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Research article

Mathematical modeling and optimal control of HPV-related cervical cancer through awareness campaigns and vaccination

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Abstract: In this research, we have derived a mathematical model to study the impact of health center-based awareness campaigns on the spread of human papillomavirus (HPV) infection to cervical cancer. In the model, the human population is divided into susceptible, vaccinated, permanently immune, infected with HPV, and recovered. Moreover, *awareness level* is assumed as a model compartment. The awareness level affects the rates of infection, vaccination, recovery, and disease progression. The basic reproduction number ($\mathcal{R}_0$) is calculated using the next-generation matrix method. The equilibrium points are determined, and their stability analysis is conducted. Disease-free equilibrium (DFE) is globally asymptotically stable for $\mathcal{R}_0 < 1$. Forward transcritical bifurcation occurs at $\mathcal{R}_0 = 1$. Furthermore, optimal control theory is employed using Pontryagin's maximum principle, with the objective of minimizing infection rates through optimal vaccination at the minimum cost of control. These results are validated by numerical simulations. The results show that effective HPV awareness campaigns can help control the spread of HPV infections, making it easier to manage cervical cancer. This study confirms the importance of awareness-induced behavioral dynamics in modeling spread of HPV for efficient disease management. Awareness campaigns with optimal vaccination can significantly lower HPV transmission and reduce the incidence of cervical cancer.

Keywords: basic reproduction number ($\mathcal{R}_0$); sensitivity analysis; equilibria and stability; forward bifurcation; optimal control approach; numerical simulations

1. Introduction

Human papillomavirus (HPV) infection is one of the most prevalent sexually transmitted diseases worldwide [1]. Moreover, it continues to be a significant causative factor in the development of cervical

cancer among women [2]. Despite the availability of effective vaccines and screening programs, the global burden of HPV-related diseases remains significant, especially in areas with low healthcare resources and awareness [3]. As a result, approximately 80% of sexually active women (and men) will contract at least one type of HPV at some point in their lifetime [4].

Persistent infection with high-risk HPV, particularly types 16 and 18, is responsible for nearly all cases of cervical cancer, accounting for approximately 70% of cases [5]. Although most HPV infections are eliminated by the immune system within one to two years, infections that persist over several years can progress to precancerous lesions and ultimately to cancer [6]. Globally, HPV infection results in substantial morbidity and mortality. This burden is particularly pronounced in low- and middle-income countries, where limited access to vaccines and screening programs exacerbates the problem [3, 4, 7]. Comprehensive public health strategies, including vaccination, screening, and education, are essential to reduce HPV prevalence and the impact of cervical cancer [8].

Medical facilities are actively addressing the rising burden of HPV-related cancers by implementing comprehensive, multifaceted awareness campaigns and prevention programs [9, 10]. These initiatives involve three key pillars, namely vaccination (primary prevention), screening (secondary prevention), and education [11, 12]. The Serum Institute of India in Kolkata organized the "Conquer HPV & Cancer Conclave 2025", which emphasized the significance of HPV vaccination and early detection techniques [12]. Similarly, experts emphasized that prompt vaccination can give up to 90% protection against HPV-induced malignancies at the free HPV vaccination clinic for girls and young women ages 9 to 26 at the Calcutta Medical Research Institute (CMRI) in Kolkata [13]. To significantly lower the incidence and death of cervical cancer, medical centers are playing a crucial role in HPV-cancer control through vaccination drives, awareness campaigns, and screening programs [14, 15].

A useful tool for studying the dynamics of HPV-related cervical cancer transmission is mathematical modeling [16]. Mathematical models provide an organized framework for assessing how interventions such as screening, vaccination, and awareness campaigns affect the prevalence of diseases [2, 17]. The relevance of ongoing public health initiatives has been highlighted by recent research that uses optimal control theory to develop solutions that minimize infection rates and lower the cost of disease management [17].

Compartmental models are effective mathematical tools for simulating the natural history of HPV infection, capturing the progression from initial infection to clearance, persistence, and eventual development of cancer [18, 19]. These models divide the population into distinct compartments (e.g., susceptible, infected, persistent, precancerous, and cancerous) and use differential equations to model the transitions between the compartments based on factors such as age, sexual activity, and vaccination status [20]. In the context of high-risk HPV infection, these models make it easier to analyze thresholds for disease control and the long-term impacts of therapies [2, 21].

Awareness programs through health centers are essential for influencing vaccination and individual behavior and encouraging early screening and treatment [10, 22, 23]. Research indicates that health education programs greatly increase adolescents' and young adults' acceptability and awareness of HPV vaccination [24]. This method highlights the potential of education-based interventions in lowering HPV prevalence and the burden of cervical cancer and offers a more practical framework for evaluating public health policies than other methods [2, 17, 25]. Recent work has highlighted the importance of awareness campaigns as dynamic control variables, showing that education-based interventions can enhance and complement biomedical measures [17, 26]. These findings underscore

the role of informed public engagement in reducing infection prevalence and mitigating the burden of cervical cancer.

Sensitivity analysis has been widely used to determine the most important parameters influencing the basic reproduction number ($\mathcal{R}_0$), which acts as a threshold for determining whether infection dies out or continues [23, 27]. The factors that are linked to HPV transmission can be determined using sensitivity analysis [23, 28]. In this study, we calculate the basic reproduction number ($\mathcal{R}_0$) and use it to offer the sensitivity analysis.

Mathematical modeling of HPV transmission and cervical cancer progression has increasingly drawn upon optimal control theory to design and evaluate intervention strategies [29]. By applying Pontryagin's maximum principle, researchers are able to construct optimal methods that balance epidemiological outcomes with economic considerations, incorporating control variables such as vaccination, screening, and awareness campaigns [17]. Optimal control approaches provide robust policy guidance, particularly in resource-constrained settings where the careful optimization of interventions is essential for maximizing impact [27, 30].

In this research, we formulate a mathematical model to examine the relationship between HPV transmission, vaccination, recovery, and the development of cervical cancer. We assess awareness impact on HPV transmission, recovery, and the development of cervical cancer by including it as a dynamic variable in a mathematical model. We determine the basic reproduction number of the system, the equilibrium, and its stability. Additionally, an optimal control problem is formulated using Pontryagin's maximum principle to minimize HPV and related cancer cases with minimum cost and shorter time. Using the model, we show that long-term illness prevalence can be considerably reduced by consistent immunization efforts in conjunction with awareness-driven behavioral adjustments. This study also emphasizes how crucial it is to incorporate optimal control theory into HPV modeling frameworks in order to direct public health decision-making and accomplish long-term decreases in the incidence of cervical cancer.

2. Model formulation

To capture the transmission dynamics of HPV infection and cervical cancer under the influence of vaccination and awareness campaigns, we stratify the total human population into six mutually exclusive compartments: (i) susceptible individuals $S(t)$, (ii) vaccinated individuals $V(t)$, (iii) permanently immunized individuals $P(t)$, (iv) HPV-infected individuals $I(t)$, (v) individuals with HPV-induced cervical cancer $C(t)$, and (vi) recovered individuals $R(t)$ at any time t. In addition, we introduce a dimensionless variable $A(t)$ to represent the awareness level in the population.

The following assumptions are made:

A1: Recruitment into the population occurs at a constant rate π, with all new entrants joining the susceptible class. Susceptible individuals may receive a first dose of vaccination at a rate $\sigma_a(A)$, which is enhanced by awareness campaigns [31], defined by

$$\sigma_a(A) = \sigma(1 + \varepsilon_\sigma A). \tag{2.1}$$

In Eq (2.1), ε_σ is used as the rate of awareness impact on vaccination. Vaccinated individuals may lose their temporary immunity at a rate δ, returning to the susceptible class, or progress to

permanent immunity at a rate θ. Permanently immunized individuals $P(t)$ are assumed to have lifelong protection against HPV infection.

A2: Transmission of HPV occurs through effective contact between susceptible and infected individuals, governed by the effective transmission rate $\beta_a(A)$ as a decreasing function of $A(t)$:

$$\beta_a(A) = \frac{\beta}{1 + \varepsilon_\beta A}. \tag{2.2}$$

Awareness campaigns reduce risky behaviors and thereby lower transmission. Here, β represents the maximum infection rate in the absence of awareness activities, and ε_β denotes the impact of awareness on transmission.

A3: Infected individuals may recover at rate $\gamma_a(A)$, with awareness increasing the likelihood of recovery through improved health-seeking behavior, thus defined as

$$\gamma_a(A) = \gamma(1 + \varepsilon_\gamma A), \tag{2.3}$$

where ε_γ is the rate of awareness impact on recovery process and γ is the recovery in absence of awareness. Infection may progress to cervical cancer at rate $\vartheta_a(A)$, which is mitigated by awareness efforts:

$$\vartheta_a(A) = \frac{\vartheta}{1 + \varepsilon_\vartheta A}, \tag{2.4}$$

where ϑ is the rate of progress to cervical cancer without awareness, and ε_ϑ is the awareness impact on the progression rate of cancer.

