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Bifurcation analysis of vertical transmission model with preventive strategy

Gosalamang Ricardo Kelatlhegile*, **Moatlhodi Kgosimore**

Department of Basic Sciences, Botswana College of Agriculture, Private Bag 0027, Gaborone, Botswana

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Abstract We formulate and analyze a deterministic mathematical model for the prevention of a disease transmitted horizontally and vertically in a population of varying size. The model incorporates prevention of disease on individuals at birth and adulthood and allows for natural recovery from infection. The main aim of the study is to investigate the impact of a preventive strategy applied at birth and at adulthood in reducing the disease burden. Bifurcation analysis is explored to determine existence conditions for establishment of the epidemic states. The results of the study showed that in addition to the disease-free equilibrium there exist multiple endemic equilibria for the model reproduction number below unity. These results may have serious implications on the design of intervention programs and public health policies. Numerical simulations were carried out to illustrate analytical results.

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1. Introduction

Mathematical models have become important tools in analyzing the spread and control of infectious diseases [1]. Understanding the transmission characteristics of infectious diseases in communities, regions and countries may lead to implementation of better approaches of mitigating against infections or epidemics. In particular, mathematical models are useful in building and testing theories, and in comparing, planning, implementing and evaluating various detection, prevention, therapeutic and control programs [1]. The results of such studies may contribute to formulation of appropriate public health policies and guide the design of other relevant studies and development of methods for data collection.

The phenomena of backward bifurcation usually expressed as a graph of the equilibrium infective population size I in terms of the basic reproduction number $\Re_0$ arise from backward

* Corresponding author. Tel.: +267 74116522; fax: +267 3928753.
E-mail address: gkelatlhegile@bca.bw, gkelatlhegile@gmail.com (G.R. Kelatlhegile).
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transfer of individuals back to the susceptible population or multiple groups and asymmetry between groups or multiple interaction mechanisms [2]. Studies of bifurcation analysis were explored in a number of epidemic models of which a few are cited herein, to characterize the behavior of equilibria, make predictions on the outcome of the epidemic and derive conditions for prevention and control of diseases [2–9]. These studies demonstrated existence of multiple positive endemic equilibria for values of the basic reproduction number below unity and a backward bifurcation when the reproduction number is one, particularly in models of vaccination [7,10–14] and/or vaccination and treatment [15].

Vertical transmission can be accomplished through transplacental transfer of disease agents (bacteria, viruses, parasites) from the mother to an embryo, fetus, or baby during pregnancy or childbirth [16]. In most studies, it is considered less important compared to horizontal transmission, because infectiousness and parturition usually occurs at different times [17]. A number of studies incorporating vertical transmission investigated the effects of various epidemiological and demographical factors on the disease transmission [2,14,17–19]. For instance, Busenbuerg and Cooke [18] constructed and analyzed various compartmental models with vertical transmission to obtain insight on the role of vertical transmission in disease epidemics. It is noted that vertical transmission alone may not cause an epidemic and there is a certain threshold above which it may or may not contribute the epidemic [19]. Thus, it is important to include vertical transmission in models to evaluate the extent to which it may contribute to the epidemic and appropriately inform design of public health policy.

Xue-Zhi et al. [8] constructed and analyzed an *SIS* model with limited resource for treatment. The study established occurrence of backward bifurcations due to insufficient capacity for treatment as well as existence of bistable endemic equilibria as a result of limited resources. Kribs-Zaleta and Velasco-Hernandez [7] analyzed a simple two dimensional SIS model with vaccination and found that the model exhibited backward bifurcation for some parameter values. Furthermore, the results indicated that a vaccination campaign meant to reduce the disease reproduction number below unity may fail to control the disease. van den Driessche and Watmough [9] found multiple stable equilibria which exhibited backward bifurcation and hysteresis for an *SIS* epidemic model with non-constant contact rate. Yicang and Hanwu [13] formulated an *SIS* model with pulse vaccination to study its dynamical behavior and established that the pulse vaccination was more effective than the proportional vaccination. Li and Ma [20] considered an *SIS* epidemic model with vaccination, temporary immunity, and varying total population size and derived three threshold parameters that govern the dynamics of the disease. Gao and Hethcote [17] considered an *SIS* model with density-dependent demographics which incorporated the effects of vertical transmission and derived contact and growth thresholds that characterized the transmission dynamics of the disease.

