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The Effect of Screening on Multiple Sexual Partners of the Dynamics of Human Papillomavirus Transmission

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Abstract
Human Papillomavirus (HPV) is an age long DNA virus from the papillomavirus family. It is a casual virus for deadly diseases, prominent among them is the cervical cancer. In this paper, the effectiveness of screening in curtailing the spread of HPV is accessed using mathematical model. Using the next generation method, the basic reproduction number R_0 is computed in terms of the model parameters. The disease free equilibrium (DFE) is obtained and found to be locally and globally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$. The stability of endemic equilibrium is examined using centre manifold theorem and showed to exhibit forward bifurcation when $R_0 > 1$ under certain condition. Numerical simulations are carried out to show how screening controls the spread of HPV disease when there are multiple sexual partners and the result indicates that effective screening must be accompanied by counseling for faithfulness to one sexual partner.

Keywords: Human Papillomavirus, Screening, Disease free equilibrium, Multiple sexual partners

1.0 Introduction

Human Papillomavirus (HPV) is a DNA virus from the papillomavirus family that is infectious to humans. It is the most common sexually transmitted infection that nearly all sexually active men and women contract at some point in their lives. HPV can cause normal cells in the skin to turn abnormal and form productive infections only in keratinocytes of the skin or mucous membrane like any other papillomavirus. Most HPV are asymptomatic and will cause no physical symptoms however, in some people asymptomatic infections will become clinical and may cause anogenital warts, squamous cell papilomas or cancers of the cervix, vulva, vagina, penis, oropharynx and anus (CDC, 2008 and Capra *et al.,* 2008). In particular, 70% of cervical cancers are caused by HPV16 and HPV18 (Schiffman *et al.,* 2007).

There are more than 100 types of HPV that have been identified but most of them are harmless while others can cause infection (Bergot *et al*., 2011). At least 30 types of it can cause infection in the genital areas and are spread from person to person through skin-to-skin sexual contact; vaginal, anal, or oral sex with someone who has the virus (Barnabas *et al*., 2006). It can be transmitted even when an infected person has no signs and symptoms. The resulting disease includes anogenital warts, respiratory papillomatosis, and cancers of the cervix, vulva, vagina, anus and the penis, as well as cancers of the head and neck.

Cervical cancer is the most common HPV-related disease and the second cause of death in women (after breast cancer). It accounts for 10% of all cancers in women. Progression to malignancy after acquisition of HPV usually takes at least 10 years. In 90% of cases the body immune system clears HPV within two years; this period is referred as the latency period.

No antiviral drug has been developed for HPV and detection has largely relied on the recommended yearly Pap smear for women of reproductive age groups, which locates cellular abnormalities that indicate that HPV may be present (Llamazares and Smith, 2008). This is mostly for cervical cancer but no routine screening tests for HPV on the vulva, vagina, anus and the penis, as well as cancers of the head and neck.

Several researchers have developed interest in HPV in order to understand and explain the dynamics and spread of the disease. Thus, many mathematical models as well as methods of analyzing them were proposed. The work by Lee and Tamaru (2012) studied the impact of treatment on African American women in the United States. Brown and White (2009) and Ribassin-Majed *et al.*(2012) considered the possibility of vaccination against the HPV, taking into consideration possible waning immunity and sex-specific immunization among males in heterosexual populations. Shaban and Mofi (2014) considered screening and vaccination as effective means of curtailing the infection of HPV in homogeneous population while Froelich *et al.*(2002) examined the impact of screening males in heterogeneous population. None of these studied the effect of multiple sexual partners on the dynamics of HPV transmission.

Although many mathematical models have considered the effect of screening, this paper will study the effect of screening considering the number of sexual partners of initially infected and chronically infected individuals. This is done using the ideal by Froelich *et al.*(2002).

The paper is organized as follows. Section 2 is the model formulation. Section 3 is analysis of the model. Numerical simulation is carried out in section 4. Finally conclusion is presented in section 5.

2.0 Model Formulation

Let $N(t)$ be the total population of size at time t. The population $N(t)$ is divided into four classes namely; Susceptible $S(t)$, Initially infected, $I(t)$, Chronically infected, $C(t)$ and Recovered R(t) individuals with natural mortality rate μ in all classes and d as the chronically infected induced death rate. We assumed that susceptible individuals are recruited at the rate μN and become infected through sexual contacts with the infected and chronically individuals $I(t)$ and $C(t)$ at rate λ_t, where

$$\lambda_t = \frac{\sigma(\beta_1 I + \beta_2 C)}{N}$$

σ represents the mean number of sexual partners of the individuals in $I(t)$ and $C(t)$ while β_1 and β_2 are the respective sexual contact rates of the $I(t)$ and $C(t)$ classes. Some initially infected class $I(t)$ progress to chronically class after going through screening at the rate $q\theta$. This may be because of late screening and poor body immune system to recover naturally, while some recovers naturally through body immune system at rate $(1-q)\theta$ where $0 < q < 1$. In addition, some initially infected class $I(t)$ progress to chronically infected class without screening at the rate $p\tau$, while some recovers naturally through body immune system or by treatment at a rate $(1-p)\tau$ where $0 < p < 1$. Furthermore, the recovered individuals become susceptible again after waning immunity at a rate α and the chronically infected people may recover with the help of treatment after screening and successful diagnosis at a rate k.