A4: Individuals with cervical cancer experience both natural mortality and disease-induced death at a rate ϕ. Recovered individuals may lose their immunity at a rate $\omega_a(A)$, returning to the susceptible class, whereas awareness reduces the rate of immunity loss:

$$\omega_a(A) = \frac{\omega}{1 + \varepsilon_\omega A}, \tag{2.5}$$

where ω is the maximum loss of immunity, and ε_ω measures the awareness impact on immunity retention.

A5: Natural death occurs uniformly across all compartments at rate μ. The awareness level $A(t)$ increases proportionally to the number of infected and cancer cases, scaled by the campaign effort parameter κ. It decays naturally at a rate η, reflecting the fading effect of campaigns over time.

The resulting system of nonlinear differential equations governing the dynamics of the model is given by:

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \mu S + \delta V + \omega_a R - \sigma_a S - \beta_a S I, \\
\frac{dV}{dt} &= \sigma_a S - (\mu + \delta + \theta)V, \\
\frac{dP}{dt} &= \theta V - \mu P, \\
\frac{dI}{dt} &= \beta_a I S - (\mu + \gamma_a + \vartheta_a)I,
\end{aligned} \tag{2.6}$$

$$\frac{dC}{dt} = \vartheta_a I - (\mu + \phi)C,$$
$$\frac{dR}{dt} = \gamma_a I - (\omega_a + \mu)R,$$
$$\frac{dA}{dt} = \kappa(I + C) - \eta A,$$

with initial values of the populations as follows:

$$S(0) \geq 0, V(0) \geq 0, P(0) \geq 0, I(0) \geq 0, C(0) \geq 0, R(0) \geq 0, A(0) \geq 0. \tag{2.7}$$

Figure 1 illustrates the transitions among the compartments of the model. It is assumed that all parameters used in the model are positive. In Table 1, we summarize the description of the parameters used in the model.

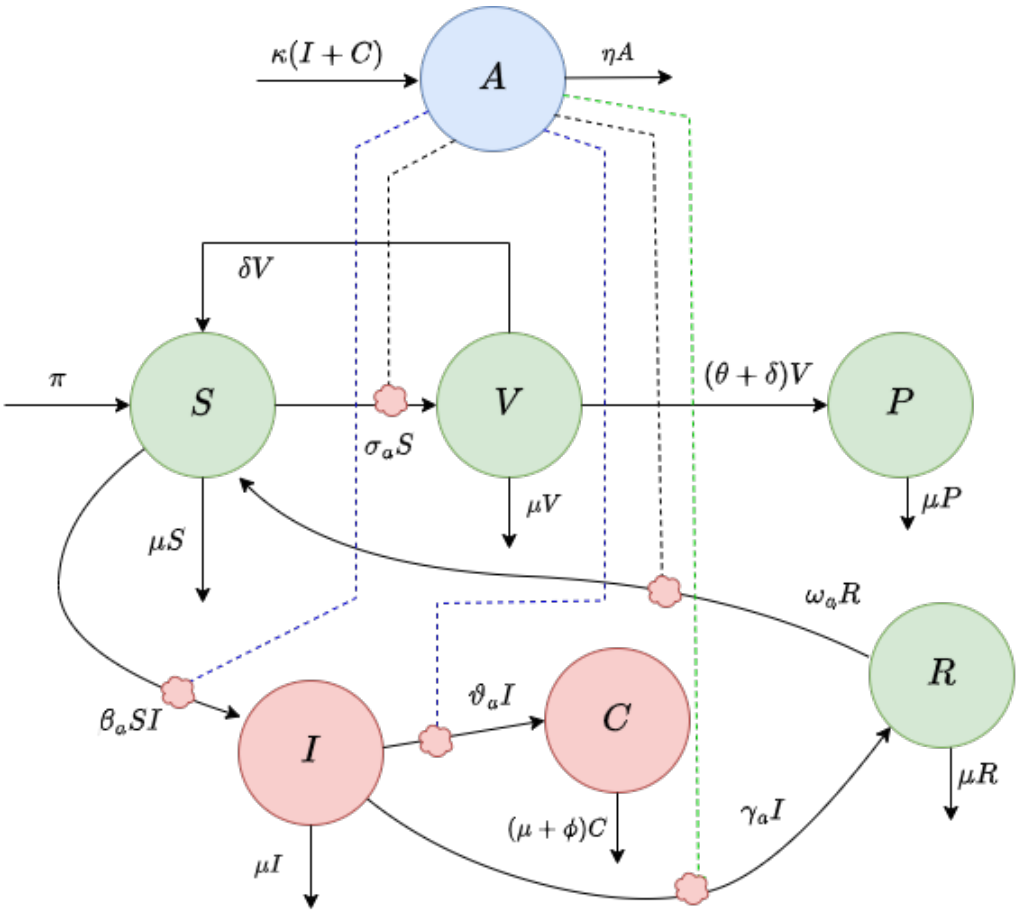

Figure 1. Schematic diagram to illustrate the transitions among compartments of the model in Eq (2.6).

Table 1. Model parameters with descriptions, typical values, and units [17, 32, 33].

Parameter	Short description	Value	Unit
π	Recruitment rate of susceptible human	45	human day^{-1}
β	Infection transmission rate	0.0005	human^{-1} day^{-1}
σ	First-dose vaccination rate	0.001	day^{-1}
δ	Waning rate of first-dose immunity	0.0003	day^{-1}
θ	Rate of transition to permanent immunity	0.0008	day^{-1}
ω	Rate of immunity loss from recovery	0.0002	day^{-1}
μ	Natural death rate	0.00004	day^{-1}
ϑ	Progression rate to cervical cancer	0.0001	day^{-1}
γ	Recovery rate from HPV infection	0.0015	day^{-1}
ϕ	Disease-induced death rate from cancer	0.0005	day^{-1}
κ	Rate of increased awareness level due to campaigns	0.01	human^{-1}day^{-1}
η	Awareness campaign decay rate	0.016	day^{-1}
ε_β	Awareness impact on transmission	0.5	dimensionless
ε_σ	Awareness impact on vaccination	0.3	dimensionless
ε_γ	Awareness impact on recovery	0.4	dimensionless
ε_ϑ	Awareness impact on progression	0.6	dimensionless
ε_ω	Awareness impact on immunity retention	0.2	dimensionless

3. Basic properties of the model

In this section, we have studied the non-negativity and boundedness of the solutions of the proposed model. The following theorems are derived for the plausibility of the model.

Theorem 1 (Non-negativity of solutions)**.** *Each solution of the system in Eq* (2.6) *with non-negative initial conditions in Eq* (2.7) *is non-negative for all* $t > 0$*.*

Proof. We proof the theorem by contradiction. Suppose there exists a first time $t^* > 0$ such that one of the state variables, say $X(t)$, becomes negative. Then we must have

(i) $X(t) \geq 0$ for all $0 \leq t < t^*$,
(ii) $X(t^*) = 0$,
(iii) and immediately after t^*, $X(t) < 0$.

At t^*, evaluating the derivative of each component at the boundary, we get

$$\left.\frac{dS}{dt}\right|_{S=0} = \pi + \delta V + \omega_a(A)R \geq 0, \qquad \left.\frac{dR}{dt}\right|_{R=0} = \gamma_a(A)I \geq 0,$$

$$\left.\frac{dV}{dt}\right|_{V=0} = \sigma_a(A)S \geq 0, \qquad \left.\frac{dA}{dt}\right|_{A=0} = \kappa(I + C) \geq 0,$$

$$\left.\frac{dP}{dt}\right|_{P=0} = \theta V \geq 0, \qquad \left.\frac{dI}{dt}\right|_{I=0} = 0,$$

$$\left.\frac{dC}{dt}\right|_{C=0} = \vartheta_a(A)I \geq 0.$$

Therefore, at the boundary $X(t^*) = 0$, the derivative is non-negative in every case. This implies that the trajectory cannot cross into the negative region, which contradicts the assumption that $X(t)$ becomes negative after t^*.

Thus, no component of the solution can ever become negative, and the system in Eq (2.6) is positively invariant in the non-negative orthant.

Theorem 2 (Boundedness of solutions)**.** *All solutions of system in Eq* (2.6) *are uniformly bounded for all* $t \geq 0$.

