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Within Host Model for Cervical Cancer Incorporating Diffusion

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Abstract
In this paper a mathematical model describing a within host cervical cancer infection with viral and cellular infection incorporating diffusion was formulated and analysed. The replenishment rate of the cells was represented by a logistic growth rate. The qualitative analysis of model showed that the infection dynamics can best be described by the thresh- old value $R0w$, in which for the value of $R0w<1$ the infection free equilibrium is globally asymptotically stable. This is theoretically in terpreted to means that cervical cancer is cleared from the body. On the other hand when $R0w>1$, the endemic equilibrium is globally asymptotically stable which implies viral persistence. The numerical results show that the movement of the virus makes the infection persist within the cells. This results in a more infected cells which implies that introduction the virus to purely uninfected cells results in propagation of the infection.

Subject Classification: *xxxxxx*
Keywords: *Diffusion, Human papilloma Virus, Reproduction Number, Stability Analysis.*

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I. Introduction

Human papilloma Virus (HPV) is transmitted sexually through skin-to-skin genital contact. Cervical cancer is mainly due to the infections of HPV though the risk due to the various HPV types has been given little attention. 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 [3]. 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.

Precancerous cells develop in the cervix as a result of the persistence of Human Papilloma Virus infection which eventually turn out to be malignant. Precancerous cells can be prevented from developing to cancer if they are properly treated. So far, it has not yet been discovered which individual who have Hu- man papilloma Virus can progress to cancer or any other health complications but studies show that persons with puny immune systems as well as those with HIV/AIDS and severe diseases might not be capable to combat off the virus. Sexually active females are at a higher risk of infection with human papilloma virus, the virus which causes cervical cancer, however no much information is there about the occurrence of the HPV infection. After development of symptoms or any other health problems is when some people will realize they have human papilloma virus but others will neither know they are infected nor develop symptoms from it [3, 9].

Within host models are also called immunological models, and they are basically tools used in understanding how to regulate viral load dynamics which is an infection progress within a single individual. In Diffusion equations will be used to develop within host model and diagnosis will be incorporated in between host model. Diffusion allows perturbation that is, it makes the sys- tem to be real by introducing movement of the Human papilloma virus (HPV) from one compartment to another. The transmission of Cervical cancer within the body is related with the spatial distribution of the high risk Human papillomavirus (consisting of viral type that may lead to cervical cancer). For example, cervical cancer is partially attributed to the mobility of high risk Human papilloma virus from one cell to the other which increases the rate of interaction and expose target cells to the virus which causes cervical cancer. The understandings on movement are hardly explored to explain the connection between the cervical cancer infection and the movement of high risk Human papilloma virus [6].

II. The model

We formulate a model composed of three variables, namely, the uninfected cells $T(t)$, cells infected with higher risk Human Papillomavirus types (viruses which leads to cervical cancer infection) $T^*(t)$, and free virus particles $V(t)$. The uninfected cells are produced at rate γ and die naturally at a rate μ. The total number of cells in the body remains bounded and thus, the growth of cells is governed by the logistic proliferation term $\left(1-\frac{T}{T_{max}}\right)$ which limits cell growth as the cell population approaches the limit $Tmax$. The uninfected cells become infected by free virus which at this instance is the lower risk Human Papillomavirus types and later develop to higher risk Human Papillomavirus types at a infection terms $\beta V T$ and $\omega T T^*$, respectively. This generates cells infected with higher risk Human Papillomavirus types, T^* (t) which dies naturally at the rate σ. The infected cells produce free viruses V at the rate δT^*. The free varions are cleared from circulation at a constant per capita rate of ϵ.

In this model, Diffusion occurs in all classes at the rate $D1$, $D2$ *and* $D3$. $D1$ is the diffusion rate for the free virus class, $D2$ is the diffusion rate forthe uninfected cells while $D3$ is the diffusion rate for the infected cells. The movement of the Human Papillomavirus from one cell to another influences the development of cervical cancer infection.