Based on the above assumptions, the governing system of differential equations for the spread of the disease is given by

$$\frac{dS}{dt} = \mu N - \sigma\left(\frac{\beta_1 SI + \beta_2 SC}{N}\right) - \mu S + \alpha R \qquad (1)$$

$$\frac{dI}{dt} = \sigma\left(\frac{\beta_1 SI + \beta_2 SC}{N}\right) - (\mu + \tau + \theta)I \qquad (2)$$

$$\frac{dC}{dt} = p\tau I - (\mu + k + d)C + \theta q I \qquad (3)$$

$$\frac{dR}{dt} = (1-p)\tau I - (\mu + \alpha)R + (1-q)\theta I + kC \qquad (4)$$

with non-negative initial conditions

$$S(0) > 0,\ I(0) \geq 0,\quad C(0) \geq 0,\ \ and\ \ R(0) \geq 0.$$

3.0 Analysis of the Model

3.0.1 Positivity and Boundedness of the Solutions

Theorem 3.1. For all time $t \geq 0$, all the solutions of the system (1) – (4) are restricted in the compact subset $\Omega = \{(S, I, C, R) \in \mathbb{R}^4_+ : N = (S(t) + I(t) + C(t) + R(t)) \leq K)\}$

Proof. Let $(S(t), I(t), C(t), R(t))$ be any solution with positive initial conditions.

We have $N = S(t) + I(t) + C(t) + R(t)$. The time derivative of $N(t)$ along the solution of the system (1) – (4) is $\frac{dN}{dt} \leq 0$. Applying theorem of differential inequality (Birkhof and Rota, 1982), we get $N \leq K$ a constant. Then $0 \leq N \leq K$ as $t \to \infty$.

Thus, it has been proved that all the solutions of (1) – (4) are bounded in the interval $[0, \infty)$. Therefore, the model can be considered as being epidemiologically and mathematically well posed.

3.0.2 Disease - Free Equilibrium (DFE)

The disease – free equilibrium is the equilibrium when there is no disease in the population. At equilibrium point, $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dR}{dt} = 0$, we have the following system of equations to be solved simultaneously for S, I, C and R.

$$\mu N - \sigma\left(\frac{\beta_1 SI + \beta_2 SC}{N}\right) - \mu S + \alpha R = 0 \qquad (5)$$

$$\sigma\left(\frac{\beta_1 SI+\beta_2 SC}{N}\right) - I(\mu+\tau+\theta) = 0 \quad (6)$$

$$p\tau I - (\mu+k+d)C + \theta qI = 0 \quad (7)$$

$$(1-p)\tau - (\mu+\alpha)R + (1-q)\theta I + kC = 0 \quad (8)$$

We have

$$\mu N - \mu S = 0$$

from which we obtain

$S = N$, provided $\mu \neq 0$

Thus, the disease – free equilibrium E_0 is

$$E_0 = (N, 0,0,0)$$

3.0.3 Stability of Disease Free Equilibrium

We shall compute the basic reproduction number R_0 using the next– generation method. The basic reproduction number is a threshold quantity used to study the prevalence of an infectious disease in epidemiological model. According to Diekmann *et al.*, (1990), the basic reproduction number R_0 is the spectral radius (i.e the dominant eigenvalue) of the next generation matrix. It is given as

$$R_0 = \rho(GU^{-1})$$

where $\rho(GU^{-1})$ is the spectral radius of the matrix GU^{-1} given as

$$GU^{-1} = \left[\frac{\partial F_i(E_0)}{\partial x_j}\right]\left[\frac{\partial V_i(E_0)}{\partial x_j}\right]^{-1}$$

F_i is rate of appearance of new infection in compartment i

V_i is the transfer of individuals in and out of compartment i by any other means and

E_0 is the disease free equilibrium.

Using the next generation method, the system of differential equation $(1)-(4)$ are rearranged in the order of the infected compartments first, then the uninfected compartment. We have two infected compartments namely, I and C whereas S and R as uninfected compartments.

