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Modelling the Impact of Vaccination and Screening on the Dynamics of Human Papillomavirus Infection

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Abstract

While human papillomavirus has been a recognized disease for a long time, the control of outbreaks remains a challenge. The aim of this study is to investigate the role of screening and vaccination as control strategies in curtailing the spread of the disease. Using the next generation matrix, the disease free equilibrium has be shown to be asymptotically stable. Furthermore, sensitivity analysis is then performed on the key parameters driving HPV dynamics in order to detrmine their relative importance and potential impact in HPV dynamics and to dertmine the impacts of vaccination and screening in the spread of HPV. Numerical results indicate that HPV infection can be reduced hwn both interventions, that is screenig and vaccination, are implemented in order to reduce the burden of the disease.

Mathematics Subject Classification: 34D20

Keywords: HPV infection, Screening, Reproduction number, Treatment, vaccination

1 Introduction

Human papillomavirus (HPV) is the name of a family of viruses that includes more than 100different types, and more than 30 of these virues are sexually transmitted ([14], [10], [4]). Most of the HPV infections are asymptomatic and can fed away without treatment over the course of a few years. Fo instance, about 70% of HPV infections fed away within a year and 90% within two years. However, in some people infection can persist for many years and can cause warts (low risk genotype of HPV), while other types lead to different

kinds of cancers (high risk genotype of HPV) including cervical cancer ([5], [4]). Although HPV itself cannot be treated, the cellular changes that come from any HPV infection can be treated. For examples, genital warts, cervical, anal, and genital cancers can be treated if the infection is diagnosed during the early stage of development. Pre-cancerous cell changes caused by HPV can be detected by Pap tests and treat individuals who are found already infected [15].

Different models have been developed to analyse the transmission dynamics of HPV infection as well as the effectiveness of some intervention strategies against the spread of HPV infection. For example, [10] developed a mathematical model to investigate the impact of vaccination against human papillomavirus, accounting for a wide-spread childhood vaccination programme that may be supplemented by voluntary adult vaccination. Also, [8] formulated an SIS model for human papillomavirus transmission with vaccination as a control strategy and [9] developed a dynamic model for the heterosexual transmission of Human Papillomavirus types 16 and 18, which are covered by available vaccines. Most studies concluded that vaccination play positive role in reducing the incidence of HPV infection in the community. However, none of them considered the mathematical model for analyzing both screening and vaccination on reducing HPV infection in the population. Although vaccination can play major role in reducing HPV infection by protecting vaccinated and unvaccinated persons, but can provide temporary immunity. Hence a combination of screening and vaccination becomes important in the effort of reducing the incidence of HPV infection in the population. In this study a mathematical model will be developed to determine the effectiveness of both screening and vaccination in attempting to reduce HPV transmission. However, the purpose of screening is to provide treatment for those who are found to have HPV infection. The rest of the paper is organised as follows. In Section 2, an HPV model with both screening and vaccination is formulated and, analysis of the model is presented in Section 3. In Section 4, numerical simulation and sensitivity analysis on the model are carried out in order to verify some analytical results. Discussion and some concluding remarks are described in Section 5.

2 Model Formulation

HPV model classifies the population at any time t, denoted by $N(t)$ in Susceptible individuals $S(t)$, Vaccinated individuals $V(t)$, Unaware infected individuals I_u, Screened infected individuals I_s, Individuals with cervical cancer C(t) and recovered individuals $R(t)$. It is assumed that susceptible individuals are recruited into the population at a constant rate Π. A proportion ω of susceptible individuals are vaccinated and the rest may acquare HPV infection at rate λ when they come into effective contact with an infectious individual at the rate β that may lead to infection. We also assume that screened individuals modify their behaviour at a rate η which results into reducing the transmission rate of HPV in the community. The force of is given by $\lambda = \frac{\beta(I_u+I_s)}{N}$. Furthermore, it is assumed that vaccinated individuals may also be infected at a reduced rate $(1-\varphi)\lambda$, where $\varphi \in (0,1)$ measures the efficacy of the vaccine. The vaccinated individuals return to the susceptible class after waning their immunity at a rate σ. The unaware infected

individuals are screened and join the screened infected class at a rate ϕ. However, some of the unaware infected individuals progress to cervical cancer at a rate ϵ and others recover naturally through body immune system at a rate δ, and the screened infected individuals are treated at a rate γ. Additionally, recovered individuals revert to the susceptible class after waning their immunity at a rate α. All individuals suffer natural mortality at a rate μ and sick individuals die of cancer at a rate ζ. The flow diagram of the model is shown in Figure 1.

