{\Lambda_m(p_1+\mu_f)}$$

$$\Phi_1 = (1-p_1)\frac{\beta_m c_f}{N_m^0}S_1^0$$

$$N_m^0 = S_m^0 = \frac{\Lambda_m}{\mu_m}$$

$$S_1^0 = \frac{\Lambda_f}{p_1+\mu_f}$$

$$\widehat{G}_1(X,I) = (1-p_1)\frac{\beta_m c_f}{N_m^0}S_1^0(\theta_m E_m + \theta_n I_m) - (\theta_m E_m + \theta_n I_m)\left((1-p_1)\frac{\beta_m c_f}{N_m}S_1 + \frac{\beta_m c_f}{N_m}k_f R_1\right)$$

$$\widehat{G}_1(X,I) = \beta_m c_f(\theta_m E_m + \theta_n I_m)\left[(1-p_1)\frac{S_1^0}{N_m^0} - (1-p_1)\frac{S_1}{N_m} - \frac{k_f R_1}{N_m}\right]$$

$$\widehat{G}_1(X,I) = \beta_m c_f(\theta_m E_m + \theta_n I_m)(1-p_1)\left[\frac{S_1^0}{N_m^0} - \frac{S_1}{N_m} - \frac{k_f R_1}{N_m(1-p_1)}\right]$$

$$\widehat{G}_1(X,I) \geq 0 \Leftrightarrow \left[\frac{S_1^0}{N_m^0} - \frac{S_1}{N_m} - \frac{k_f R_1}{N_m(1-p_1)}\right] \geq 0$$

$$\widehat{G}_1(X,I) \geq 0 \Leftrightarrow \frac{S_1}{N_m} + \frac{k_f R_1}{N_m(1-p_1)} \leq \frac{S_1^0}{N_m^0}$$

$$\widehat{G}_1(X,I) \geq 0 \Leftrightarrow \frac{S_1}{N_m} \leq \frac{S_1^0}{N_m^0}$$

$$\widehat{G}_1(X,I) \geq 0 \Leftrightarrow S_1 \leq S_1^0\frac{N_m}{N_m^0}$$

$$like \quad N_m \leq \frac{\Lambda_m}{\mu_m} \quad , \quad N_m^0 = S_m^0 = \frac{\Lambda_m}{\mu_m}$$

$$then \quad N_m \leq N_m^0 \quad and \quad \frac{N_m}{N_m^0} \leq 1$$

$$\widehat{G}_1(X,I) \geq 0 \Leftrightarrow S_1 \leq S_1^0$$

$$\widehat{G}_3(X,I) = \theta_m\Phi_2 E_m + \theta_n\Phi_2 I_m - \frac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m}(S_2 + k_f R_2) + B_f N_f\left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right)$$

$$\widehat{G}_3(X,I) = \theta_m\Phi_2 E_m + \theta_n\Phi_2 I_m - \frac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m}S_2 - \frac{\beta_m c_f(\theta_m E_m + \theta_n I_m)}{N_m}k_f R_2 + B_f N_f\left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right)$$

$$\widehat{G}_3(X,I) = \Phi_2(\theta_m E_m + \theta_n I_m) - (\theta_m E_m + \theta_n I_m)\left(\frac{\beta_m c_f}{N_m}S_2 + \frac{\beta_m c_f}{N_m}k_f R_2\right) + B_f N_f\left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right) \tag{49}$$

$$\Phi_2 = \frac{\beta_m c_f}{N_m^0} S_2^0$$

$$N_m^0 = S_m^0 = \frac{\Lambda_m}{\mu_m}$$

$$S_2^0 = \frac{p_1 \Lambda_f}{\mu_f \left(p_1 + \mu_f\right)}$$

$$\widehat{G}_3\left(X, I\right) = \frac{\beta_m c_f}{N_m^0} S_2^0 \left(\theta_m E_m + \theta_n I_m\right) - \left(\theta_m E_m + \theta_n I_m\right) \left(\frac{\beta_m c_f}{N_m} S_2 + \frac{\beta_m c_f}{N_m} k_f R_2\right) + B_f N_f \left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right)$$

$$\widehat{G}_3\left(X, I\right) = \beta_m c_f \left(\theta_m E_m + \theta_n I_m\right) \left[\frac{S_2^0}{N_m^0} - \frac{S_2}{N_m} - \frac{k_f R_2}{N_m}\right] + B_f N_f \left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right)$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow \beta_m c_f \left(\theta_m E_m + \theta_n I_m\right) \left[\frac{S_2^0}{N_m^0} - \frac{S_2}{N_m} - \frac{k_f R_2}{N_m}\right] + B_f N_f \left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right) \geq 0$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow \left[\frac{S_2^0}{N_m^0} - \frac{S_2}{N_m} - \frac{k_f R_2}{N_m}\right] \geq 0 \quad and \quad \left(\frac{S_2^0}{N_f^0} - \frac{S_2}{N_f}\right) \geq 0$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow \frac{S_2^0}{N_m^0} \geq \frac{S_2}{N_m} + \frac{k_f R_2}{N_m} \quad and \quad \frac{S_2^0}{N_f^0} \geq \frac{S_2}{N_f}$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow \frac{S_2^0}{N_m^0} \geq \frac{S_2}{N_m} \quad and \quad \frac{S_2^0}{N_f^0} \geq \frac{S_2}{N_f}$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow \frac{S_2}{N_m} \leq \frac{S_2^0}{N_m^0} \quad and \quad \frac{S_2}{N_f} \leq \frac{S_2^0}{N_f^0}$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow S_2 \leq \frac{S_2^0 N_m}{N_m^0} \quad and \quad S_2 \leq \frac{S_2^0 N_f}{N_f^0}$$