$$\frac{dI}{dt} = \sigma\left(\frac{\beta_1 SI+\beta_2 SC}{N}\right) - I(\mu+\tau+\theta) \quad (9)$$

$$\frac{dC}{dt} = p\tau I - (\mu+k+d)C + \theta qI \quad (10)$$

$$\frac{dS}{dt} = \mu N - \sigma\left(\frac{\beta_1 SI+\beta_2 SC}{N}\right) - \mu S + \alpha R \quad (11)$$

$$\frac{dR}{dt} = (1-p)\tau I - (\mu+\alpha)R + (1-q)\theta I + kC \quad (12)$$

from which F, V, G, U, and GU^{-1} are given as

$$F = \begin{bmatrix} \sigma\left(\frac{\beta_1 SI+\beta_2 SC}{N}\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} I(\mu+\tau)+\theta I \\ -p\tau I + (\mu+k+d)c - \theta qI \\ -\mu N + \sigma\left(\frac{\beta_1 SI+\beta_2 SC}{N}\right) + \mu S - \alpha R \\ -(1-p)\tau I + (\mu+\alpha)R - (1-q)\theta I - kC \end{bmatrix}$$

$$G = \begin{bmatrix} \frac{\partial F_1(E_0)}{\partial I} & \frac{\partial F_1(E_0)}{\partial c} \\ \frac{\partial F_2(E_0)}{\partial I} & \frac{\partial F_2(E_0)}{\partial c} \end{bmatrix} = \begin{bmatrix} \sigma\beta_1 & \sigma\beta_2 \\ 0 & 0 \end{bmatrix}$$

$$U = \begin{bmatrix} \frac{\partial V_1(E_0)}{\partial I} & \frac{\partial V_1(E_0)}{\partial c} \\ \frac{\partial V_2(E_0)}{\partial I} & \frac{\partial V_2(E_0)}{\partial c} \end{bmatrix} = \begin{bmatrix} \mu + \tau + \theta & 0 \\ -p\tau - \theta q & \mu + k + d \end{bmatrix}$$

and

$$GU^{-1} = \begin{bmatrix} \frac{\sigma\beta_1}{\mu+\tau+\theta} + \frac{\sigma\beta_2(p\tau+\theta q)}{(\mu+\tau+\theta)(\mu+k+d)} & \frac{\sigma\beta_2}{\mu+k+d} \\ 0 & 0 \end{bmatrix}$$

We find the eigenvalue of GU^{-1} as

$$|GU^{-1} - \lambda \mathrm{I}| = 0$$

This gives

$$\lambda_1 = 0 \ or \ \lambda_2 = \frac{\sigma\beta_1(\mu+k+d)+\sigma\beta_2(p\tau+\theta q)}{(\mu+\tau+\theta)(\mu+k+d)}$$

Since the spectral radius of GU^{-1} is given by

$R_0 = max[|\lambda_1|, |\lambda_2|]$,

we have

$$R_0 = \frac{\sigma\beta_1(\mu+d+k)+\sigma\beta_2(p\tau+\theta q)}{(\mu+\tau+\theta)(\mu+k+d)} \qquad (13)$$

If $R_0 < 1$, the disease dies out, otherwise the disease will be maintained in the population. From the equation (13), we have R_0 increases as the number of sexual partners of $I(t)$ and $C(t)$ increase. This implies that R_0 can be kept at minimum if there is a restriction on the number of sexual partners of $I(t)$ and $C(t)$.

Theorem 2: The disease – free equilibrium of the system of ODE $(9) - (12)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

The proof of theorem 2 is done by linearization method. The Jacobian matrix associated with the system (9)-(12) at the DFE $E_0 = (0,0,N,0)$ is

$$J(E_0) = \begin{bmatrix} \sigma\beta_1 - (\mu+\tau) - \theta & \sigma\beta_2 & 0 & 0 \\ p\tau + \theta q & -(\mu+k+d) & 0 & 0 \\ -\sigma\beta_1 & -\sigma\beta_2 & -\mu & \alpha \\ (1-p)\tau + (1-q)\theta & k & 0 & -(\mu+\alpha) \end{bmatrix}$$

and the characteristics of equation corresponding to $J(E_0)$ is given by

$$\rho(\lambda) = (-\mu - \lambda)(-(\mu+\alpha) - \lambda)[\lambda^2 + A\lambda + \mathrm{B}] = 0$$

where

$$A = -\big[\big(\sigma\beta_1 - (\mu+\tau+\theta)\big) - (\mu+k+d)\big]$$

$$B = -\big[\sigma\beta_2(p\tau+\theta q) + (\mu+k+d)\big(\sigma\beta_1 - (\mu+\tau+\theta)\big)\big]$$

Using Routh–Hurwitz criteria, E_0 is locally asymptotically stable if $A > 0$ and $B > 0$.

We have

$$A = -\big[\big(\sigma\beta_1 - (\mu+\tau+\theta)\big) - (\mu+k+d)\big] > 0$$

$$B = -\left[\sigma\beta_2(p\tau + \theta q) + (\mu + k + d)\left(\sigma\beta_1 - (\mu + \tau + \theta)\right)\right] > 0$$

This implies that

$$\sigma\beta_2(p\tau + \theta q) + (\mu + k + d)\left(\sigma\beta_1 - (\mu + \tau + \theta)\right) < 0 \text{ or } \left(\sigma\beta_1 - (\mu + \tau + \theta)\right) - (\mu + k + d) < 0$$

and gives

$$\frac{\sigma\beta_1(\mu+k+d)+\sigma\beta_2(p\tau+\theta q)}{(\mu+\tau+\theta)(\mu+k+d)} < 1 \quad \text{or} \quad \frac{\sigma\beta_1}{(2\mu+\tau+\theta+k+d)} < 1 \qquad (*)$$

Comparing (*) with (13), we have $R_0 < 1$.This proves the theorem 2.

