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Mathematical study of the impact of vaccination on the dynamics of human papillomavirus infection using homotopy perturbation method

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ABSTRACT

This study presents a mathematical model to assess the impact of vaccination on the dynamics of human papillomavirus (HPV) infection. A compartmental model is developed to describe the transmission dynamics of HPV, incorporating vaccination as a control measure. The system of equations describing the transmission dynamics was obtained. The homotopy perturbation method (HPM) was applied to obtain the analytical solution of the model's equations. Numerical simulations of the model solution were carried out, and graphical responses of the system were presented. Numerical simulations demonstrate the effectiveness of vaccination in reducing HPV prevalence. The results show that increasing the vaccination rate slows the rate of decline in the susceptible individual and leads to a substantial reduction in the number of infected individuals. For higher vaccination rates, the infection curve flattens significantly, showing the effectiveness of vaccination in curbing HPV spread. The results highlight the importance of optimal vaccination strategies in controlling HPV transmission. This study provides valuable insights for policymakers and public health officials in designing effective HPV vaccination programs, which helps to reduce the number of health problems and subsequent deaths attributed to the infection.

Highlights

- A mathematical model for the impact of vaccination on the dynamics of human Papillomavirus is proposed.
- Susceptible population decreases rapidly when the vaccination rate ψ is low.
- Increasing the vaccination rate leads to a substantial reduction in the number of infections.
- Infection can be effectively controlled by vaccination.

1. Introduction

Human papillomavirus (HPV) is a sexually transmitted infection that affects close to 70% of individuals during their lifetime (World Health Organization, 2024). HPV is connected with different types of cancers, including penile, anal, head and neck, and cervical cancer, with cervical cancer being the most common HPV-related cancer (WHO, 2024). Globally, the prevalence of HPV infection is estimated to be between 11% and 12%, with Nigeria reporting an overall HPV prevalence of up to 26.3% (Umeh et al., 2016). In Nigeria, the second most common type

of cancer among women is cervical cancer, with about 47million women aged 15 years and older at risk of developing the disease.

About 14,089 Nigerian women are diagnosed annually with cervical cancer, and the number of deaths due to the disease is approximately 8,240 (Umeh et al., 2016). Efforts to control the spread of the disease led to the introduction of HPV vaccination, which is the first preventive measure recommended to combat infection. The quadrivalent vaccine, authorized by the Centre for Disease Control and Prevention (CDC) in 2006, targets HPV types 16 and 18, which are responsible for 70% of cervical cancer, as well as HPV types 6 and 11, which cause benign anogenital warts and recurrent respiratory papillomatosis (Rosenthal et

al., 2011). To target HPV types 16 and 18, a bivalent vaccine was also introduced in 2009 (Rosenthal et al., 2011). The two vaccines are most effective when administered before sexual debut, as HPV infection typically occurs shortly after sexual initiation (World Health Organization, 2013). According to the report of the CDC, which stated that increase in the administration of HPV vaccination rates among susceptible individuals under 12 to 80% can prevent close to 53,000 cervical cancer cases in their lifetimes (CDC, 2013). For those who start the vaccination series later, between the ages of 15 and 26, three doses are required (Bell, 2018).

Despite enormous progress in prevention and treatment, cervical cancer remains a major cause of death among women, particularly in Low and Middle Income Countries (LMICs). Over 90% of the 604,000 cases and deaths that occurred globally in 2020 were recorded in low- and middle-income nations (Kumar et al., 2015). In Nigeria, cervical cancer is the second most common cancer among women of reproductive age (15--45 years), with an estimated 12,075 new cases annually as of 2020 (Ezechi et al., 2023). HPV types 16 and 18 are responsible for 66.9% of invasive cervical cancers, and these types are expected to be found in 3.5% of women with cervical cancer in Nigeria (Ezechi et al., 2023). The lack of impact of vaccines on the spread of HPV among adolescents and young adults in Nigeria is a fundamental barrier to prevention efforts.

To solve the problem, several mathematical models were developed to assess the impact of vaccination as a control measure in the prevention of the spread of the HPV infection in the population. Tokose (2022) developed a mathematical model of Cervical Cancer caused by HPV dynamics in the presence of immunization. The model's feasible region, solution set positivity, fundamental reproduction number (R_0), equilibrium points, and stability analysis were all examined. The author came to the conclusion that if immunization and the right treatment are carefully combined, the number of infected people will continue to decline.