For this reason, the following model with spatial diffusion is developed to capture the mobility of the Human Papillomavirus within an individual.

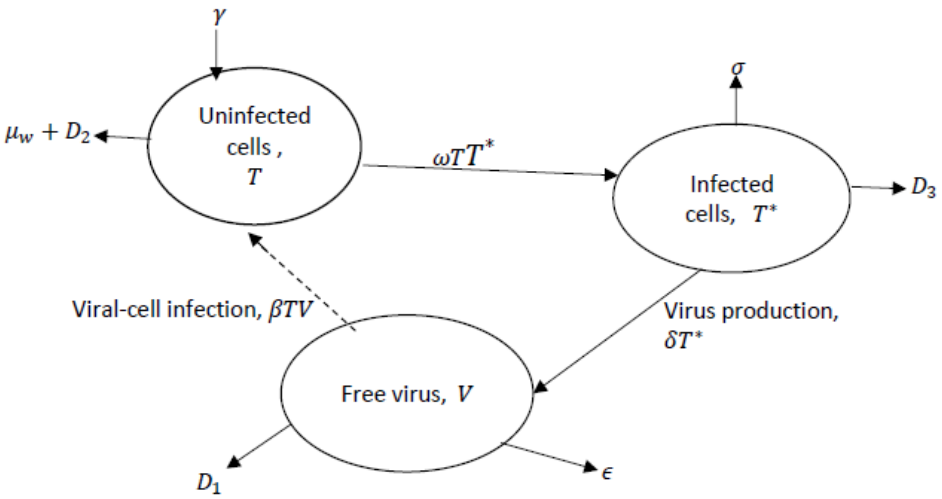

Figure 1: Within-Host Model flow diagram

$$\frac{dT(t,x)}{dt} = \gamma T\left(1 - \frac{T}{T_{max}}\right) - \beta TV - \omega TT^* - \mu_w - \frac{D_2 d^2T}{dx^2}$$
$$\frac{dT^*(t,x)}{dt} = \beta TV + \omega TT^* - \sigma T^* - \frac{D_3 d^2T}{dx^2}$$
$$\frac{dV(t,x)}{dt} = \delta\sigma T^* - \epsilon V - \frac{D_1 d^2T}{dx^2} \quad (1)$$

Where D_1, D_2, D_3 is the diffusion coefficient with homogeneous Neumann boundary condition

$$\frac{\partial T}{\partial t} = \frac{\partial T^*}{\partial t} = \frac{\partial V}{\partial t} = 0 \quad (2)$$

On $\partial\Omega \times (0, +\infty)$. And initial conditions

$$T(0) = T_0 > 0,\ T^*(0) = T^*{}_0 \geq 0, V(0) = V_0 > 0 \quad (3)$$

The region is assumed to be whole $(-\infty, +\infty)$

3 Model analysis

The basic reproduction number R_{0w} is defined as the average number of secondary infections produced by one infectious virion over the course of their infectious period in uninfected cell population. The basic reproduction number, R_{0w}, for model (1) was computed using the next generation matrix methodas used in [2, 8]. From Model (1) and at diffusion free state $(D = 0)$, the basic reproduction number R_{0w} was computed as

$$R_{0w} = T_{max}\left(1 - \frac{\mu_w}{\gamma}\right)\frac{\omega}{\sigma} + T_{max}\left(1 - \frac{\mu_w}{\gamma}\right)\frac{\beta\delta}{\epsilon} \quad (4)$$

4 Local stability of the Disease free equilibrium

We linearize the system (1) around the arbitrary spatially homogeneous fixed point $E_{0w}(T, T^*, V)$ for small space and time.