Proof. Let us define $N(t)$ as follows:

$$N(t) = S(t) + V(t) + P(t) + I(t) + C(t) + R(t).$$

Differentiating, we obtain

$$\begin{aligned}\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dV}{dt} + \frac{dP}{dt} + \frac{dI}{dt} + \frac{dC}{dt} + \frac{dR}{dt}\\ &= \pi - \mu S + \delta V + \omega_a(A)R - (\sigma_a(A) + \beta_a(A)I)S\\ &\quad + \sigma_a(A)S - (\mu + \delta + \theta)V + \theta V - \mu P\\ &\quad + \beta_a(A)IS - (\mu + \gamma_a(A) + \vartheta_a(A))I\\ &\quad + \vartheta_a(A)I - (\mu + \phi)C + \gamma_a(A)I - (\omega_a(A) + \mu)R.\end{aligned}$$

After simplifying the calculations, we finally obtain

$$\frac{dN}{dt} = \pi - \mu(S + V + P + I + C + R) - \phi C.$$

This gives

$$\frac{dN}{dt} \leq \pi - \mu N(t). \tag{3.1}$$

By the comparison principle, we have

$$N(t) \leq \max\left\{N(0), \frac{\pi}{\mu}\right\}. \tag{3.2}$$

Therefore, $N(t)$ is uniformly bounded for all $t \geq 0$. Finally, because $A(t)$ satisfies

$$\frac{dA}{dt} = \kappa(I + C) - \eta A \leq \kappa N(t) - \eta A,$$

and using Eq (3.2), $N(t)$ is bounded. Thus, standard comparison arguments show that $A(t)$ is also bounded. Hence, all components S, V, P, I, C, R, A remain bounded for all $t \geq 0$.

4. Dynamics of the system

In this section, we find the equilibria and present their stability by finding Jacobian matrices. Also, the basic reproduction number is derived at the disease-free equilibrium (DFE).

4.1. Equilibrium points

Equating the right sides of the model in Eq (2.6) to zero , we have the following equilibrium points:
(i) The DFE is $E_0 = (\bar{S},\ \bar{V},\ \bar{P},\ 0,\ 0,\ 0,\ 0)$, where

$$\begin{aligned} \bar{S} &= \frac{\pi(\mu+\delta+\theta)}{(\sigma+\mu)(\mu+\delta+\theta)-\delta\sigma}, \\ \bar{V} &= \frac{\sigma\pi}{(\sigma+\mu)(\mu+\delta+\theta)-\delta\sigma}, \\ \bar{P} &= \frac{\sigma\theta\pi}{\mu[(\sigma+\mu)(\mu+\delta+\theta)-\delta\sigma]}. \end{aligned} \tag{4.1}$$

(ii) The endemic equilibrium $E^*\,(S^*, V^*, P^*, I^*, C^*, R^*, A^*)$, where

$$\begin{aligned} S^* &= \frac{\mu+\gamma_a(A^*)+\vartheta_a(A^*)}{\beta_a(A^*)}, \\ V^* &= \frac{\sigma_a(A^*)}{\mu+\delta+\theta}\,S^*, \\ P^* &= \frac{\theta}{\mu}\,V^*, \\ I^* &= \frac{\pi+\{-\mu-\sigma_a(A^*)+B_1\}\,B_2}{(\mu+\gamma_a(A^*)+\vartheta_a(A^*))-B_3}, \\ C^* &= \frac{\vartheta_a(A^*)}{\mu+\phi}\,I^*, \\ R^* &= \frac{\gamma_a(A^*)}{\omega_a(A^*)+\mu}\,I^*, \end{aligned}$$

where

$$B_1 = \frac{\delta\,\sigma_a(A^*)}{\mu+\delta+\theta}, B_2 = \frac{\mu+\gamma_a(A^*)+\vartheta_a(A^*)}{\beta_a(A^*)}, B_3 = \frac{\omega_a(A^*)\,\gamma_a(A^*)}{\omega_a(A^*)+\mu},$$

and the equation that determines A^* is given below:

$$F(A^*) = A^* - \frac{\kappa}{\eta}\,I^*(A^*)\times\left(1+\frac{\vartheta_a(A^*)}{\mu+\phi}\right) = 0. \tag{4.2}$$

This equation can be further simplified to the following quintic polynomial equation:

$$c_5A^5 + c_4A^4 + c_3A^3 + c_2A^2 + c_1A + c_0 = 0, \tag{4.3}$$

where

$$\begin{aligned} c_0 &= -\pi(\omega+\mu)(\mu+\phi)\eta \ - \ \kappa\mu(\mu+\delta+\theta)(\mu+\gamma+\vartheta), \\ c_1 &= \varepsilon_\sigma\sigma(\mu+\delta+\theta)(\mu+\gamma+\vartheta) + \varepsilon_\beta\beta(\mu+\delta+\theta)(\omega+\mu) + \varepsilon_\gamma\gamma(\mu+\delta+\theta)(\omega+\mu) \\ &\quad + \varepsilon_\vartheta\vartheta(\mu+\delta+\theta)(\omega+\mu) + \varepsilon_\omega\omega(\mu+\delta+\theta)(\mu+\gamma+\vartheta), \\ c_2 &= \frac{\kappa}{\eta}(\mu+\delta+\theta)(\mu+\phi)\Big[\varepsilon_\sigma\varepsilon_\beta\sigma\beta + \varepsilon_\sigma\varepsilon_\gamma\sigma\gamma + \varepsilon_\sigma\varepsilon_\vartheta\sigma\vartheta + \varepsilon_\sigma\varepsilon_\omega\sigma\omega \end{aligned}$$

$$+ \varepsilon_\beta\varepsilon_\gamma\beta\gamma + \varepsilon_\beta\varepsilon_\vartheta\beta\vartheta + \varepsilon_\beta\varepsilon_\omega\beta\omega + \varepsilon_\gamma\varepsilon_\vartheta\gamma\vartheta + \varepsilon_\gamma\varepsilon_\omega\gamma\omega + \varepsilon_\vartheta\varepsilon_\omega\vartheta\omega\Big],$$

$$c_3 = \frac{\kappa}{\eta}(\mu+\delta+\theta)(\mu+\phi)\Big[\varepsilon_\sigma\varepsilon_\beta\varepsilon_\gamma\sigma\beta\gamma + \varepsilon_\sigma\varepsilon_\beta\varepsilon_\vartheta\sigma\beta\vartheta + \varepsilon_\sigma\varepsilon_\beta\varepsilon_\omega\sigma\beta\omega$$
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$$c_4 = \frac{\kappa}{\eta}(\mu+\delta+\theta)(\mu+\phi)\Big[\varepsilon_\sigma\varepsilon_\beta\varepsilon_\gamma\varepsilon_\vartheta\sigma\beta\gamma\vartheta + \varepsilon_\sigma\varepsilon_\beta\varepsilon_\gamma\varepsilon_\omega\sigma\beta\gamma\omega$$
$$+ \varepsilon_\sigma\varepsilon_\beta\varepsilon_\vartheta\varepsilon_\omega\sigma\beta\vartheta\omega + \varepsilon_\sigma\varepsilon_\gamma\varepsilon_\vartheta\varepsilon_\omega\sigma\gamma\vartheta\omega + \varepsilon_\beta\varepsilon_\gamma\varepsilon_\vartheta\varepsilon_\omega\beta\gamma\vartheta\omega\Big],$$

$$c_5 = \frac{\kappa}{\eta}(\mu+\delta+\theta)(\mu+\phi)\,\sigma\beta\gamma\vartheta\omega\,\varepsilon_\sigma\varepsilon_\beta\varepsilon_\gamma\varepsilon_\vartheta\varepsilon_\omega.$$

Here, Eq (4.3) is a quintic equation with $c_0 < 0$ and all other coefficients are positive. Thus, using Descartes' rule of signs, Eq (4.3) has one positive root. Also, for the feasibility of E^*, I^* should be positive.

4.2. The basic reproduction number

The basic reproduction number is obtained via the next-generation method. We consider the infected subsystem with state vector x. We write the dynamics in the form

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x),$$

where $\mathcal{F}$ collects new infections, and $\mathcal{V}$ collects transitions out of infected states (recovery, progression, natural death).