3.0.4 Global Stability of the Disease-Free Equilibrium

Using the approach by Castillo – Chavez *et al.* (2002), the system of equations (9) - (12) can be rewritten as

$$\frac{\mathrm{dx}}{\mathrm{dt}} = \mathrm{F}(\mathbf{x}, \mathbf{I}), \qquad (14)$$

$$\frac{\mathrm{dI}}{\mathrm{dt}} = \mathrm{G}(\mathbf{x}, \mathbf{I}), \qquad \mathrm{G}(\mathbf{x}, 0) = 0 \qquad (15)$$

Where $\mathbf{x} \in \mathbb{R}^2 = (\mathrm{S}, \mathrm{R})$ denotes the number of uninfected individuals (susceptible and recovered) and $\mathbf{I} \in \mathbb{R}^2 = (\mathrm{I}, \mathrm{C})$ denotes the number of infected individuals (infected and chronically infected). $\mathrm{E}_0 = (0,0,\mathrm{N},0)$ as the disease free equilibrium of the system (9) – (12).

The condition for global stability for E_0 is given by

(H1) For $\frac{\mathrm{dx}}{\mathrm{dt}} = \mathrm{F}(\mathbf{x}, \mathbf{0})$, x^* is globally asymptotically stable,

(H2) $\mathrm{G}(\mathbf{x}, \mathbf{I}) = W\boldsymbol{I} - \tilde{G}(\mathbf{x}, \boldsymbol{I}), \quad \tilde{G}(\mathbf{x}, \boldsymbol{I}) \geq 0 \quad for \quad (\mathbf{x}, \boldsymbol{I}) \in \Omega \qquad (16)$

where $W = D_I G(\mathbf{x}, 0)$ is an M-matrix (i.e. the off diagonal elements of W are nonnegative) and Ω is the region where the system of equations of the model makes epidemiological meaningful.

If the system (9)-(12) satisfies the above condition then the following theorem holds:

Theorem 3: The disease-free equilibrium $\mathrm{E}_0 = (0,0,\mathrm{N},0)$ is globally asymptotically stable if $R_0 < 1$ and that condition (16) is satisfied.

From (15) and (16), we have

$$\tilde{G}(\mathbf{x}, \boldsymbol{I}) = (G - U)\boldsymbol{I} - \frac{d\boldsymbol{I}}{dt}, \quad \text{where } (G - U) = W$$

so, we have

$$(G - U) = \begin{bmatrix} \sigma\beta_1 - (\mu + \tau + \theta) & \sigma\beta_2 \\ p\tau + \theta q & -(\mu + k + d) \end{bmatrix}, \quad \tilde{G}(\mathbf{x}, \boldsymbol{I}) = \begin{bmatrix} \sigma\beta_1 I\left(1 - S/N\right) + \sigma\beta_2 C\left(1 - S/N\right) \\ 0 \end{bmatrix}$$

since $S \leq N$, it is clear that

$$\tilde{G}(\mathbf{x}, \boldsymbol{I}) \geq 0$$

This implies that

$$\frac{d\boldsymbol{I}}{dt} \leq (G - U)\boldsymbol{I} \qquad (17)$$

All the eigenvalues of $(G - U)$ has negative real parts if $\frac{\sigma\beta_1}{(2\mu+\tau+\theta+k+d)} < 1$ and $R_0 < 1$. It follows that for $R_0 < 1$, the inequality (17) is stable and it results as $t \to \infty$, $(I, C) \to (0,0)$. Then, the DFE $\mathrm{E}_0 = (0,0,\mathrm{N},0)$ is globally asymptotically stable if $R_0 < 1$.

3.0.5 Local Stability of Endemic Equilibrium

The local stability of endemic equilibrium is determined by finding the eigenvalues of the Jacobian Matrix evaluated at the endemic equilibrium. Sometimes, this approach can be mathematically complicated. Here, we recourse to the approach of centre manifold theory described by Castillo - Chavez and Song (2004) to investigate the stability of endemic equilibrium. Centre Manifold theory is used to investigate the existence of backward and forward bifurcation at $R_0 = 1$ (Arion *et al.*(2003); Huang *et a;.*,(1992) and Xue and Wang, (2012)).When the bifurcation is forward, it implies that disease free equilibrium is locally asymptotically stable for $R_0 < 1$ and there is no disease in the population and also endemic equilibrium is locally asymptotically stable for $R_0 > 1$.Backward bifurcation occurs when the endemic equilibrium exists for $R_0 < 1$ and disease free equilibrium may exists when $R_0 > 1$.