Theorem 4.1. *The infection free equilibrium E_{0w} is locally asymptoticallystable if and only if $R_{0w} < 1$*

Proof. The Jacobian matrix of Equation (1) is given by

$$J = \begin{pmatrix} \mu_w - \gamma - D & -\omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) & -\beta T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) \\ 0 & \omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) - \sigma - D & \beta T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) \\ 0 & \sigma\delta & -\epsilon - D \end{pmatrix} \quad (5)$$

Clearly $\lambda_1 = \mu_w - \gamma - D$ is an eigenvalue. The eigenvalue λ_1 is negative since for any population whose growth is positive; the production rate is greater than the mortality rate, that is $\gamma > \mu_w$. We analyse the reduced matrix of equation (5)

$$J = \begin{pmatrix} \omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) - \sigma - D & \beta T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) \\ \sigma\delta & -\epsilon - D \end{pmatrix} \quad (6)$$

Applying the Routh-Hurwitz criterion, for stability analysis, then matrix J in equation (6) will have negative real roots if and only if the $tr(J) < 0$ and $det(J) > 0$, and thus

$$tr(J) = \omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) - \sigma - \epsilon - 2D < 0 \quad (7)$$

Provided that $\omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) < 0$ and

$$det(J) = \left\{-\omega T_{max}\left(1 - \frac{\mu_w}{\gamma}\right)\frac{\beta\delta}{\epsilon} - T_{max}\left(1 - \frac{\mu_w}{\gamma}\right)\frac{\omega}{\sigma} + 1\right\} - 2D \quad (8)$$

Using equation (4) equation (7) reduces to

$$det(J) = \{R_{0w} - 1\} - 2D \quad (9)$$

The $det(J)$ is positive when $R_{0w} < 1$ and $\{R_{0w} - 1\} > 2D$. Therefore, the DFE is locally asymptotically stable whenever $R_{0w} < 1$ and unstable when $R_{0w} > 1$. This implies that for any perturbation of the model by the introduction of the virus, the solutions of the model (1) will converge to the DFE when $R_{0w} < 1$. Biologically, if a few virion enters the blood stream, then there is a high chance of infecting less than one susceptible cells in its entire period of infectivity whenever $R_{0w} < 1$. Thus the virus will clear from the human body when $R_{0w} < 1$.

5 Global stability of the disease-free equilibrium

The Castillo Chavez theorem [1] is applied to study the global stability of the disease-free equilibrium. We rewrite model (1) with $D1 = D2 = D3 = 0$ in the form;

$$\frac{dX}{dt} = H(X, Z)$$
$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \qquad (10)$$

Where $X \in \mathbb{R}$ denotes the number of susceptible cells and $Z \in \mathbb{R}^2$ denotes the number of actively infected cells and free virions respectively. At disease Free Equilibrium (DFE)

$$E_{0w} = (X^0, 0), X^0 = T_{max}\left(1 - \frac{\mu_w}{\gamma}\right) \qquad (11)$$

The conditions below must be met to guarantee global asymptotic stability

$\frac{dX}{dt} = H(X, 0)X^0$ is globally Asymptotically stable (GAS)

$$G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0, \text{ for } (X, Z) \in \Omega \qquad (12)$$

Where $P = D_z G(X^0, 0)$ is an M- matrix (the off diagonal elements of P are nonnegative)

and Ω is the region where the model makes biological sense. If system (10) satisfies conditions in (12) then the following theorem holds:

Theorem 5.1. *The fixed point $E_0 = (X^0, 0)$ is a Globally Asymptotically Stable equilibrium point of model (1) provided that $R_{0w} < 1$. and the conditions in (12) are satisfied*

Proof.

$$H(X, 0) = \gamma T\left(1 - \frac{T}{T_{max}}\right) - \omega T \qquad (13)$$

And $G(X, Z) = PZ - \hat{G}(X, Z)$ where

$$P = \begin{pmatrix} -\sigma & 0 \\ \sigma\delta & -\epsilon \end{pmatrix} \qquad (14)$$

$$\widehat{G}(X,Z) = \begin{pmatrix} \widehat{G}_1(X,Z) \\ \widehat{G}_2(X,Z) \end{pmatrix} = \begin{pmatrix} \beta TV + \omega TT^* \\ 0 \end{pmatrix} \quad (15)$$

Considering the Jacobian matrix, and replacing $T(t) = T_{max}\left(1 - \frac{\mu_w}{\gamma}\right)$, $T^* = 0$ and $V(t) = 0$ we obtain $\widehat{G}_1(X,Z) = 0$ and so the conditions in (12) are satisfied, therefore, E_{0w} is globally asymptotically stable whenever $R_{0w} < 1$.