$$\widehat{G}_3\left(X, I\right) \geq 0 \Leftrightarrow S_2 \leq S_2^0 \times 1 \quad because \quad \frac{N_m}{N_m^0} \leq 1 \quad and \quad \frac{N_f}{N_f^0} \leq 1$$

$$\widehat{G}_7\left(X, I\right) = m_1 \theta_m E_m + m_2 \theta_n I_m - \frac{\lambda_f N_f}{\beta_f c_m} \left[-\Phi_3 + \frac{\beta_f c_m}{N_f} \left(k_m R_m + S_m\right)\right] - \frac{\left(m_1 \theta_m E_m + m_2 \theta_n I_m\right)}{N_m} S_m$$

$$\widehat{G}_7\left(X, I\right) = m_1 \theta_m E_m + m_2 \theta_n I_m - \frac{\lambda_f N_f}{\beta_f c_m} \left[-\Phi_3 + \frac{\beta_f c_m}{N_f} k_m R_m + \frac{\beta_f c_m}{N_f} S_m\right] - \frac{\left(m_1 \theta_m E_m + m_2 \theta_n I_m\right)}{N_m} S_m$$

$$\Phi_3 = \frac{\beta_f c_m \mu_f S_m^0}{\Lambda_f}$$

$$S_m^0 = \frac{\Lambda_m}{\mu_m}$$

$$\widehat{G}_7\left(X, I\right) = \left(m_1 \theta_m E_m + m_2 \theta_n I_m\right) \left(1 - \frac{S_m}{N_m}\right) + \frac{\lambda_f N_f}{\beta_f c_m} \left[\Phi_3 - \frac{\beta_f c_m}{N_f} k_m R_m - \frac{\beta_f c_m}{N_f} S_m\right]$$

$$\widehat{G}_7\left(X, I\right) \geq 0 \Leftrightarrow \left[\Phi_3 - \frac{\beta_f c_m}{N_f} k_m R_m - \frac{\beta_f c_m}{N_f} S_m\right] \geq 0$$

(50)

$$
\begin{aligned}
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow\left[\frac{\beta_f c_m\mu_f S_m^0}{\Lambda_f}-\frac{\beta_f c_m}{N_f}k_m R_m-\frac{\beta_f c_m}{N_f}S_m\right]\geq 0\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow\beta_f c_m\left[\frac{\mu_f S_m^0}{\Lambda_f}-\frac{k_m R_m}{N_f}-\frac{S_m}{N_f}\right]\geq 0\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow\left[\frac{\mu_f S_m^0}{\Lambda_f}-\frac{k_m R_m}{N_f}-\frac{S_m}{N_f}\right]\geq 0\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow\frac{\mu_f S_m^0}{\Lambda_f}-\frac{k_m R_m}{N_f}\geq\frac{S_m}{N_f}\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow\frac{\mu_f S_m^0}{\Lambda_f}\geq\frac{S_m}{N_f}\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow S_m\leq\frac{S_m^0 N_f\mu_f}{\Lambda_f};\quad we\quad have\quad N_f\leq\frac{\Lambda_f}{\mu_f}\Rightarrow\frac{N_f\mu_f}{\Lambda_f}\leq 1\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow S_m\leq S_m^0\times 1\\
&\widehat{G}_7\left(X,I\right)\geq 0\Leftrightarrow S_m\leq S_m^0
\end{aligned}
\tag{51}
$$

From the above, we can say that: $\widehat{G}\left(X,I\right)\geq 0\Leftrightarrow S_1\leq S_1^0$,$S_2\leq S_2^0$ and $S_m\leq S_m^0$.Therefore, the conditions (H_1) and (H_2) above are satisfied. Thus, we can use the CASTILLO CHAVEZ technique to conclude that if $\mathcal{R}_0<1$, then the DFE point is globally asymptotically stable. □

3.5. **Endemic equilibruim point ε^*.**

3.5.1. *Existence of ε^*.*

Theorem 3.4. *The system (1) admits a unique positive endemic equilibrium* $\varepsilon^*=(S_1^*,S_2^*,E_1^*,I_1^*,R_1^*,E_2^*,I_2^*,R_2^*,P^*,Q^*,C^*,R_C^*,S_m^*,E_m^*,I_m^*,R_m^*)$ *whenever* $\mathcal{R}_0>1$.

