On a new SIMVW epidemic model with vaccination and endemic testing

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Abstract. A new epidemic model referred to as SIMVW is proposed which includes the following subpopulations: susceptible human, infectious human, susceptible vector, infectious vector and vaccinated human. The transmission vector is the anopheles mosquito for the malaria disease. The disease-free equilibrium points concerning the absence of infection in humans and /or mosquitoes are defined and discussed from a stability point of view. A set of simulated examples are performed mainly related to typical endemic situations which are tested for a malaria case study in the absence and in the presence of vaccination efforts. The vaccination efforts delete susceptible numbers which are transferred to the vaccinated subpopulation and include online feedback information on the susceptible subpopulation to adjust the control effort. The vaccination effects are mainly to improve the convergence rates to the equilibrium point, to decrease the numbers of susceptible individuals while increasing those of recovered ones and to reduce the basic reproduction number so that the range of stability of the disease-free equilibrium points is larger compared to the case of absence of vaccination.

1. Introduction

In this work, a study of the stability and equilibrium points of an epidemic model has been carried out by extending a previous one in [1]. The new proposed epidemic model is referred to as an SIMVW epidemic model which includes the subsequent subpopulations: susceptible human, infectious human, susceptible vector, infectious vector and vaccinated human. The transmission vector in our investigation is the anopheles mosquito for the malaria disease. The model is analyzed and discussed using theoretical results of [2], that is, the numerical value that influences the evolution of the populations and determines whether or not the disease will spread. In particular, the values of the parameters used in the epidemic model have been based on known data on the malaria disease so that there are two populations involved: the mosquito vectors and the humans, each of them with the relevant subpopulations integrated within the whole epidemic model [3-6] and controlled via feedback of the susceptible subpopulation as it has been proposed in the literature for previous epidemic models [7-12]. In order to take into account a potential vaccination, the model used in the reference [1] has been modified by implementing in the basic differential equations a vaccination feedback effort and adding a new population to the model, namely, the subpopulation of the vaccinated humans. These facts imply the modification of the equilibrium points of the system related to the vaccination-free case, and, therefore, the need to calculate the new equilibrium points and the evaluation of their associated influence in the model dynamics. The vaccination controls involve the use of proportional feedback information on the susceptible so that the susceptible variation rate is decreased to improve in parallel that of the recovered by the same amount. Also, the basic reproduction number is below unity for larger values of the coefficient transmission rate related to the case of absence of vaccination

so that the disease-free equilibrium points have a larger stability domain for higher values of such a coefficient and small numbers at the equilibrium point.

2. The model
The basic model of Kermack and McKendrick is well- known given by:

$$
\begin{cases}
\dfrac{dS(t)}{dt} = -\,\beta S(t)I(t) \\[2mm]
\dfrac{dI(t)}{dt} = \beta S(t)I(t) - \dfrac{1}{\tau}I(t) \\[2mm]
\dfrac{dR(t)}{dt} = \dfrac{1}{\tau}I(t)
\end{cases}
$$

where $\beta > 0$ is the disease transmission rate and $\frac{1}{\tau} > 0$ is the recovery rate, with all the variables having non-negative values under non-negative initial conditions. The so-called SIMVW epidemic model that we propose is given below while it is an extension of that discussed in [1] including vaccination with feedback information:

$$
\begin{cases}
S' = \dfrac{dS(t)}{dt} = B(N)N - \mu S - b\beta VS + \gamma I - u(t), & S(0) = S_0 \geq 0 \\[2mm]
I' = \dfrac{dI(t)}{dt} = b\beta VS - (\mu + \gamma + d)I, & I(0) = I_0 \geq 0 \\[2mm]
M' = \dfrac{dM(t)}{dt} = F(T)T - \varepsilon M - b\alpha IM, & M(0) = M_0 \geq 0 \\[2mm]
V' = \dfrac{dV(t)}{dt} = b\alpha IM - \varepsilon V, & V(0) = V_0 \geq 0 \\[2mm]
W' = \dfrac{dW(t)}{dt} = u(t) - \mu W, & W(0) = W_0 \geq 0
\end{cases}
\qquad (1)
$$

where S, I, M, V y W are respectively, the subpopulations: susceptible human, infectious human, susceptible vector, infectious vector and vaccinated human subpopulations. Those subpopulations define the acronym SIMVW used to name the epidemic model, and the prime prime subscripts of the model denote time-derivatives. N is the total human population ($N = S + I + W$) and T is the total vector population ($T = M + V$). $B(N)$ and $F(T)$ are the non-linear so-called Ricker birth functions of human and vector which together with the remaining parameters ($\mu,\varepsilon,d,\alpha,\beta,\gamma,b,u(t)$) are:
- $B(N)$: nonlinear birth rate of humans
- $F(T)$: nonlinear birth rate of vectors
- μ : natural human mortaslity rate
- ε : natural vector mortality rate
- b : per-capita amount of contacts of vectors to humans
- α : transmission probability from infectious human to non-infectious vector
- β : transmission probability of infectious vector to non-infectious
- d : human disease-due mortality rate
- γ : recovery rate of infectious human
- W : vaccinated human subpopulation
- $u(t) = gS(t)$: vaccination feedback control effort
- g: control gain parameter
- $k = \dfrac{\mu}{\mu + g}$: auxiliary control gain

Summing-up separately the two subpopulation groups time-derivatives leads to
$N' = B(N)N - \mu N - dI,\ T' = F(T)T - \varepsilon T$
The nonlinear birth rates satisfy the subsequent conditions:
- $B(N) > 0; F(T) > 0.$
- $B(N),\ F(T)$ are continously- differentiable with $B'(N), F'(T) < 0.$

-   $B(0^+) > \mu > B(\infty); F(0^+) > \varepsilon > F(\infty)$.

