MATHEMATICAL MODEL OF CERVICAL CANCER INCORPORATING PROTECTION MEASURES AGAINST THE DISEASE

Abstract

Cervical cancer caused by human papillomavirus (HPV) has attracted more attention due to its social economic ramifications and its complex behavior. Even with the introduction of routine screening programs and vaccination, the disease prevalence remains high especially in Sub-saharan Africa. However, Cervical cancer is a major preventable public health problem. Due to the high cost of treatment, protection against the infection may be preferable in scarce resource settings. In this paper a deterministic model incorporating protection against cervical cancer infection is considered. Specifically the model considers maximum protection against the infection. The model is shown to be positively invariant as well as bounded. The endemic states are shown to exist provided that the reproduction number is greater than unity $R_0 > 1$. By use of Routh-Hurwitz criterion and suitable Lyapunov functions, the endemic states are shown to be locally and globally asymptotically stable respectively. This implies that disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrences. Numerical simulations indicate that enhanced protection against the disease lowers new incidences and hence low disease prevalence rates. Therefore, public awareness campaign efforts on protective measures against cervical cancer should be enhanced.

Keywords: Protection, cervical cancer, Human papilloma Virus, Reproduction Number.

1 Introduction

HPV is the world's most widespread sexually transmitted infection, affecting 80 percent of people worldwide at least once in their lives. Typically, the body naturally clears the infection. However, in some cases, the infection becomes persistent. When you contract HPV, it takes 10 to 15 years to develop into

cancer, but during this period, precancerous lesions may appear. Over one hundred dissimilar strains of HPV being identified and classified with HPV types 16, 18, 31 and 45 been classified as "high-risk". Approximately 85 percentage cancer of the cervix are reported to be as a result of these four strains alone. Africa, in particular, bears a significant burden, contributing 21 percent of global cervical cancer deaths in 2020, largely due to the prevalence of HIV, which compromises patients' immune systems, leaving them more vulnerable to the cancer-causing human papilloma virus (HPV). However, if prevented cervical cancer is a form of cancer that can be successfully managed [5].

There is no treatment for HPV but in most cases it disappears naturally. However, with persistent infections the high risk strains may become chronic and shed HPV virions. Cervical cancer treatment is dependent on a multiplicity of factors. Key among them include the stage of the cancer when it was initially diagnosed, age of the patient and the overall health status of the person in question. The type of treatment administered is dependent on the stage where the specific cancer manifests itself. Treatment options include but not limited to chemotherapy, surgery and radiography. Palliative care is a key plank in management and containment of cancer, especially in the assuaging of severe pains and mollifying the suffering of cancer patients [2].

According to the World Health Organization (WHO) and recent data, in 2023, cervical cancer is estimated to have caused around 660,000 new cases and 350,000 deaths globally, making it the fourth most common cancer among women worldwide, the highest rates of incidence and mortality are observed in low and middle-income countries due to limited access to HPV vaccination and screening services [11].

Numerous mathematical models have been developed to explore transmission dynamics and treatment of cervical cancer and their intervention strategies by many researchers. Investigations have been done on the dynamics of HPV infections among women despite of the presence of vaccination [14]. A mathematical model exploring the transmission dynamics of human papilloma virus (HPV) was formulated by [9]. In their model, infected individuals can recover with a limited immunity that results in a lower probability of being infected again. In practice, it is necessary to revaccinate individuals within a period after the first vaccination to ensure immunity against HPV infection. A between host model for cervical cancer infection incorporating diagnosis was formulated and analysed by [12, 13]. Results showed that,the disease related mortality is eradicated if diagnosis is done at an early stage hence late diagnosis increases the risk of cervical cancer infection among the infected individuals.