This implies that given a large perturbation of the DFE by the introduction of free virus particles, the solutions of model (1) will eventually converge to the DFE whenever $R_{0w} < 1$.

6 Existence of Endemic Equilibrium

Theorem 6.1. *A positive Endemic Equilibrium exist provided* $R_{0w} > 1$, $T_e^* \neq 0$ *and* $V_e \neq 0$

Proof. The endemic equilibrium $E_{ew} = (T_e, T_e^*, V_e)$ satisfies:

$$\gamma T_e\left(1 - \frac{T_e}{T_{max}}\right) - \beta T_e V_e - \omega T_e T_e^*, -\mu_w T_e = 0 \quad (16)$$

$$\beta T_e V_e + \omega T_e T_e^*, -\sigma T_e^*, = 0 \quad (17)$$

$$\delta\sigma T_e^*, -\epsilon V_e = 0 \quad (18)$$

From equation (18) we have

$$V_e = \frac{\delta\sigma T_e^*}{\epsilon} \quad (19)$$

Substituting equation (19) in equation (17) and making T_e the subject of the formula we get;

$$T_e = \frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}} \quad (20)$$

Substituting V_e and T_e and equation (4) in equation (16) we obtain:

$$T_e^* = \frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)(\gamma-\mu_w)(R_{0w}-1)}{\sigma R_{0w}^2} \quad (21)$$

The endemic equilibrium E_e will thus be given by

$$EE = \left\{\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}}, \frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)(\gamma-\mu_w)(R_{0w}-1)}{\sigma R_{0w}^2}, \frac{\delta\sigma T_e^*}{\epsilon}\right\} \quad (22)$$

Upon simplification we get

$$E_{ew} = \left\{\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}}, \frac{T_e(\gamma-\mu_w)(R_{0w}-1)}{\sigma R_{0w}}, \frac{\delta\sigma T_e^*}{\epsilon}\right\} \quad (23)$$

$T_e^* > 0$ if and only if $R_{0w} > 1$ and thus $V_e > 0$

7 Local Stability of the Endemic Equilibrium

To investigate the local stability at endemic equilibrium $E_{ew} = (T_e, T_e^*, V_e)$ Turing stability concept was used

Theorem 7.1. *The endemic equilibrium point* E_{ew} *is locally asymptotically stable when* $R_{0w} > 1$ *otherwise unstable*

Proof. Consider the Jacobin matrices evaluated at endemic state

$$J = \begin{pmatrix} \gamma - \frac{2\gamma T_e}{T_{max}} - \beta V_e - \omega\, T_e^* - \mu_w & -\omega T_e & -\beta T_e \\ \omega T_e + \beta T_e & \omega T_e - \sigma & \beta T_e \\ 0 & \sigma\delta & -\epsilon \end{pmatrix} \quad (24)$$

This simplifies to

$$J = \begin{pmatrix} \frac{\mu_w-\gamma}{R_{0w}} & -\omega\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}} & -\beta\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}} \\ \frac{(\gamma-\mu_w)(R_{0w-1})}{R_{0w}} & \omega\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}}-\sigma & \beta\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}} \\ 0 & \sigma\delta & -\epsilon \end{pmatrix} \quad (25)$$

The characteristic equation can be found using the formula

$$-\lambda^3 + tr(J_e)\lambda^2 = \frac{1}{2}\{tr(J_e)^2 - tr\,(J_e)^2\}\lambda + \det J_e$$