In particular, we will use the particular functions commonly referred to as Ricker functions:

$$B(N) = c_1 \cdot e^{-a_1 N} ; F(T) = c_2 \cdot e^{-a_2 T}$$

where c_1, c_2, a_1 y a_2 are real constants subject to $c_1 > \mu$; $c_2 > \varepsilon$; $a_1 > 0$; $a_2 > 0$. The above functions offer a consistent population model since the populations sizes are taken into account while taking care of an uncontrolled growth of them and their possible incoherent negative values. The flow diagram of the model is shown in Figure 1.
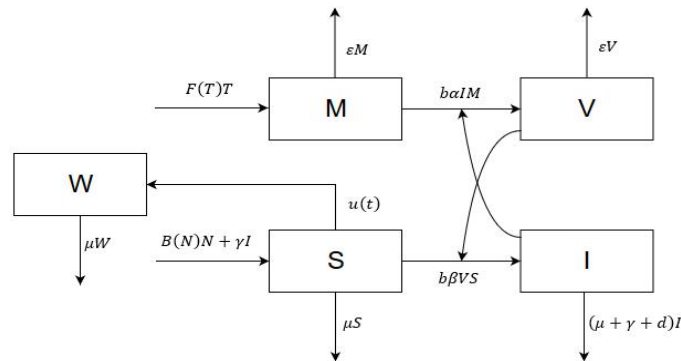

Figure 1. Flow diagram of the epidemic model.

This model is able to describe, the malaria. Now, we proceed to establish the basic reproduction number R_0 which depends on the disease parameters and the control gain g. Consider the following functions for secondary infections (F) and for the transfer rate of individuals ($\mathcal{V}$):

$$\mathcal{F} = \begin{pmatrix} \mathcal{F}_{I'} \\ \mathcal{F}_{V'} \end{pmatrix} = \begin{pmatrix} b\beta VS \\ b\alpha IM \end{pmatrix}; \mathcal{V} = \begin{pmatrix} \mathcal{V}_{I'} \\ \mathcal{V}_{V'} \end{pmatrix} = \begin{pmatrix} (\mu + \gamma + d)I \\ \varepsilon V \end{pmatrix}$$

Since $N = S + I + W = \left(1 + \frac{g}{\mu}\right)S = N^c$, then one gets $S = \frac{\mu N^c}{(\mu+g)}$ and $T = M + V = M = T^c$, where N^c and T^c are, respectively, the human and vector disease-free equilibrium values. After using a linearization around such an equilibrium point, one gets:

$$F = \frac{\partial F_i}{\partial x_j} = \begin{pmatrix} \frac{\partial F_{I'}}{\partial I} & \frac{\partial F_{I'}}{\partial V} \\ \frac{\partial F_{V'}}{\partial I} & \frac{\partial F_{V'}}{\partial V} \end{pmatrix} = \begin{pmatrix} 0 & b\beta \frac{\mu N^c}{(\mu+g)} \\ b\alpha T^c & 0 \end{pmatrix}; V = \frac{\partial \mathcal{V}_i}{\partial x_j} = \begin{pmatrix} \frac{\partial \mathcal{V}_{I'}}{\partial I} & \frac{\partial \mathcal{V}_{I'}}{\partial V} \\ \frac{\partial \mathcal{V}_{V'}}{\partial I} & \frac{\partial \mathcal{V}_{V'}}{\partial V} \end{pmatrix} = \begin{pmatrix} \mu + \gamma + d & 0 \\ 0 & \varepsilon \end{pmatrix}$$

leading to

$$FV^{-1} = \begin{pmatrix} 0 & \frac{b\beta\mu N^c}{\varepsilon(\mu+g)} \\ \frac{b\alpha T^c}{\mu+\gamma+d} & 0 \end{pmatrix}$$

and the basic reproduction number becomes:

$$R_0 = \rho(FV^{-1}) = \sqrt{\frac{b^2\alpha\beta\mu N^c T^c}{\varepsilon(\mu + \gamma + d)(\mu + g)}}$$

The value of this equation is of vital importance since it indicates the total number of secondary cases that will occur in a population of humans from a single infectious vector. In particular, an infectious individual generates, in average, less than another infectious individual if $R_0 < 1$. However, if $R_0 > 1$ then an infectious individual generates more than new one infectious individual. Note also the relevance of the vaccination control gain g (zero in the vaccination- free case) to the value of the basic reproduction number.