Cervical cancer diagnosis sounds like a death sentence to many in Kenya,

though the cancer is among the most treatable. It's crucially also almost totally vaccine-preventable. The fourth most common cancer among women worldwide, cervical cancer is the second most prevalent in Kenya, trailing only behind breast cancer. However, with 5,200 fatalities annually, it's the primary cause of cancer-related deaths in the country, claiming approximately nine Kenyan women's lives each day. In many developing nations, the public health goals that can help prevent and control the spread of cancer among potential patients include, absolute adherence to vaccination, regular cervical screening and treatment arrangement [7]. However, they are not 100 percent effective and the disease prevalence remains high. Therefore, maximum protection against HPV may help prevent the rapid progression of the cervical cancer infection especially in scarce resource setting where treatment is not readily available. Protection involves limiting exposure to risk factors that can lead to HPV infection. The most important and widely contributing risk factor is long-term use of oral contraceptives. If oral contraceptives are used by girls at the same time having first sexual intercourse before the age of 17 years, this may increase the risk of cervical cancer more. These two risks are associated with high hormone levels and thus exaggerating the progression of cervical cancer. Other risk factors include; Sexual history (becoming sexually active at a young age especially younger than 18 years old, having many sexual partners, having one partner who is considered high risk), smoking, having a weakened immune system, having multiple full-term pregnancies, economic status and having a diet low in fruits and vegetables [8].

2 The model

We formulate a model in which the total human population at any time t denoted by N is subdivided into classes, $S(t)$ the class of individuals susceptible to cervical cancer infection. Recruitment into susceptible class is done at a rate $(1-\theta)\Lambda$. The class $P(t)$ consist of individuals protected against HPV infection. Recruitment into the protected class is done at the rate $\theta\Lambda$. The class $I(t)$ consists of individuals who are asymptomatically infected with HPV infection, this infection occurs at the rate λ. Most HPV infected Individuals recover from the infection at a rate α and slide back to the $S(t)$ class, ρ is the rate of progression to the cervical cancer $C(t)$ class due to persistence of the HPV infection. Mortality occurs among cervical cancer patients at the rate ν while natural death is assumed to occur in all classes at the rate μ.

The rate at which the susceptible individuals acquire HPV infection is defined as

$$\lambda = \frac{\kappa\omega I(t)}{N(t)} \tag{1}$$

Where κ is the effective contact rate with HPV infected individuals while ω is the probability rate of acquiring HPV infection. With the assumption that, high viral load increases the probability of persistence of the HPV infection and thus exposes the individuals to cervical cancer. Let δ be the probability of success of protection against HPV infection, thus the effective force of infection λ^p becomes

$$\lambda^p = \frac{\kappa\omega(1-\delta)I(t)}{N(t)} \tag{2}$$

From the above definitions, the resulting diagram for the model is given below.

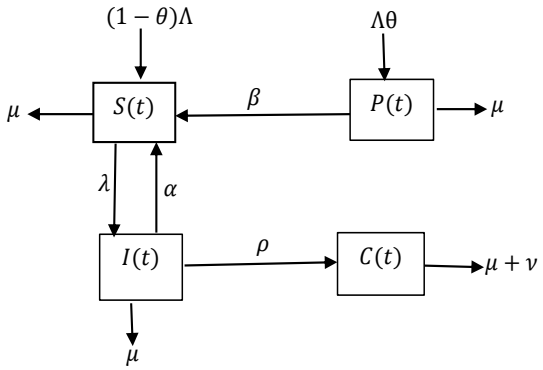

Figure 1: Model flow diagram

The dynamics described can be represented mathematically as:

$$\begin{aligned}
\dot{S}(t) &= (1-\theta)\Lambda + \alpha I(t) + \beta P(t) - \frac{\kappa\omega(1-\delta)I(t)}{N(t)}S(t) - \mu S(t) \\
\dot{P}(t) &= \theta\Lambda - (\beta+\mu)P(t) \\
\dot{I}(t) &= \frac{\kappa\omega(1-\delta)I(t)}{N(t)}S(t) - (\rho+\alpha+\mu)I(t) \\
\dot{C}(t) &= \rho I(t) - (\nu+\mu)C(t)
\end{aligned} \tag{3}$$

3 Analysis of the Model

Since the model describes a human population, all population compartments will be non negative $t > 0$ in the feasible region Ω where $S(t), P(t), I(t), C(t) \in \Omega \subset R^4_+$. It can be shown that all the solutions are bounded in Ω, $\forall t > 0$ such that $0 \leq N \leq \frac{\Lambda}{\mu}$. Thus the model is epidemiologically well posed in the region Ω and can be analysed.