Zhang et al (2020) carried out a study on the best course of treatment and sensitivity analysis of the model of HPV transmission dynamics. The investigation came to the conclusion that the sickness disappears when the value of the parameter R_0 is changed to $R_0 < 1$. There will be an epidemic of the infection, which will thereafter turn into an endemic illness when $R_0 > 1$. The outcomes also demonstrated that a sound treatment plan can successfully stop the disease from spreading.

A mathematical model was developed by Saldana et al. (2022) as an optimal control strategy to study HPV infection dynamics and vaccination techniques. The fundamental reproduction number R_0 was analysed using the next-generation matrix. The local stability of the Disease-Free Equilibrium point (DFE) was examined for $R_0 > 1$; the model shows a singular Endemic Equilibrium point (EE) that is locally asymptotically stable. Additionally, the model incorporated vaccination rates over time. The results therefore indicate that even if males are not given the vaccine, vaccination strategies for girls alone combined with catch-up vaccination for adult females can assist in eradicating HPV-related malignancies as long as high female coverage is maintained for several years. Further, HPV eradication can be accomplished much more quickly if both sexes are involved. The author also reported that the best way to distribute vaccines is to give them out in large quantities at the beginning of an outbreak and then gradually reduce immunization rates until they are nil. Despite all of this research, it is still difficult and expensive to eradicate HPV in the population. An optimal control analysis of a mathematical model of HPV with vaccination that included optimal control analysis was therefore carried out to bridge the gap.

Okware et al (2023) proposed a mathematical model of Human Papillomavirus (HPV) dynamics with vaccination incorporating optimal control analysis. The model equations were derived, and two equilibrium states were obtained. The stability of the two equilibrium states was determined and found to be asymptotically stable. Using MATLAB software, the optimality system was mathematically solved before being plotted against time for several interventions and their graphs described. Their findings revealed that when three interventions are combined, the infection begins to decrease considerably and eventually dies out in the community when the interventions are intensified.

This study seeks to improve on the work of Okware et al. (2023) by solving the model equations analytically by homotopy perturbation method (HPM), obtain the analytical solution and carry out numerical simulation of the solution that would be obtained using values of variables and parameters from verifiable sources. The parameter value of the rate of vaccination would be varied in the three compartments: susceptible, infected, and recovered. The graphical responses of the system would be obtained and results presented.

This paper is organized as follows: Section 1 is the introduction, and Section 2 is the methodology of the solution. In Section 3, the analytical solution of the model equation is obtained. Section 4 is the numerical simulation and discussion of results. Section 5 is the conclusion.

2. Methodology

In recent years, the application of the HPM in nonlinear problems has been developed by scientists and engineers because this method continuously deforms the difficult problem under study into a simple problem which is easier to solve. The homotopy perturbation method was proposed by He (1999) and was developed and improved by He (2000, 2006). Homotopy is an important part of differential topology. Actually, the homotopy perturbation method is a coupling of the

2.1 Basic idea of homotopy perturbation method (HPM)

To illustrate the ideas of this method, the following nonlinear differential equation was considered:

$$\text{Let } A(u) - f(r) = 0, \quad r \in \Omega \quad (1)$$

subject to the boundary condition of:

$$B(u, \frac{\partial u}{\partial n}) = 0, \quad r \in \Gamma \quad (2)$$

where A is a general differential operator, B is a boundary operator, $F(v)$ is known as an analytic function, and Γ is the boundary of the domain Ω .

The operator A can be divided into two parts, L and N , where L is the linear part, while N is a nonlinear one. Equation (1) can therefore be rewritten as follows:

$$L(u + N(u) - f(v)) = 0, \quad v \in \Omega \quad (3)$$

By homotopy techniques, we construct a homotopy defined by:

$$V(r, p): \Omega \times [0,1] \rightarrow \mathbb{R}, \quad v \in \Omega \rightarrow \mathbb{R}, \text{ which satisfies}$$

$$H(V, P) = L(v) - L(u_0 + PL(v_0)) + P[N(V) - F(r)] = 0 \quad (4)$$

where $P \in [0,1]$ is an embedding parameter, while u_0 is an initial approximation of Equation (2) which satisfies the boundary condition, $P = 0$ and $P = 1$, Equation (4) becomes:

$$H(v, 0) = L(v) - L(u_0) = 0 \tag{5}$$

$$H(v, 1) = A(v) - F(r) = 0 \tag{6}$$

The changing process of P from zero to one is just a change of $v(r, p)$ from u_0 to $u(r)$. In topology, the process is called deformation, $L(v) - L(u_0)$ and $A(v) - f(r)$ are known as homotopy. According to HPM, we first use the embedding parameter ‘P’ as a small parameter and assume that the solution of Equation (4) can be written as a power series in ‘P’ as:

$$V = V_0 + PV_1 + p^2V_2 + \dots + p^nV_n \tag{7}$$

Setting $P = 1$ results in the approximate solution of Equation (1) that is:

$$U \lim_{p \rightarrow 1} v = v_0 + v_1 + v_2 + \dots + v_n \tag{8}$$

Now the combination of the perturbation method and the Homotopy is called the HPM. This method eliminates the setback of the traditional perturbation method while keeping all its advantages. The series in Equation (7) is convergent for most cases. However, the convergence rate depends on the nonlinear parameter A(v). Moreover, the following suggestion is to be considered: the derivatives of N(v) with respect to v must be small because the parameter p may be large, i.e., $p \rightarrow 1$. The norm of $L^{-1} \frac{\partial N}{\partial v}$ must be smaller than one so that the series converges.

3. Model Formulation

The total population N(t) was subdivided into five compartments; S(t) Susceptible to HPV infection, V(t) Individuals that are vaccinated against HPV, I(t) Individuals infected with HPV without CC, C(t) Individuals that have developed cervical cancer due to HPV infection, R(t) Individuals that are permanently recovered due to vaccination and body immune system.

The total population $N(t) = S(t) + V(t) + I(t) + R(t) + C(t)$. The model was described by the following system of ODEs:

$$\frac{dS}{dt} = (1 - \psi)\Lambda - (a + \mu + \beta I)S + \sigma I + BV \tag{9}$$

$$\frac{dV}{dt} = \psi\Lambda + aS - (b + \mu + K)V \tag{10}$$

$$\frac{dI}{dt} = \beta SI - (\sigma + \mu + \alpha + \gamma)I \tag{11}$$

$$\frac{dC}{dt} = I\alpha - (\mu + \delta)C \tag{12}$$

$$\frac{dR}{dt} = \gamma I + kV - \mu R \tag{13}$$

Parameters used in the model include: Λ Recruitment rate, $(1 - \psi)\Lambda$ likelihood recruitment into susceptible, $\psi\Lambda$ likelihood recruitment into vaccinated, a rate of vaccination for those who are at risk, b how quickly those who have had vaccinations are vulnerable, κ rate for vaccine-protected people to fully recover, β rate of HPV infection among those

who are susceptible, σ rate at which infected individuals go back to being susceptible, α rate at which CC develops in those who have HPV infection, δ mortality due to CC, μ natural mortality rate, γ rate of recovery for infected people.

The study was based on the following assumptions:

- i. Recruitment into the population is by females becoming sexually active and other factors like immigration.
- ii. HPV recoveries acquire permanent immunity and are not susceptible to cervical cancer infection.

Rearranging the model Equations (9)–(13), we have:

$$\frac{dS}{dt} = \theta\Lambda - \pi S + \sigma I + BV \tag{14}$$

$$\frac{dV}{dt} = \rho\Lambda + aS - \phi V \tag{15}$$

$$\frac{dI}{dt} = \beta SI - \tau I \tag{16}$$

$$\frac{dC}{dt} = I\alpha - \omega C \tag{17}$$

$$\frac{dR}{dt} = \gamma I + kV - \mu R \tag{18}$$

where $\theta = 1 - \psi$

$$\pi = a + \mu + \beta I \tag{19}$$

$$\phi = b + \mu + K \tag{20}$$

$$\tau = \sigma + \mu + \alpha + \gamma \tag{21}$$

$$\omega = \mu + \delta \tag{22}$$

3.1 Analytical solution of the model equation using homotopy perturbation method (HPM)

Constructing the homotopy for Equations (14)–(22), we have:

$$S: (1 - p)\left(\frac{dS}{dt} - \frac{\theta}{\Lambda}\right) + p\left(\frac{dS}{dt} - \frac{\theta}{\Lambda} + \pi S - \sigma I - \beta V\right) = 0 \tag{23}$$

$$V: (1 - p)\left(\frac{dV}{dt} - \rho\Lambda\right) + p\left(\frac{dV}{dt} - \rho\Lambda + aS + \Phi V\right) = 0 \tag{24}$$

$$I: (1 - p)\left(\frac{dI}{dt}\right) + p\left(\frac{dI}{dt} - \beta SI + \tau I\right) = 0 \tag{25}$$

$$C: (1 - p)\left(\frac{dC}{dt}\right) + p\left(\frac{dC}{dt} - I\alpha + \omega C\right) = 0 \tag{26}$$

$$R: (1 - p)\left(\frac{dR}{dt}\right) + p\left(\frac{dR}{dt} - \gamma I - kV + \mu R\right) = 0 \tag{27}$$