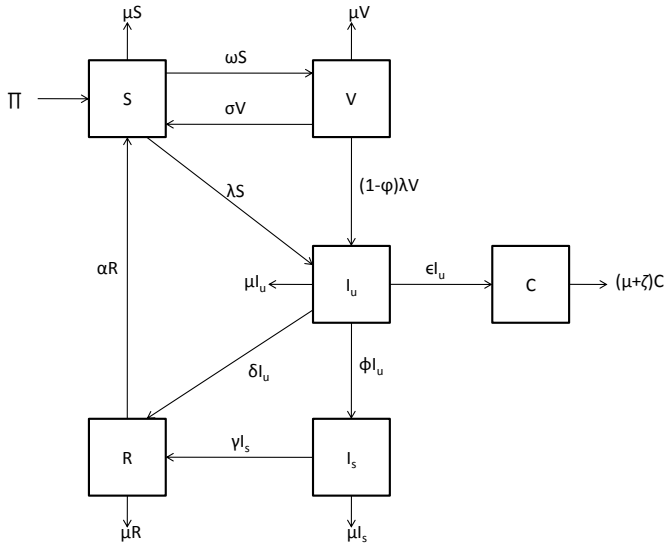


Figure 1: Flow diagram for HPV model with screening and vaccination

In view of the assumptions and the flow diagram we obtain the following system of non linear ordinary differential equations;

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - (\lambda + \omega + \mu)S + \alpha R + \sigma V \\
\frac{dV}{dt} &= \omega S - [(1-\varphi)\lambda + \sigma + \mu)]V \\
\frac{dI_u}{dt} &= \lambda S + (1-\varphi)\lambda V - (\epsilon + \phi + \delta + \mu)I_u \\
\frac{dI_s}{dt} &= \phi I_u - (\gamma + \mu)I_s \\
\frac{dC}{dt} &= \epsilon I_u - (\mu + \zeta)C \\
\frac{dR}{dt} &= \delta I_u + \gamma I_s - (\alpha + \mu)R.
\end{aligned} \tag{2.1}$$

The non- negative initial conditions of the model system (2.1) are $S(0) > 0$, $V(0) \geq 0$, $I_u(0) \geq 0$, $I_s(0) \geq 0$, $C(0) \geq 0$, $R(0) \geq 0$.

3 Model Analysis

We begin by showing that all feasible solutions are uniformly bounded in a proper subset of $\Omega \in \mathbb{R}^6_+$. The feasible region Ω with $\Omega = \{(S, V, I_u, I_s, C, R) \in \mathbb{R}^6_+; N \leq \frac{\Pi}{\mu}\}$ is considered. Summation of all the equations of the model (2.1) gives

$$\begin{aligned} \frac{dN(t)}{dt} &= \Pi - \mu N(t) - \zeta C(t) \\ &\leq \Pi - \mu N(t). \end{aligned} \tag{3.1}$$

Applying [6] on a differential inequalities on (3.1) we obtain

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Pi}{\mu}(1 - e^{-\mu t}), \tag{3.2}$$

where N(0) represents the initial values of the respective variables. Then $0 \leq N \leq \frac{\Pi}{\mu}$ as $t \to \infty$. Therefore, $\frac{\Pi}{\mu}$ is an upper bound of $N(t)$ provided $N(0) \leq \frac{\Pi}{\mu}$. Hence feasible solution of the model system (2.1) enters the region Ω which is positively invariant set. Thus the system is biologically meaningful and mathematically well posed in the domain of Ω. In this domain, it is sufficient to consider the dynamics of the flow generated by the model system (2.1). We therefore, summarize the result in the following lemma, The closed set $\{(S, V, I_u, I_s, C, R) \in \mathbb{R}^6_+; N \leq \frac{\Pi}{\mu}\}$ is positively invariant and attracting with respect to the model (2.1).