3. Equilibrium points

The equilibrium points of the form $P_i = (S,I,M,V,W)$ are calculated by zeroing the time-derivatives of the various subpopulations of the whole model, that is:

$$\begin{cases} B(N)N - \mu S - b\beta VS + \gamma I - gS = 0 \\ b\beta VS - (\mu + \gamma + d)I = 0 \\ F(T)T - \varepsilon M - b\alpha IM = 0 \\ \quad b\alpha IM - \varepsilon V = 0 \\ \quad gS - \mu W = 0 \end{cases}$$

The simplest equilibrium point is given by $S = I = M = V = W = 0 \;\rightarrow P_0 = (0,0,0,0,0)$. The disease-free equilibrium points occur when the infectious subpopulations are zeroed, that is,

$$\begin{cases} B(N)N - (\mu + g)S = 0 \\ F(T)T - \varepsilon M = 0 \\ gS - \mu W = 0 \end{cases} \qquad (2)$$

By using the Ricker function $B(N)$ into the first above equation and using $N = S + I + W = S + W = \left(1 + \frac{g}{\mu}\right)S = N^c$ where N^c is such that $B(N^c) = \mu$ holds, one gets:

$$c_1 \cdot e^{-a_1 N^c} N^c - \mu N^c = 0 \;\rightarrow\; e^{-a_1 N^c} = \frac{\mu}{c_1} \;\rightarrow\; N^c = -\frac{1}{a_1}\ln\left(\frac{\mu}{c_1}\right) \geq 0$$

In order to reach non-negative subpopulations, $\ln\left(\frac{\mu}{c_1}\right) < 0$ so that $c_1 > \mu$ and $a_1 > 0$. A similar procedure is performed for the second equation by replacing $F(T)$ by its Ricker-function and noting that $T = M + V = M = T^c$, where T^c satisfies $F(T^c) = \varepsilon$, so that:

$$c_2 \cdot e^{-a_2 T^c} T^c - \varepsilon T^c = 0 \;\rightarrow\; e^{-a_2 T^c} = \frac{\varepsilon}{c_2} \;\rightarrow\; T^c = -\frac{1}{a_2}\ln\left(\frac{\varepsilon}{c_2}\right) \geq 0$$

Again for non-negativity, $c_2 > \varepsilon$ and $a_2 > 0$. As a result, we get the four subsequent disease-free equilibrium points:

$$P_0 = (0,0,0,0,0), \quad P_1 = \left(\frac{\mu N^c}{(\mu+g)},0,0,0,\frac{gN^c}{(\mu+g)}\right), P_2 = (0,0,T^c,0,0), \quad P_3 = \left(\frac{\mu N^c}{(\mu+g)},0,T^c,0,\frac{gN^c}{(\mu+g)}\right)$$

where P_0 describes extinction of both human and vector populations, P_1 has uniquely susceptible and vaccinated human subpopulations, P_2 has only vector susceptible subpopulation while P_3 describes the coexistence of human subpopualtions (both susceptible and vaccinated) and a susceptible vector subpopulation. The reach ability and stability of those points is now investigated. Remember that $N = S + I + W = I + (1 + \frac{g}{\mu})S$ and $T = M + V$. After some calculations using the model equations yields:

$$\begin{cases} N' = S' + I' + W' = B(N)N - \mu N - dI \\ I' = b\beta V(N-I)\frac{\mu}{(\mu+g)} - (\mu + \gamma + d)I \\ T' = M' + V' = F(T)T - \varepsilon T \\ V' = b\alpha I(T-V) - \varepsilon V \\ W' = g(N-I) - (\mu + g)W \end{cases} \qquad (3)$$

- If $B(0^+) \leq \mu$ y $F(0^+) \leq \varepsilon$ then P_0 is locally asymptoically stable in R_+^5.
- If $B(0^+) \leq \mu$ y $F(0^+) > \varepsilon$ then P_2 is locally asymptotically stable.
- If $B(0^+) > \mu$ and $F(0^+) \leq \varepsilon$ then P_1 is locally asymptotically stable.
- For the case when $B(0^+) > \mu$ and $F(0^+) > \varepsilon$ our system can have any of the equilibria $(P_0, P_1, P_2 \; y \; P_3)$.

It is now studied how the disease-free equilibrium point behaved for values of R_0 larger than and less than unity by assuming that $B(0^+) > \mu$ and $F(0^+) > \varepsilon$. To this end, the following set of differential equations, which is equivalent to that of the basic model, is used:

$$
\begin{cases}
S' = B(N)N - (\mu + g)(N - I - W) - b\beta VS + \gamma I \\
I' = b\beta V(N - I - W) - (\mu + \gamma + d)I \\
M' = F(T)T - b\alpha IM - \varepsilon(T - V) \\
V' = b\alpha IM - \varepsilon V \\
W' = g(N - I) - (\mu + g)W
\end{cases}
\tag{4}
$$

By calculating the Jacobian matrix of (4) with respect to the equilibrium point P_3, we get the Jacobian $J_{P_3}(S,I,M,V,W)$ and its eigenvalues:

$$
\begin{pmatrix}
B'(N^c)N^c & B'(N^c)N^c + \mu + \gamma + g & 0 & -kb\beta N^c & B'(N^c)N^c + \mu + g \\
0 & -(\mu + \gamma + d) & 0 & kb\beta N^c & 0 \\
0 & -b\alpha T^c & F'(T^c)T^c & F'(T^c)T^c + \varepsilon & 0 \\
0 & b\alpha T^c & 0 & -\varepsilon & 0 \\
0 & -g & 0 & 0 & -(\mu + g)
\end{pmatrix}
$$