Proof. By setting the right-hand side of system (1) to zero and assuming all variables are non-zero $(S_1\neq 0,S_2\neq 0,E_1\neq 0,I_1\neq 0,\ldots,R_m\neq 0)$ we obtain:

$$
\begin{aligned}
&S_1^*=\frac{\Lambda_f-\rho_1 I_2^*-\rho_2 P^*-\rho_3 Q^*}{(1-p_1)\lambda_m^*+p_1+\mu_f};\quad S_2^*=\frac{p_1 S_1^*}{\lambda_m^*+B_f^*+\mu_f}\\
&E_1^*=\frac{(1-p_1)\lambda_m^*\left(S_1^*+k_f R_1^*\right)+\rho_1 I_2^*+\rho_2 P^*+\rho_3 Q^*}{\sigma_i+\mu_f};\quad I_1^*=\frac{\tau_1\sigma_i E_1^*}{(\sigma_1+\mu_f)};\quad R_1^*=\frac{r_1\sigma_1 I_1^*}{\lambda_m^* k_f+\mu_f}\\
&E_2^*=\frac{\lambda_m^*\left(S_2^*+k_f R_2^*\right)+(1-\tau_1)\sigma_i E_1^*+B_f^* S_2^*}{(\tau_2+\mu_f)};\quad I_2^*=\frac{\tau_2 E_2^*+(1-r_1)\sigma_1 I_1^*}{(\sigma_2+\mu_f)}\\
&R_2^*=\frac{r_2\sigma_2 I_2^*+r_3\sigma_3 P^*+r_5\sigma_5 T^*}{\lambda_m^* k_f+\mu_f};\quad P^*=\frac{(1-r_2)\sigma_2 I_2^*}{(\sigma_3+\mu_f)};\quad Q^*=\frac{(1-r_3)\sigma_3 P^*}{(\sigma_4+\mu_f+\mu_q)}\\
&T^*=\frac{r\sigma_4 Q^*}{(\sigma_5+\mu_f+\mu_t)};\quad C^*=\frac{(1-r)\sigma_4 Q^*+(1-r_5)\sigma_5 T^*}{(r_4+\mu_f+\mu_c)};\quad R_C^*=\frac{r_4 C^*}{\mu_f};\quad S_m^*=\frac{\Lambda_m}{\lambda_f^*+B_m^*+\mu_m}\\
&E_m^*=\frac{\lambda_f^*\left(k_m R_m^*+S_m^*\right)+B_m^* S_m^*}{(\tau_m+\mu_m)};\quad I_m^*=\frac{\tau_m E_m^*}{(r_m+\mu_m)};\quad R_m^*=\frac{r_m I_m^*}{\lambda_f^* k_m+\mu_m}
\end{aligned}
\tag{52}
$$

□

3.5.2. *Global stability of ε^*.* By constructing an appropriate Lyapunov function, we will demonstrate the global asymptotic stability of the endemic equilibrium point.

Theorem 3.5. *If $\mathcal{R}_0 > 1$, the global endemic equilibrium point ε^* of system (1) is globally asymptotically stable.*

Proof. When $\mathcal{R}_0 > 1$, the basic reproduction number is positive, confirming the existence of the endemic equilibrium ε^*. To demonstrate global asymptotic stability, construct the Lyapunov function [4, 14–17]:

$$L = (S_1 - S_1^*) + (S_2 - S_2^*) + (E_1 - E_1^*) + (I_1 - I_1^*) + (R_1 - R_1^*) + (E_2 - E_2^*) + (I_2 - I_2^*) + (R_2 - R_2^*) + (P - P^*) + (Q - Q^*) + (T - T^*) + (C - C^*) + (R_C - R_C^*) + (S_m - S_m^*) + (E_m - E_m^*) + (I_m - I_m^*) + (R_m - R_m^*) - (S_1^* + S_2^* + E_1^* + I_1^* + R_1^* + E_2^* + I_2^* + R_2^* + P^* + Q^* + C^* + R_C^* + S_m^* + E_m^* + I_m^* + R_m^*) \times \ln\left(\frac{S_1 + S_2 + E_1 + I_1 + R_1 + E_2 + I_2 + R_2 + P + Q + T + C + R_C + S_m + E_m + I_m + R_m}{S_1^* + S_2^* + E_1^* + I_1^* + R_1^* + E_2^* + I_2^* + R_2^* + P^* + Q^* + T^* + C^* + R_C^* + S_m^* + E_m^* + I_m^* + R_m^*}\right)$$

Let's define:

$$N = S_1 + S_2 + E_1 + I_1 + R_1 + E_2 + I_2 + R_2 + P + Q + T + C + R_C + S_m + E_m + I_m + R_m$$

$$N^* = S_1^* + S_2^* + E_1^* + I_1^* + R_1^* + E_2^* + I_2^* + R_2^* + P^* + Q^* + T^* + C^* + R_C^* + S_m^* + E_m^* + I_m^* + R_m^*$$

The Lyapunov function can be rewritten as:

$$L = N - N^* - N^* \ln \frac{N}{N^*}$$

$$L = N^*\left(\frac{N}{N^*} - 1 - \ln \frac{N}{N^*}\right)$$

We will use the Volterra-type Lyapunov function family defined by $g(x) = x - 1 - \ln(x), x \in \mathbf{R}^+$ which has a global minimum at $x = 1$ and satisfies $g(1) = 0$.