3.1 Local stability of the disease-free equilibrium, E_0

The disease-free equilibrium of the model system (2.1) is given by

$$E_0 = (S^0, V^0, 0, 0, 0) = \left(\frac{\Pi(\sigma + \mu)}{\mu(\sigma + \omega + \mu)}, \frac{\Pi\omega}{\mu(\sigma + \omega + \mu)}, 0, 0, 0, 0\right).$$

The stability of this eguilibrium will be inverstigate using the next generation (see [13], [12]). Using the notations as in [12] for the model system (2.1), the associated matrices F and V for the new infectious terms and the remaining transition terms are respectively given by

$$F = \begin{bmatrix} \frac{\beta(\sigma+\mu+(1-\varphi)\omega)}{(\mu+\sigma+\omega)} & \frac{\eta\beta(\sigma+\mu+(1-\varphi)\omega)}{(\mu+\sigma+\omega)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \epsilon + \phi + \delta + \mu & 0 & 0 \\ -\phi & (\gamma + \mu) & 0 \\ -\epsilon & 0 & (\mu + \zeta) \end{bmatrix}. \tag{3.3}$$

It follows that the effective reproduction number R_e is given by

$$R_e = \rho(FV^{-1}) = \frac{\beta(\sigma + \mu + (1 - \varphi)\omega)(\gamma + \mu) + \phi\eta\beta(\sigma + \mu + (1 - \varphi)\omega)}{(\mu + \sigma + \omega)(\epsilon + \phi + \delta + \mu)(\gamma + \mu)}, \tag{3.4}$$

where $\rho(FV^{-1})$ is the spectral radius of the matrix FV^{-1}. The effective reproduction number measures the average number of new infectious denerated by a single HPV infectious individual in a community where vaccination and screening are used as control strategies.

3.2 Global stability of the disease-free equilibrium, E_0

The disease free equilibrium E_0 of the model system (2.1) is globally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$. *Proof:* By the comparison theorem, the rate of change of the variables representing the infected components of model system (2.1) can be re-written as,

$$\begin{bmatrix} I_u' \\ I_s' \\ C' \end{bmatrix} = (F - V) \begin{bmatrix} I_u \\ I_s \\ C \end{bmatrix} - M_1\vartheta_1 \begin{bmatrix} I_u \\ I_s \\ C \end{bmatrix} - M_2\vartheta_2 \begin{bmatrix} I_u \\ I_s \\ C \end{bmatrix}, \tag{3.5}$$

where the matrices F and V are as defined by the expressions (3.3). $M_1 = 1 - \frac{S^0}{N^0}$ and $M_2 = 1 - \frac{V^0}{N^0}$, ϑ_1 and ϑ_2 are non negative matrices. But, $S^0 = \frac{\Pi(\sigma+\mu)}{\mu(\mu+\sigma+\omega)}$, and $V^0 = \frac{\Pi\omega}{\mu(\mu+\sigma+\omega)}$. Since $S^0 \leq N^0$ then $V^0 \leq N^0$. From the system (3.5) we get,

$$\begin{bmatrix} I_u' \\ I_s' \\ C' \end{bmatrix} \leq (F - V) \begin{bmatrix} I_u \\ I_s \\ C \end{bmatrix}. \tag{3.6}$$

Therefore,

$$(F - V) = \begin{bmatrix} A_{11} & A_{12} & 0 \\ \phi & -(\gamma + \mu) & 0 \\ \epsilon & 0 & -(\mu + \zeta) \end{bmatrix}, \tag{3.7}$$

where

$$A_{11} = \frac{\beta(\sigma + \mu + (1 - \varphi)\omega)}{(\mu + \sigma + \omega)} - (\epsilon + \phi + \delta + \mu),$$
$$A_{12} = \frac{\eta\beta(\sigma + \mu + (1 - \varphi)\omega)}{(\mu + \sigma + \omega)}.$$

From the matrix (3.7), let ψ be the eigenvalue. Then we have $|(F - V) - I\psi| = 0$. Matrix (3.6) gives the characteristic equation

$$\psi^3 + b_2\psi^2 + b_1\psi + b_0 = 0 \tag{3.8}$$

where,

$$\begin{aligned} b_2 &= -(A_{11} - (\gamma + 2\mu + \zeta)), \\ b_1 &= -(A_{11} - (\gamma + 2\mu + \zeta) - (\gamma + \mu)(\mu + \zeta)), \\ b_0 &= -A_{11}(\gamma + \mu)(\mu + \zeta). \end{aligned}$$