Most studies reviewed in this paper did not consider multiple group targeted interventions and also ignored effects of vertical transmission. In this paper, we modify the models in [2,3] to formulate and analyze a mathematical model appropriate for the implementation of prevention strategy at birth and susceptible stage for a disease transmitted horizontally and vertically. We assume that the disease upon recovery does not induce permanent immunity and recovered individuals become susceptible. Following [2], we employ elementary methods to establish bifurcation behavior based on the number of solutions of a quadratic equation. The major distinction between our work and those reviewed are: (i) the incorporation of vertical transmission and (ii) the implementation of preventive strategies targeting two groups of susceptibles (newly borns and susceptible adults).

The organization of this paper is as follows. Section 2 provides model formulation and analysis. In Section 3, we investigate the existence of equilibria of the model. In Section 4, we investigate the stability analysis of the model. In Section 5 some numerical simulations are displayed in detail and close with a discussion in Section 6.

2. Model formulation and analysis

2.1. Model formulation

We consider an epidemic model with preventive strategies (e.g. vaccination, educational campaigns) [10] that incorporates the effects of vertical transmission. We formulate a mathematical model consisting of four compartments of susceptibles ($S(t)$), vaccinated susceptibles ($V(t)$), (comprising individuals protected against infection), infectives ($I(t)$) (assumed to be infectious) and the recovered ($R(t)$). The total host population is given by $N(t) = S(t) + V(t) + I(t) + R(t)$. The susceptible class is replenished through recruitment or births of unvaccinated individuals at a constant rate $(1-\theta\epsilon)\pi_s$ and through births or recruitment of infection-free individuals from infected individuals at a rate $b\omega(1-\theta\epsilon)$. A proportion $\theta, (0 \le \theta \le 1)$ of new susceptible individuals are vaccinated and the vaccine produces a protective immunological response at rate ϵ, $(0 \le \epsilon \le 1)$ of those vaccinated. This process results in a fraction $1-\theta\epsilon$ of new susceptibles entering the susceptible population, while the fraction $\theta\epsilon$ enters the vaccinated (protected) population. The susceptible individuals are vaccinated at a constant rate ψ and also enters the vaccinated compartment. The susceptible population acquires infection at the rate βIS, where β is the infectivity rate. A proportion ω $(0 \le \omega \le 1)$ of new births are born infected through mother-to-child transmission (MTCT) and the remaining $(1-\omega)$ infection-free newly borns are subjected to vaccination at a rate $\theta\epsilon$. Since vaccination induces protection among those vaccinated, we assume that vaccinated individuals acquire infection at a discounted rate $\rho\beta I$, $(0 \le \rho \le 1)$, where $\rho = 0$ means vaccine is perfect and $\rho = 1$ means that the vaccine is useless. We assume vaccine-induced immunity decays exponentially at a constant rate σ. All the compartments are subjected to natural mortality at per capita rate μ. Infected individuals further experience excess mortality due to infection at a constant rate δ. Infected class recover with temporary immunity at a constant rate γ and join a class of the recovered. Individuals in the recovered class loose their immunity at a constant rate α and return to the pool of susceptibles. The above description leads to the following system of differential equations:

$$\begin{aligned}
\dot{S}(t) &= \pi_s(1-\theta\epsilon) + b(1-\omega)(1-\theta\epsilon)I \\
&\quad -\beta IS - (\mu+\psi)S + \alpha R + \sigma V, \\
\dot{V}(t) &= \pi_s\theta\epsilon + \psi S + b(1-\omega)\theta\epsilon I - \rho\beta IV - (\mu+\sigma)V, \\
\dot{I}(t) &= b\omega I + \beta IS + \rho\beta IV - (\mu+\gamma+\delta)I, \\
\dot{R}(t) &= \gamma I - (\mu+\alpha)R.
\end{aligned} \tag{1}$$

Adding the equations of system (1), we obtain an equation describing changes in the total population over time t given by

$$\dot{N}(t) = \pi_s - \mu N - \tau I, \tag{2}$$

where $\tau = \delta - b > 0$, is the net growth rate of the infectives. For biological considerations, we study a problem governed by the invariant set Ω, defined below.