Since $(S_1(t) > 0, \quad S_2(t) > 0, \quad E_1(t) > 0, \quad I_1(t) > 0, \quad R_1(t) > 0, \quad E_2(t) > 0, \quad I_2(t) > 0, \quad R_2(t) > 0, \quad P(t) > 0, \quad Q(t) > 0, \quad T(t) > 0, \quad C(t) > 0, \quad R_C(t) > 0, \quad S_m(t) > 0, \quad E_m(t) > 0, \quad I_m(t) > 0, \quad R_m(t) > 0,$

One can obtain the following: $L = N^*\left(\frac{N}{N^*} - 1 - \ln \frac{N}{N^*}\right) \geq 0$

Therefore, the derivative of the Lyapunov function L is given by:

$$\frac{dL}{dt} = \frac{dN}{dt}\left(1 - \frac{N^*}{N}\right) \tag{53}$$

Considering system (1) , we have:

$$\begin{aligned}\frac{dN}{dt} &= \Lambda_f - \mu_f\left(S_1 + S_2 + E_1 + I_1 + R_1 + E_2 + I_2 + R_2 + P + Q + T + C + R_C\right) \\ &\quad - \mu_q Q - \mu_t T - \mu_c C + \Lambda_m - \mu_m\left(S_m + E_m + I_m + R_m\right) \\ \frac{dN}{dt} &= \Lambda_f + \Lambda_m - \mu_f N_f - \mu_m N_m - \mu_q Q - \mu_t T - \mu_c C\end{aligned} \tag{54}$$

At the endemic equilibrium point where $\frac{dN}{dt} = 0$, we have:

$$\begin{aligned} 0 &= \Lambda_f + \Lambda_m - \mu_f N_f^* - \mu_m N_m^* - \mu_q Q^* - \mu_t T^* - \mu_c C^* \\ \Lambda_f + \Lambda_m &= \mu_f N_f^* + \mu_m N_m^* + \mu_q Q^* + \mu_t T^* + \mu_c C^* \end{aligned} \tag{55}$$

According to (53), (54), and (55), assuming $N - N^* \geq 0, \quad N_f - N_f^* \geq 0, \quad N_m - N_m^* \geq 0, \quad Q - Q^* \geq 0, \quad T - T^* \geq 0, \quad C - C^* \geq 0$, we have:

$$\begin{aligned} \frac{dL}{dt} &= \frac{dN}{dt}\left(1 - \frac{N^*}{N}\right) \\ \frac{dL}{dt} &= \left(1 - \frac{N^*}{N}\right)\left(\mu_f N_f^* + \mu_m N_m^* + \mu_q Q^* + \mu_t T^* + \mu_c C^* - \mu_f N_f - \mu_m N_m - \mu_q Q - \mu_t T - \mu_c C\right) \\ \frac{dL}{dt} &= -\left(\frac{N - N^*}{N}\right)\left(\mu_f\left(N_f - N_f^*\right) + \mu_m\left(N_m - N_m^*\right) + \mu_q\left(Q - Q^*\right) + \mu_t\left(T - T^*\right) + \mu_c\left(C - C^*\right)\right) \\ \frac{dL}{dt} &\leq 0 \end{aligned} \tag{56}$$

According to (56) and using the fact thate $\frac{dL}{dt} = 0$ if and only if $S_1 = S_1^*, \quad S_2 = S_2^*, \quad E_1 = E_1^*, \quad I_1 = I_1^*, \quad R_1 = R_1^*, \quad E_2 = E_2^*, \quad I_2 = I_2^*, , \quad R_2 = R_2^*, \quad P = P^*, \quad Q = Q^*, \quad T = T^*, \quad C = C^*, \quad R_C = R_C^*, \quad S_m = S_m^*, \quad E_m = E_m^*, \quad I_m = I_m^*, \quad R_m = R_m^*,$
then $\frac{dL}{dt}$ converges as $t \to \infty$. By LaSalle's invariance principle, the endemic equilibrium point ε^* is said to be globally asymptotically stable when $\mathcal{R}_0 > 1$ [4, 18–20, 22]. □

3.6. **Sensitivity analysis of $\mathcal{R}_0$.** To assess the impact of model parameters on the transmission dynamics of the HPV virus, a sensitivity analysis of model (1) is conducted. This analysis aims to quantify how variations in each parameter affect $\mathcal{R}_0$, the basic reproduction number, while excluding parameters μ_m and μ_f, as these cannot be targeted through intervention strategies. The standard equation for the sensitivity index of a parameter α with respect to $\mathcal{R}_0$ is given by equation (57) [24] [25, 26]:

$$\chi_\alpha^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0}. \tag{57}$$

Given the complexity of $\mathcal{R}_0$, numerical differentiation was employed to compute the sensitivity indices. The numerical results for these indices are summarized in Table 3. Figure 2 presents the results of the sensitivity analysis.