4 The basic reproduction number

The dynamics of the model are highly dependant on the basic reproduction number. The basic reproduction R_0 is the average number of secondary infections produced by a single infectious individual during his or her entire life time as an infective when introduced into a purely susceptible population. The basic reproduction number, R_0, for model (3) is computed using the next generation matrix method as used in [4, 18]. It is the spectral radius of a matrix.

$$FV^{-1} \tag{4}$$

Where F is the Jacobian of f and f is the rate of appearance of new infections in compartment. V is the Jacobian of v where v is the rate of transfer of individuals into and out of compartment. From the model,

$$f := \begin{pmatrix} \frac{\kappa\omega(1-\delta)IS}{N} \\ 0 \end{pmatrix} \tag{5}$$

$$v := \begin{pmatrix} (\rho + \alpha + \mu)I \\ -\rho I + (\mu + \nu)C \end{pmatrix} \tag{6}$$

The Jacobian of f at disease free equilibrium $N = S)$ denoted as F is given by

$$F = \begin{pmatrix} \kappa\omega(1-\delta) & 0 \\ 0 & 0 \end{pmatrix} \tag{7}$$

Similarly, the Jacobian of v at the disease free equilibrium is denoted by V and is given by

$$V = \begin{pmatrix} \alpha+\rho+\mu & 0 \\ -\rho & \mu+\nu \end{pmatrix} \tag{8}$$

On computing V^{-1} we have

$$V^{-1} = \begin{pmatrix} \frac{1}{\alpha+\rho+\mu} & 0 \\ \frac{\rho}{(\mu+\nu)(\alpha+\rho+\mu)} & \frac{1}{(\mu+\nu)} \end{pmatrix} \tag{9}$$

thus

$$FV^{-1} = \begin{pmatrix} \frac{\kappa\omega(1-\delta)}{(\alpha+\rho+\mu)} & 0 \\ 0 & 0 \end{pmatrix} \tag{10}$$

. The reproduction number R_0 which is the spectral radius of the matrix FV^{-1} is given by ;

$$R_0 = \frac{\kappa\omega(1-\delta)}{(\alpha+\rho+\mu)} \tag{11}$$

which is the measure of the severity of an epidemic and one of the most important concern parameter for the disease to invade a population.

5 Disease-free Equilibrium point (DFE)

The disease-free equilibrium point, denoted by E_o is a steady-state solution for which there is no disease or infection in the population [15]. To obtain the disease-free equilibrium point we set the normalized model system (3) equal to zero. Since there are no infections in the human populations, we set $P(t) = I(t) = C(t) = 0$. This implies that $E_0 = \{S_0, P_0, I_0, C_0\} = \{\frac{\Lambda(\mu+\beta-\theta\mu)}{\mu(\mu+\beta)}, \frac{\Lambda\theta}{\mu+\beta}, 0, 0\}$

6 Local Stability Analysis of the Disease Free Equilibrium

The model in Equation (1) has disease free equilibrium (DFE) given by $E_0 = \{S_0, P_0, I_0, C_0\} = \{\frac{\Lambda(\mu+\beta-\theta\mu)}{\mu(\mu+\beta)}, \frac{\Lambda\theta}{\mu+\beta}, 0, 0\}$

Theorem 6.1. *If $R_0 < 1$ then $E_0 = \{S_0, P_0, I_0, C_0\} = \{\frac{\Lambda(\mu+\beta-\theta\mu)}{\mu(\mu+\beta)}, \frac{\Lambda\theta}{\mu+\beta}, 0, 0\}$ is an equilibrium state in Ω and is locally asymptotically stable otherwise unstable.*

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Proof. Consider the Jacobian matrix of Equation (3) given by

$$J = \begin{pmatrix} -\frac{\kappa\omega(1-\delta)I(t)}{N(t)} - \mu & \beta & -\frac{\kappa\omega(1-\delta)S(t)}{N(t)} + \alpha & 0 \\ 0 & -(\beta+\mu) & 0 & 0 \\ \frac{\kappa\omega(1-\delta)I(t)}{N(t)} & 0 & \frac{\kappa\omega(1-\delta)S(t)}{N(t)} - (\rho+\alpha+\mu) & 0 \\ 0 & 0 & \rho & -(\mu+\nu) \end{pmatrix} \quad (12)$$

The Jacobian matrix of Equation (3) at DFE is given by

$$J_{E_0} = \begin{pmatrix} -\mu & \beta & -\kappa\omega(1-\delta)+\alpha & 0 \\ 0 & -(\beta+\mu) & 0 & 0 \\ 0 & 0 & \kappa\omega(1-\delta) - (\rho+\alpha+\mu) & 0 \\ 0 & 0 & \rho & -(\mu+\nu) \end{pmatrix} \quad (13)$$