Therefore, all eigenvalues of matrix (3.7) have negative real part, showing that matrix (3.7) is stable for $R_e < 1$. Consequently, using the equation (3.1), $(I_u, I_s, C) \Rightarrow (0, 0, 0)$ as $t \Rightarrow \infty$. Thus by comparison theorem according to [16], $(I_u, I_s, C) \Rightarrow (0, 0, 0)$ as $t \Rightarrow \infty$. Evaluating the system (2.1) at $I_u = I_s = C = 0$ gives $S^0 \Rightarrow \frac{\Pi(\sigma+\mu)}{\mu(\mu+\sigma+\omega)}$, $V^0 \Rightarrow \frac{\Pi\omega}{\mu(\mu+\sigma+\omega)}$ and $R \Rightarrow 0$ for $R_e < 1$. Hence the disease-free equilibrium E_2 is globally asymptotically stable if $R_e < 1$. ◇

3.3 The Endemic Equilibrium Point, E_1

Endemic equilibrium point E_1 is steady state solution where the disease persists in the population. For the existence and uniqueness of endemic equilibrium $E_1 = (S^*, V^*, I_u^*, I_s^*, C^*, R^*)$, its coordinates should satisfy the conditions; $E_1 = (S^*, V^*, I_u^*, I_s^*, C^*, R^*) \neq 0$, where $S^* > 0$, $V^* > 0$, $I_u^* > 0$, $I_s^* > 0$, $C^* > 0$, $R^* > 0$. The endemic equilibrium point is obtained by setting equation of the system (2.1) to zero. We then solve for state variables in terms of the force of infection, λ and obtain the following;

$$\begin{aligned}
S^* &= \frac{(FG\Pi + \alpha H I_u^*)((1-\varphi)\lambda^* + K)}{P},\\
V^* &= \frac{\omega(FG\Pi + \alpha H I_u^*)}{P},\\
I_u^* &= \frac{FG\Pi\lambda^*((1-\varphi)\lambda^* + K + (1-\varphi)\omega)}{MP - H\alpha\lambda^*((1-\varphi)\lambda^* + K + (1-\varphi)\lambda^*\omega)},\\
I_s^* &= \frac{\phi I_u^*}{F},\\
C^* &= \frac{\epsilon I_u^*}{E},\\
R^* &= \frac{H I_u^*}{FG},
\end{aligned} \tag{3.9}$$

where,

$$\begin{aligned}
&E = \mu + \zeta, \quad F = \gamma + \mu, \quad G = \alpha + \mu,\\
&H = \delta F + \gamma\phi, \quad J = \omega + \mu,\\
&K = \sigma + \mu, \quad L = F + \phi\eta, \quad M = \epsilon + \phi + \delta + \mu,\\
&Z = EFG + \phi EG + \epsilon FG + EH,\\
&P = GF(\lambda^* + J)((1-\varphi)\lambda + K) - FG\sigma\omega.
\end{aligned}$$

We substitute $S^*, V^*, I_u^*, I_s^*, C^*$ and R^* in $\lambda^* = \frac{\beta(I_u^* + \eta I_s^*)}{N}$ which simplifies to the polynomial,

$$\lambda^* f(\lambda^*) = \lambda^* \left(h_5\lambda^{*4} + h_4\lambda^{*3} + h_3\lambda^{*2} + h_2\lambda^* + h_1\right) = 0, \tag{3.10}$$

where

$$\begin{aligned}
h_5 &= ZGF(1-\varphi)^2,\\
h_4 &= ZGF(1-\varphi)((1-\varphi)\omega + 2K + (1-\varphi)J) + EG^2F^2M(1-\varphi)^2 - EGFH\\
&\quad \alpha\omega(1-\varphi)^2 - EG^2F\beta L(1-\varphi)^2,\\
h_3 &= ZGF((1-\varphi)J + K)(\omega(1+\omega) + K) + ZGF(1-\varphi)(KJ - \sigma\omega) + EG^2F^2\\
&\quad M(1-\varphi)((1-\varphi)J + K) - EG^2F\beta L(1-\varphi)((1-\varphi)J + K) - EFGHK\\
&\quad \alpha\omega(1-\varphi) + EG^2F^2M(1-\varphi)(K+\omega)(1-R_e),\\
h_2 &= ZGF((1-\varphi)\omega + K) + EG^2F^2JM(1-\varphi)(K+\omega+1) - EG^2F^2M(1-\varphi)\\
&\quad + EGFHK\alpha\omega((1-\varphi)+1) + EG^2F^2MK(K+\omega)(1-R_e),\\
h_1 &= EG^2F^2M(K+\omega)(JK - \sigma\omega) + EG^2F\beta L(K + (1-\varphi)\omega)(JK - \sigma\omega).
\end{aligned}$$