2.1.1. Invariant region

The vertical transmission model (1) shall be analyzed in a biologically meaningful feasible region given by

$$\Omega = \left\{ (S,\ V,\ I,\ R) \in \mathbf{R}_+^4 \ :\ N \ \leq \ \frac{\pi_s}{\mu} \right\}.$$

To show that all solutions of system (1) are uniformly bounded in a proper subset $\Omega \subset \mathbf{R}_+^4$. Let $(S,\ V,\ I,\ R) \in \mathbf{R}_+^4$, be any solution with a non-negative initial conditions. Using the theorem of differential inequality cited in [21],

$$\limsup_{t\to\infty} S(t) \leq \frac{\pi_s}{\mu},$$

the time derivative of the total population size N along the solution path

$$\dot{N}(t) = \pi_s - \mu N - \tau I,$$

where

$$\tau = \delta - b,$$

reduces to

$$\dot{N} = \pi_s - \mu N - \tau I \leq \pi_s - \mu N,$$

which upon integration yields,

$$N \leq \frac{\pi_s}{\mu} + N_0 e^{-\mu t},$$

resulting in

$$0 \leq N \leq \frac{\pi_s}{\mu}$$

as $t \to \infty$.

Therefore, the problem is well-posed in the invariant set

$$\Omega = \left\{ (S,\ V,\ I,\ R) \in \mathbf{R}_+^4 \ :\ N \ \leq \ \frac{\pi_s}{\mu} = k \right\}.$$

Thus, Ω is positively invariant and it is sufficient to consider solutions in Ω. Hence, all solutions of system (1) starting in Ω remain in Ω for all $t \geq 0$. All parameters and state variables for model system (1) are assumed to be non-negative for $t \geq 0$ since it monitors changes in the human population.

3. Equilibria and bifurcation analysis

To obtain the steady state solutions of system (1), we set the right hand side of the system to zero, so that

$$\begin{aligned}
&\pi_s(1-\theta\epsilon) + b(1-\omega)(1-\theta\epsilon)I^* - (\mu+\psi) \\
&\quad +\beta I^*)S^* + \alpha R^* + \sigma V^* = 0, \\
&\pi_s\theta\epsilon + \psi S^* + b(1-\omega)\theta\epsilon I^* - \rho\beta I^* V^* - (\mu+\sigma)V^* = 0, \\
&b\omega I^* + \beta I^* S^* + \rho\beta I^* V^* - (\mu+\gamma+\delta)I^* = 0, \\
&\gamma I^* - (\mu+\alpha)R^* = 0.
\end{aligned} \tag{3}$$

Assuming

$$\mu + \gamma + \delta - b\omega > 0, \quad \text{and defining} \quad R_{0V} = \frac{\beta}{\mu+\gamma+\delta-b\omega},$$

as the basic reproductive number under vertical transmission (demographic replacement number) [12,19] or the average number of secondary cases caused by one primary case through birth during its infectiousness, then from the third equation of (3), we have

$$I^* = 0, \tag{4}$$

or

$$R_{0V}S^* + \rho R_{0V}V^* = 1 \quad \text{or simply} \quad R_{0V}S^* = 1 - \rho R_{0V}V^*. \tag{5}$$

3.1. Disease-free equilibrium

Solution (4) leads to the disease-free equilibrium with coordinates

$$E_0 \quad = \quad \left[\left(\frac{\sigma + \mu(1-\theta\epsilon)}{\mu+\psi+\sigma} \right)k,\ \left(\frac{\psi + \mu\theta\epsilon}{\mu+\psi+\sigma} \right)k,\ 0,\ 0 \right]. \tag{6}$$

3.1.1. The model reproduction number

The fact that we have a population which is not completely susceptible, we shall speak of an effective reproduction number instead of the basic reproduction number. We shall use $\Re_{0P}$ denote the effective reproduction number of model (1) and the effective reproduction number is given by

$$\Re_{0P} = \left(\frac{\mu+\sigma+\rho\psi}{\mu+\sigma+\psi} \right)(1-\theta\phi)kR_{0V}, \tag{7}$$

where $\phi = \dfrac{\mu\epsilon(1-\rho)}{\mu+\sigma+\rho\psi}$.

From Eq. (7) we have determined the amplification factors and thresholds ϕ, $\Re_{0P}$, and R_{0V}, characterizing and defining the generation of secondary infections, and the impact of preventive strategies in the disease transmission dynamics. The threshold quantity $\Re_{0P}$ defines the effective reproduction number, which is the average number of secondary infectives produced by an infected individual either horizontally or vertically in the presence of prevention programs. The parameter ϕ is commonly called the prevention impact and it summaries the measure of

prevention imperfections [11]. The critical prevention coverage necessary for elimination of the disease is given by

$$\theta_c = \left(\frac{1}{\chi\phi}\right)\left(\chi - \frac{1}{kR_{0V}}\right).$$

In the absence of vertical transmission result (7) reduces to

$$\Re_{0P}^{V} = \chi(1-\theta\phi)R_0,$$

which yields a critical prevention coverage level for elimination of the disease

$$\theta^* = \left(\frac{1}{\phi}\right)\left(\chi - \frac{1}{R_0}\right),$$

where $\chi = (\frac{\mu+\sigma+\rho\psi}{\mu+\sigma+\psi})$ and $R_0 = \frac{k\beta}{\mu+\gamma+\delta}$.

