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IMPACT OF HUMAN BEHAVIOR ON ITNs CONTROL STRATEGIES TO PREVENT THE SPREAD OF VECTOR BORNE DISEASES

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ABSTRACT. The use of insecticide-treated nets (ITNs) is known to be one of the best preventive mechanisms for mosquito-borne human diseases by avoiding infective mosquito bites. However, the effective use of them is affected by human behavior which is further fueled by the persuasive power of those who object their use. Taking this into account, we propose a mathematical model for mosquito-borne diseases transmission that considers the effect of information dependent human behavior in the use of ITNs. The model is mathematically analyzed to determine the basic reproduction number, and to study equilibria and their stability. Moreover, optimal control theory is applied to the model and optimal strategy for implementing health-promotion campaigns is derived. Numerical simulations to the optimal control problem show that as the power of persuasion of the anti-ITNs use group becomes larger than those of the pro–ITNs use group in the population, the burden of the disease becomes more challenging and its control becomes more costly.

1. Introduction

In many countries in the world, the burden of infectious diseases in general and of mosquito-borne diseases in particular remains very important and constitutes one of the most significant public health problem. Mosquito-borne diseases are human illnesses caused by an infectious microbe that is transmitted to human by blood-sucking mosquitoes. Malaria, dengue fever, yellow fever, zika, encephalitis, filariasis, West Nile fever and chikungunya are some of such diseases known to affect human population (Himeidan *et al.* 2012).

The major vector-borne diseases, together, account for more than 17% of all infectious diseases, causing more than 700,000 deaths annually worldwide; but the highest incidence and mortality rates are reported in sub-Saharan Africa. More than 3.9 billion people in over 128 countries are at risk of contracting dengue, with 96 million cases estimated per year. Malaria causes more than 400,000 deaths every year globally, most of them children under 5 years of age. Many of these diseases are preventable through informed protective measures (*World Health Organization. Vector-borne diseases, Fact sheet* n.d.).

For most of the mosquito-borne diseases, such as West Nile fever, dengue, and chikungunya, no vaccines are available to prevent disease and no specific drugs are available for treatment. Therefore, preventing infective mosquito bites, is the best way to prevent

infection (*CDC. Joint Statement on Insect Repellents from the Environmental Protection Agency and the Centers for Disease Control and Prevention* 2014). Until better vaccine and vector-control options are available, the best way to prevent most mosquito-borne diseases is to avoid mosquito bites, mainly through the use of insecticide-treated nets (ITNs). The use of ITNs for protection against mosquito bites have proven to be a practical and cost-effective intervention with high impact in malaria prevention (Nevill *et al.* 1996). ITNs are virtually side-effect free and can be used at any geographical place (Lengeler 2004; Hanson *et al.* 2009). According to the WHO, more people at risk of malaria in Africa are sleeping under an ITN. In 2016, 54% of the population was protected by ITNs, with an increase from 30% in 2010 (*World Health Organization. World malaria report* 2017).

Even though the distribution of ITNs to the population has been scaled up due to the universal access policy of the WHO, it seems that awareness creation is not made at the same scale. Some researchers have indicated that in some places there is an insufficient knowledge in the population of the link between mosquito bites and a mosquito-borne disease, and as to who should be the main users of nets (Belay and Deressa 2008).

Among the general reasons for not using ITNs, especially for young children, there are hot weather, a tendency to sleep outdoors and lack of mosquito nuisance (Frey *et al.* 2006). Moreover, many care takers believe that children get too hot or fear sleeping under the nets or the use of the net disturbs their sleep (Alaii *et al.* 2003) and therefore, try to influence others not to use ITNs. Mild and reversible paraesthesia has regularly been reported from persons having been in unprotected contact with the insecticide or with ITNs (Barlow *et al.* 2001). In some places there are also complaints regarding burning sensation experienced upon sleeping under the net at night, thereby discouraging people from patronizing the ITNs (*Ghana Web. Health news from Ghana and health information,* Dry insecticide mosquito nets before usage 2015). Moreover, some debate on the safety of frequent exposure to low concentrations of pyrethroids continued, especially after evidence for an irreversible and cumulative effect of pyrethroid on nerve tissue in animal models was published in 1984 (Kolaczinski and Curtis 2004).

In summary, many studies have found evidence that the effectiveness of ITNs is largely influenced by human behavioral factors. Therefore, a more realistic modeling approach to ITN use should include the role of human behavior (and misbehaviors).

In this paper we introduce a behavioral change model (BCM) to assess the impact of human behavior on ITNs use. The BCMs are a major tool in the new field of *behavioral epidemiology* (Manfredi and d'Onofrio 2013; Wang *et al.* 2016), where the key issue is the investigation of the interplay between human behavior and the spread of infectious diseases.