From the polynomial (3.10), one of the solution is $\lambda^* = 0$ corresponding to the disease free equilibrium point. The other solution $h_5\lambda^{*4} + h_4\lambda^{*3} + h_3\lambda^{*2} + h_2\lambda^* + h_1 = 0$ corresponds the situation where the disease persists. The polynomial (3.10) has at least one positive root if $h_2 < 0$ and $h_3 < 0$. This implies that there exists at least one endemic equilibrium point. Hence, the existence of the endemic equilibrium point will be governed by the following theorem. The model system (2.1) has at least one endemic equilibrium point if $h_2 < 0$ and $h_3 < 0$. Then the theorem below gives a condition for the existence of the endemic equilibrium point for HPV model with screening and vaccination. The endemic equilibrium point of HPV model with screening and vaccination exists if and only if $R_e > 1$.

4 Numerical Simulations

Numerical simulations of the model are carried out using a set of reasonable parameter values given in Table 1. Some parameter values were obtained from different literatures and other were estimated. We simulate the model system by using ODE solver coded in Matlab program language by using the parameter values shown in Table 1 and the following initial conditions; $S(0) = 600000, V(0) = 350000, I_u(0) = 200000, I_s = 120000, C(0) = 50000$ and $R(0) = 105000$.

Table 1: Parameter values used in numerical simulations

Parameter	value$(\text{yr})^{-1}$	Source
Π	700	Estimated
β	0.8	[15]
φ	0.7	Estimated
μ	0.1	[10]
ω	0.4	[10]
α	0.2	Estimated
γ	0.3	[17]
δ	0.2	Estimated
ϕ	0.8	Estimated
η	0.2	Estimated
ϵ	0.05	Estimated
σ	0.2	Estimated
ζ	0.03	Estimated

4.1 Numerical results in the presence of screening and vaccination

Here we show the trend of the state variables of the modified HPV model. Due to the presence of interventions, the number of susceptible individuals decreases implying that,