Expanding Equations (23)–(27), gives:

$$S(t) = S_0(t) + pS_1(t) + p^2S_2(t) + \dots \tag{28}$$

$$V(t) = V_0(t) + pV_1(t) + p^2V_2(t) + \dots \tag{29}$$

$$I(t) = I_0(t) + pI_1(t) + p^2I_2(t) + \dots \tag{30}$$

$$C(t) = C_0(t) + pC_1 + p^2R_2(t) + \dots \tag{31}$$

$$R(t) = R_0(t) + pR_1(t) + p^2R_2(t) + \dots \tag{32}$$

Solving For S_0, V_0, I_0, C_0, R_0

Substituting $p = 0$ we get:

$$\frac{dS_0}{dt} = \theta\Lambda \tag{33}$$

$$\frac{dV_0}{dt} = \rho\Lambda \tag{34}$$

$$\frac{dI_0}{dt} = 0 \tag{35}$$

$$\frac{dC_0}{dt} = 0 \tag{36}$$

$$\frac{dR_0}{dt} = 0 \tag{37}$$

Solving the initial approximation Equations (33)–(37):

$$S_0(t) = S_0(0) + \theta\Lambda t \tag{38}$$

$$V_0(t) = V_0(0) + \rho\Lambda t \tag{39}$$

$$I_0(t) = I_0(0) \tag{40}$$

$$R_0(t) = R_0(0) \tag{41}$$

Substituting the series expansion into the homotopy equations and equating coefficients of P^1 , we get :

$$\frac{dS_1}{dt} = -\pi S_0 + \sigma I_0 + \beta \tag{42}$$

$$\frac{dV_1}{dt} = aS_0 - \phi \tag{43}$$

$$\frac{dI_1}{dt} = \beta S_0 I_0 - \tau \tag{44}$$

$$\frac{dC_1}{dt} = I_0\alpha - \omega \tag{45}$$

$$\frac{dR_1}{dt} = \gamma I_0 + kV_0 - \mu \tag{46}$$

Solving for S_1, V_1, I_1, C_1, R_1

Solving the first-order differential equation: solving Equations (38)–(42):

$$S_1(t) = \int(-\pi S_0 + \sigma I_0 + BV_0) dt \tag{47}$$

$$V_1(t) = \int(aS_0 - \phi V_0) dt \tag{48}$$

$$I_1(t) = \int(\beta S_0 I_0 - \tau I_0) dt \tag{49}$$

$$C_1(t) = \int(I_0\alpha - \omega C_0) dt \tag{50}$$

$$R_1(t) = \int(\gamma I_0 + kV_0 - \mu R_0) dt \tag{51}$$

Evaluating the integrals of Equations (47)–(51) gives:

$$\begin{aligned} S_1(t) &= \int(-\pi S_0 + \sigma I_0 + BV_0) dt \\ S_1(t) &= \int(-\pi(S_0(0) + \theta\Lambda t) + \sigma I_0(0) + B(V_0(0) + \rho\Lambda t)) dt \\ S_1(t) &= -\pi S_0(0)t - \frac{\pi\theta\Lambda t^2}{2} + \sigma I_0(0)t + BV_0(0)t + \frac{B\rho\Lambda t^2}{2} \end{aligned} \tag{52}$$

$$\begin{aligned} V_1(t) &= \int(aS_0 - \phi V_0) dt \\ V_1(t) &= \int(a(S_0(0) + \theta\Lambda t) - \phi(V_0(0) + \rho\Lambda t)) dt \\ V_1(t) &= aS_0(0)t + \frac{a\theta\Lambda t^2}{2} - \phi V_0(0)t - \frac{\phi\rho\Lambda t^2}{2} \end{aligned} \tag{53}$$

$$\begin{aligned} I_1(t) &= \int(\beta S_0 I_0 - \tau I_0) dt \\ I_1(t) &= \int(\beta(S_0(0) + \theta\Lambda t)I_0(0) - \tau I_0(0)) dt \\ I_1(t) &= \beta S_0(0)I_0(0)t + \frac{\beta\theta\Lambda I_0(0)t^2}{2} - \tau I_0(0)t \end{aligned} \tag{54}$$