$\lambda_1 = B'(N^c)N^c < 0$; $\lambda_2 = F'(T^c)T^c < 0$, $\lambda_3 = -(\mu + g) < 0$,

$$
\lambda_{4,5} = \frac{-(\varepsilon + x) \pm \sqrt{(\varepsilon + x)^2 - 4\varepsilon x(1 - (R_0)^2)}}{2}, \lambda_4 \lambda_5 = \varepsilon x(1 - (R_0)^2)
$$

If $R_0 > 1$ then $\lambda_4 \lambda_5 < 0$. Thus, one of the eigenvalues has a positive real part. Therefore, the equilibrium point P_3 is hyperbolically unstable. If $R_0 < 1$ then $\lambda_4 < 0$ y $\lambda_5 < 0$ and P_3 is locally asymptotically stable. On the other hand, by summing-up all the equations (1) $F(T) = \varepsilon$, with $F(0^+) > \varepsilon$, $F(\infty) < \varepsilon$ and $F'(T) < 0$. Then, $T = T^c$ since T has a unique positive root. By noting that $N = S + I + W$ by summing-up the equations referred to the human subpopulations), one gets:

$$
S = \frac{(\mu + d - B(N))N}{d}, I = \frac{(B(N) - \mu)N}{d}
\tag{5}
$$

where $\mu < B(N) < \mu + d$ to ensure that $S, I > 0$. By taking into account the Ricker functions $B(N)$, one gets $z < N < N^c$, where $z = \frac{-1}{a_1} \ln\left(\frac{\mu + d}{c_1}\right)$. After some simple calculations, one gets also for $T = T^c$:

$$
M = T^c - V, V = \frac{(\mu + d + \gamma)(B(N) - \mu)}{b\beta(\mu + d - B(N))}
$$

$$
f(N) = (\mu + d + \gamma)[(B(N) - \mu)Nb\alpha + d\varepsilon] - T^c b^2 \alpha \beta(\mu + d - B(N))N = 0
$$

$$
f'(N) = [(\mu + d + \gamma)b\alpha + T^c b^2 \alpha \beta]B'(N)N - \frac{(\mu + d + \gamma)\varepsilon d}{N} < 0
$$

since $f'(N) < 0$. To get the solution of (1), one considers the positive range of solutions (z, N^c):

$$
f(z) = \begin{cases} (\mu + d + \gamma)d[\varepsilon + b\alpha z] > 0, si\ B(0^+) > (\mu + d) \\ (\mu + d + \gamma)d\varepsilon > 0, si\ B(0^+) \le (\mu + d) \end{cases}, f(N^c) = (\mu + d + \gamma)d\varepsilon(1 - R_0)
$$

for $R_0 < 1$, $f(N^c) > 0$. Therefore, there is no positive root in the interval (z, N^c). That means that the system has no positive equilibrium point of infectious population. If $R_0 > 1$ then $f(N^c) < 0$. This implies that the system has a unique positive root in the interval (z, N^c). Therefore, there is a unique equilibrium point which is given by $P_4 = (S^*, I^*, M^*, V^*, W^*)$, where S^*, I^*, M^*, V^* y W^* are:

$$
S^* = \frac{(\mu + d - B(N^*))N^*}{d}, I^* = \frac{(B(N) - \mu)N^*}{d}, W^* = \frac{g(\mu + d - B(N^*))N^*}{\mu d}, M^* = T^c - V^*, V^* = \frac{(\mu + d + \gamma)(B(N^*) - \mu)}{b\beta(\mu + d - B(N^*))}
$$

Note that the equilibrium values of the various subpopulations depend on the control parameter g. Therefore, they are dependent on the existence of vaccination effort ($g \ne 0$) or not ($g = 0$).

4. Simulated examples

The subsequent simulated study is based on the endemic malaria disease evolution. The model parameters are given in $days^{-1}$. In particular, $\mu = \frac{1}{83 \cdot 365} \approx 3.30 \cdot 10^{-5}$ which corresponds to an average life expectation of 83.09 years (Spain, 2017). There are more than two thousands of mosquito

species. In average, they live from one week to a month. We consider here, an average of two weeks of life for a mosquito leading to $\varepsilon = \frac{1}{2 \cdot 7} \approx 7.14 \cdot 10^{-2}$. One chooses the following transmission probabilities from human infectious to non-infectious vector (α) and from infectious vector to non-infectious human (β): $\alpha = 0.50, \beta = 0.50$. For instance in the dengue case, the typical values would be $\alpha = \beta = 0.75$. The amount of per-capita contacts are in the range $b \in (0.2, 0.8)$. The chosen value is $b = 0.80$. Some simulatons have been performed by varying this value. The infectious-human mortality rate (d) and infectious-human recovery rate (γ), the last one varies in the range $\gamma \in (0.01, 0.05)$. The used values are $d = 0.002$ and $\gamma = 0.05$. The relative values of mosquito to human have been taken from two to one. The initial conditions are $S(0) = 1$, $I(0) = 0$ and $W(0) = 0$. That is, we do not have initially infectious and vaccinated individuals. The initial subpopulations of mosquito are $M(0) = 1.99$ with a single infectious individual, $V(0) = 0.01$. In order that the growing human and mosquito rates $B(N)$ and $F(T)$ agree with values found in the background literature. For mosquito the birth rates are $F(T) \in (0.036, 42.5)$ per day. For humans, the fertility rate in Spain, according to data published by the World Health Organization (WHO), Spanish acronym: OMS, for 2017, was 1.3 per person and year. Then, c_1, c_2, a_1 and a_2 are chosen such that the population model is appropriate so that they approach initially $B(N)$ and $F(T)$ to their respective estimated rates, so that $c_1 = 0.003$; $c_2 = 0.537$; $a_1 = a_2 = 1$. The ranges of values of the control gain from no vaccination to full vaccination effort is $g \in [0,1]$. Table 1 gives the values of parameters in en $days^{-1}$ and the initial conditions:

Table 1. Parameter values and initial conditions.

subpopulation numbers	Value
susceptible human S(0)	1
Infectious human I(0)	0
susceptible vector M(0)	1.99
infectious vector V(0)	0.01
vaccinated human W(0)	0
human birth rate B(N)	$1.10 \cdot 10^{-3}$
vector birth rate F(T)	$7.27 \cdot 10^{-2}$
human mortality rate μ	$3.30 \cdot 10^{-5}$
vector mortality rate ε	$7.14 \cdot 10^{-2}$
transmission probability: infectious human–healthy vector α	0.50
transmission probability: infectious vector healthy-human β	0.50
human recovery propability γ	0.05
infectious human mortality probability d	0.002
per capita contacts b	0.80
Vaccination gain g	Varying

The values of $B(N)$ and $F(T)$ in Table 1 are the initial ones. Then, they follow their expressions accordingly to the evolution of the subpopulations. The simulations are performed via the Scilab package. Unless some particular specification be given, the parameters and initial values are those of Table 1 and the time range is ($t = 0:0.01:100$) implying 10^4 iterations. In Figure 2, there is no vaccination, i.e. $g = 0$. We observe that for a single infectious vector, the human/vector infectious grow very fast. This is expected since the basic reproduction number is $R_0 \approx 19.74 > 1$. In order to observe the values of the equilibrium subpopulations, the simulation is repeated with more iterations, (10^6). See Figure 3. It is seen that both infectious subpopulations reach endemic equilibria, the human equilibrium population been small due to the low values of $B(N)$ comapred to the mortality rate d. Next, we apply vaccination effort with $g = 0.05$. See Figure 4. Note that the infectious human subpopulations reduce significantly. By comparing Figures 2 and 3, note that the infectious vector subpopulation also becomes reduced.
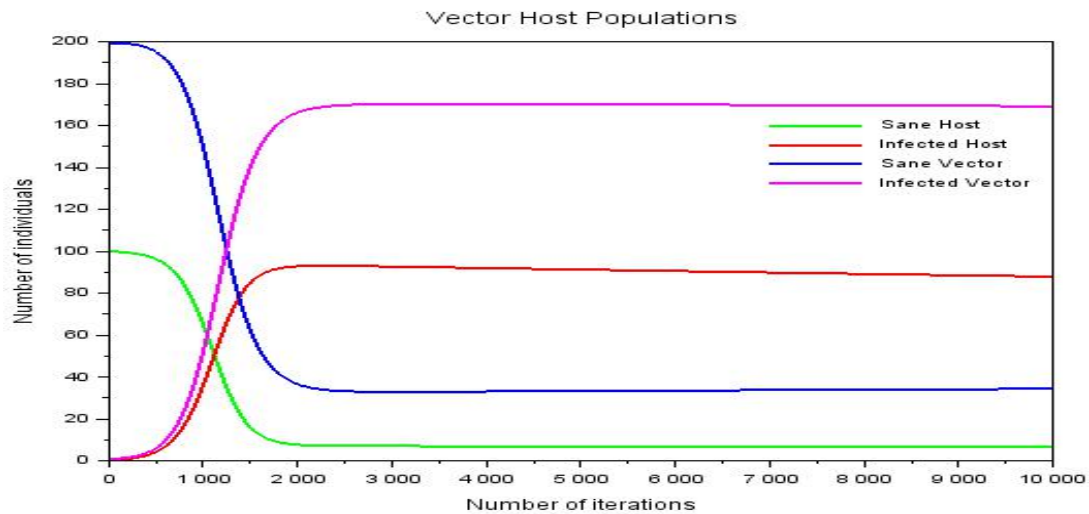
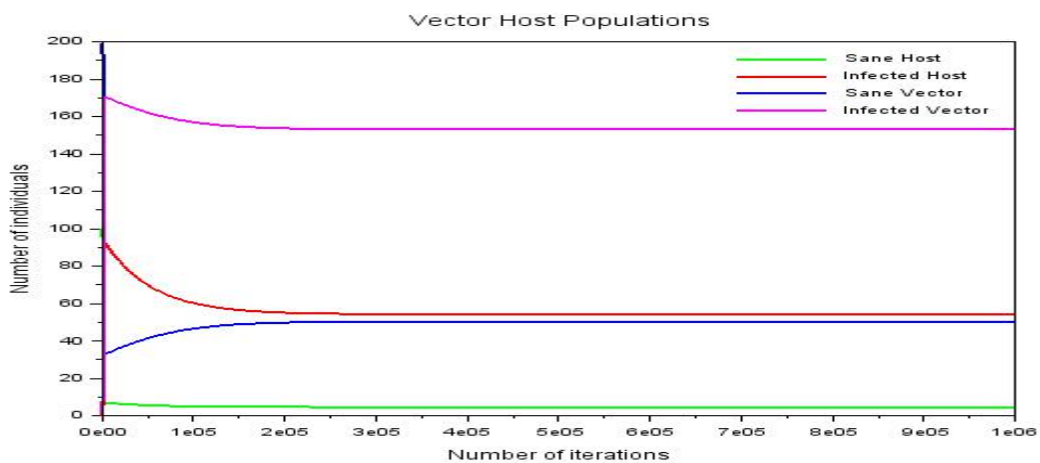**Figure 2.** Subpopulations evolution in the vaccination-free case.