Theorem 3: *Centre manifold theory (Castillo – Chavez and Song, 2004).*

Consider a general system of ODEs with the parameter β:

$$\frac{dx}{dt} = \boldsymbol{f}(\boldsymbol{x}, \beta) \qquad (18)$$

$\boldsymbol{f}: R \to R^n$ *and* $\boldsymbol{f} \in C^2(R^2 \times R)$

Where 0 *is an equilibrium point for the system (18) for all values of the parameter* β, *that is* $\boldsymbol{f}(0, \beta) \equiv 0$ *for all* β *and*

$$A = D_*\boldsymbol{f}(0,0) = \left[\frac{df_i}{dx_j}(0,0)\right]$$

is the linearization point 0 *with* β *evaluate at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts. Matrix A has a non negative right eigenvector* $\boldsymbol{w} = (w_1, w_2, w_3, w_4)$ *and a left eigenvector* $\boldsymbol{v} = (v_1, v_2, v_3, v_4)$ *corresponding to the zero eigenvalue.*

Let f_k *be the* k^{th} *component of* f *and*

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(0,0)$$

the local dynamics of the system (13) around the equilibrium point 0 *is totally determined by the signs of* a *and* b.

1. $a > 0$, $b > 0$ *when* $\beta < 0$ *with* $|\beta| \ll 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < \beta \ll 1$, 0 *is unstable and there exists a negative and locally asymptotically stable equilibrium.*
2. $a < 0$, $b < 0$, *when* $\beta < 0$ *with* $|\beta| \ll 1$, 0 *unstable; when* $0 < \beta \ll 1$, *asymptotically stable, and there exists a positive unstable equilibrium;*
3. $a > 0$, $b < 0$, *when* $\beta < 0$ *with* $|\beta| \ll 1$, 0 *unstable; and there exists a locally asymptotically stable negative equilibrium; when* $0 < \beta \ll 1$, 0 *is stable and a positive unstable equilibrium appears;*
4. $a < 0$, $b > 0$, *when* $\beta < 0$ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Corresponding to a negative equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a < 0$ *and* $b > 0$, *then a forward bifurcation occurs at* $\beta = 0$.

Applying the theorem 3 involves the following change of variables; Let

$$I = x_1, \quad C = x_2, \quad S = x_3, \quad R = x_4$$

Let $\boldsymbol{x} = (x_1, x_2, x_3, x_4)^T$ be the vector written so that the model can be re-written in the form $\frac{dx}{dt} = \boldsymbol{f}(x)$, where $\boldsymbol{f} = (f_1, f_2, f_3, f_4)^T$ as follows

$$\frac{dx_1}{dt} = f_1(x) = \sigma\left[\frac{\beta_1 x_1 x_3 + \beta_2 x_2 x_3}{N}\right] - (\theta + \mu + \tau)x_1 \quad (19)$$

$$\frac{dx_2}{dt} = f_2(x) = (p\tau + \theta q)x_1 - (\mu + k + d)x_2 \quad (20)$$

$$\frac{dx_3}{dt} = f_3(x) = \pi N - \sigma\left[\frac{\beta_1 x_1 x_3 + \beta_2 x_2 x_3}{N}\right] - \mu x_3 + \alpha x_4 \quad (21)$$

$$\frac{dx_4}{dt} = f_4(x) = \big((1-p)\tau + (1-q)\theta\big)x_1 - (\mu + \alpha)x_4 + kx_2 \quad (22)$$

The Jacobian matrix of the equations $(9) - (12)$ at the disease-free equilibrium $J(E_0)$ is defined in previous section. Taking $\beta_1 = \beta$ and $\beta_2 = r\beta$, where β is chose as the bifurcation parameter and the bifurcation occurs at $R_0 = 1$, we consider the case $R_0 = 1$ and solve for the bifurcation parameter β.

We have

$$R_0 = \frac{\sigma\beta(\mu + k + d) + \sigma r\beta(p\tau + \theta q)}{(\mu + \tau + \theta)(\mu + k + d)} = 1$$

from which we obtain

$$\beta = \frac{(\mu+\tau+\theta)(\mu+k+d)}{\sigma[(\mu+k+d)+r((p\tau+\theta q))]}$$

The linearized system of the system $(9) - (12)$ with $\beta_1 = \beta$ and $\beta_2 = r\beta$ at $R_0 = 1$ has a simple zero eigenvalue. Using the Centre Manifold theory, the Jacobian matrix of $(9) - (12)$ has right eigenvector associated with the zero eigenvalue as

$$\begin{bmatrix} \sigma\beta - (\mu + \tau + \theta) & \sigma r\beta & 0 & 0 \\ p\tau + \theta q & -(\mu + d) & 0 & 0 \\ -\sigma\beta & -\sigma r\beta & -\mu & \alpha \\ (1-p)\tau + (1-q)\theta & k & 0 & -(\mu + \alpha) \end{bmatrix} \begin{bmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (21)$$

where $\boldsymbol{w} = (w_1, w_2, w_3, w_4)^T$ is the right eigenvector.