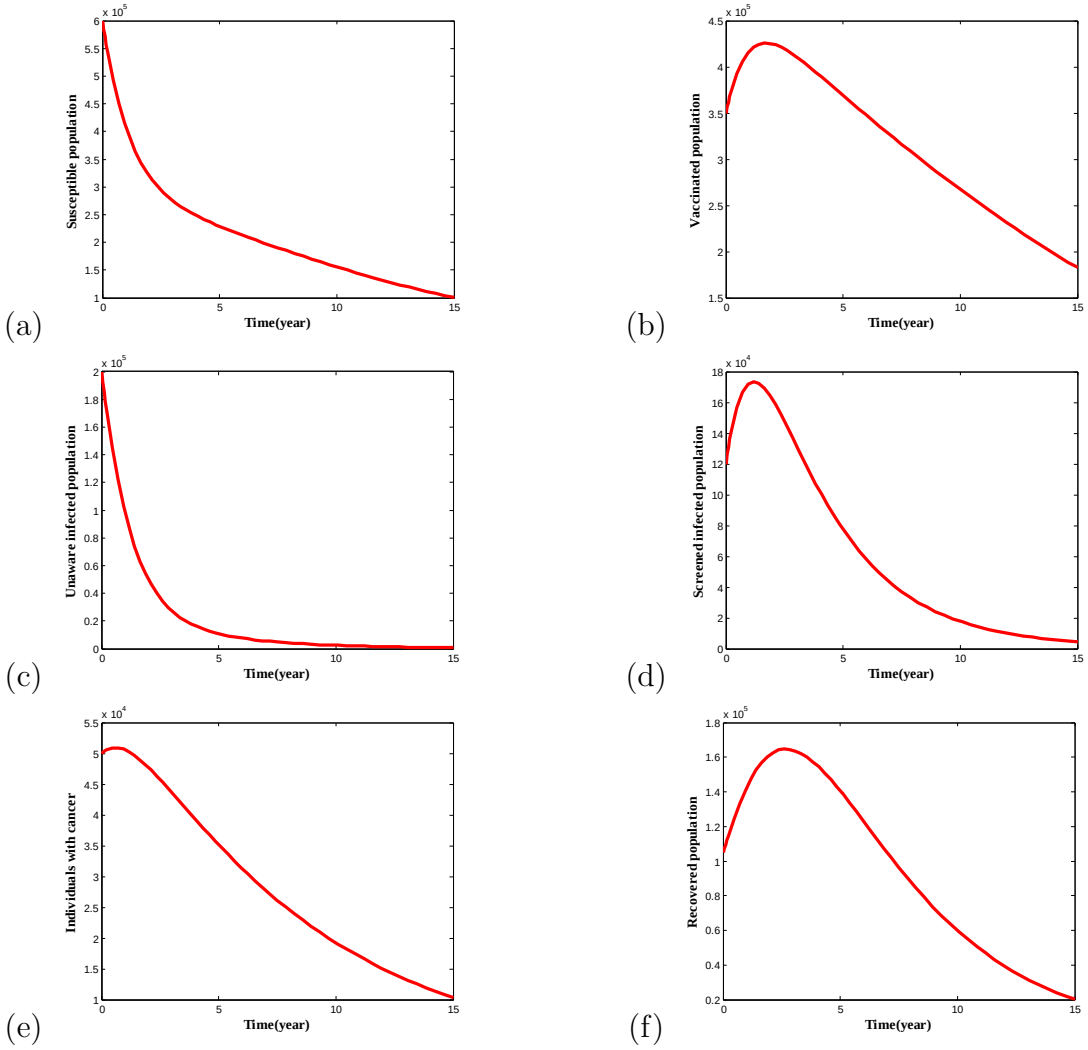


Figure 2: Simulation results showing the trends of the state variables of the HPV model with screening and vaccination for (a) Susceptible population, (b)Vaccinated population, (c) Unaware Infected, (d) Screened infected , (e)Individuals with cancer and (f) Recovered population of the model system (2.1) where $R_0 = 2.28571$ and $R_e = 0.584348$

most of the susceptible individuals are vaccinated as in Figure 2(a). The vaccinated individuals increase up to the equilibrium point and start to decrease as shown in Figure 2(b). Similarly, Figure 2(c) shows that, the number of unaware infected individuals decreases. This happens because unaware infected individuals become aware after screening. The screened infected population initially increases and then starts to diminish after the equilibrium point. This is because many people from screened class recover through treatment as shown in Figure 2(d). Also, Figure 2(e) indicates that, the number of individuals with cancer decreases and this may be due to disease induced death. Furthermore, the number of recovered individuals increases because there are two ways of recovering, through immune system or treatment as shown in Figure 2(f). With $R_e = 0.584348$, implies that, screening and vaccination can reduce the transmission of the disease in the population when $R_e < 1$.

4.2 Variation of population under different vaccination rates

In Figures 3,we show the role of vaccination in reducing HPV infection in the population. Figure 3(a) shows that, when the vaccination rate, ω increases the susceptible population decreases rapidly. This implies that most of the susceptible individuals become vaccinated. Initially, when the value of ω increases, the vaccination population increases with steep slope as shown in Figure 3(b). We noted that in Figure 3(c), the number of unaware infected individuals decreases when the value of ω increases because a few individuals get infected. Similarly, Figure 3(d) shows that, the number of individuals with cancer decreases as ω increases. This implies that only a few unaware infected individuals will progress to cancer. Generally, as vaccination rate increases the number of unaware infected individuals as well as individuals with cancer decreases.

4.3 Variation of population under different screening rates

Figure 4, illustrates the importance of screening in reducing the incidence of HPV infection in the population. The number of unaware infected decreases fast as shown in Figure 4(a), when the screening rate, ϕ increases. This means that unaware infected individuals become aware of their infectious status after screening. In Figure 4(b), the number of screened individuals initially increases rapidly as ϕ increases and then starts to decrease. This implies that most of the screened individuals recovered through treatment. Figure 4(c) indicates that the number of individuals with cancer decreases when ϕ increases. This happens because only a few of unaware infected individuals progress to the cancer. The recovered population initially increases as ϕ increases as shown in Figure 4(d). This implying that screening infected population recover through treatment and unaware infected population recover naturally (by body immune system). Finally, we conclude that, when the screening rate increases the population of unaware infected individuals and individuals with cancer decreases, which implies that, the chance of the disease to persist also decreases.