For the equation of the infected class I,

$$\frac{dI}{dt} = \underbrace{\beta_a S\, I}_{\mathcal{F}(I)} - \underbrace{(\mu+\gamma_a+\vartheta_a)\, I}_{\mathcal{V}(I)}.$$

Computing the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at E_0, we have

$$F = \left.\frac{\partial \mathcal{F}}{\partial x}\right|_{E_0} = \beta_a \bar{S}, \qquad V = \left.\frac{\partial \mathcal{V}}{\partial x}\right|_{E_0} = \mu+\gamma_a+\vartheta_a.$$

The next generation matrix is $K = FV^{-1}$. Because this is a scalar system,

$$\mathcal{R}_0 = \rho(K) = \frac{\beta_a \bar{S}}{\mu+\gamma_a+\vartheta_a}, \tag{4.4}$$

where $\bar{S}$ is given in Eq (4.1). At the DFE, $A = 0$, and $S = \bar{S}$. Substituting these values, we have

$$\mathcal{R}_0 = \frac{\beta\pi}{(\mu+\gamma+\vartheta)\left(\sigma+\mu-\dfrac{\delta\sigma}{\mu+\delta+\theta}\right)}.$$

Remark 1. *Detailed numerical simulations show that the endemic equilibrium E^* exists when the basic reproduction number, $\mathcal{R}_0$, is greater than unity, i.e., $\mathcal{R}_0 > 1$ (see Figure 2).*

Remark 2. *The basic reproduction of the proposed system $\mathcal{R}_0$ is a decreasing function of vaccination rate σ. Thus, the disease can be managed using vaccination. Also, if we lower the infection rate β_a by raising awareness among the population, as β_a depends on awareness level $A(t)$, the size of the epidemic can be reduced.*

Remark 3. *Explicit steady-state values are difficult to obtain. In particular, I^* depends on A^*, a root of a fifth-degree polynomial. Hence, unlike SIR-type models [34, 35], the existence condition of endemic equilibrium E^* or the positivity of I^* cannot be expressed in terms of $\mathcal{R}_0$. Nevertheless, numerical simulations confirm that E^* exists whenever $\mathcal{R}_0 > 1$ (see Figure 2).*

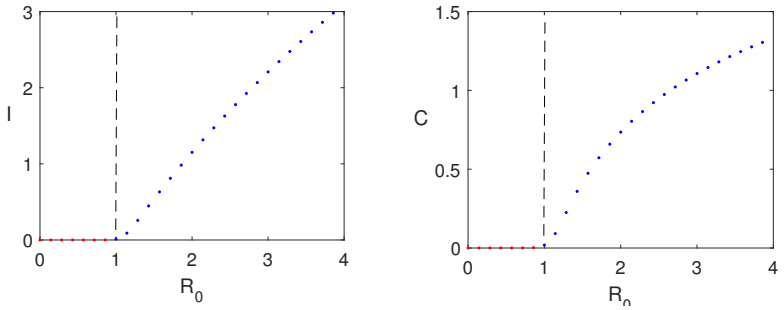

Figure 2. Forward bifurcation at $\mathcal{R}_0 = 1$: Steady-state values of the infected populations are plotted with respect to $\mathcal{R}_0$. We have varied the infection rate β with other parameters as in Table 1.

4.3. Stability of equilibria

This section contains the analysis of the stability of equilibria. We determine the Jacobian matrix and determine the sign of the real parts of the eigenvalues of the Jacobian matrix to determine the stability nature.

4.3.1. Stability of the DFE

Local stability. Let us introduce $\sigma_0 = \sigma_a(0)$, $\beta_0 = \beta_a(0)$, $\gamma_0 = \gamma_a(0)$, $\vartheta_0 = \vartheta_a(0)$, $\omega_0 = \omega_a(0)$, and similarly $\sigma_0' = \sigma_a'(0)$, $\beta_0' = \beta_a'(0)$, $\gamma_0' = \gamma_a'(0)$, $\vartheta_0' = \vartheta_a'(0)$, $\omega_0' = \omega_a'(0)$. Evaluating at $E_0 = (\bar{S}, \bar{V}, \bar{P}, 0, 0, 0, 0)$, Then the Jacobian matrix at E_0 is:

$$J(E_0) = \begin{bmatrix} -\mu-\sigma_0 & \delta & 0 & -\bar{S}\,\beta_0 & 0 & \omega_0 & -\bar{S}\,\sigma_0' \\ \sigma_0 & -(\mu+\delta+\theta) & 0 & 0 & 0 & 0 & \bar{S}\,\sigma_0' \\ 0 & \theta & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \bar{S}\,\beta_0-\mu-\gamma_0-\vartheta_0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \vartheta_0 & -(\mu+\phi) & 0 & 0 \\ 0 & 0 & 0 & \gamma_0 & 0 & -\mu-\omega_0 & 0 \\ 0 & 0 & 0 & \kappa & \kappa & 0 & -\eta \end{bmatrix}.$$

The seven eigenvalues of the Jacobian matrix are obtained as $-\mu, -(\mu+\delta+\theta), -\mu, \bar{S}\,\beta_0 - \mu - \gamma_0 - \vartheta_0, -(\mu+\phi), -(\mu+\omega_0)$, and $-\eta$. Using the definition of the basic reproduction number, we can write $\bar{S}\,\beta_0 - \mu - \gamma_0 - \vartheta_0, -(\mu+\phi) < 0$ if and only if $\mathcal{R}_0 < 1$. Thus, we have the following result.

Theorem 3. *If $\mathcal{R}_0 < 1$, the DFE E_0 is locally asymptotically stable. If $\mathcal{R}_0 > 1$, E_0 is unstable. When $\mathcal{R}_0 = 1$, transcritical bifurcation occurs.*

Proof of the last part of Theorem 3 is given in **Appendix**.

Global stability. We now establish the global stability of the DFE. We established the following theorem.

Theorem 4. *The DFE of System* (2.6) *is globally asymptotically stable when $\mathcal{R}_0 < 1$.*

Proof. At the DFE, there are no infections or cancer cases. This means that at the DFE, we have

$$\bar{I} = 0, \quad \bar{C} = 0, \quad \bar{R} = 0, \quad \bar{A} = 0,$$

and the equilibrium values of the remaining compartments are

$$\bar{S} = \frac{\pi}{\mu+\sigma}, \quad \bar{V} = \frac{\sigma\bar{S}}{\mu+\delta+\theta}, \quad \bar{P} = \frac{\theta\bar{V}}{\mu}.$$

Using the next-generation matrix approach, the basic reproduction number is computed as

$$\mathcal{R}_0 = \frac{\beta\bar{S}}{\mu+\gamma+\vartheta}.$$

To analyze global stability, we take the form of the Lyapunov function, $\mathcal{L}$, as

$$\mathcal{L}(I,C) = I + \frac{\vartheta}{\mu+\phi}C. \tag{4.5}$$

Clearly, $\mathcal{L}(I,C) \geq 0$, and $\mathcal{L}(I,C) = 0$ if and only if $I = 0$ and $C = 0$. Differentiating $\mathcal{L}$ along the trajectories of the system, we get

$$\frac{d\mathcal{L}}{dt} = (\beta S - (\mu+\gamma+\vartheta))\,I - \vartheta C.$$

At the DFE, $S = \bar{S}$. Now, if $\mathcal{R}_0 < 1$, then $\beta\bar{S} < \mu+\gamma+\vartheta$, which implies

$$\frac{d\mathcal{L}}{dt} \leq 0,$$

and equality holds when $I = 0$ and $C = 0$. Therefore, by LaSalle's invariance principle [36], the DFE is globally asymptotically stable for $\mathcal{R}_0 < 1$.

4.3.2. Stability of E^*

The Jacobian matrix at E^* is determined:

$$J(E) = \begin{bmatrix} J_{11} & \delta & 0 & -S\,\beta_a(A) & 0 & \omega_a(A) & J_{16} \\ \sigma_a(A) & -(\mu+\delta+\theta) & 0 & 0 & 0 & 0 & S\,\sigma_a'(A) \\ 0 & \theta & -\mu & 0 & 0 & 0 & 0 \\ I\,\beta_a(A) & 0 & 0 & J_{44} & 0 & 0 & J_{46} \\ 0 & 0 & 0 & \vartheta_a(A) & -(\mu+\phi) & 0 & I\,\vartheta_a'(A) \\ 0 & 0 & 0 & \gamma_a(A) & 0 & -\mu-\omega_a(A) & J_{67} \\ 0 & 0 & 0 & \kappa & \kappa & 0 & -\eta \end{bmatrix},$$

where $J_{11} = -\mu - \sigma_a(A) - \beta_a(A)I$, $J_{44} = S\,\beta_a(A) - \mu - \gamma_a(A) - \vartheta_a(A)$, $J_{16} = R\,\omega_a'(A) - S\,(\sigma_a'(A) + I\,\beta_a'(A))$, $J_{46} = IS\,\beta_a'(A) - I(\gamma_a'(A) + \vartheta_a'(A))$, and $J_{67} = I\,\gamma_a'(A) - R\,\omega_a'(A)$.