Figure 3. Subpopulations evolution in the vaccination-free case for a larger observation time.
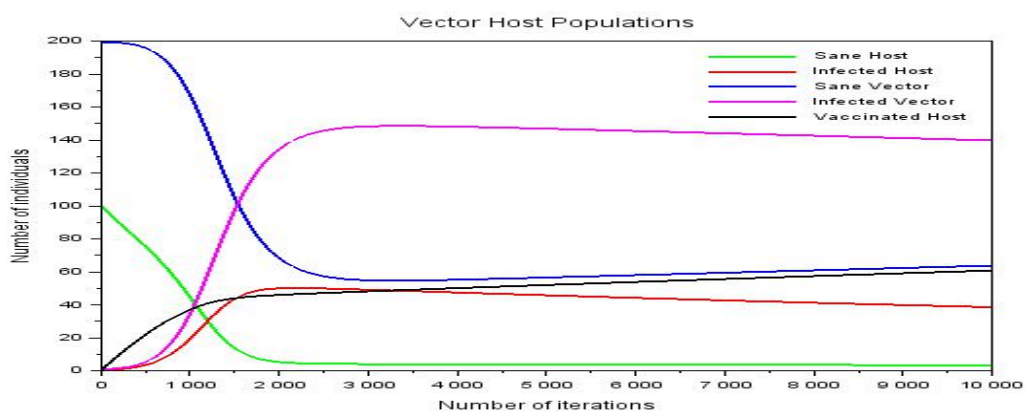

Figure 4. Subpopulations evolution with a vaccination effort of 5%.

Figure 5 is appropriate to compare the subpopulations of vector and host without and with vaccination. R_0 decreases with a vaccination effort of 5% to yield $R_0 \approx 1.60 > 1$. Still the disease becomes endemic in this case but with a lower intensity exhibition. See Figure 5 (continuous portraits).

Figure 5. Subpopulations evolution with a vaccination effort of 5% (continuous portrait) and in the vaccination-free case (discontinuous portrait).
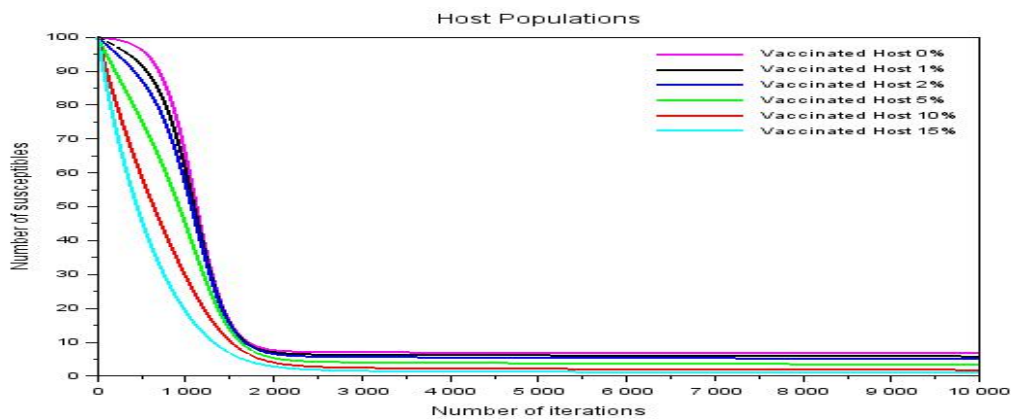

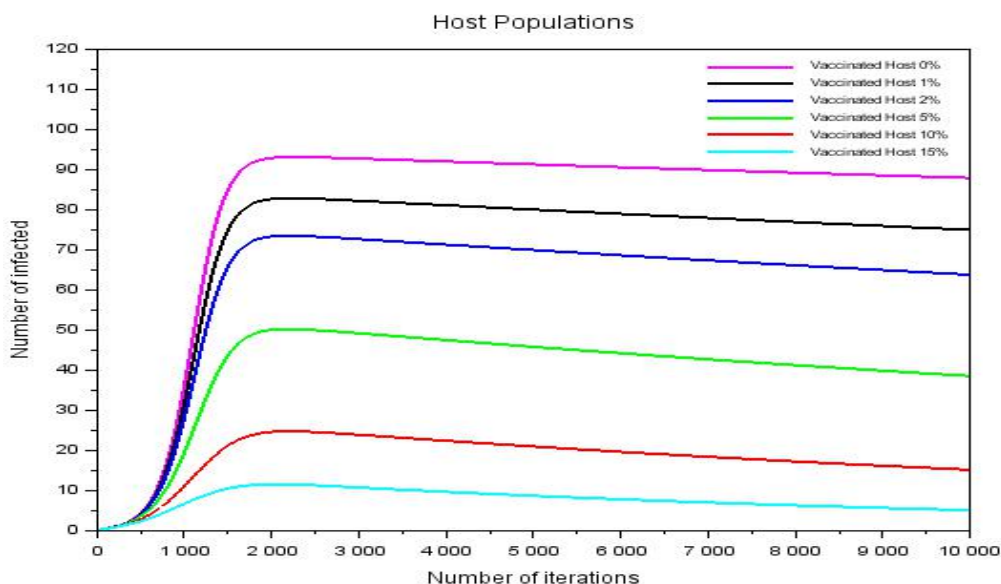**Figure 6.** Human susceptible subpopulation versus vaccination effort.