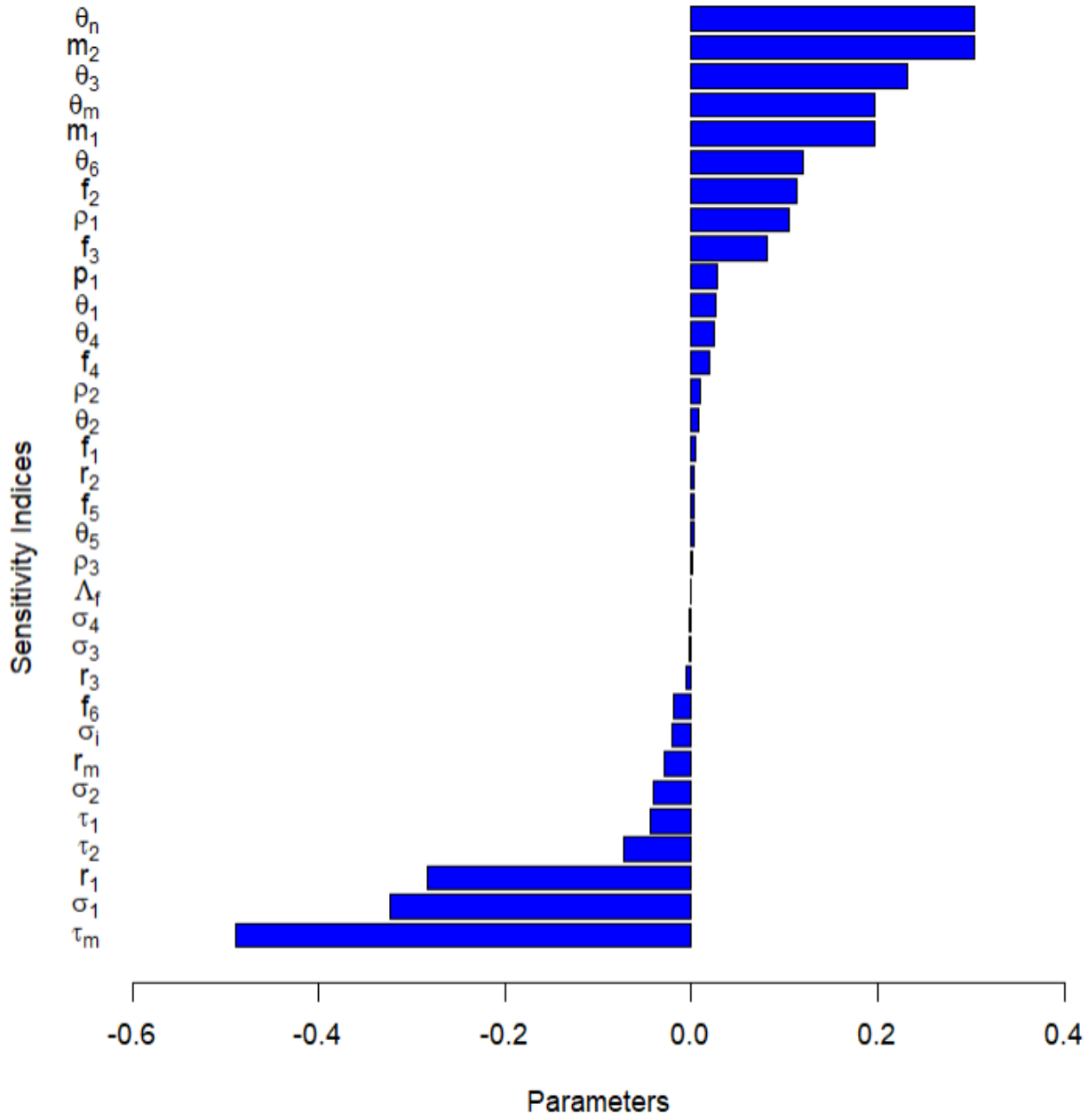

FIGURE 2. Sensitivity analysis of the model parameters

Sensitivity Index	Values
$\chi_{\rho_1}^{\mathcal{R}_0}$	0.105097
$\chi_{\rho_2}^{\mathcal{R}_0}$	0.008929
$\chi_{\rho_3}^{\mathcal{R}_0}$	0.000877
$\chi_{p_1}^{\mathcal{R}_0}$	0.027623
$\chi_{\tau_1}^{\mathcal{R}_0}$	-0.045038
$\chi_{\tau_2}^{\mathcal{R}_0}$	-0.072378
$\chi_{\tau_m}^{\mathcal{R}_0}$	-0.489685
$\chi_{m_1}^{\mathcal{R}_0}$	0.196375
$\chi_{m_2}^{\mathcal{R}_0}$	0.303625
$\chi_{\theta_1}^{\mathcal{R}_0}$	0.026503
$\chi_{\theta_m}^{\mathcal{R}_0}$	0.196375
$\chi_{\theta_n}^{\mathcal{R}_0}$	0.303625
$\chi_{\theta_2}^{\mathcal{R}_0}$	0.007454
$\chi_{\theta_3}^{\mathcal{R}_0}$	0.231115
$\chi_{\theta_4}^{\mathcal{R}_0}$	0.024067
$\chi_{\theta_5}^{\mathcal{R}_0}$	0.002397
$\chi_{\theta_6}^{\mathcal{R}_0}$	0.119413
$\chi_{f_1}^{\mathcal{R}_0}$	0.004695
$\chi_{f_2}^{\mathcal{R}_0}$	0.113392
$\chi_{f_3}^{\mathcal{R}_0}$	0.081847
$\chi_{f_4}^{\mathcal{R}_0}$	0.019575
$\chi_{f_5}^{\mathcal{R}_0}$	0.002423
$\chi_{f_6}^{\mathcal{R}_0}$	-0.018402
$\chi_{r_1}^{\mathcal{R}_0}$	-0.282710
$\chi_{r_2}^{\mathcal{R}_0}$	0.003304
$\chi_{r_m}^{\mathcal{R}_0}$	-0.029699
$\chi_{r_3}^{\mathcal{R}_0}$	-0.005802
$\chi_{\sigma_i}^{\mathcal{R}_0}$	-0.020580
$\chi_{\sigma_1}^{\mathcal{R}_0}$	-0.323786
$\chi_{\sigma_2}^{\mathcal{R}_0}$	-0.041608
$\chi_{\sigma_3}^{\mathcal{R}_0}$	-0.003241
$\chi_{\sigma_4}^{\mathcal{R}_0}$	-0.003241

TABLE 3. Sensitivity indices of the model parameters

Interpretation of sensitivity analysis results.