$$\begin{aligned} C_1(t) &= \int(I_0\alpha - \omega C_0) dt \\ C_1(t) &= \int(I_0(0)\alpha - \omega C_0(0)) dt \\ C_1(t) &= I_0(0)\alpha t - \omega C_0(0)t \end{aligned} \tag{55}$$

$$\begin{aligned} R_1(t) &= \int(\gamma I_0 + kV_0 - \mu R_0) dt \\ R_1(t) &= \int(\gamma I_0(0) + k(V_0(0) + \rho\Lambda t) - \mu R_0(0)) dt \\ R_1(t) &= \gamma I_0(0)t + kV_0(0)t + \frac{k\rho\Lambda t^2}{2} - \mu R_0(0)t \end{aligned} \tag{56}$$

The solution of Equations (9)–(13) are stated in Equations (56)–(61):

$$S_1(t) = -\pi S_0(0)t - \frac{\pi\theta\Lambda t^2}{2} + \sigma I_0(0)t + BV_0(0)t + \frac{B\rho\Lambda t^2}{2} \tag{57}$$

$$V_1(t) = aS_0(0)t + \frac{a\theta\Lambda t^2}{2} - \phi V_0(0)t - \frac{\phi\rho\Lambda t^2}{2} \tag{58}$$

$$I_1(t) = \beta S_0(0)I_0(0)t + \frac{\beta\theta\Lambda I_0(0)t^2}{2} - \tau I_0(0) \tag{59}$$

$$C_1(t) = I_0(0)\alpha t - \omega C_0(0) \tag{60}$$

$$R_1(t) = \gamma I_0(0)t + kV_0(0)t + \frac{k\rho\Lambda t^2}{2} - \mu R_0(0) \tag{61}$$

4. Numerical Simulation and Discussion of Results

This section presents the numerical simulations and graphical results of the mathematical model formulated for the dynamics of HPV infection with vaccination. The numerical simulation aims to analyze the behavior of each compartment of the model under varying parameter values, particularly the vaccination rate ψ . The simulations were carried out using MATLAB software. For the initial conditions. We assume: $S(0) = 900, I(0) = 20, R(0) = 10$.

Table 1: Parameter values used in simulations.

Parameter	Description	Value	Source
Λ	Recruitment Rate	1000	Okware et al., 2023
ψ	Vaccination Rate	0.05	Okware et al., 2023
b	How quickly those who are vaccinated are vulnerable	0.05	Okware et al., 2023
k	Rate for vaccine-protected people to fully recover	0.2	Okware et al., 2023
β	Rate of HPV infection among those who are susceptible	0.001	Okware et al., 2023
σ	Rate at which infected individuals go back to being susceptible	0.2	Okware et al., 2023
γ	Rate of recovery for infected people	0.2	Okware et al., 2023
μ	Natural mortality rate	0.07	Okware et al., 2023
δ	Mortality rate due to cervical cancer	0.4	Okware et al., 2023

From Figure 1, it is observed that the susceptible population decreases rapidly when the vaccination rate ψ is low. However, as the vaccination rate increases, the decline in the susceptible population becomes slower. This implies that vaccination protects a greater proportion of the population from becoming infected, thereby maintaining a higher number of susceptible individuals.

From Figure 2, the results show that increasing the vaccination rate leads to a substantial reduction in the number of infected individuals. When $\psi = 0$, the infection grows rapidly and reaches a peak before declining. For higher vaccination rates, the infection curve flattens significantly, showing the effectiveness of vaccination in curbing HPV spread.

Effect of vaccination rate ψ on susceptible population

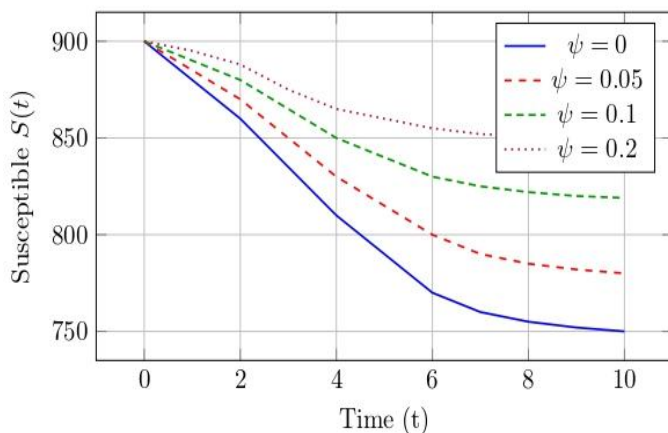


Figure 1: Effect of vaccination rate ψ on susceptible population $S(t)$.