From equation (25) we obtain the characteristic equation in the form

$$P(\lambda) = \lambda^3 + a\lambda^2 + b\lambda + c = 0 \quad (26)$$

Thus, the number of possible negative real roots of equation (26) depends on the signs of *a, b* and *c* . This can be established by applying the Descartes Rules of Signs of the polynomial given in [7]:

$$P(\lambda) = a\lambda^2 = b\lambda + c \quad (27)$$

The number of negative real zeros of P is either equal to the number of variations in sign of $P(\lambda)$ or less than this by an even number. Thus, the possibilities of negative roots of Equation (27) is as summarized in the Table.

Cases	a	b	c	R_{0v}	No. of changes	No. of negative roots
1	+	+	+	R_{0v}	0	0
2	+	-	+	R_{0v}	2	2,0
3	+	-	-	R_{0v}	1	0
4	+	+	-	R_{0v}	1	0
5	-	-	+	R_{0v}	1	0
6	-	+	+	R_{0v}	1	0
7	-	+	-	R_{0v}	2	2,0
8	-	-	-	R_{0v}	0	0

From the table the maximum number of variations of signs in $P(-\lambda)$ is two, hence the characteristic polynomial in equation (27) has two negative roots. Thus,

$$P(-\lambda) = -\lambda^3 + a\lambda^2 - b\lambda + c = 0$$

Where

$$a = \sigma + \epsilon - \frac{\mu_w-\gamma}{R_{0w}} - \omega\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}}$$

$$b = \frac{\mu_w-\gamma}{R_{0w}}\left\{\frac{T_{max}\left(1-\frac{\mu_w}{\gamma}\right)}{R_{0w}} + \frac{(\gamma-\mu_w)(R_{0w-1})}{R_{0w}}\right\} + \frac{\mu_w-\gamma}{R_{0w}}(\epsilon-\sigma) + \epsilon\sigma - 1$$

$$c = \frac{(\gamma-\mu_w)(R_{0w-1})}{R_{0w}} + \frac{\mu_w-\gamma}{R_{0w}}(1-\sigma\epsilon)$$

has negative roots and thus if $\gamma > \mu_w$ and if cases $1-8$ in the table are satisfied, then model (1) is locally asymptotically stable if $R_{0w} > 1$.

This means that, given a small number of free virus particle, each virus in the entire period of infectivity will produce on average more than one infected cells when $R_{0w} > 1$. This shows that persistence of the virus occurs whenever.

8 Global Stability of the Endemic Equilibrium

Here we study the global stability of model (1) with homogeneous boundary conditions

$\frac{\partial u}{\partial n} = 0$ on $\partial\Omega \times (0, +\infty)$ and initial conditions

$T(x,0) = T_0(x) \geq 0, T^*(x,0) = T_0^* \geq 0, V(x,0) = V_0(x) \geq 0 \; in \; \Omega$

Where $\frac{\partial}{\partial n}$ is the outward normal derivative on $\partial\Omega$.

Let $u(x,t) = \{T(x,t), T^*(x,t), V(x,t)\}$ be any solution of the model (1) and

$$W = \int_\Omega V\big(u(t,x)\big)dx \tag{28}$$

be a lyapunov functional for model (1). Calculating the time derivative of W along the positive solution of the model (1),

$$\frac{dW}{dt} = \int_\Omega \nabla V(u).\big(D\Delta u + f(u)\big)dx$$

$$= \int_\Omega \nabla V(u).f(u)dx + \int_\Omega \Delta V(u).D\Delta u\, dx$$

$$\frac{dW}{dt} = \int_\Omega \nabla V(u).f(u)dx + \sum_{i=1}^{4} D_i \int_\Omega \frac{\partial V(u)}{\partial u_i}\Delta u_i\, dx \tag{29}$$