(1) Parameters with a Significant Impact on $\mathcal{R}_0$: The parameters τ_m (fraction of infectious men), m_1, and m_2 (fractions of latent and infected men capable of transmitting the virus) play a crucial role, with sensitivity indices of -0.489685, 0.196375, and 0.303625, respectively. This means that infected men, particularly in the latent and infectious classes, have a significant influence on the spread of the virus. Additionally, a decrease in the fraction of infectious men (τ_m) leads to a substantial reduction in $\mathcal{R}_0$.

(2) Impact of Vertical Transmission:
The parameters ρ_1, ρ_2, and ρ_3 (vertical transmission from classes I_2, P, and Q) show positive sensitivity indices, but of different magnitudes, with $\chi_{\rho_1}^{\mathcal{R}_0} = 0.105095$, $\chi_{\rho_2}^{\mathcal{R}_0} = 0.008929$, and $\chi_{\rho_3}^{\mathcal{R}_0} = 0.000880$. Vertical transmission from I_2 has the most significant influence on $\mathcal{R}_0$.

(3) Parameters of Progression in Infectious Classes for Women:
The sensitivity indices for the progression rates τ_1 (infectious women aged 9 to 15 years) and τ_2 (infectious women over 15 years) are negative, -0.045039 and -0.072376, respectively. This indicates that faster progression through these classes results in a lower $\mathcal{R}_0$. In other words, if individuals exit the infectious classes more quickly, it reduces the spread of the infection.

(4) Impact of Healing Rates:
The parameter r_1, which represents the healing fraction of women infected in class I_1, has a significant impact with $\chi_{r_1}^{\mathcal{R}_0} = -0.282780$.

(5) Contamination Between Genders:
The parameters λ_f and λ_m, representing the force of infection in women and men, respectively, do not have direct sensitivity indices indicated but are indirectly related to transmission parameters such as θ_m, θ_n, and θ_1 to θ_6. These show relatively high sensitivity indices, with $\chi_{\theta_3}^{\mathcal{R}_0} = 0.231111$ and $\chi_{\theta_6}^{\mathcal{R}_0} = 0.119414$, indicating the importance of individuals in the latent and pre-cancerous classes for viral transmission.

In Summary: The analysis reveals that the fractions of latent and infected men capable of transmitting the virus play an important role and highlights the need for special attention to these groups to better control the transmission of the virus. The results also show that the speed of healing and transition out of the infectious state, particularly among young girls, is crucial for reducing $\mathcal{R}_0$. Efforts to reduce $\mathcal{R}_0$ should focus on reducing transmission among infectious men, improving healing rates in women, and decreasing vertical transmission, particularly from the I_2 class. Measures such as vaccination, early screening, and treatment of latent infections could have a significant impact on reducing $\mathcal{R}_0$.

4. Numerical Simulations

In this section, we present numerical results that illustrate the theoretical conclusions established in the previous sections. To achieve this, we perform numerical simulations using MATLAB, applying the fourth-order Runge-Kutta method . This method was selected for its accuracy and stability in solving systems of nonlinear ordinary differential equations, such as those used to model the dynamics of human papillomavirus (HPV) and its associated complications.

The goal of these simulations is not only to validate the theoretical results but also to provide deeper insights into the system's dynamic behavior, particularly concerning parameter sensitivities and equilibrium points. With the parameters specified in (2), the basic reproduction number is calculated as $\mathcal{R}_0 = 1.0173$. We set $N_f = 7\,718\,366$ and $N_m = 5\,004\,502$, representing the total population of females aged over 9 years and males aged over 15 years in Burkina Faso in 2020. Therefore, $N = 12\,722\,868$ [21]. Initial conditions are: $S_1(0) = 1\,500\,000$; $S_2(0) = 6\,106\,666$; $S_m(0) = 4\,959\,502$; $E_1(0) = 10\,000$; $R_1(0) = 5\,000$; $I_1(0) = 7\,000$; $E_2(0) = 30\,000$; $I_2(0) = 20\,000$; $P(0) = 15\,000$; $Q(0) = 5\,000$; $T(0) = 3\,000$; $C(0) = 1\,000$; $R_C(0) = 700$; $E_m(0) = 20\,000$, $I_m(0) = 15\,000$; $R_m(0) = 10000$; $R_2(0) = 15\,000$

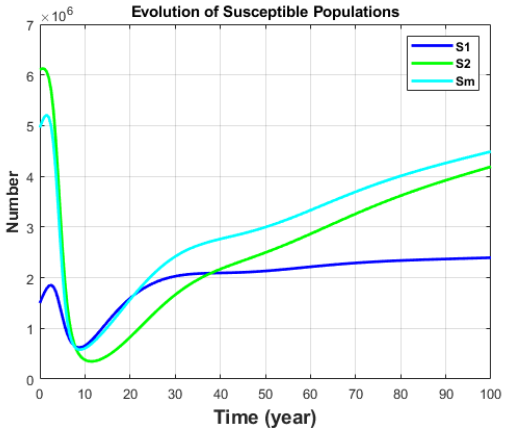

Figure 3. Evolution of the susceptible population.