Figure 3 shows that as the vaccination rate increases, the recovered population rises more rapidly and stabilizes at a higher level. This shows that vaccination enhances immunity, to avoid going back to susceptible. Summarily, these results suggest that the disease management strategies are effective in controlling the spread of the disease, and the population is recovering over time. The trends observed in the calculated values

provide valuable insights into the population’s dynamics and the impact of interventions on disease transmission.

Effect of vaccination rate ψ on infection

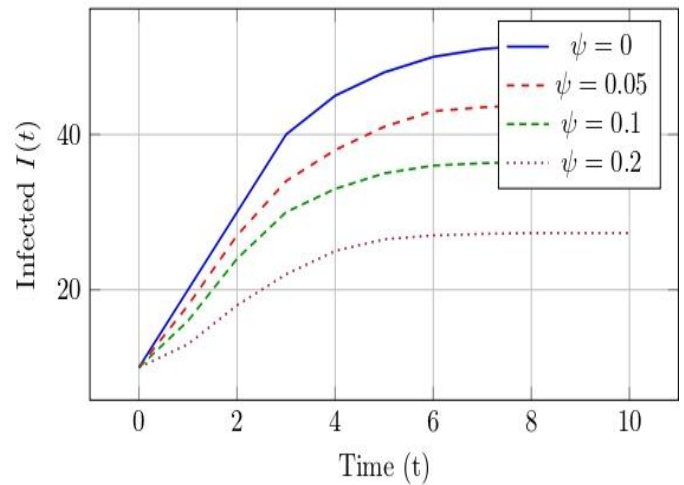


Figure 2: Effect of vaccination rate ψ on susceptible population $S(t)$.

Effect of vaccination rate ψ on recovered population

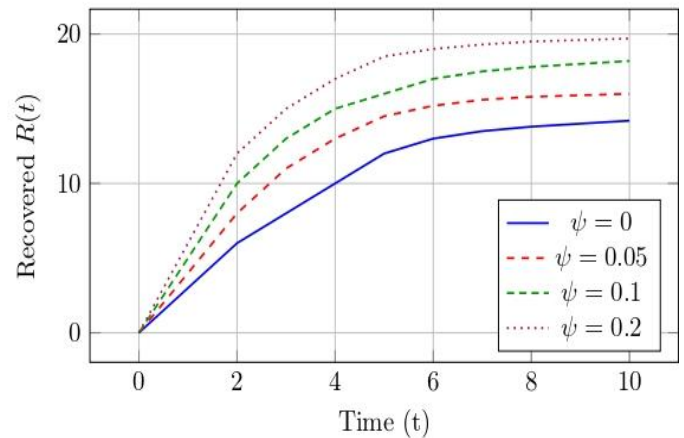


Figure 3: Effect of vaccination rate ψ on susceptible population $S(t)$.

5. Conclusion

This study proposed a mathematical model to assess the impact of vaccination on the dynamics of HPV infection. The model equations describing the dynamics of HPV were derived, and it was solved analytically by HPM. Numerical Simulation of the analytical solution obtained by homotopy perturbation was carried out, and graphical responses of the system were presented. The results of the numerical simulation were presented graphically in order to have a better understanding of the models. The model described well the dynamics of Human Papillomavirus and the effectiveness of vaccination in controlling the spread of the infection. The results highlight the importance of optimal vaccination strategies in controlling HPV transmission. This study provides valuable insights for policymakers and public health officials in designing effective HPV vaccination programs, which helps to reduce the number of health problems and subsequent deaths attributed to the infection.

6. Recommendations

1. The study recommends the introduction of a government policy of a compulsory vaccination programme to prevent the disease from spreading.
2. The government should put in place a medical support programme that will help people who are suffering from the disease to access medical facilities for treatment.
3. The study suggests that future work should focus on other effective prevention measures that are affordable to low-income individuals.
4. A mathematical model on the dynamics of HPV infection should aggregate all the control measures that will enhance prevention and proper management of the infection in the population.

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Competing Interest

The author declared that there are no identifiable conflicts of interest that could be perceived as influencing this research work.

Data Availability Statement

All data generated or analyzed during this study are included in this published article.

Ethics Approval

This study did not involve human participants or animals and therefore did not require ethics approval.

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