Evaluating the system in (21) gives

$$w_2 = \frac{p\tau + \theta q}{(\mu + d)} w_1, w_3 = \frac{m_1}{\alpha m_2} w_1, w_4 = \frac{m_3}{b m_2} w_1$$

Where

$$m_1 = (p\tau + \theta q)\big(k - \sigma r\beta(\mu + \alpha)\big) + (\mu + k + d)\big((1-p)\tau + (1-q)\theta\big) - \sigma\beta(\mu + \alpha),$$

$$m_2 = (\mu + k + d)(\mu + \alpha),$$

$$m_3 = \big((1-p)\tau + (1-q)\theta\big)(\mu + k + d),$$

The left eigenvector of the Jacobian $J(E_0)$ associated with the zero eigenvalue is given by $\boldsymbol{v} = (v_1, v_2, v_3, v_4)^T$. Transposing Jacobian $J(E_0)$ first and multiply by $\boldsymbol{v}$, we have

$$\begin{bmatrix} \sigma\beta - (\mu + \tau + \theta) & p\tau + \theta q & -\sigma\beta & (1-p)\tau + (1-q)\theta \\ \sigma r\beta & -(\mu + d) & -\sigma r\beta & k \\ 0 & 0 & -\mu & 0 \\ 0 & 0 & \alpha & -(\mu + \alpha) \end{bmatrix} \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

from which we get

$$v_3 = v_4 = 0, v_2 = \frac{\sigma r\beta}{\mu + d} v_1$$

Using the property $\boldsymbol{w}.\boldsymbol{v} = 1$, we obtain

$\boldsymbol{w} = \left(\frac{(\mu+k+d)^2}{\sigma r\beta(p\tau+\theta q)+(\mu+k+d)^2}, \frac{(p\tau+\theta q)(\mu+k+d)}{\sigma r\beta(p\tau+\theta q)+(\mu+k+d)^2}, \frac{m_1}{\alpha m_2}\frac{(\mu+k+d)^2}{\sigma r\beta(p\tau+\theta q)+(\mu+k+d)^2}, \frac{m_3}{bm_2}\frac{(\mu+k+d)^2}{\sigma r\beta(p\tau+\theta q)+(\mu+k+d)^2}\right)$ and

$\boldsymbol{v} = \left(1, \frac{\sigma r\beta}{\mu+k+d}, 0, 0\right)$

Computations of a and b

From the system (19) - (22), the associated non-zero partial derivative of F at DFE for v_1, v_2 are given by

$\frac{\partial^2 f_1}{\partial x_1 \partial x_3} = \frac{\sigma\beta}{N}$, $\frac{\partial^2 f_1}{\partial x_2 \partial x_3} = \frac{\sigma r\beta}{N}$,

Since $v_3 = 0$ and $v_4 = 0$

It follows that

$$a = v_1\left[w_1 w_3 \frac{\sigma\beta}{N} + w_2 w_3 \frac{\sigma r\beta}{N}\right]$$

or

$$a = [{w_1}^2 \frac{m_1}{\alpha m_2}\frac{\sigma\beta}{N} + {w_1}^2 \frac{m_1}{\alpha m_2}\frac{p\tau+\theta q}{(\mu+k+d)}\frac{\sigma r\beta}{N}].$$

This gives

$$a = [{w_1}^2 \frac{1}{\alpha m_2}\frac{\sigma\beta}{N} + {w_1}^2 \frac{1}{\alpha m_2}\frac{p\tau+\theta q}{(\mu+k+d)}\frac{\sigma r\beta}{N}]m_1$$

For b, we have

$$b = v_1\left[w_1 \frac{\partial^2 f_1}{\partial x_1 \partial\beta}(0,0) + w_2 \frac{\partial^2 f_1}{\partial x_2 \partial\beta}(0,0)\right]$$

Substituting $\beta_1 = \beta$ and $\beta_2 = r\beta$ into $\boldsymbol{f}$ and differentiating, we have

$$b = w_1\left[\frac{\sigma}{N} + \frac{p\tau+\theta q}{(\mu+k+d)}\frac{\sigma r}{N}\right] > 0.$$

Theorem 4: *The system (9) – (12) exhibits a backward bifurcation at* $R_0 = 1$ *if* $m_1 > 0$ and $a > 0$. *If* $\beta < 0$, *there exists a positive unstable endemic equilibrium point and when* β *changes from negative to positive, a positive stable endemic equilibrium point exists. Therefore, the endemic equilibrium point is locally asymptotically stable for* $R_0 > 1$ *but close to 1.*

4.0 Numerical Result

To examine the dynamics of the model numerically, the system is solved using the fourth-order Runge-Kutta method with the following values for the parameters $\mu = 0.1, \sigma = 2, \beta_1 = 0.4,\ \beta_2 = 0.05,\ \theta = 0.6, p = 0.2, q = 0.3, k = 0, \alpha = 0.2, \tau = 0.3, d = 0.03$ and initial conditions $S(0) = 120000, I(0) = 50000, C(0) = 25000, R(0) = 5000$ for the period of 15 years. The result are displayed graphically in figures $1(a) - 3(c)$. Figures $1(a) - 1(c)$ show the effect of the number of sexual partners on the initially infected, chronically infected and recovered individuals. As the number of sexual partner increases, the number of people that are initially infected with HPV and the number of people that recovered among these initially infected HPV individuals increase. Similarly, the number of people that progress to chronically infected class among those initially infected with HPV also increases. This is because in about 90% of those who are initially infected with HPV, their body's immune system is able to clear the infection within two years (Medical News Today, 2014).