In Agusto *et al.* (2013), a malaria model including ITNs use was proposed, where the dynamics of human and vector populations are described by two linked SIR epidemic models and the ITNs use is represented by a constant coverage. A BCM version of the model in Agusto *et al.* (2013) was later on considered in Buonomo (2014, 2015) by employing the idea of information–dependent epidemic models (Manfredi and d'Onofrio 2013).

More precisely, it is assumed that the contact rate depend on a *goodwill* index $w(t)$, which can be interpreted as concern or the willingness to use ITNs. The goodwill, in turn, depends on the current and the past history of disease prevalence in the community. Therefore, the goodwill index is an analogous of the *information index*, often employed in epidemic models including self–protective actions (as vaccination or social distancing) (d'Onofrio

et al. 2007; Buonomo *et al.* 2008; d'Onofrio *et al.* 2008; d'Onofrio and Manfredi 2009; Buonomo *et al.* 2012, 2013; d'Onofrio and Manfredi 2016).

However, as it has been pointed out in Buonomo *et al.* (2018a), the BCMs that are based on the information index do not take into account the two important and opposite phenomena that are widely investigated in the public health and epidemiology literature on vaccination (and that may be relevant also for ITNs adoptions). From one hand, the awareness concerning the status of the disease in the community and the benefits of adopting ITNs, which increase the propensity to use ITNs. On the other hand, the information and rumors on the ITNs, which produces a propensity reduction.

A consequence is that, from the mathematical point of view, the propensity to use self–protections (as vaccine or, in this paper, ITNs) has to be a state variable (Bauch 2005; d'Onofrio *et al.* 2011, 2012; Buonomo *et al.* 2018a).

Therefore, in this paper we modify the malaria model with ITNs use considered in Agusto *et al.* (2013) by following the imitation–game approach as described in Buonomo *et al.* (2018a). We consider a population where it is possible to distinguish among individuals who are willing to use ITNs and, in fact, they adopt it, and individuals that are against ITNs use (we denote the fraction of the two groups at time t with $w(t)$ and $a(t)$, respectively). The *imitation* game follows a double *contagion of ideas* process (Wang *et al.* 2016) and includes also the action of Public Health (PH) agencies in influencing the perceptions regarding both ITNs use and disease consequence. The game produces a dynamic equation for $w(t)$.

The paper is organized as follows: The model is introduced in Section **2**. Section **3** is devoted to equilibria existence and stability. A sensitivity analysis is performed in **4** and the optimal control problem regarding the actions enacted by PH systems is formulated in Section **5**. Conclusions are given in Section **6**.

2. The model

2.1. Host–vector model with ITN usage. The basic system we consider in this paper is the classical host–vector epidemic model in the version presented in Agusto *et al.* (2013), where the effects of ITN usage on the transmission of the disease is taken into account. The human population N_h, and vector populations N_v, are both divided into two disjoint compartments, given by susceptible and infectious individuals. Therefore, for $t \geq 0$,

$$N_h(t) = S_h(t) + I_h(t), \quad N_v(t) = S_v(t) + I_v(t), \tag{1}$$

where S_h, I_h, S_v and I_v denote, respectively, susceptible humans, infectious humans, susceptible vectors and infectious vectors. The dynamics is ruled by the following system of nonlinear ordinary differential equations:

$$\begin{aligned} \dot{S}_h &= \Lambda_h - \lambda_h(b)S_h - \mu S_h + \delta I_h \\ \dot{I}_h &= \lambda_h(b)S_h - (\alpha_d + \mu + \delta)I_h \\ \dot{S}_v &= \Lambda_v - \lambda_v(b)S_v - \eta(b)S_v \\ \dot{I}_v &= \lambda_v(b)S_v - \eta(b)I_v, \end{aligned} \tag{2}$$

where the upper dot denotes the time derivative. The terms λ_h and λ_v denote the *forces of infection* on humans and on vectors, respectively. That is, the per capita rate at which susceptible individuals contract the infection (Keeling and Rohani 2011). The infection rate

TABLE 1. Description of parameters in model (2) and baseline values (taken from Agusto *et al.* (2013)).

Parameter	Description	Baseline value
Λ_h	Immigration rate in humans	$10^3/(70\times 365)$
Λ_v	Immigration rate in mosquitoes	$10^4/21$
μ	Natural mortality rate in humans	$1/(70\times 365)$
η	Natural mortality rate in mosquitoes	$1/21$
η_{bn}	Maximum ITN-induced death rate in mosquitoes	$1/21$
α_d	Disease–induced death rate in humans	10^{-3}
p_1	Prob. of disease transm. from mosquito to human	variable
p_2	Prob. of disease transm. from human to mosquito	variable
$\beta_{\max}$	Maximum host–vector contact rate	0.6
$\beta_{\min}$	Minimum host–vector contact rate	0.005
δ	Recovery rate of infectious humans to be susceptible	1/4
α	Rate of persuasion by the anti-ITNs groups	variable
θ	Rate of persuasion by the pro-ITNs groups	variable

per susceptible human and per susceptible vector are given, respectively, by

$$\lambda_h(b) = p_1\beta(b)\frac{I_v}{N_h}, \quad \text{and} \quad \lambda_v(b) = p_2\beta(b)\frac{I_h}{N_h}, \tag{3}$$

where $\beta(b)$ represents the human–mosquito contact rate and the parameter $b \in [0,1]$ is the proportion of ITNs usage. The functions β and η are specified below. All the other parameters in (2) are positive constants and their meaning is described in Table 1.