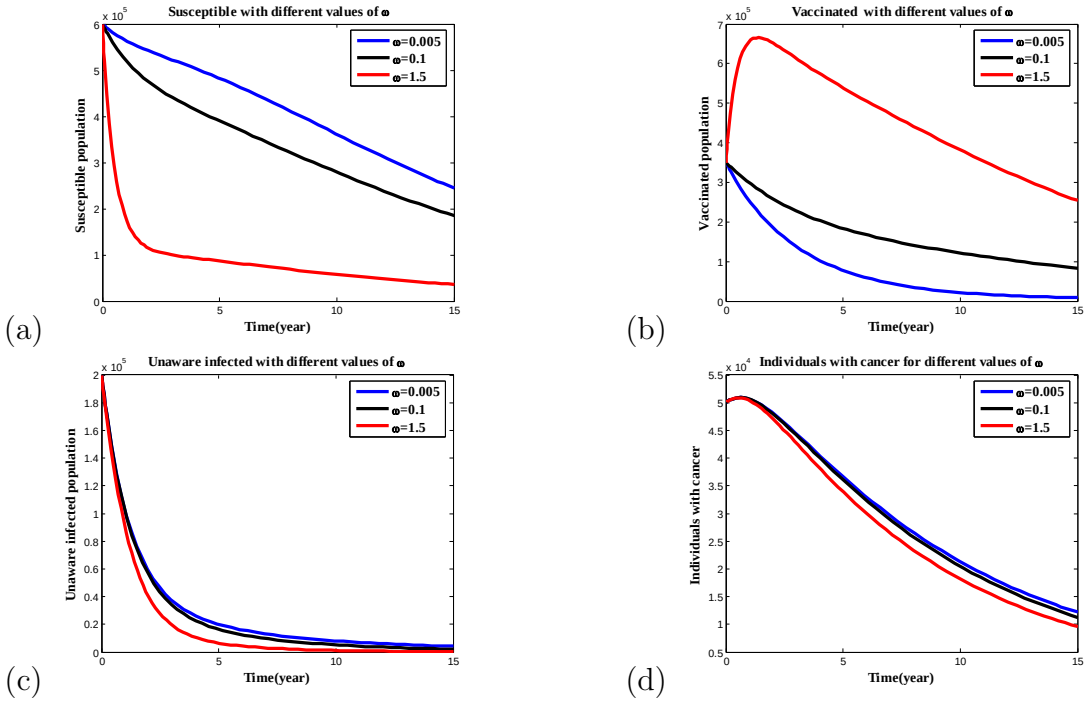


Figure 3: Simulation results showing the effect of varying vaccination rate (ω) on (a)Susceptible population, (b)Vaccinated population (c)Unaware infected and (d)Individual with cancer