Figures $2(a) - 2(c)$ show the effect of screening on multiple sexual partners of the initially infected, chronically infected and recovered individuals. As the rate of screening increases, the number of people that are initially infected with HPV and the number of people that recovered among these initially infected HPV individual decreases, while the number of people that progress to chronically infected class among those initially infected with HPV increases. This is perhaps as a result of multiple sexual partners of the initially and chronically infected individuals.

Figures $3(a) - 3(c)$ show the effect of screening on a single sexual partner of the initially infected, chronically infected and recovered individuals. As the rate of screening increases, the number of people that are initially infected with HPV decreases, while the number of people that progress to chronically infected class and the number of people that recovered among these initially infected HPV individual increases. This is because the initially and chronically infected are faithful to one sexual partner.

Therefore, in order to reduce the spread of HPV, effective screening must be accompanied by counselling for faithfulness to one sexual partner.

5.0 CONCLUSION

A variate of the model by Froelich *et al.*(2002) is proposed to incorprorate mean numbers of sexual partners for initially and chronically infected HPV infectives in a homogeneously varying population. Intervention such as screening was considered in model in order to know the effect on the number of sexual partners of the initially and chronically infected HPV individuals. The model is investigated to exhibit local and global asymptotic stabilty at DFE provided $R_0 < 1$. While, the stability of the endemic equilibrium is examined using bifurcation analysis (centre manifold approach) and proved to be backward bifurcation based on certain conditions. Results from numerical simulations indicate that an increase in the rate of screening will reduce the number of initially infected HPV and increase the number of chronically infected and recovered HPV indivdiuals if the HPV infected indivduals stick to one sexual partner if at all they will have sexual partners.

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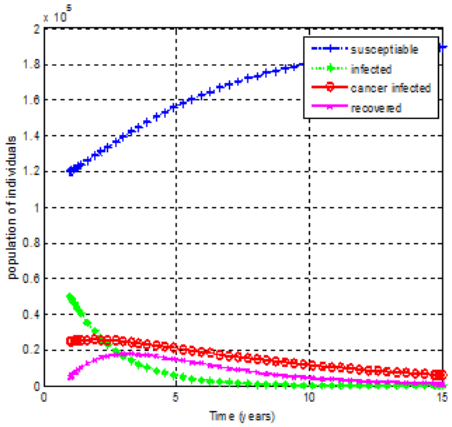

Figure 1(a). Variation of Population in different classes for $\sigma = 0, \theta = 0.15$

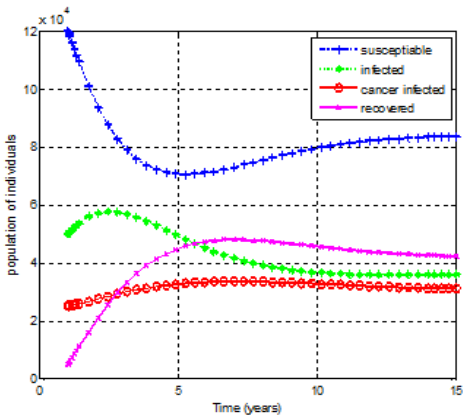

Figure 1(b). Variation of Population in different classes for $\sigma = 3, \theta = 0.15$.

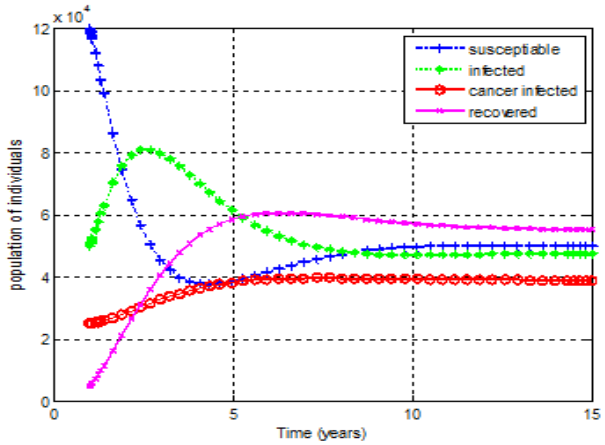

Figure 1(c). Variation of Population in different classes for $\sigma = 5, \theta = 0.15$