Then, investigate the stability of equation (13) and its effect due to the reproduction number R_0

$$J_{E_0} = \begin{pmatrix} -\mu & \beta & -\kappa\omega(1-\delta)+\alpha & 0 \\ 0 & -(\beta+\mu) & 0 & 0 \\ 0 & 0 & (\rho+\alpha+\mu)(R_0-1) & 0 \\ 0 & 0 & \rho & -(\mu+\nu) \end{pmatrix} \quad (14)$$

Using Routh-Hurwitz criterion [16], to analyzing the stability of the Jacobian at DFE, compute the trace (Tr) and the determinant (Det) and set the conditions. The Trace at DFE, is given by

$$Tr(J_{E_0}) = -\mu - (\beta+\mu) + (\rho+\alpha+\mu)(R_0-1) - (\mu+\nu) \quad (15)$$

which is negative provided that $R_0 < 1$, and the determinant is given by

$$Det(J_{E_0}) = \mu^2(\beta+\mu)(1-R_0) \quad (16)$$

The determinant of the Jacobian matrix at DFE remains positive provide that $R_0 < 1$. Therefore, by Routh-Hurwitz criterion [16], the disease-free equilibrium of model (3) is locally asymptotically stable. Given a small initial infective population, each infected individual in the entire period of infectivity will produce less than one infected individual on average if $R_0 < 1$. □

6.1 Global stability of Disease-free Equilibrium point

For global stability of the DFE, the technique by Castillo [3] is used. There are two conditions that if met guarantee the global asymptotic stability of the disease free state. Equation (3) may be written in the form

$$\frac{dX}{dt} = H(X, Z),$$
$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \tag{17}$$

where $X = \{S(t), P(t)\}$ with $X \in \mathbb{R}^{2\text{‘}}$ denoting the number of uninfected compartments and $Z \in \mathbb{R}^2$ where $Z = (I(t), C(t))$ denotes the number of infected individuals. $E_0 = \{S_0, P_0, I_0, C_0\} = \{\frac{\Lambda(\mu+\beta-\theta\mu)}{\mu(\mu+\beta)}, \frac{\Lambda\theta}{\mu+\beta}, 0, 0\}$ denotes the disease free equilibrium point of this system. Conditions below must be met to guarantee a local asymptotic stability:

$$\frac{dX}{dt} = H(X, 0), X^* \text{ is globally asymptotically stable (GAS)}$$
$$G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 for (X, Z) \in \Omega \tag{18}$$

Where $P = D_z G(X^*, 0)$ is an M-matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes medical sense.

Theorem 6.2. *If system* (17) *satisfies conditions* (18)*, then the fixed point* $E_0 = (X^*, 0)$ *is a globally asymptotically stable equilibrium provided that* $R_0 < 1$ *and the assumptions in* (18) *are satisfied.*

Proof. Consider
$H(X, O) = 1 - \theta\Lambda - \mu S(t), \theta\Lambda - (\beta + \mu)P(t)$ and $G(X, Z) = PZ - \hat{G}(X, Z)$

Where $P = \begin{pmatrix} -(\alpha + \rho + \mu) & 0 \\ \rho & -(\mu + \nu) \end{pmatrix}$

And
$$G(X, Z) = \begin{pmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \end{pmatrix} = \begin{pmatrix} -\kappa\omega(1 - \delta)I(t) \\ 0 \end{pmatrix}$$

Considering the Jacobian matrix, and replacing $I(t) = 0$ $C(t) = 0$, we obtain $\hat{G}_1(X, Z) = 0$ and so the conditions in (18) are met and therefore, E_0 is globally asymptotically stable when $R_0 < 1$. Epidemiologically, any perturbation of the model by the introduction of infectives shows that the model solutions will converge to the DFE whenever $R_0 < 1$.