Using Green's formula, we obtain

$$\frac{\partial V(u)}{\partial u_i}\Delta u_i dx = \int_\Omega \Omega \frac{\partial V(u)}{\partial u_i}\frac{\partial u_i}{\partial n}dx - \int_\Omega \nabla u_i.\nabla\left(\frac{\partial V}{\partial u_i}\right)dx \tag{30}$$

$\frac{\partial u}{\partial n} = 0$ on $\partial\Omega$, then

$$\frac{\partial V}{\partial u_i}\Delta u_i dx = -\int_\Omega \nabla u_i.\nabla\frac{\partial V}{\partial u_i}\,dx$$

$$-\int_\Omega \nabla u_i.\nabla\frac{\partial V}{\partial u_i}\,dx = -\int_\Omega \frac{|\nabla V|^2}{V}dx \tag{31}$$

Thus the Lyapunov function of the model (1) at E_{we} is given by

$$V = T - T_e In\, T + T^* - T_e^* In\, T^* + V - V_e In\, V \tag{32}$$

$$\nabla V(u).f(u) = \left\{\gamma - \left(\frac{2\gamma}{T_{max}} + \beta V + \omega\, T^* + \mu_w\right)\right\} T\left(1 - \frac{T_e}{T}\right)$$
$$+ (\beta TV + \omega\, TT^* - \delta T^*)\left(1 - \frac{T_e^*}{T^*}\right) + (\delta\sigma T^* - \epsilon V)\left(1 - \frac{V_e}{V}\right)$$

$$\nabla V(u).f(u) = \gamma - \gamma\frac{T_e}{T} - \left(\frac{2\gamma}{T_{max}} + \beta V + \omega\, T^* + \mu_w\right)T + \left(\frac{2\gamma}{T_{max}} + \beta V + \omega\, T^* + \mu_w\right)T_e$$
$$+ \beta TV - \beta TV\frac{T_e^*}{T^*} - (\delta - \omega T)T_e^* + \delta\sigma T^* - \delta\sigma T^*\frac{V_e}{V} - \epsilon V + \epsilon V_e$$

Thus at endemic state

$$\frac{dW}{dt} = \int_\Omega \left\{\left(\frac{2\gamma}{T_{max}} + \beta V + \omega\, T^* + \mu_w\right)T_e\left(2 - \frac{T}{T_e} - \frac{T_e}{T}\right) + \beta T_e V_e\left(1 - \frac{T}{T_e}\frac{V}{V_e}\frac{T_e^*}{T^*}\right) + \sigma\delta T_e^*\left(1 - \frac{V_e}{V}\frac{T^*}{T_e^*}\right)\right\} -$$
$$\int_\Omega \frac{|\nabla V|^2}{V}dx$$

The inequality $\frac{dW}{dt} = 0$ holds if and only if (T, T^*, V) takes the equilibrium values (T_e, T_e^*, V_e). Hence all the solution of model (1) are positive where the largest invariant subset of the set $\left(\frac{dW}{dt} = 0\right)$. Thus the endemic equilibrium point E_{we} is globally asymptotically stable.

This implies that regardless of any starting solution, the solution of the model will converge to E_{we} whenever $R_{0w} > 1$. This means that any perturbation of the equilibrium point as a result of the introduction of the free virus particles, the model solutions will converge to the endemic state.

8 Numerical Simulation

In this section, numerical simulations were carried out to graphically illustrate the behavior of model (1). To do this, some parameter values were used as indicated in table (1).