Here, the primes denote derivatives with respect to A. Also, we have

$$\sigma_a'(A) = \frac{d}{dA}\sigma_a(A),$$

and similarly for $\beta_a, \gamma_a, \vartheta_a, \omega_a$, we obtain

$$\beta_a'(A) = -\frac{\beta\,\varepsilon_\beta}{(1+\varepsilon_\beta A)^2}, \quad \sigma_a'(A) = \sigma\,\varepsilon_\sigma, \quad \gamma_a'(A) = \gamma\,\varepsilon_\gamma,$$
$$\vartheta_a'(A) = -\frac{\vartheta\,\varepsilon_\vartheta}{(1+\varepsilon_\vartheta A)^2}, \quad \omega_a'(A) = -\frac{\omega\,\varepsilon_\omega}{(1+\varepsilon_\omega A)^2}.$$

The characteristic equation is given as

$$p(\lambda) = (\lambda+\mu)(\lambda+\mu+\phi)(\lambda+\eta)\,q(\lambda) = 0, \tag{4.6}$$

where

$$q(\lambda) = \det(M(\lambda)), \tag{4.7}$$

with

$$M(\lambda) = \begin{bmatrix} J_{11}-\lambda & \delta & -\beta_a S & \omega_a \\ \sigma_a & J_{22}-\lambda & 0 & 0 \\ \beta_a I & 0 & J_{44}-\lambda & J_{46} \\ 0 & 0 & \gamma_a & J_{66}-\lambda \end{bmatrix}.$$

We determine $q(\lambda)$ as

$$q(\lambda) = \lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0,$$

where the coefficients are

$$c_3 = -(J_{11} + J_{22} + J_{44} + J_{66}),$$
$$c_2 = (J_{11}+J_{22})(J_{44}+J_{66}) + (J_{11}J_{22} - \sigma_a\delta) + (J_{44}J_{66} - J_{46}\gamma_a) - J_{14}J_{41},$$
$$c_1 = -\big[(J_{11}J_{22} - \sigma\delta)(J_{44}J_{66}) + (J_{44}J_{66} - J_{46}\gamma_a)(J_{11}+J_{22})\big] + J_{14}J_{41}(J_{22}+J_{66}),$$
$$c_0 = (J_{11}J_{22} - \sigma\delta)(J_{44}J_{66} - J_{46}\gamma_a) - J_{14}J_{41}J_{22}J_{66}.$$

Thus, the three eigenvalues of J are

$$\lambda_1 = -\mu < 0, \quad \lambda_2 = -(\mu + \phi) < 0, \quad \lambda_3 = -\eta < 0,$$

and the remaining eigenvalues are the four roots of the quartic polynomial $q(\lambda)$. Hence, the Routh–Hurwitz stability conditions for E^* are

$$\begin{cases} c_3 > 0, \quad c_2 > 0, \quad c_1 > 0, \quad c_0 > 0, \\ c_3c_2 - c_1 > 0, \\ c_3c_2c_1 - c_1^2 + c_3^2c_0 > 0. \end{cases} \tag{4.8}$$

5. Optimal control analysis

Here, we propose an optimal control problem to optimally manage the occurrence of HPV-related cancer. We consider three control variables, $u_i(t), i = 1, 2, 3$ with $0 \le u_i(t) \le u_{\max}^i < 1$ aiming to reduce the incidence of HPV infection using optimal vaccination effort (u_1), transmission reduction (u_2) and recovery improvement (u_3). The objective function is formulated as

$$\min_{u \in \mathcal{U}} J(u) = \int_0^T \left(q_1 I(t) + q_2 C(t) + \tfrac{\alpha_1}{2} u_1^2(t) + \tfrac{\alpha_2}{2} u_2^2(t) + \tfrac{\alpha}{2} u_3^2(t) \right) dt, \tag{5.1}$$

where $u = (u_1, u_2, u_3)$. Also, $q_1, q_2 > 0$ are weighting parameters, and α is the penalty multiplier. Further, $\mathcal{U}$ is defined as

$$\mathcal{U} = \{(u_1, u_2, u_3) : \ 0 \le u_i(t) \le u_{\max}^i < 1, \ t \in [0, T]\}, i = 1, 2, 3.$$

The state system is given by

$$\begin{aligned} \frac{dS}{dt} &= \pi + \delta V + \omega_a R - (u_1(t)\sigma_a + \mu + u_2(t)\beta_a I)S, \\ \frac{dV}{dt} &= u_1(t)\sigma_a S - (\mu + \delta + \theta)V, \\ \frac{dP}{dt} &= \theta V - \mu P, \\ \frac{dI}{dt} &= u_2(t)\beta_a IS - (\mu + u_3(t)\gamma_a + \vartheta_a)I, \\ \frac{dC}{dt} &= \vartheta_a I - (\mu + \phi)C, \\ \frac{dR}{dt} &= u_3(t)\gamma_a I - (\omega_a + \mu)R, \\ \frac{dA}{dt} &= \kappa(I + C) - \eta A, \end{aligned} \tag{5.2}$$

with the initial conditions given in Eq (2.7) and the functions $\beta_a(A), \sigma_a(A), \gamma_a(A), \vartheta_a(A)$, and $\omega_a(A)$ are defined in Eqs (2.1)–(2.5).

5.1. Characterize the optimal control

We formulate the Hamiltonian as

$$\mathcal{H} = q_1 I(t) + q_2 C(t) + \tfrac{\alpha_1}{2} u_1^2(t) + \tfrac{\alpha_2}{2} u_1^2(t) + \tfrac{\alpha_3}{2} u_3^2(t) + \xi_S \dot{S} + \xi_V \dot{V} + \xi_P \dot{P} + \xi_I \dot{I} + \xi_C \dot{C} + \xi_R \dot{R} + \xi_A \dot{A},$$

where $\xi_S, \xi_V, \xi_P, \xi_I, \xi_C, \xi_R, \xi_A$ are the adjoint variables. The Pontryagin maximum principle gives the following relations:

$$\dot{\xi}_X = -\frac{\partial \mathcal{H}}{\partial X}, \qquad \xi_X(T) = 0, \quad X \in \{S, V, P, I, C, R, A\}. \tag{5.3}$$

Using Eq (5.3), the adjoint system is obtained as

$$\begin{aligned}
\frac{\xi_S}{dt} &= [\sigma_a(A) + \mu + \beta_a(A)I]\,\xi_S - \sigma_a(A)\,\xi_V - \beta_a(A)I\,\xi_I, \\
\frac{\xi_V}{dt} &= -\delta\,\xi_S + (\mu + \delta + \theta)\,\xi_V - \theta\,\xi_P, \\
\frac{\xi_P}{dt} &= \mu\,\xi_P, \\
\frac{\xi_I}{dt} &= -q_1 + \beta_a(A)S\,\xi_I + (\mu + \gamma_a(A) + \vartheta_a(A))\,\xi_I - \vartheta_a(A)\,\xi_C \\
&\quad -\gamma_a(A)\,\xi_R - \beta_a(A)S\,\xi_S - \kappa\,u\,\xi_A, \\
\frac{\xi_C}{dt} &= -q_2 + (\mu + \phi)\,\xi_C - \kappa\,u\,\xi_A, \\
\frac{\xi_R}{dt} &= -\omega_a(A)\,\xi_S + (\omega_a(A) + \mu)\,\xi_R, \\
\frac{\xi_A}{dt} &= -\Big[\omega_a'(A)\,R\,\xi_S - \sigma_a'(A)\,S\,\xi_S - \beta_a'(A)\,I\,S\,\xi_S - \gamma_a'(A)\,I\,\xi_R - \vartheta_a'(A)\,I\,\xi_C - \eta\,\xi_A\Big].
\end{aligned} \tag{5.4}$$

The transversality conditions are $\xi_X(T) = 0$ for all X, as no terminal penalties are included. According to the maximum principle, the optimal control minimizes the Hamiltonian, i.e.,

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0, i = 1, 2, 3. \tag{5.5}$$

Using the above equation and the boundary of the controls, we finally obtain

$$\begin{aligned}
u_1^*(t) &= \min\left\{u_{1,\max}, \max\left\{0, \frac{(\xi_S(t) - \xi_V(t))\,\sigma_a(A)\,S(t)}{\alpha_1}\right\}\right\}, \\
u_2^*(t) &= \min\left\{u_{2,\max}, \max\left\{0, \frac{(\xi_S(t) - \xi_I(t))\,\beta_a(A)\,I(t)\,S(t)}{\alpha_2}\right\}\right\}, \\
u_3^*(t) &= \min\left\{u_{3,\max}, \max\left\{0, \frac{(\xi_I(t) - \xi_R(t))\,\gamma_a(A)\,I(t)}{\alpha_3}\right\}\right\}.
\end{aligned} \tag{5.6}$$

We have the following theorem from the above discussion.