□

7 Existence of Endemic Equilibrium

At the Endemic equilibrium point, we have persistence of infection thus at least one of the infected classes is greater than zero. The positive endemic equilibrium of model (3) is denoted by

$$E_e(S^*(t), P^*(t), I^*(t), C^*(t)). \tag{19}$$

Theorem 7.1. *Cervical cancer infections exist and persist in the population where* $I_h^* > 0$ *and* $C^* > 0$ *whenever* $R_0 > 1$

Proof. Using mathematica software, the endemic states were given as

$$\begin{aligned} S^*(t) &= \frac{N(\alpha+\mu+\rho)}{\kappa\omega(1-\delta)} \\ P^*(t) &= \frac{\Lambda\theta}{(\mu+\beta)} \\ I^*(t) &= \frac{1}{\mu+\rho}\{\Lambda - \frac{\Lambda\mu\theta}{\beta+\mu} - \frac{N\mu(\alpha+\mu+\rho)}{(1-\delta)\kappa\omega}\} \\ C^*(t) &= -\frac{\rho\{N\mu(\beta+\mu)(\alpha+\mu+\rho) - \omega\Lambda\kappa(1-\delta)(\beta+\mu-\mu\theta)\}}{\kappa\omega(1-\delta)(\beta+\mu)(\mu+\nu)(\mu+\rho)} \end{aligned} \tag{20}$$

□

7.1 Local stability of endemic equilibrium point

Theorem 7.2. *If* $R_0 > 1$*, then the endemic equilibrium* $E_e(S^*(t), (P^*(t), I^*(t), C^*(t))$*, is locally asymptotically stable*

Proof. The Jacobian of Equation (3) at endemic state is given by

$$J_e = \begin{pmatrix} -\frac{\kappa\omega(1-\delta)I^*(t)}{N(t)} - \mu & \beta & -\frac{\kappa\omega(1-\delta)S^*(t)}{N(t)} + \alpha & 0 \\ 0 & -(\beta+\mu) & 0 & 0 \\ \frac{\kappa\omega(1-\delta)I^*(t)}{N(t)} & 0 & \frac{\kappa\omega(1-\delta)S^*(t)}{N(t)} - (\rho+\alpha+\mu) & 0 \\ 0 & 0 & \rho & -(\mu+\nu) \end{pmatrix} \tag{21}$$

Where $I^*(t) = \frac{1}{\mu+\rho}\{\Lambda - \frac{\Lambda\mu\theta}{\beta+\mu} - \frac{N\mu(\alpha+\mu+\rho)}{(1-\delta)\kappa\omega}\}$ and $S^*(t) = \frac{N(\alpha+\mu+\rho)}{\kappa\omega(1-\delta)}$

Using Routh-Hurwitz criterion [16], the Trace (Tr) at E_e is given by

$$Tr(J_e) = -\frac{\kappa\omega(1-\delta)I^*(t)}{N(t)} - \mu - (\beta+\mu) + \frac{\kappa\omega(1-\delta)S^*(t)}{N(t)} - (\rho+\alpha+\mu) - (\mu+\nu)$$

upon substitution

$$Tr(J_e) = -(\beta + \mu) - (\mu + \nu) - \mu R_o \tag{22}$$

Then the trace of matrix J_e is negative provided that $R_o > 1$ and the determinant will be given by

$$Tr(Det_e) = \mu^2(\mu + \beta)(\mu + \rho + \alpha)(R_o - 1) \tag{23}$$

Clearly the determinant of the matric is positive provided that $R_o > 1$. This implies that the Routh-Hurwitz criterion holds and thus the endemic Equilibrium (E_e) of model (3) is locally asymptotically stable otherwise unstable.

□

7.2 Global stability of endemic equilibrium point

The global stability of the equilibrium is obtained by means of Lyapunov's direct method and LaSalle's invariance principle De Leon [6].

Theorem 7.3. *The endemic equilibrium E_e of model (1) is globally asymptotically stable in Ω whenever $R_0 > 1$.*

Proof. Consider the non-linear Lyapunov function
$V : (S(t), P(t), I(t), C(t)) \in \Omega \subset \mathbb{R}^4_+ : S(t), P(t), I(t), C(t) > 0$

defined as

$$V = S - S^* \ln S + P - P^* \ln P + I - I^* \ln I + C - C^* \ln C \tag{24}$$

where V is in the interior of the region Ω. E_e is the global minimum of V on Ω and $V : \{S(t), P(t), I(t), C(t)\} = 0$. Differentiating V with respect to time gives

$$\frac{dV}{dt} = \dot{V} = \dot{S}(1 - \frac{S^*}{S}) + \dot{P}(1 - \frac{P^*}{P}) + \dot{I}(1 - \frac{I^*}{I}) + \dot{C}(1 - \frac{C^*}{C}) \tag{25}$$

Replacing $\dot{S}, \dot{P}, \dot{I}, \dot{C}$ from equation (3) in equation (25) we obtain