The parameter R_0, denotes the basic reproductive number, which is the average number of secondary cases caused by one primary case introduced into a population that is wholly susceptible [12,22].

In absence of vaccination at the susceptible stage ($\psi = 0$) the effective reproduction number (7) reduces to

$$\hat{\Re}_{0P} = (1-\theta\phi)R_0,$$

which yields the critical prevention coverage level in the absence of vaccination

$$\hat{\theta}^* = \left(\frac{1}{\phi}\right)\left(1 - \frac{1}{R_0}\right). \tag{8}$$

This result is analogous to the findings in [7,11,12].

3.2. Endemic equilibrium

Solution (5) leads to the endemic equilibrium in terms of I^* given by

$$E_1 = \left[\frac{1}{R_{0V}} - \rho V^*, V^*, I^*, \frac{\gamma I^*}{\mu+\alpha}\right], \tag{9}$$

where

$$V^* = \frac{b(1-\omega)R_{0V}\theta\epsilon I^* + \pi_s\theta\epsilon R_{0V} + \psi}{R_{0V}(\mu+\sigma+\rho\omega+\rho\beta I^*)}.$$

To obtain a closed form solution for the endemic equilibrium, we eliminate S^* and V^* in result (5). This yields in term of R_{0V} and $\Re_{0P}$ the quadratic polynomial in I given by

$$-k_2R_{0V}I^{*2} + k_1(R_{0V}-\varphi)I^* + k_0(\Re_{0P}-1) = 0, \tag{10}$$

where $k_2 = \rho\beta[\mu(\mu+\gamma+\alpha) + (\mu+\alpha)\tau]$, $k_1 = (\mu+\alpha)[\mu\rho\beta k + b\theta\epsilon(1-\omega)(\sigma+\rho\mu+\rho\psi)]$,

$k_0 = \mu(\mu+\alpha)(\mu+\sigma+\rho\psi)$ and

$$\varphi = \frac{\mu\rho\beta}{\mu\rho\beta k + b\theta(1-\omega)(\mu+\rho\mu+\rho\psi)}.$$

The threshold parameter φ can be interpreted as the critical value at which the severity effect of vertical transmission changes.

From result (10), we obtain

$$I^* = F_{\pm}(R_{0V}, \Re_{0P}) = F_{\pm}(\bullet), \tag{11}$$

$$F_+(\bullet) = \frac{k_1(R_{0V}-\varphi) + \sqrt{k_1^2(R_{0V}-\varphi)^2 + 4k_0k_2R_{0V}(\Re_{0P}-1)}}{2k_2R_{0V}} \tag{12}$$

and

$$F_-(\bullet) = \frac{k_1(R_{0V}-\varphi) - \sqrt{k_1^2(R_{0V}-\varphi)^2 + 4k_0k_2R_{0V}(\Re_{0P}-1)}}{2k_2R_{0V}}. \tag{13}$$

We want to establish the feasibility of the steady states (11) in the regions $\Re_{0P} \le 1$ and $\Re_{0P} > 1$.

It is evident that as $\Re_{0P}$ approaches one, the system settles to either the disease-free state governed by

$$I^* = F_-(\bullet) = 0, \tag{14}$$

irrespective of the values of R_{0V} or a unique endemic steady state governed by

$$I^* = F_+(\bullet) = \frac{k_1(R_{0V}-\varphi)}{k_2R_{0V}}, \text{ provided } R_{0V} > \varphi. \tag{15}$$

It is worth-noting that when $R_{0V} = \varphi$, solution (13) collapses to the disease-free equilibrium to coalesce with solution (12). In the case $\Re_{0P} > 1$, the discriminant dominates the term $k_1(R_{0V}-\varphi)$, leading to a positive solution characterized by $I^* = F_+(\bullet)$ and a negative solution given by $I^* = F_-(\bullet)$, thus there exists a unique endemic equilibrium for the model. However, if $\Re_{0P} < 1$ and $R_{0V} > \varphi$ then Eq. (10) has two positive solutions $I^* = F_{\pm}(\bullet)$, while if $R_{0V} < \varphi$ there are two negative solutions. This result can be summarized as follows:

Theorem 3.1. *Consider Eq. (10):*

1. *If $\Re_{0P} > 1$ there exists a unique endemic equilibrium state corresponding to $I^* = F_+(\bullet)$ for all values of $R_{0V} > 0$.*
2. *If $\Re_{0P} = 1$ there exists a unique endemic equilibrium state corresponding to $I^* = F_+(\bullet)$ satisfying $R_{0V} > \varphi$ and coalesce with the disease-free equilibrium at $R_{0V} = \varphi$.*
3. *On the other hand, if $R_{0P} < 1$ and $R_{0V} > \varphi$ then there exist two endemic equilibrium points corresponding to $I^* = F_{\pm}(\bullet)$. Otherwise, there are none.*

3.3. Backward bifurcation

Consider Eq. (10), we think of R_{0V} as a variable with the other parameters and thresholds as constants. Here we wish to carry out qualitative analysis of the equilibria curve in the neighborhood of the critical threshold $\Re_{0_p} = 1$. Through implicit

differentiation of (10) with respect to R_{0V}, we get

$$(k_1(R_{0V}-\varphi)-2k_2R_{0V}I)\frac{dI^*}{dR_{0V}} = k_2I^2 - k_1I^* - k_0k\chi(1-\theta\phi). \tag{16}$$

At $I^*=0$, we have

$$k_1(R_{0V}-\varphi)\frac{dI^*}{dR_{0V}} = -k_0k\chi(1-\theta\phi) < 0. \tag{17}$$

If the sign of $[\frac{dI^*}{dR_{0V}}]_{I^*=0}$ and $R_{0V}-\varphi) > 0$ then backward bifurcation will occur, while if $(R_{0V}-\varphi) < 0$ a unique endemic equilibrium will exist (forward bifurcation). It worth noting that I^* is a linear function in R_{0V}, hence the bifurcation curve increases as R_{0V} increases. The bifurcation curve has a positive slope at equilibrium values with $(R_{0V}-\varphi) > 0$ and negative slope if $(R_{0V}-\varphi) < 0$. To gain more insight into the transmission dynamics of the disease, we derive the critical value, $\Re^*_{0P}$ for the saddle-node bifurcation, which relates to the appearance or disappearance of two positive equilibria in the backward bifurcation framework. To compute this critical value, we solve the equation

$$k_1^2(R_{0V}-\varphi)^2 + 4k_0k_2R_{0V}(\Re_{0P}-1) = 0,$$

which is equivalent to

$$\Re_{0P} = 1 - \frac{k_1}{4k_0R_{0V}}(R_{0V}-\varphi)^2 = \Re^*_{0P} < 1. \tag{18}$$

Qualitative bifurcation diagram describing the backward bifurcation for Eq. (10) are depicted in Fig. 1 for selected parameter values. The critical value $\Re^*_{0P}$ is nonlinear function of R_{0V}. The dependence of bifurcation parameter $\Re_{0P}$ on R_{0V} (linear dependence) demonstrate complications that may result in persistence of the epidemic in the host population under conditions where the disease should be clearing from the population. The results show that backward bifurcation occurs when $\Re_{0P}$ is below unity. Implications of this result is, the existence of the equilibrium state depends on the sizes of the different sub populations of the model, leading to persistence of the disease in the community. Meanwhile, reducing $\Re_{0P}$ below the saddle-node bifurcation value , may lead to the reduction of the disease. Therefore, determining this sub-thresholds may have a crucial significance in prevention of the disease in the host population.

Implication of these results demonstrate that in the region $\Re_{0P} < \Re^*_{0P}$ the disease clears from the population, while in the region $\Re_{0P} \geq \Re^*_{0P}$ the disease persists. The study reveals that either $\Re_{0P} < \Re^*_{0P}$ or $R_{0V} = \varphi$ and $\Re_{0P} = 1$ are sufficient conditions for the elimination of the disease from the host population. These results show that vertical transmission plays a crucial role in the persistence of the epidemic, that is, even under preventive strategies capable of reducing the reproduction number below unity, the epidemic may fail to be controlled. Hence, these results confirms the findings in [2,3,7].