Theorem 5. *Assume the state system has a unique solution for any admissible control pair $u \in \mathcal{U}$, and the parameters are positive with bounded initial condition in Eq* (2.7)*. Then, there exists an optimal control $u^* \in \mathcal{U}$ minimizing $J(u)$. Moreover, the pair $(u^*(t), X(t))$, with $X = (S, V, P, I, C, R, A)$, satisfies the state system in Eq* (5.2)*, the adjoint system in Eq* (5.4)*, and the control triplet*

$$u_1^*(t) = \min\left\{u_{1,\max}, \max\left\{0, \frac{(\xi_S(t) - \xi_V(t))\,\sigma_a(A)\,S(t)}{\alpha_1}\right\}\right\},$$

$$u_2^*(t) = \min\left\{u_{2,\max}, \max\left\{0, \frac{(\xi_S(t) - \xi_I(t))\,\beta_a(A)\,I(t)\,S(t)}{\alpha_2}\right\}\right\},$$

$$u_3^*(t) = \min\left\{u_{3,\max}, \max\left\{0, \frac{(\xi_I(t) - \xi_R(t))\,\gamma_a(A)\,I(t)}{\alpha_3}\right\}\right\}.$$

Remark 4. *The optimal system is a two-point boundary value problem. The state system in Eq* (5.2) *is an initial value, whereas the adjoint system in Eq* (5.4) *is a boundary value problem. For the simulations, we use a standard forward–backward sweep algorithm. We integrate the state system forward with an initial guess for $u_i, i = 1, 2, 3$, and integrate the adjoint system backward using the resulting state trajectory. The control variables are updated in each iteration via the iteration scheme. This process is continued until convergence is achieved.*

6. Sensitivity analysis

Sensitivity analysis provides a systematic approach to quantifying the relative influence of model parameters on disease-free dynamics, as measured by the basic reproduction number, $\mathcal{R}_0$. This quantity is a key threshold parameter in infectious disease modeling [37, 38]. The normalized forward sensitivity index of $\mathcal{R}_0$ with respect to a parameter p is defined as

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p}\frac{p}{\mathcal{R}_0}.$$

This index measures the relative change in the basic reproduction number due to a relative change in the parameter p. A positive sensitivity index indicates that increasing the parameter will increase $\mathcal{R}_0$, thereby enhancing disease transmission, whereas a negative index implies that increasing the parameter will reduce $\mathcal{R}_0$ and help control the infection.

The sensitivity indices of $\mathcal{R}_0$ with respect to the model parameters are computed as follows:

$$\begin{aligned}
\Upsilon_\pi^{\mathcal{R}_0} &= 1, \qquad \Upsilon_\beta^{\mathcal{R}_0} = 1,\\
\Upsilon_\gamma^{\mathcal{R}_0} &= -\frac{\gamma}{\mu+\gamma+\vartheta}, \qquad \Upsilon_\vartheta^{\mathcal{R}_0} = -\frac{\vartheta}{\mu+\gamma+\vartheta},\\
\Upsilon_\mu^{\mathcal{R}_0} &= -\frac{\mu}{\mu+\gamma+\vartheta} - \frac{\mu}{\sigma+\mu-\frac{\delta\sigma}{\mu+\delta+\theta}},\\
\Upsilon_\sigma^{\mathcal{R}_0} &= -\frac{\sigma}{\sigma+\mu-\frac{\delta\sigma}{\mu+\delta+\theta}},\\
\Upsilon_\delta^{\mathcal{R}_0} &= \frac{\delta\sigma}{(\mu+\delta+\theta)\left(\sigma+\mu-\frac{\delta\sigma}{\mu+\delta+\theta}\right)},
\end{aligned}$$

$$\Upsilon_{\theta}^{\mathcal{R}_0} = \frac{\delta\sigma\theta}{(\mu+\delta+\theta)^2\left(\sigma+\mu-\frac{\delta\sigma}{\mu+\delta+\theta}\right)}.$$

To find the influence of model parameters on the transmission HPV and related cancer, we provide the sensitivity analysis using the basic reproduction number $\mathcal{R}_0$. The forward sensitivity index is employed to quantify the relative change in $\mathcal{R}_0$ resulting from a relative change in a particular parameter. Positive sensitivity indices of parameters indicate an increase in $\mathcal{R}_0$, thereby promoting disease transmission, whereas a negative index implies a reduced $\mathcal{R}_0$, that contributing to infection control.

7. Numerical simulations

Numerical results are presented in this section. First, we plot the figures for the system without control. Then, we solve the optimal system numerically. Parameter values are taken from Table 1. In our simulations, the recruitment rate of $\pi = 45$ individuals per day was chosen to represent a population of approximately N individuals. For example, a population size of about 100,000 corresponds to an annual recruitment of 16,425 individuals, which is consistent with small to medium-scale demographic settings.

7.1. Dynamics without control

In Figure 3, the sensitivity analysis illustrates the influence of parameters on $\mathcal{R}_0$. Parameters associated with disease transmission exert a positive effect on $\mathcal{R}_0$. From this figure, we conclude that π, β, and δ act as amplifiers of HPV transmission, whereas σ, γ, ϑ, and μ function as suppressors. This analysis underscores that strengthening vaccination programs (increasing σ and θ), enhancing recovery rates (γ), and reducing waning immunity (δ) are critical strategies for lowering $\mathcal{R}_0$ and mitigating the spread of HPV and cervical cancer. Among these, vaccination emerges as the most important control mechanism, as it substantially reduces $\mathcal{R}_0$ and decreases both HPV prevalence and cervical cancer incidence.

Forward bifurcation between E_0 and E^* is observed in Figure 2. One can see that the DFE is stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$. Also, E^* is feasible for $\mathcal{R}_0 > 1$. Figure 4 shows that a unique positive root exists for the set of parameters in Table 1.

The numerical solution of the proposed mathematical model is presented in Figure 5. In this case, the basic reproduction number $\mathcal{R}_0$ is greater than one, indicating that the system is endemic. The equilibrium E^* is asymptotically stable. The number of infected cases increases initially and then converges to a fixed level.

Figure 6 is plotted to show that the endemic equilibrium E^* is nonlinearly asymptotically stable. Trajectories from different initial conditions converge to the same steady state. This shows the nonlinear stability of endemic equilibrium E^*.

Figure 7 illustrates the impact of the awareness campaign. By varying the awareness rate κ, changes in the steady-state values of the system population are observed. The figure shows that awareness campaigns play a crucial role in controlling HPV infection and related cancers. As the awareness rate κ increases, the number of infected cases decreases significantly, whereas both the vaccinated and permanently immune populations increase correspondingly. For sufficiently large values of κ, the

infected cases can be eradicated.

Figure 8 illustrates the effect of vaccination. As the vaccination rate α increases, both the vaccinated and permanently immune populations rise significantly. At the same time, the number of infected cases decreases markedly, demonstrating the effectiveness of higher vaccination rates.

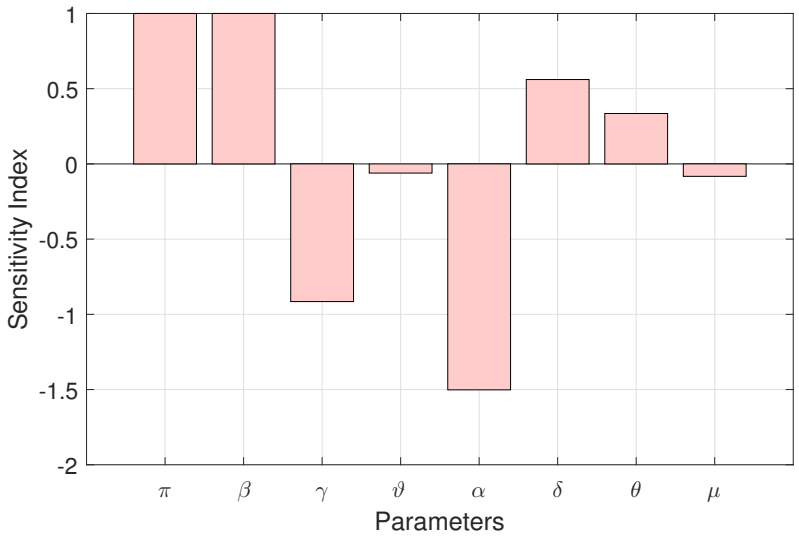

Figure 3. Sensitivity index of the model parameters.

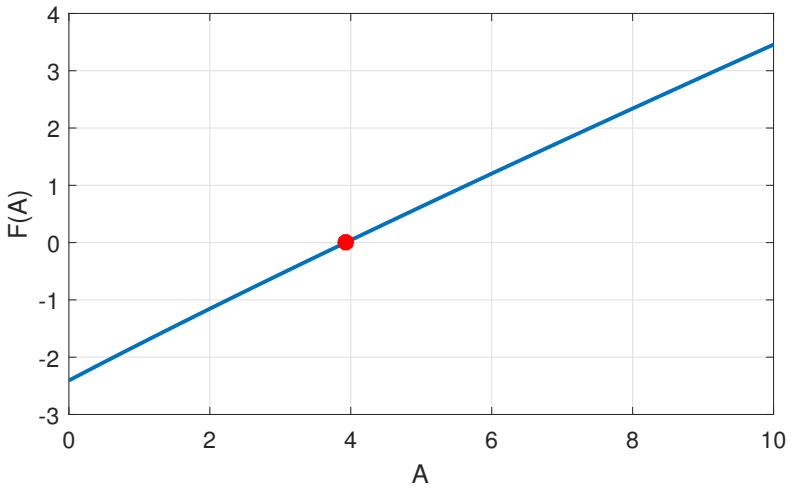

Figure 4. Existence of unique positive root of Eq (4.2). Parameter values as in Table 1.