Figure 7. Human infectious subpopulation versus vaccination effort.

In the subsequent Figures 6, 7 and 8 the evolution of the percentages of the subpopulations is represented versus the vaccination effort in the endemic situation.

In Figure 6, it is seen how the susceptible numbers decrease faster as the vaccination effort increases to get smaller equilibrium numbers at the equilibrium steady-state. Figure 7 indicates that

larger vaccination efforts give smaller amount of infectious humans as expected. Figures 8 and 9 display, respectively, the growing rates of the human vaccinated populations versus vaccination efforts and some mixed human- vector subpopulations depending on the vaccination efforts.
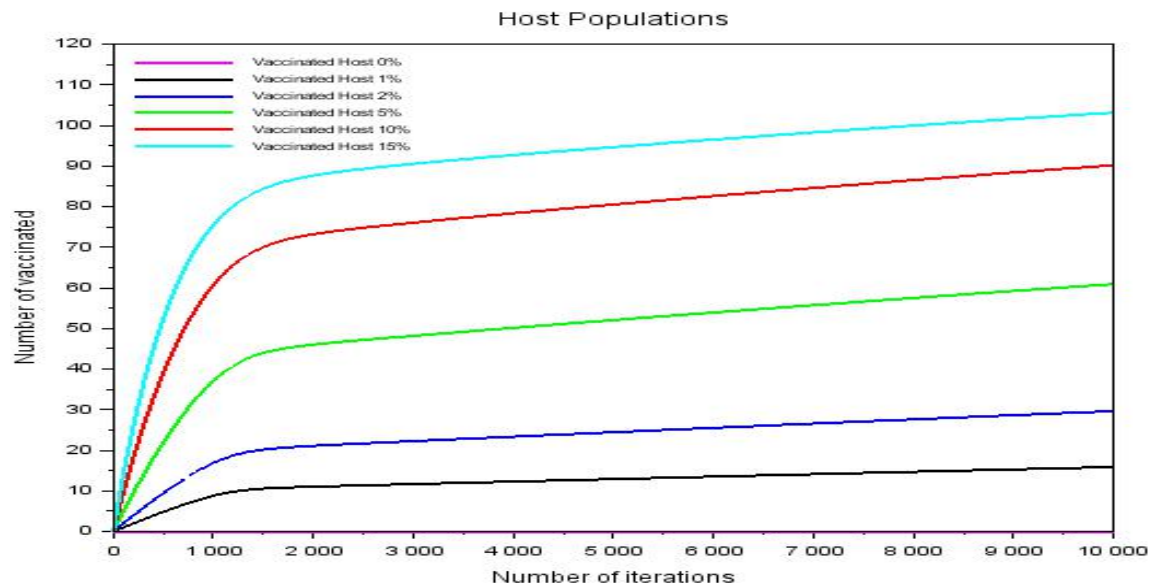

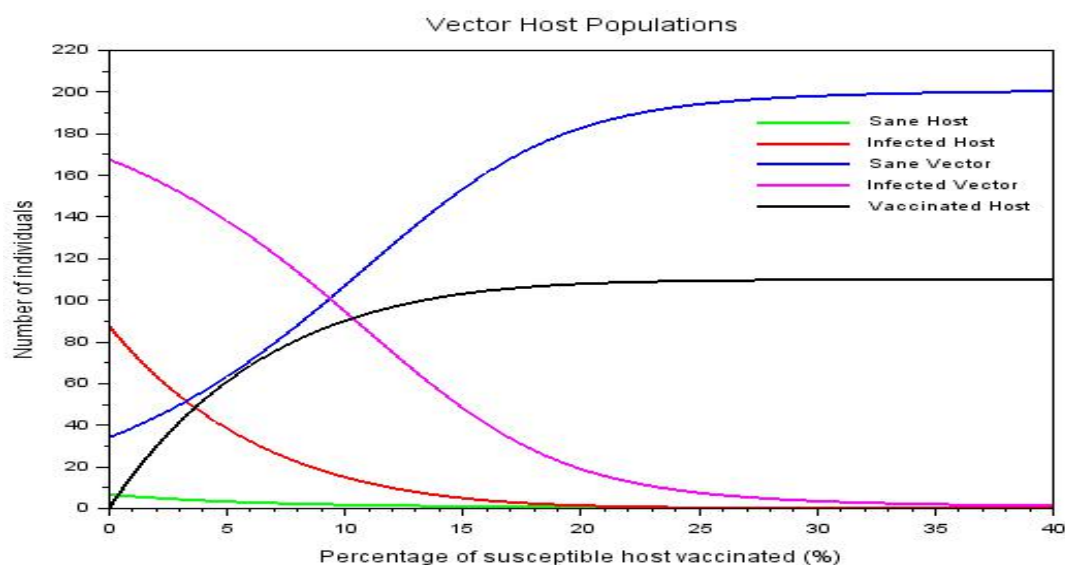**Figure 8.** Human vaccinated subpopulation versus vaccination effort.