4. Stability analysis

The Jacobian matrix of system (1) at the disease-free equilibrium, $E_0 = (S^*, V^*, 0, 0)$, is given by

$$J_{E_0} = \begin{pmatrix} A & B \\ \mathbf{0} & C \end{pmatrix} \tag{19}$$

with

$$A = \begin{pmatrix} -(\mu+\psi) & \sigma \\ \psi & -(\mu+\sigma) \end{pmatrix},$$

$$B = \begin{pmatrix} b(1-\omega)(1-\theta\epsilon) - \beta S^* & \alpha \\ b(1-\omega)\theta\epsilon - \rho\beta V^* & 0 \end{pmatrix}$$

and

$$C = \begin{pmatrix} -(\mu+\gamma+\delta-b\omega)(1-R_{0V}) & 0 \\ \gamma & -(\mu+\alpha) \end{pmatrix},$$

where S^* and V^* are as given in (5). The eigenvalues of J_{E_0} are obtained by solving the characteristic equations of matrices A and C. Thus, we solve $|A-\lambda I| = 0$ and $|C-\lambda I| = 0$, yielding $\lambda_1 = -\mu$, $\lambda_2 = -(\mu+\sigma+\psi)$, $\lambda_3 = -(\mu+\alpha)$ and $\lambda_4 = -(\mu+\gamma+\delta-b\omega)(1-R_{0V})$, respectively. Hence, $\lambda_4 < 0$ when $R_{0V} < 1$.

Clearly, all eigenvalues of J_{E_0} have negative real parts only if $R_{0V} < 1$. The stability of the disease-free equilibrium point can be summarized with the following theorem.

Theorem 4.1. *The disease-free equilibrium point is locally asymptotically stable if* $R_{0V} < 1$ *and unstable if* $R_{0V} > 1$.

5. Numerical simulation

In order to support and complement the analytical results, we simulate system (1) through the implementation of different vaccination programs, by considering different assumptions on

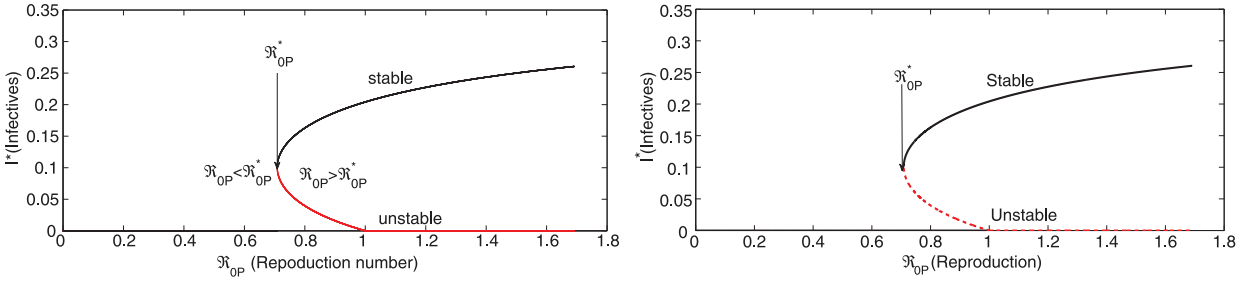

Fig. 1 Qualitative bifurcation diagrams for the backward bifurcation. The bifurcation parameter is the control reproduction number $\Re_{0P}$.

Table 1 Parameter values.

Parameters	Parameters values	Citation
π_s	10000	Estimated
ψ	0.05	[11]
ρ	0–1	[11]
β	0.012	[11]
σ	0.8	Estimated
δ	0.5	[11]
μ	0.02	[23]
ϵ	0.7	[19]
γ	0.5	Estimated
b	0.003	Estimated
α	0.5	Estimated
ω	0.04	[11]
θ	0.08	[11]

measures of efficacy (the degree of protection), the rates of recovery and the role of vertical transmission. We considered a prevention strategy that targeted new recruits (or newly born individuals) and susceptible population (adulthood). The parameter values in Table 1 below were used in the simulation. This parameter values were extracted from the existing literature and those not available were estimated.

Variation on the level of protection ρ, induced by the preventive strategy shows that increasing ρ has the effect of increasing the proportion vaccinated while reducing the proportion of the infected population. Increasing the proportion of newly born individuals infected through vertical transmission has the effect of increasing endemicity. The results of the study further show that in the early stages of infection the disease rapidly develops into an epidemic Fig. 2(a) before settling at an endemic equilibrium level. Furthermore, the study shows that the epidemic can be sustained through vertical transmission. Thus, vertical transmission has the potential of increasing the transmission. The result depicted in Fig. 2(b) reveals that an increase in ρ has the effect of reducing the proportion of the infected population and increasing the vaccinated population.