Using bed nets reduces the probability for humans to be bitten. Moreover, the nets are treated with insecticide. Therefore, in Agusto *et al.* (2013) the following two main assumptions are made:
(i) ITNs usage reduces the human–mosquito contact rate and this is described by the relation

$$\beta(b) = \beta_{\max} - b\,(\beta_{\max} - \beta_{\min})\,, \tag{4}$$

where $\beta_{\max}$ and $\beta_{\min}$ are the maximum and the minimum contact rate, respectively.
(ii) ITNs usage increases the mosquito death rate η. This is modeled by

$$\eta(b) = \eta + \eta_{bn}b, \tag{5}$$

where η_{bn} is a non negative constant and $\eta_{bn}\,b$ represents the death rate due to insecticide on treated bed-nets.

2.2. Imitation dynamics. Following the imitation–game approach described in Buonomo *et al.* (2018a), we consider a population where it is possible to distinguish among individuals who are willing to use ITNs and, in fact, they adopt it, and individuals that are against ITNs use. We denote the measure of such individuals at time t with $W(t)$ and $A(t)$, respectively. The fractions of the two groups at time t is denoted with $w(t) = W(t)/N_h(t)$

and $a(t) = A(t)/N_h(t)$, respectively, where N_h is given in (1), and therefore $w(t) + a(t) = 1$ for all t.

The *imitation* game is a double *contagion of ideas* process (Wang *et al.* 2016):

$$\begin{aligned} \dot{w} &= -\alpha a w + \theta w a \\ \dot{a} &= \alpha a w - \theta w a. \end{aligned} \tag{6}$$

In practice, the opinions of the anti-ITNs group exert an influence on the other group described by a *force of persuasion* of the type

$$F_a = \alpha\, a,$$

and those of the ITNs favourable group have a force of persuasion on the anti-ITNs group of the type:

$$F_w = \theta\, w.$$

The transmission rates from one group to the other, α and θ, are positive constants.
In a seminal paper by Bauch (Bauch 2005), this imitation game approach is applied to the reciprocal influence of pro- and anti-vaccination groups. Bauch writes directly an imitation game equation in which it is implicitly assumed that the transmission rate from group A to W is amplified by the perception of the disease-related adverse events, which results in the assumption that θ is in reality an increasing function of the prevalence of the disease, I_h. In Bauch (2005), the rate α is assumed to be constant (but it could depend on w, instead).

The action of Public Health (PH) authorities can be modeled as convincing people in the anti-ITNs group to use bed-nets. As first approximation, it can be modeled as an *additional* transfer rate from the group that has no propensity to use ITNs to the group that has propensity to use it, yielding:

$$\begin{aligned} \dot{w} &= -\alpha a w + \theta w a + \gamma(t) a \\ \dot{a} &= \alpha a w - \theta w a - \gamma(t) a, \end{aligned} \tag{7}$$

where $\gamma(t)$ is a positive function that, generally speaking, captures the effectiveness of actions enacted by the PH agencies (as information, education, distribution of ITNs, etc.) in influencing the perceptions regarding both ITNs and disease consequences. These various actions enacted by the PHS induce a flux (from the group A to the group W) which is different from the one generated by the exchange of ideas typical of the imitation-games.

Since $a = 1 - w$, one can write down the following extension of an imitation-game equation:

$$\dot{w} = w(1-w)(\theta - \alpha) + \gamma(t)(1-w), \tag{8}$$

We will analyze the model in the simplest case where $\gamma(t) = \gamma > 0$, whereas in Section **5** the function $\gamma(t)$ will be assumed to be a control variable and obtained as output of an optimal control problem.