$\dot{V} = [(1-\theta)\Lambda + \alpha I(t) + \beta P(t) - \frac{\kappa\omega(1-\delta)I(t)}{N(t)}S(t) - \mu S(t)](1 - \frac{S^*}{S}) + [\theta\Lambda - (\beta + \mu)P(t)](1 - \frac{P^*}{P}) + [\frac{\kappa\omega(1-\delta)I(t)}{N(t)}S(t) - (\rho + \alpha + \mu)I(t)](1 - \frac{I^*}{I}) +$
$[\rho I(t) - (\nu + \mu)C(t)](1 - \frac{C^*}{C})$

At boundary $N \leq \frac{\Lambda}{\mu}$, we let $N = \frac{\Lambda}{\mu}$

$\dot{V} = [(1-\theta)\Lambda + \alpha I(t) + \beta P(t) - \frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) - \mu S(t)](1 - \frac{S^*}{S}) + [\theta\Lambda - (\beta+\mu)P(t)](1 - \frac{P^*}{P}) + [\frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) - (\rho+\alpha+\mu)I(t)](1 - \frac{I^*}{I}) + [\rho I(t) - (\nu+\mu)C(t)](1 - \frac{C^*}{C})$

At steady state the following results from model (3) were obtained

$$\begin{aligned} (1-\theta)\Lambda &= \frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) + \mu S(t) - \alpha I(t) - \beta P(t) \\ \theta\Lambda &= (\beta+\mu)P(t) \\ \frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) &= (\rho+\alpha+\mu)I(t) \\ \rho I(t) &= (\nu+\mu)C(t) \end{aligned} \tag{26}$$

Thus we have

$\dot{V} = [\frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) + \mu S(t) - \alpha I(t) - \beta P(t) + \alpha I(t) + \beta P(t) - \frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) - \mu S(t)](1 - \frac{S^*}{S}) + [(\beta+\mu)P(t) - (\beta+\mu)P(t)](1 - \frac{P^*}{P}) + [\frac{\kappa\mu\omega(1-\delta)I(t)}{\Lambda}S(t) - (\rho+\alpha+\mu)I(t)](1 - \frac{I^*}{I}) + [\rho I(t) - (\nu+\mu)C(t)](1 - \frac{C^*}{C})$

$\dot{V} = [\frac{\kappa\mu\omega(1-\delta)I^*S^*}{\Lambda} + \mu S^* - \alpha I^* - \beta P^*](2 - \frac{S}{S^*} - \frac{S^*}{S}) + (\beta+\mu)P^*(2 - \frac{P}{P^*} - \frac{P^*}{P}) + \frac{\kappa\mu\omega(1-\delta)I^*S^*}{\Lambda}(1 - \frac{S}{S^*}\frac{I^*}{I}) + \rho I^*(1 - \frac{I}{I^*}\frac{C^*}{C})$

At $S = S^*, P = P^*, I = I^*, C = C^*$ and from the property that the geometric mean is less than or equal to the arithmetic mean, the inequality $\dot{V} \leq 0$ holds iff $(S(t), P(t), I(t), C(t))$ takes the equilibrium values $S^*(t), P^*(t) I^*(t), C^*(t)$. Thus, by LaSalle's invariance principle [6], the endemic equilibrium E_e is globally asymptotically stable.

Epidemiologically, any perturbation of the model by the introduction of infectives, the model solutions will converge to the E_e whenever $R_o > 1$. This implies that the disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrence

□

If DFE and EE are locally and globally asymptotically stable, then all the epidemiological situation different from the given stable equilibria $t \to 0$ evolve to the equilibrium points. This is significant to epidemiologists, as the conditions required for stability of the model when $R_o < 1$, will provide a basis for the necessary indicators to be controlled in the reduction of the transmission of Human papilloma virus.

8 Numerical simulation

Numerical simulations were carried out to graphically illustrate the effect of protection on the dynamics of infection. To do this, some parameter values were used as indicated in table (1).