6. Discussion

To discuss the findings of this paper we make reference to two key threshold parameters that describe the transmission dynamics of the disease. The effective reproduction number, $\Re_{0P}$, which measures the average number of secondary cases generated by an infectives individual introduced in a population where a proportion of individuals are subjected to preventative strategy at birth as well as at adulthood. The threshold parameter, R_{0V} characterizing the generation of secondary infectives due to demographic replacement of infected individuals due to the presence of vertical transmission.

The results of this study can provide an insights on resurgence of some diseases such as STI, HIV/AIDS despite the low incidence of diseases. The study further, demonstrates the potential for resurgence of traces of epidemic in situations at which the preventive programs are expected to mitigate against the epidemics. The study revealed that even if $\Re_{0P}$ is reduced below unity, there still can exist an endemic equilibrium mainly characterized by the presence of vertical transmission. Implications of the study are that there is a critical threshold, parameter φ below which the role vertical transmission is insignificant and above which the role of vertical transmission is significantly increasing the burden of disease infection. The study identified two sufficient conditions for the elimination of the disease in the host population: (*i*) the effective reproduction number $\Re_{0P}$ below the critical value for the saddle-node bifurcation ($\Re_{0P} < \Re^*_{0P}$) and (*ii*) the effective reproduction number at unity, coupled with demographic replacement threshold at some critical value ($R_{0V} = \varphi$). These results concurs with most of backward bifurcation analysis studies, except for an additional condition emanating from vertical transmission [2,3,7]. These results advocates for multiple interventions on the control of infections transmitted both horizontally and vertically. The study reveals challenges that blanket implementation of preventive strategies and pauses a critical question of targeted intervention on the mode of transmission. The other critical question is the level of intervention: target susceptible population visa infected population. For instance, in case of HIV treatment of infectives

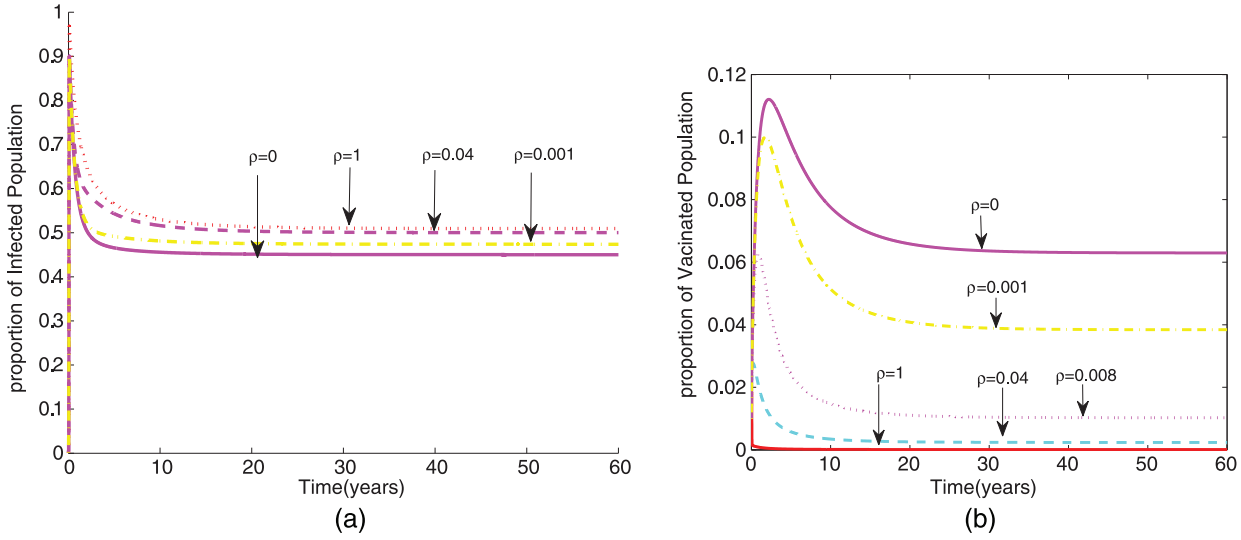

Fig. 2 We vary ρ and fixed the other parameter values: $\beta = 0.012$; $b = 0.03$; $\gamma = 0.5$; $\pi = 10000$; $\sigma = 0.5$; $\delta = 0.3$; $\epsilon = 0.5$; $\alpha = 0.5$; $\omega = 0.4$; $\psi = 0.5$; $\theta = 0.8$; $\mu = 0.02$.

results have been found to induce prevention on the population at risk, while educational campaigns and male circumcision render protection to the susceptible population.