Figure 9. The various subpopulations versus the vaccination effort.

As it has been observed, at higher values of vaccination we would see populations of healthy mosquitoes increasing, since the transmission of infection between vectors will be slower. This occurs since the human population is rapidly vaccinated and a large number of infectious humans are not generated. In Figure 10, the case of convergence to a disease- free steady- state is displayed In this case the reproduction number is less than unity and in particular the coefficient transmission rate takes values under a critical values which corresponds to a basic reproduction number equalizing unity.
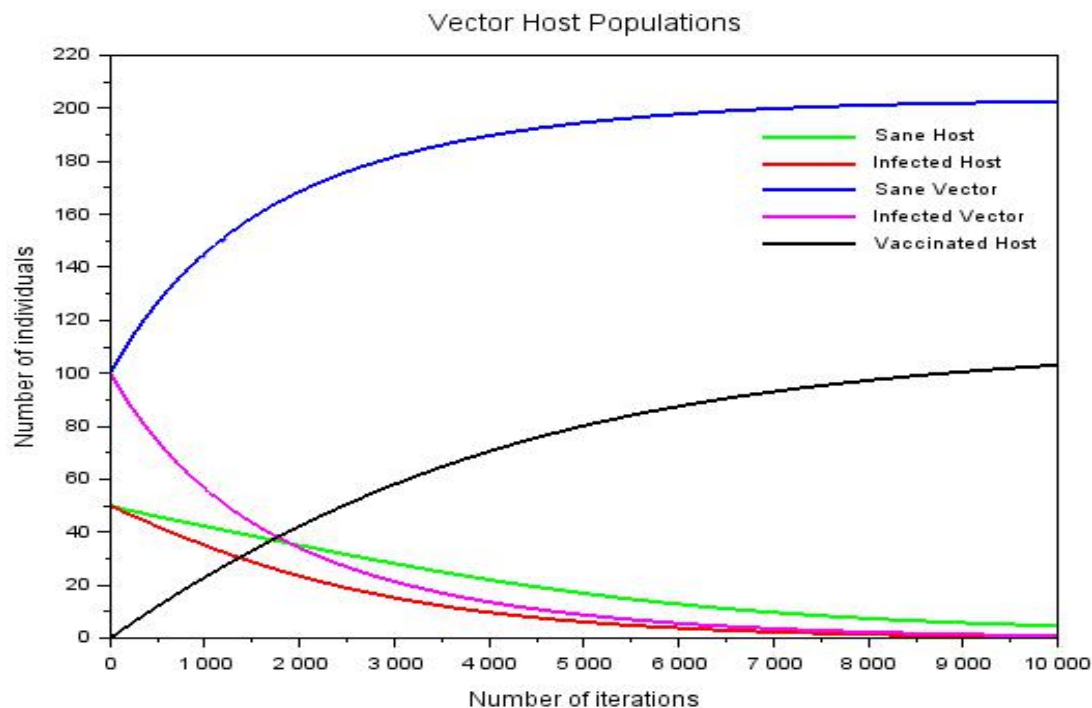
Figure 10. Convergence to the Disease- free equilibrium with =b/2=0.1, g=0.05.

5. Results and Discussion

The basic reproduction number of our model has been calculated while demonstrating that it depends on the vaccination control used, its value being lower the greater the control parameter used. This fact allows the infection to be eliminated asymptotically with higher tolerated virulence rates of the infection if it is vaccinated than if it is not. It has been concluded that, if both vector and human mortality rates are greater than their respective birth rates then a stable equilibrium is reached in which populations disappear. It has also been demonstrated analytically that a disease-free equilibrium will be reached as long as the number of reproduction is less than than one that leading to the disease-free equilibrium point to be asymptotically stable. The latter has also been tested experimentally. For values of this number exceeding the unity value, it has been found that there is a positive balance since a single infectious individual will be replaced by more than one new infectious case. This endemic equilibrium state exists and it is stable, presenting non-zero values of infectious and susceptible populations in equilibrium. For small coefficient transmission rates under a certain critical value which corresponds to the basic reproduction number equalizing unity, the deqauiolibrium state is disease-free and the infectious number reach the zero value.

6. Conclusion

In this paper, we have studied a deterministic epidemic model that allows to representing the transmission dynamics of infectious diseases between two species. In particular, the relationship between human populations and vectors against the disease of malaria has been analyzed mainly in the endemic steady-state. A vaccine-dependent function of the population of susceptible humans has also been incorporated into the model which has a term of proportional feedback information to the susceptible. The proportionality constant is a control parameter which can be used for design so as to improve the equilibrium numbers and the convergence rates. The simulation results have displayed endemic equilibrium situations with nonzero numbers of infectious subpopulations if the basic reproduction number exceeds unity and disease- free equilibrium steady- states if such a number is below unity.

Acknowledgments

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