Table 1: Parameter values used in simulation of model (1)

Parameter	**description**	**Value**	**Source**
S(t)	Susceptible individuals	3000	Estimate
$P(t)$	Protected individuals	1000	Estimate
$I(t)$	HPV infected individuals	500	Estimate
C(t)	Cervical cancer infected individuals	100	Estimate
Λ	Recruitment rate	149 per year	[2]
β	Loss of protection	0.001	Estimate
θ	Adjustment parameter	0.001	Estimate
ω	probability rate of acquiring HPV infection	0.31 per year	[1, 10]
κ	Contact rate with HPV infective	0.80 per year	[10, 14]
μ	Natural mortality rate	0.05393 per year	[10]
ν	Cervical cancer related death rate	0.61325 per year	[2]
α	Recovery rate of HPV infection	0.70 per year	[10]
ρ	Rate of progression to Cervical cancer	0.1271 per year	[10, 14, 17]
δ	Modification parameter	$0 < \delta < 1$	Estimate

Based on the initial conditions and parameter values in table (1), the following graphs were obtained;

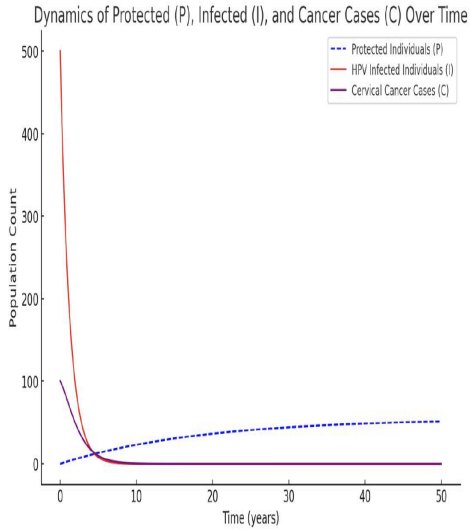

Figure 2: *Simulation of Equation (3) showing the evolution of the infection against time.*

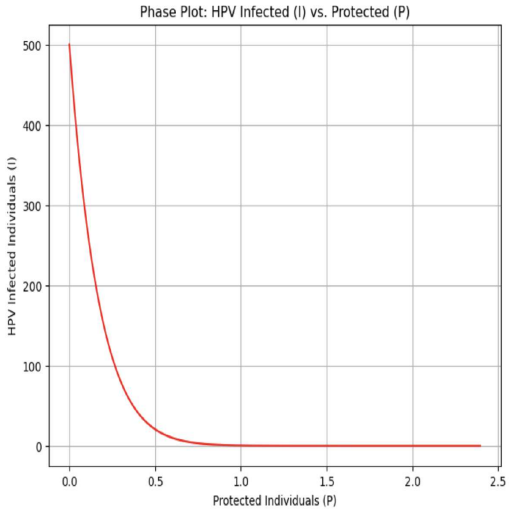

Figure 3: *The effect of varying the protection term on HPV.*

Figure 2 shows the evolution of cervical cancer infection in the presence of protection against time in days. With high success of protection, there is low contact rate and low prevalence rate hence the HPV infected and Cervical cancer individuals in the population decreases sharply over time. With low protection there is high contact rate and hence a high disease prevalence in the population.

From Figure 3, we observe that the number of individuals infected with HPV reduces with increased protection. On the contrary, when the protection rate is low, the number of HPV infected individuals will be high. This is consistent with reality.

This is in agreement with the mathematical analysis which showed that the disease free equilibrium point for Equation (3) is locally and globally asymptotically stable when $R_0 < 1$. The endemic states are shown to exist provided that the reproduction number is greater than unity. By use of Routh-Hurwitz criterion and suitable Lyapunov functions, the endemic states are shown to be locally and globally asymptotically stable respectively.

In order to eliminate cervical cancer infections, there is need to employ strategies such as increasing the public awareness drive to behaviour change, promoting increased condom use to reduce the spread of HPV infection, avoid use of oral contraceptives especially by girls before the age of 17 years, being faithful to one partner and avoid smoking. These strategies will help in reducing the economic burden that are borne by a country in giving care and treating the infected individuals. As evidenced from these results, it is indeed true that prevention is better than cure.

9 Conclusion

Effective control of HPV infection prevents progression to cervical cancer especially in scarce resource setting where treatment is not readily available. Screening and vaccination are effective prevention measures. Moreover, increasing public awareness drive to behaviour change, promoting increased condom use to reduce the spread of HPV infection, avoid use of oral contraceptives especially by girls before the age of 17 years, being faithful to one partner, avoid smoking, will reduce the probability of the infection hence resulting to less people contacting the infection. Protection against Hpv infection is perceived to yield better results in the reduction of cervical cancer mortality rate.

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