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

Parameter	Descriptions	Values	Resource
T(t)	Uninfected cells	50	Estimate
$T^*(t)$	Infected cells	100	Estimate
V(t)	Free virus	97	Estimate
T_{max}	Maximum cell population level	$1500\ cells\ mm^{-3}$	[4]
γ	Production rate of uninfected T cells	$0.05\ cells\ days^{-1}$	[4]
μ_w	Mortality rate of uninfected cells	$0.02\ cells\ days^{-1}$	[4]
σ	Mortality rate of infected cells	$0.25\ cells\ days^{-1}$	[4]
β	Viral infection rate by free virions	$2.4 \times 10^{-5}\ mm^{-3}$	[5]
ϵ	Shedding rate of virions	$2.4\ days^{-1}$	[5]
δ	Viral production rate	$\delta \geq 0$	Estimate
ω	Cellular infection rate	$2.4 \times 10^{-5}\ mm^{-3}$	[5]

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

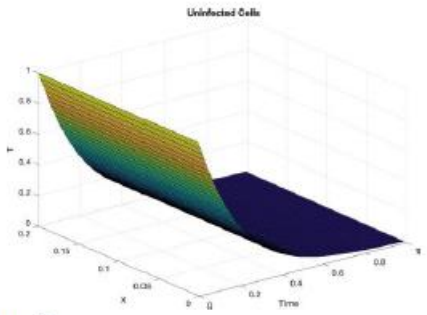

Figure 2:

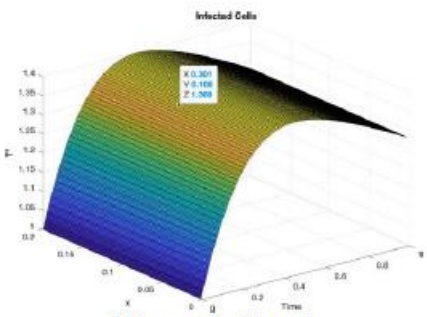

Figure 3: *Graph*

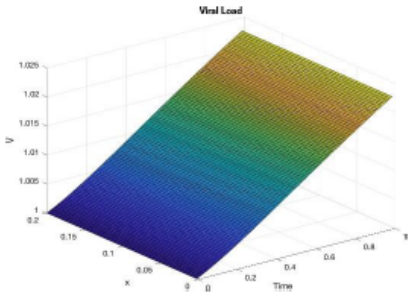

Figure 4

The viral load increase exponentially with the passage of time and space, figure (4), while the uninfected cells decrease exponentially, figure (2). Correspondingly, the infected cells increase with increase in time and space, figure (3).This agrees with reality, in that as the viral load increases, more uninfected cells are recruited into the infected compartment. This explains why the infected cells are increasing. Therefore, the increase in viral load has a corresponding increase in infected cells and decrease in uninfected cells.

The virus will diffuse to regions where there is no infection till an equilibrium is achieved. The numerical results show that the movement of the virus makes the infection persist within the cells. This results in a more infected cells which implies that introduction the virus to purely uninfected cells results in propagation of the infection.

It can be deduced that the mobility of HPV from one cell to the other has an impact on the number of infected cells as well as viral load. With minimal mobility of HPV leads to decrease in the number of infected cells and the viral load.

8 Discussion

In this study, a mathematical model describing a within host cervical cancer infection with viral and cellular infection incorporating diffusion was formulated and analysed. The replenishment rate of the cells was represented by a logistic growth rate. The qualitative analysis of model (1) shows that the infection dynamics can best be described by the threshold value R_{0w} , in which for the value of $R_{0w} < 1$ the infection free equilibrium is globally asymptotically stable. This is theoretically interpreted to means that cervical canceris cleared from the body. On the other hand when $R_{0w} > 1$, the endemic equilibrium is globally asymptotically stable which implies viral persistence.

8 Conclusion

The disease persists in the cells if there exist movement of the virus from one cell to the other which increases the spread of the high risk Human Papilloma Virus in a high risk domain. Introduction of the Virus at one end of the equilibrium results in a wide spread to the infected cells. This shows that the mobility of the virus from one cell to the other increases the spread and hence higher riskof developing cervical cancer. Hence, the spread of the infection is dependenton the movement patterns of the virus. This result are in agreement with a study done by [10], they argued that the spread of the disease is dependent on the interaction between the uninfected cells and the virus and the spreading speed of the virus.

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