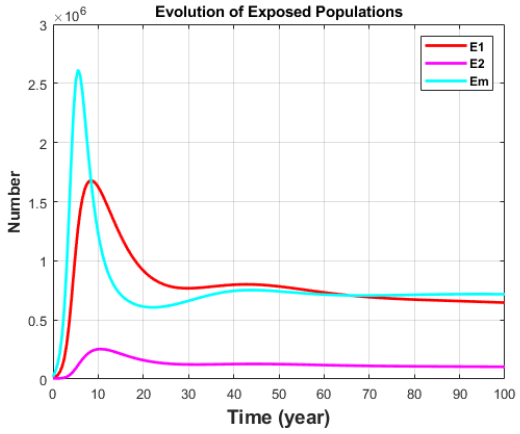

Figure 4. Evolution of the exposed population.

- Susceptible : As shown in Figure 3, the susceptible population starts high, mainly comprising women over 15. A gradual decline occurs over time as individuals move to latent or infected states, particularly noticeable among young girls.
- Exposed : Figure 4 illustrates the exposed population, with higher prevalence among men and a significant increase in girls aged 9 to 15 during the first decade. This prevalence decreases as they progress to infected or recovered states.

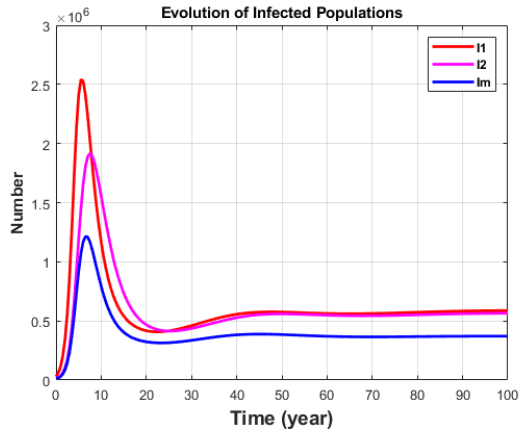

FIGURE 5. Evolution of the infected population.

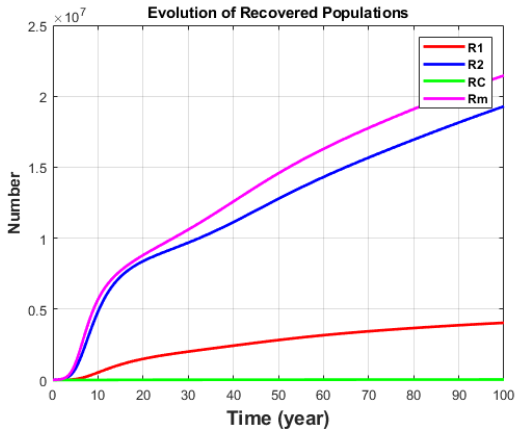

FIGURE 6. Evolution of the recovered population.

- Infected: Figure 5 shows an exponential growth in the infected population during the first decade, especially among young girls, indicating rapid transmission, with men playing a crucial role.
- Recovered: Figure 6 depicts a steady increase in the recovered population, contributing to an overall decrease in infections after the initial decade.

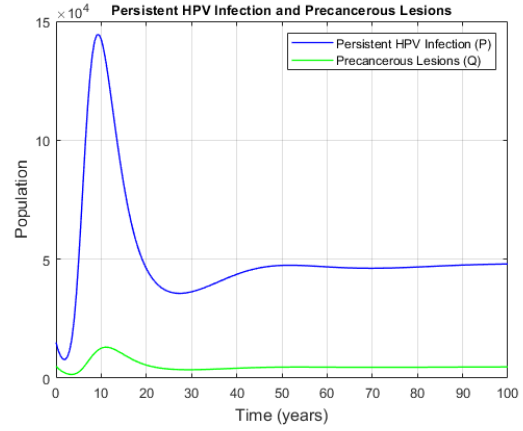

FIGURE 7. Trends in persistent infections and precancerous lesions.

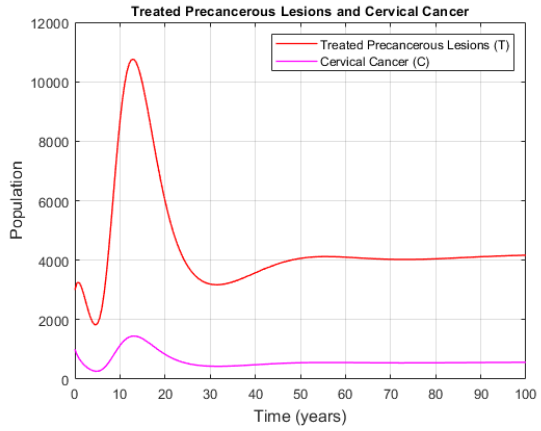

FIGURE 8. Precancerous lesions treatment and cervical cancer.

Persistent Infections and Precancerous Lesions

Figure 7 shows a significant increase in persistent infections and precancerous lesions during the first decade, followed by stabilization.

- Persistent Infections: This initial rise indicates unresolved cases, contributing to the progression towards precancerous lesions and potentially cervical cancer.