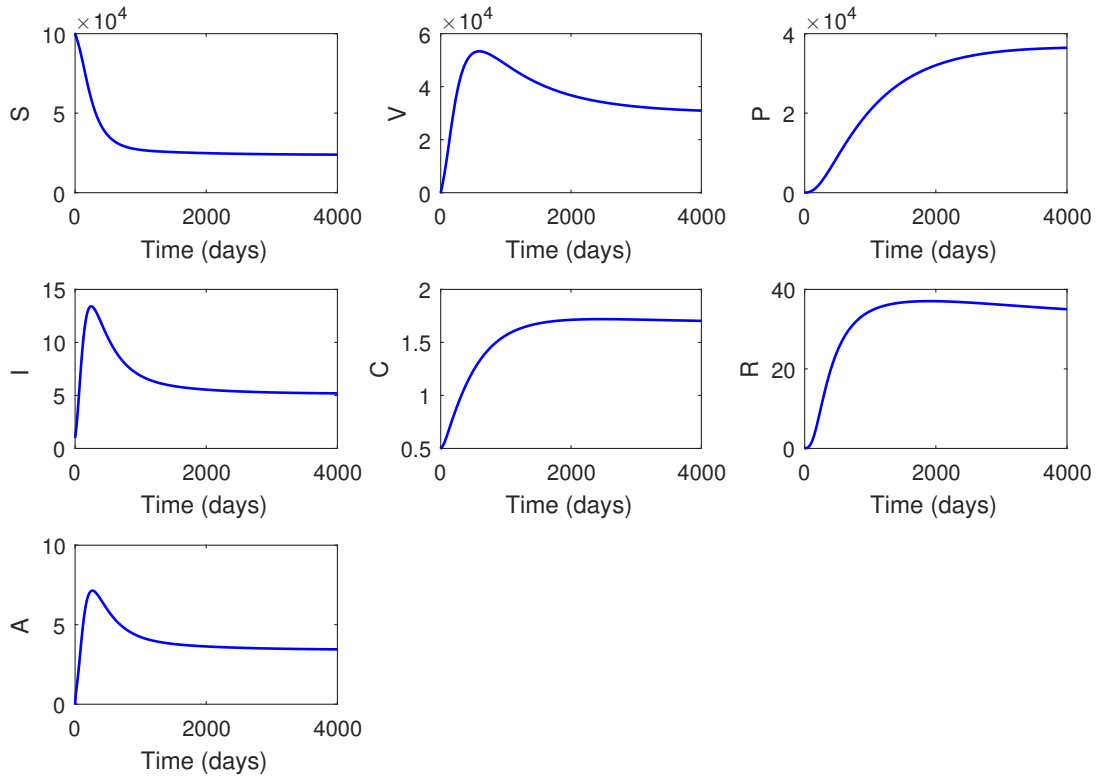

Figure 5. Time series solution of the model in Eq (2.6) with parameter values as in Table 1.

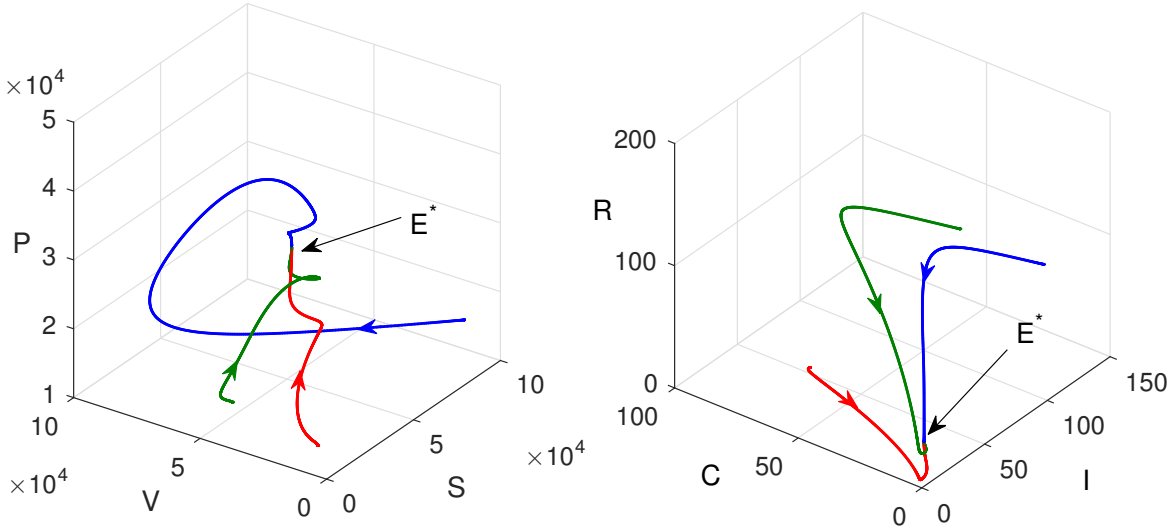

Figure 6. Nonlinear stability: Phase portraits are plotted in S-V-P planes. Parameter values are the same as in Figure 4. Phase trajectories are plotted using different initial values of the populations.

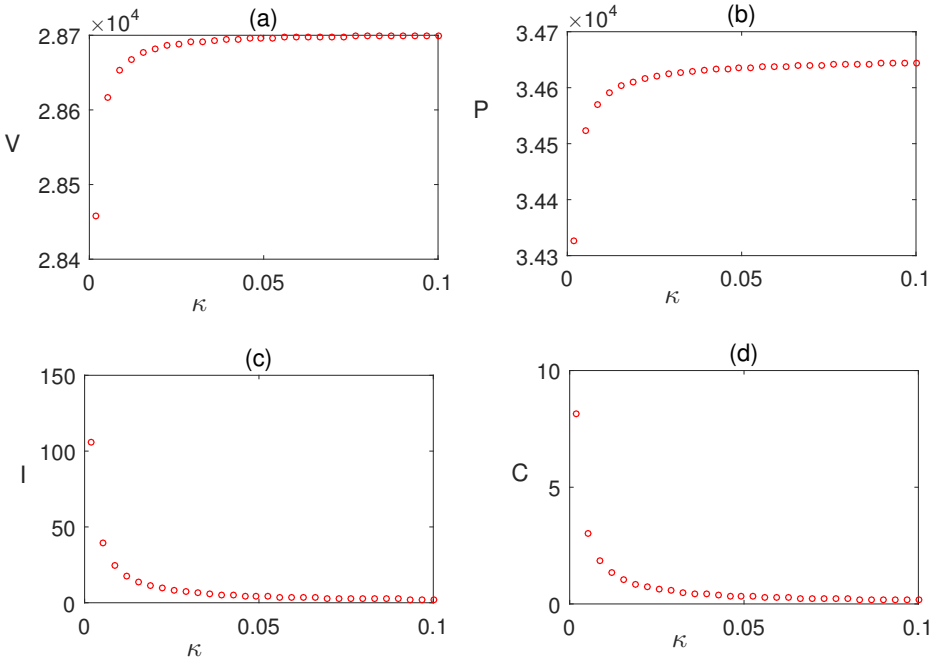

Figure 7. Effect of the rate of awareness level κ on the dynamics of the infection. We plotted the steady state values of the populations as a function of κ.

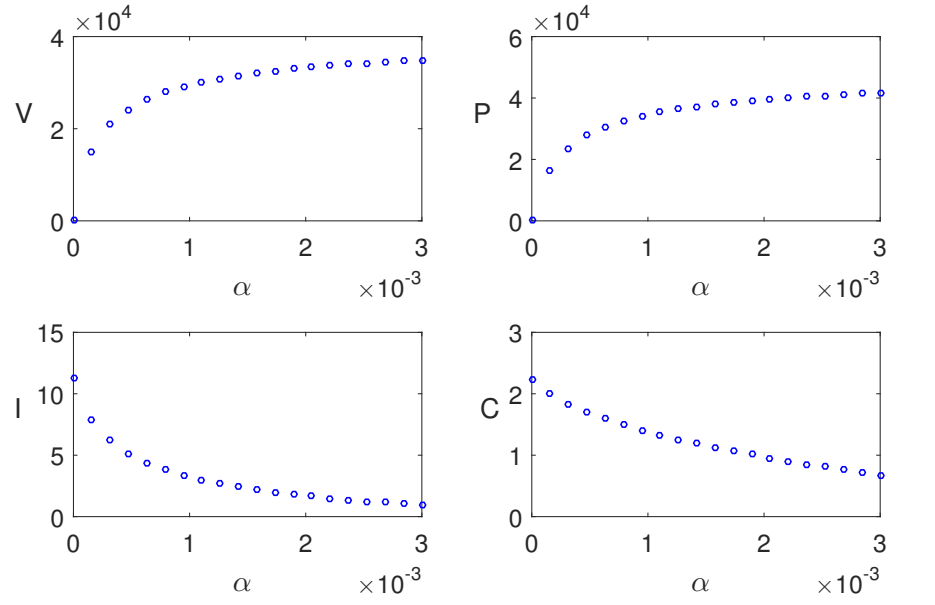

Figure 8. Effect of the rate of vaccination α on the dynamics of the system in Eq (2.6). Other parameters are taken from Table 1.