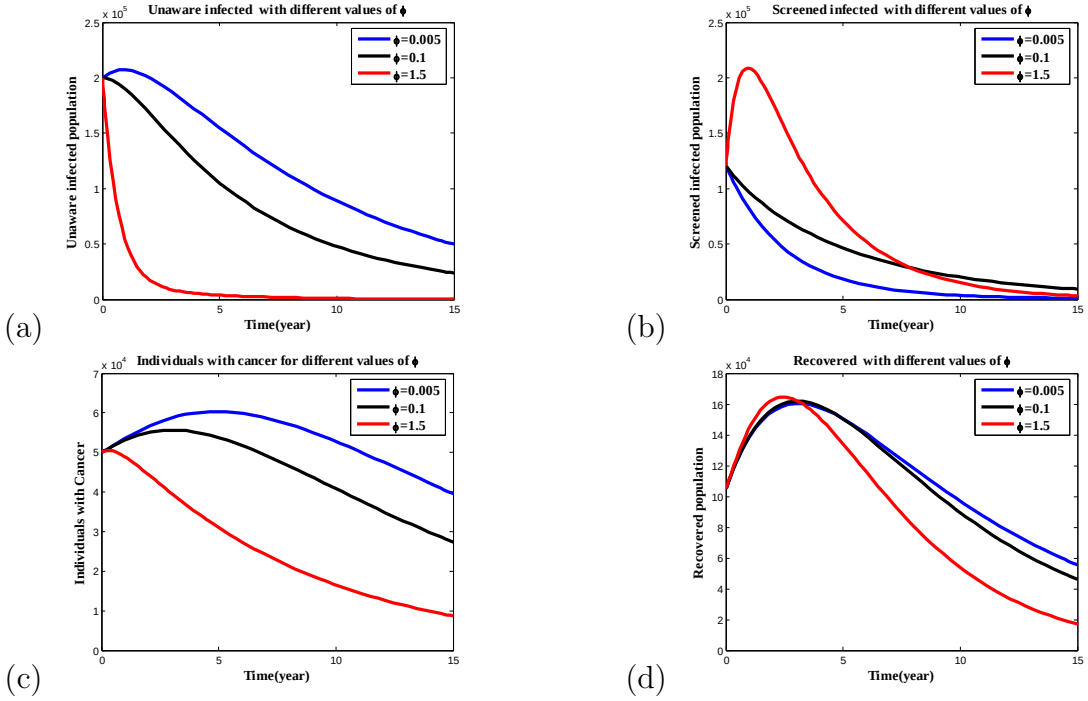


Figure 4: Simulation results showing the effect of varying screening rate (ϕ) on (a)Unaware infected, (b)Screened infected, (c)Individual with cancer and (d)Recovered population

4.4 Sensitivity Analysis

We perform sensitivity analysis in order to determine the relative importance of model parameters on disease transmission. The analysis will enable us to find out parameters that have high impact on the effective reproduction number and which should be targeted by intervention strategies. We perform sensitivity analysis by calculating the sensitivity indices of the effective reproduction number R_e in order to determine whether HPV can be spread in the population or not. These indices tell us how crucial each parameter is on the transmission of the HPV. To investigate which parameters in the model system (2.1) have high impact on the R_e, we apply the approach presented by [11].

Definition 1 *The normalized forward sensitivity index of a variable, τ that depends differentiable on index on a parameter p is defined as* $r_\tau^p = \dfrac{\partial p}{\partial \tau} \times \dfrac{\tau}{p}$.

We derive analytical expression for the sensitivity of R_e as $r_\tau^{R_e} = \dfrac{\partial R_e}{\partial \tau} \times \dfrac{\tau}{R_e}$, where τ denoting the parameter. We compute the sensitive indices of the model system (2.1) for each parameter involves in R_e. Then we use parameter values displayed in the Table 1 to get numerical value. The sensitivity indices results of R_e are given in Table 2 and are arranged from the highest sensitivity value to the lowest value. The indices with positive signs show that the value of R_e increases when the corresponding parameters are increased and those having negative signs indicate that the value of R_e decreases when the parameters are increased.

Table 2: Numerical values of sensitivity indices of R_e

Parameter symbol	Sensitivity Index
β	+1.0000000
φ	-0.6666670
ω	-0.4173910
ϕ	-0.4099380
η	+0.2857140
γ	-0.2142860
σ	+0.1904760
δ	-0.1739130
μ	-0.0631470
ϵ	-0.0434783

5 Discussion

In the present paper we have studied the impacts of screening and vaccination as control strategies against the transmission dynamics of HPV infection. Vaccination takes place when screened individuals are still susceptible to HPV, and those who are diagnosed

to have been infected undergo treatment. The reproduction numbers for vaccination and screening were derived and the usefulness of the strategies was assessed numerically by using some estimated parameters. The model analysis showed that there exists a domain where the model is epidemiologically and mathematically well-posed. The threshold parameter that governs the disease transmission was computed by the next generation operator approach as described by [12]. Then the model was analysed qualitatively for the existence and stability of disease free and endemic equilibrium. It was proved that the disease free equilibrium is locally asymptotically stable under certain conditions.

The model we developed here is not fully realistic, but we believe that it can capture some relevant properties also valid in more complex communities. For instance, to make the model more realistic the community can be extended to incorporate individuals of different types assuming that HPV transmission depends on the type of individuals. For example, it would be interesting to investigate the performance of the two strategies (screening and vaccination) in the community made up of individuals of varying infectivity and susceptibility to the disease.

Although eradication of HPV infection remain a challenge especially in developing countries, but from results of this study we recommend that, the Government should introduce education programmes on the importance of voluntary and routinely screening on HPV infection. There is a need to use vaccines with strong efficacy in order to reduce the transmission of HPV infection in the community, since vaccination has significant impact on the reduction of the disease, but it depends on the vaccine efficacy. Also, there is need to increase the number of hospitals to deal with HPV infection as well as cancers to ensure that, many people have access to the facilities, because HPV infection in long run results into different types of human cancers which pose serious health problem. Morever, the future work should consider, a cost effectiveness analysis of screening and vaccination against HPV transmission dynamics in the model or an investigation on the impact of public health education programmes against HPV transmission dynamics.

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