- Precancerous Lesions: Highlighted in Figure 7, this trend is closely tied to persistent infections, with untreated cases often advancing to this stage.
- Treatments: Figure 8 illustrates effective management of precancerous lesions, which helps keep cervical cancer incidence relatively low, emphasizing the importance of ongoing treatment and vaccination.
- Cervical Cancer: This figure demonstrates the initial increase in cervical cancer cases, mitigated by effective treatments, underscoring the need for sustained prevention and management strategies.

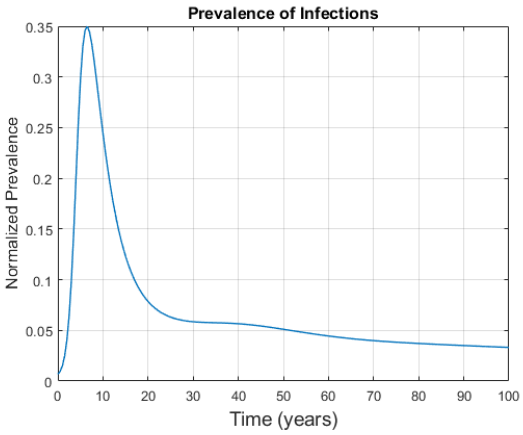

FIGURE 9. Prevalence of infections.

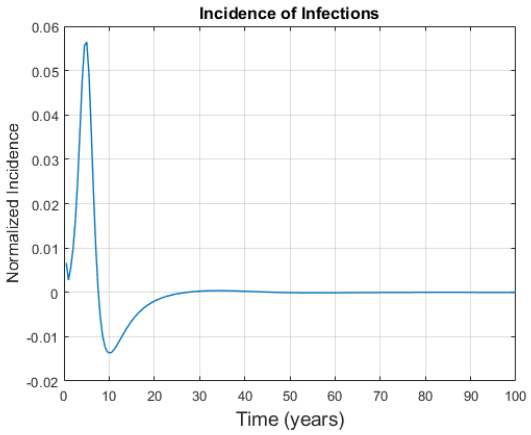

FIGURE 10. Incidence of infections.

Analysis of Prevalence and Incidence

- Prevalence: Figure 9 indicates that prevalence is initially driven by latent cases, peaking among young girls before stabilizing with the increase in recoveries.
- Incidence: Figure 10 mirrors prevalence trends, with early increases followed by declines due to recovery and effective treatment interventions.

Perspectives on Solutions

- Strengthening Vaccination Programs: Intensify efforts among young girls to reduce both latent and infected cases.
- Improving Treatments: Enhance access to healthcare and treatments, particularly in underserved areas.
- Education and Awareness: Implement campaigns to raise awareness of HPV risks, treatment options, and vaccination benefits.

- Prenatal Screening and Vertical Transmission: Promote screening and vaccination prior to reproductive age to mitigate vertical transmission.
- Enhancing Medical Infrastructure: Strengthen infrastructure and training to improve infection management and recovery rates.

Confirmation of Theoretical Results: The simulations confirm the accuracy of the theoretical equilibrium points, indicating that infection persists in the population due to $\mathcal{R}_0 > 1$.

Dynamic Behaviors of the System: The simulations reveal clear trends, such as the rapid increase in infections followed by stabilization post-treatment introduction, underscoring the importance of early intervention to curb the virus's spread.

Equilibrium Points: The simulations provide insights into how quickly the system converges to the equilibrium, crucial for understanding intervention effects. They show that a stable endemic equilibrium can be achieved with continuous strategies.

In Summary: Numerical simulations enrich the theoretical analysis by providing insights into infection dynamics and supporting the proposed interventions against HPV and its complications.

5. Conclusion

This study developed a mathematical model that uniquely incorporates **same-sex transmission** and **vertical transmission** to better capture the full dynamics of HPV transmission in Burkina Faso. By extending the standard SEIR framework, the model offers a more comprehensive understanding of how these additional pathways contribute to the spread of HPV and cervical cancer. The existence and global stability of disease-free and endemic equilibria, determined by the basic reproduction number $\mathcal{R}_0$, provide valuable insights into the conditions required for controlling HPV. Sensitivity analysis revealed that parameters such as the fraction of infectious men, the rate of vertical transmission, and progression through different infectious stages play pivotal roles in shaping HPV's dynamics. Importantly, our results emphasize the necessity of addressing both same-sex and vertical transmission pathways in public health strategies to effectively reduce HPV incidence and mortality from cervical cancer. Numerical simulations, using data from Burkina Faso, support these findings and highlight the practical relevance of the model in informing local prevention efforts. Future research should aim to refine the model by incorporating more detailed demographic and behavioral data, as well as exploring the impact of integrated intervention strategies, including vaccination and screening, across various subpopulations. Overall, this work offers a solid foundation for advancing HPV control efforts in regions with high disease burden.

Authors' Contributions

The authors contributed to this work in the following ways:

- JOEL LANKOANDE: Conceptualization, Methodology, Software, Formal Analysis, Data Curation, Investigation, Writing - Original Draft, Writing - Review & Editing, Suggestion, Validation.
- WENDDABO OLIVIER SAWADOGO: Conceptualization, Methodology, Investigation, Software, Data Curation, Review & Editing, Visualization, Suggestion, Supervision, Project Administration, Validation
- ADAMA KIEMTORE: Conceptualization, Methodology, Software, Software, Investigation, Review & Editing, Validation.

All authors discussed the results and contributed to the final manuscript.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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