7.2. *Dynamics of the optimal system*

We have used the forward–backward sweep method to solve the optimal system, which is actually a two-point boundary value problem. Results obtained from the optimally controlled system are shown in Figures 9 and 10.

Using optimal vaccination strategies leads to better outcomes. The proportion of vaccinated individuals and the permanently immune population increases significantly compared to scenarios without an optimal control system. With this approach, the number of susceptible individuals increases, and the infected populations decrease. Thus, the application of optimal control theory is significant for HPV management.

Figure 10 presents the profiles of the optimal control triplet $u^*(t)$. The figure indicates that the vaccination, treatment, and process should initially be implemented at a higher rate, which can later be reduced in order to minimize the overall cost.

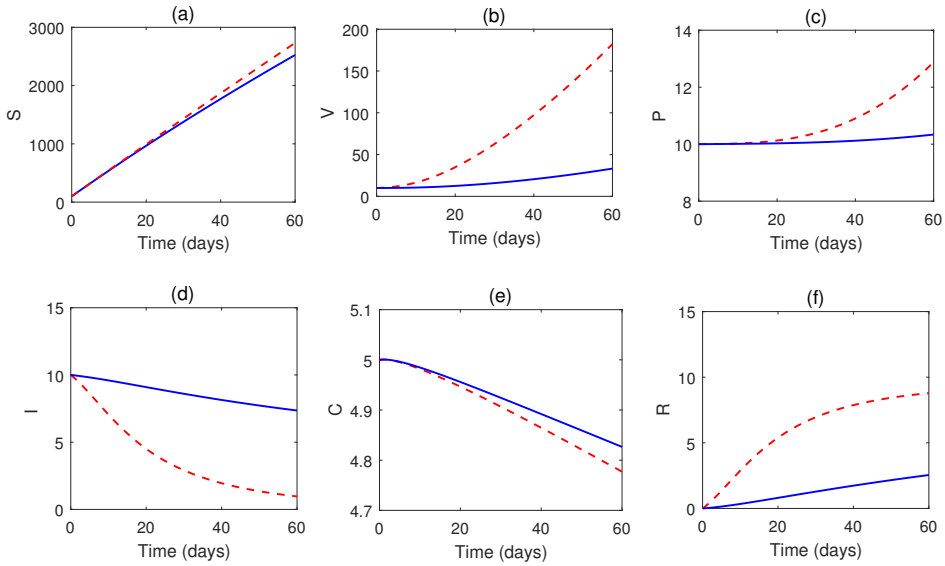

Figure 9. The optimal system is plotted using parameter values as in Table 1.

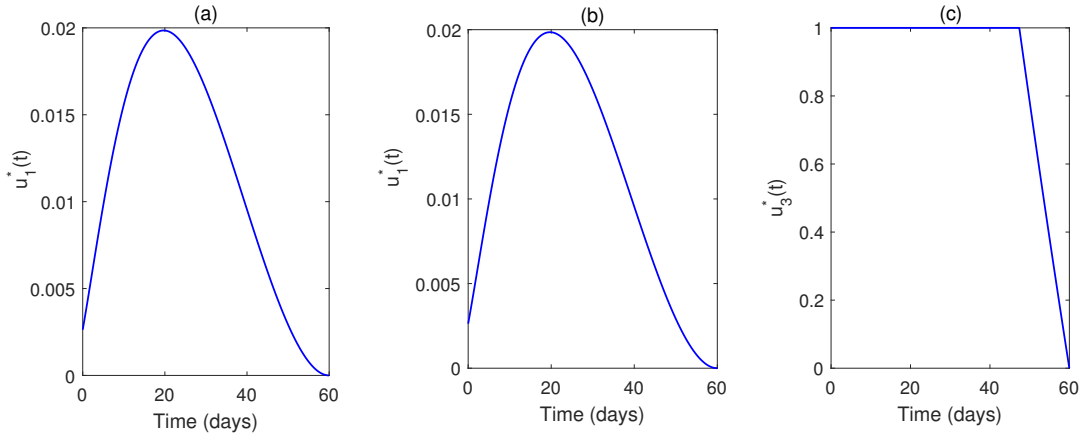

Figure 10. The optimal controls are plotted taking parameter values from Figure 9.

8. Discussion and conclusions

HPV is recognized as a major contributing factor to cervical cancer, which remains the second-most prevalent malignancy among women worldwide. Mathematical modeling is an effective tool for comprehending the process of disease transmission and developing efficient strategies for controlling cancer. In this paper, we develop a population model with the six compartments: Susceptible, HPV-infected, cancer-infected, recovered, and vaccinated and the impact of awareness.

The proposed model effectively captures the dynamic process of awareness campaigns and their impact on HPV transmission and cervical cancer development. Awareness modifies key epidemiological parameters by reducing transmission, disease progression, and immunity loss while enhancing vaccination uptake and recovery rates. The awareness variable, $A(t)$, acts as a behavioral intermediary, which decreases the transmission rate and increases the rate of vaccination and recovery. This model enables the simulation of various awareness programs and their long-term effects on the disease, thus providing information on effective and sustainable approaches to the prevention and control of cervical cancer.

The inclusion of awareness campaigns in mathematical models of epidemiology offers a practical approach to the evaluation of public health interventions. This model illustrates that awareness campaigns can greatly decrease the prevalence of HPV and the incidence of cervical cancer, thus establishing the need for the development of behavioral and biomedical interventions.

This study demonstrates that HPV transmission dynamics are highly sensitive to key epidemiological parameters. Vaccination, recovery, and awareness campaigns serve as the most effective suppressors of $\mathcal{R}_0$. The forward bifurcation analysis confirms that HPV persists endemically when $\mathcal{R}_0 > 1$, but targeted interventions can shift the system toward disease elimination.

Numerical simulations validate the reliability of the analytical findings. Awareness programs and vaccination policies play a pivotal role in controlling disease spread. Most importantly, the optimal control analysis highlights that large-scale vaccination strategies, initiated early and implemented gradually, deliver the most effective outcomes.

In a nutshell, building upon the strength of vaccination efforts, developing awareness campaigns, and increasing recovery rates are some of the major strategies that can help reduce the transmission of HPV and cervical cancer. This study provides a holistic framework for public health policymakers to launch effective campaigns against HPV and cervical cancer.

In our model, we assume that permanent immunity $P(t)$ provides lifelong protection. In reality, however, the HPV vaccine typically offers protection for only 10–20 years. This difference is an important limitation of the current work. As a next step, we plan to refine the model by incorporating more realistic assumptions about the duration of vaccine-induced immunity. One possible approach is to develop a delay model that explicitly accounts for the finite period of protection.

Use of AI tools declaration

No artificial intelligence tools were used in the preparation of this article.

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Conflict of interest

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Author contributions

Writing–original draft: F. A. B.; A Formal analysis: K. A., F. A. B; Investigation: F. A. B, K. A.; Project administration: K. A.; Software: K. A., F. A. B.; Supervision: F. A. B.; Writing–review & editing: K. A., F. A. B.

Appendix

Bifurcation analysis at $\mathcal{R}_0 = 1$. To study the local bifurcation near the DFE, $E_0 = (\bar{S}, \bar{V}, \bar{P}, 0, 0, 0, 0)$, we apply the center manifold theory. The Jacobian matrix at E_0 has a simple zero eigenvalue when $\mathcal{R}_0 = 1$. The basic reproduction number is:

$$\mathcal{R}_0 = \frac{\beta_a \bar{S}}{\mu + \gamma_a + \vartheta_a}.$$

Let $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$ be the right eigenvector associated with the zero eigenvalue. From the Jacobian matrix, the infected compartments dominate, so we obtain

$$w = (0, 0, 0, w_4, w_5, w_6, w_7)^T, \quad w_4 > 0.$$

Let $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$ be the left eigenvector satisfying $v^T J(E_0) = 0$. We normalize such that $v \cdot w = 1$. Following Castillo-Chavez and Song [39], the coefficients are defined as:

$$a = \sum_{i,j,k} v_i w_j w_k \frac{\partial^2 f_i}{\partial x_j \partial x_k}(E_0), \qquad b = \sum_{i,j} v_i w_j \frac{\partial^2 f_i}{\partial x_j \partial \beta_a}(E_0),$$

where f_i are the components of the system in Eq (2.6). Computing the second derivatives, we obtain:

$$a = -\frac{\beta_a \bar{S}\, v_4 w_4^2}{\mu + \gamma_a + \vartheta_a} < 0,$$

and

$$b = \frac{\bar{S}\, v_4 w_4}{\mu + \gamma_a + \vartheta_a} > 0.$$

Because $a < 0$, and $b > 0$, a forward transcritical bifurcation occurs at $\mathcal{R}_0 = 1$.

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