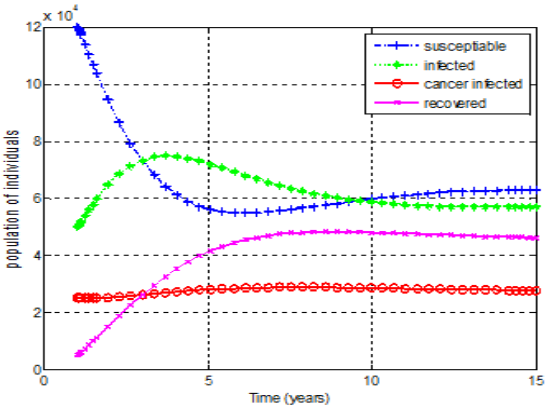

Figure 2(a). Variation of Population in different classes for $\sigma = 3, \theta = 0$.

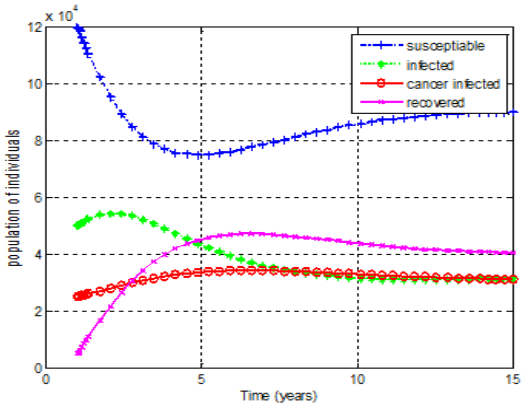

Figure 2(b). Variation of Population in different classes for $\sigma = 3, \theta = 0.2$

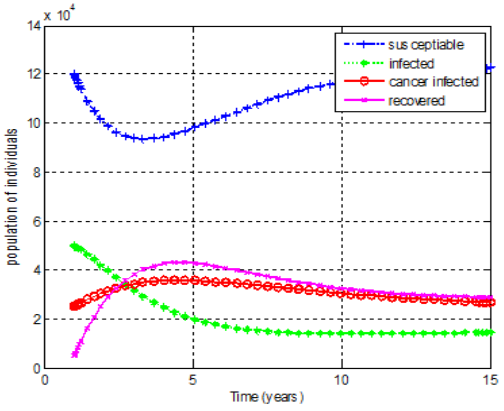

Figure 2(c). Variation of Population in different classes for $\sigma = 3, \theta = 0.5$

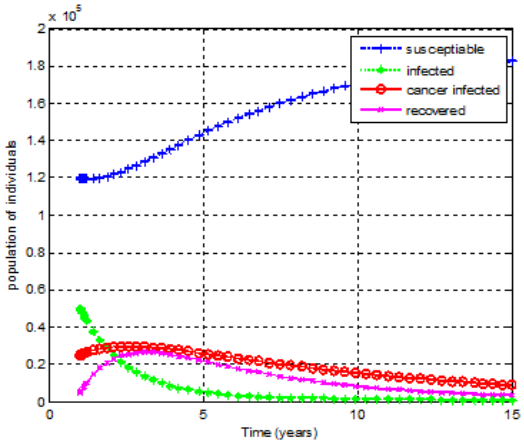

Figure 3(a). Variation of Population in different classes for $\sigma = 0, \theta = 0.15$

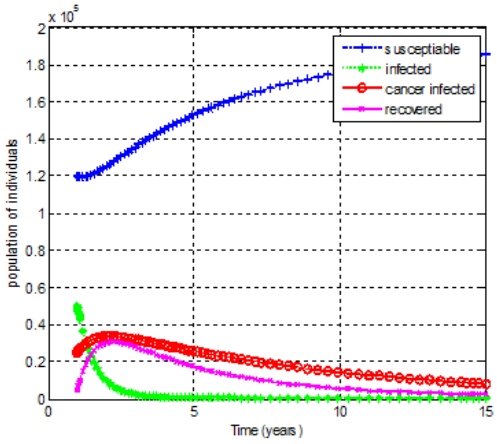

Figure 3(b). Variation of Population in different classes for $\sigma = 1, \theta = 1.5$

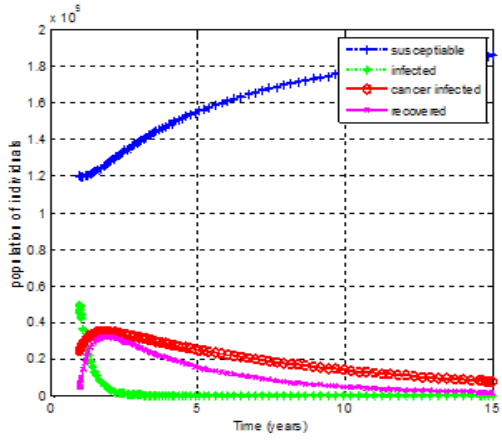

Figure 3(c). Variation of Population in different classes for $\sigma = 1, \theta = 2.5$

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