The result have implications regarding the complexity control strategies, that are non-discriminating and are able to bring down the epidemic levels to zero, while maintaining the pool of infected individuals in the population with less negative impact in disease transmission dynamics, particularly on diseases without cure.

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References

[1] H.W. Hethcote, A thousand and one epidemic models, Front. Math. Biol. (1994) 505–515.
[2] F. Brauer, Backward bifurctions in simple vaccination/treatment models, J. Biol. Dyn. 5 (2011) 410–418.
[3] B. Buonomo, D. Lacitignola, On the backward bifurcation of a vaccination model with nonlinear incidence, Nonlinear Anal. Model. Control 16 (2011) 30–46.
[4] G. Li, Z. Jin, Bifurcation analysis in models for vector-borne diseases with logistic growth, Sci. World J. (2014) 9.
[5] B. Buonomo, C. Vargas-De-Leon, Stability and bifurcation analysis of a vector-bias model of malaria transmission, Math. Biosci. 242 (2013) 59–67.
[6] D.H. Knipl, G. Rost, Backward bifurcation in SIVS model with immigration of non-infectives, Biomath 2 (2013) 1312051. http://dx.doi.org/10.11145/j.biomath.2013.12.051.
[7] M.C. Kribs-Zaleta, X.J. Velasco-Hernandez, A simple vaccination model with multiple endemic states, Math. Biosci. 164 (2000) 183–201.
[8] X.-Z. Li, W.-S. Li, M. Ghosh, Stability and bifurcation of an SIS epidemic model with treatment, Chaos Solitons Fractals 42 (2009) 2822–2832.
[9] P. van den Driessche, J. Watmough, A simple epidemic model with backward bifurcation, J. Math. Biol. 40 (2000) 525–540.
[10] S. Blower, K. Koelle, J. Mills, Modeling: epidemic control, HIV vaccines, and risky behavior, in: E.H. Kaplan, R. Brookmeyer (Eds.), Qualitative Evaluation of HIV Prevention Programs, Yale University Press, 2002, pp. 260–289.
[11] A.R. McLean, S.M. Blower, Imperfect vaccines and herd immunity to HIV, Proc. Biol. Sci. 253 (1993) 9–13.
[12] A. Scherer, A. McLean, Mathematical models of vaccination, Br. Med. Bull. 62 (2002) 187–199.
[13] Z. Yicang, L. Hanwu, Stability of periodic solutions for an SIS model pulse vaccination, Math. Comput. Model. 38 (2003) 299–308.
[14] D. Long, Z. Xiang, On the study of an SEIV epidemic model concerning vaccination and vertical transmission, J. Appl. Math. Bioinform. 1 (2011) 21–30.
[15] Z. Hu, W. Ma, S. Ruan, Analysis of SIR epidemic models with nonlinear incidence rate and treatment, Math. Biosci. 238 (2012) 12–20.
[16] M.Y. Li, H.L. Simth, L. Wang, Global dynamics of an SEIR epidemic model with vertical transmission, SIAM J. Appl. Math. 62 (2001) 58–69.
[17] L.Q. Gao, H.W. Hethcote, Disease transmission models with desity-dependent demographics, Math. Biol. 30 (1992) 717–731.
[18] S. Busenberg, K. Cooke, Vertically transmitted diseases, Models and Dynamics: Biomathematics, 23, Springer-Verlag, Berlin, 1993.
[19] M. Kgosimore, E.M. Lungu, The effects of vertical transmission on the spread of HIV/AIDS in the presence of treatment, Math. Biosci. Eng. 23 (2006) 297–312.
[20] J. Li, Z. Ma, Qualitative analyses of SIS epidemic model with vaccination and varying total population size, Math. Comput. Model. 20 (2002) 1235–1243.
[21] C.P. Bhunu, W. Garira, Z. Mukandvire, Modeling HIV/AIDS and tuberculosis co-infection, Bull. Math. Biol. 71 (2009) 1745–1780.
[22] R.M. Anderson, R.M. May, Infectious Disease of Humans: Dynamics and Control, Oxford Univeristy Press, Oxford, UK, 1991.
[23] R. Naresh, A. Tripathi, Modelling and analysis of HIV–TB co-infection in a variable size population, Math. Model. Anal. 10 (2005) 275–286.