We now couple the equation (8) with the model (2) to obtain:

$$\begin{aligned} \dot{S}_h &= \Lambda_h - \lambda_h(w) S_h - \mu S_h + \delta I_h \\ \dot{I}_h &= \lambda_h(w) S_h - (\alpha_d + \mu + \delta) I_h \\ \dot{S}_v &= \Lambda_v - \lambda_v(w) S_v - \eta S_v \\ \dot{I}_v &= \lambda_v(w) S_v - \eta I_v, \\ \dot{w} &= (\theta - \alpha) w(1-w) + \gamma(1-w). \end{aligned} \tag{9}$$

where

$$\lambda_h(w) = p_1\beta(w)\frac{I_v}{N_h}; \qquad \lambda_v(w) = p_2\beta(w)\frac{I_h}{N_h}, \tag{10}$$

and

$$\beta(w) = \beta_{\max} - w(\beta_{\max} - \beta_{\min}). \tag{11}$$

The initial conditions for the system at time $t = 0$ are all non-negative and such that $w(0) \in (0,1]$.

3. Equilibria and stability

First of all, note that from (1) and (9) it follows that $\dot{N}_h = \Lambda_h - \mu N_h - \alpha_d I_h$, and $\dot{N}_v = \Lambda_v - \eta N_v$, for $t \geq 0$. Since $\alpha_d I_h \geq 0$, it follows that $\dot{N}_h \leq \Lambda_h - \mu N_h$. Then it can be shown by comparison theorem that $N_h(t) \leq \tilde{N}_h$, and $N_v(t) \leq \tilde{N}_v$, for all $t \geq 0$, where $\tilde{N}_h = \Lambda_h/\mu$ and $\tilde{N}_v = \Lambda_v/\eta$ are the carrying capacities. It can be also immediately checked that $w(0) \in (0,1)$ implies $w(t) \in (0,1)$ for all $t \geq 0$.

Therefore, the set

$$\Omega = \{(S_h, I_h, S_v, I_v, w) \in \mathbf{R}^5 \mid 0 \leq N_h \leq \tilde{N}_h,\ 0 \leq N_v \leq \tilde{N}_v,\ 0 < w \leq 1\},$$

is positively invariant and attractive.

Depending on the coverage of the use of the ITNs in the human population and the impact of the already adopted individuals, model (9) may admit two disease–free equilibria (DFE): a *disease-free and full–ITNs utilization equilibrium*,

$$\mathscr{E}_{o1} = \left(\frac{\Lambda_h}{\mu}, 0, \frac{\Lambda_v}{\eta}, 0, 1\right),$$

where everyone in the population adopts the ITNs perfectly and a *disease-free and negative-impact–ITNs equilibrium*,

$$\mathscr{E}_{o2} = \left(\frac{\Lambda_h}{\mu}, 0, \frac{\Lambda_v}{\eta}, 0, \frac{\gamma}{\alpha - \theta}\right),$$

where $0 < \gamma < \alpha - \theta < 1$.

The Jacobian matrix corresponding to system (9) is

$$J = \begin{pmatrix} -\frac{p_1\beta(w)I_vI_h}{N_h^2} - \mu & \frac{p_1\beta(w)I_vS_h}{N_h^2} + \delta & 0 & -\frac{p_1\beta(w)S_h}{N_h} & \frac{p_1\tilde{\beta}I_vS_h}{N_h} \\ \frac{p_1\beta(w)I_vI_h}{N_h^2} & -\frac{p_1\beta(w)I_vS_h}{N_h^2} - \alpha_o & 0 & \frac{p_1\beta(w)S_h}{N_h} & -\frac{p_1\tilde{\beta}I_vS_h}{N_h} \\ \frac{p_2\beta(w)S_vI_h}{N_h^2} & -\frac{p_2\beta(w)S_vS_h}{N_h^2} & -\frac{p_2\beta(w)I_h}{N_h} - \eta & 0 & \frac{p_2\tilde{\beta}I_hS_v}{N_h} \\ -\frac{p_2\beta(w)S_vI_h}{N_h^2} & \frac{p_2\beta(w)S_vS_h}{N_h^2} & \frac{p_2\beta(w)I_h}{N_h} & -\eta & -\frac{p_2\tilde{\beta}I_hS_v}{N_h} \\ 0 & 0 & 0 & 0 & (\theta-\alpha)(1-2w) - \gamma \end{pmatrix}, \tag{12}$$

where $\alpha_o = \alpha_d + \mu + \delta$ and $\tilde{\beta} = \beta_{\max} - \beta_{\min}$.

Now, let us introduce the following quantity:

$$\mathscr{R}_0 = \frac{p_1 p_2 \beta_*^2 \mu \Lambda_v}{\alpha_o \eta^2 \Lambda_h}, \tag{13}$$

where

$$\beta_* = \begin{cases} \beta_{\min}, & \text{for } w^* = 1; \\ \beta_{\max} - w^*(\beta_{\max} - \beta_{\min}), & \text{for } w^* = \dfrac{\gamma}{\alpha - \theta} \text{ and } \alpha - \theta > 0. \end{cases} \tag{14}$$

Then the local stability of the two DFE is investigated in the following Theorem.

Theorem 3.1. *If the internal influence parameters α and θ satisfy the condition that*

1. $0 < \gamma \leq \alpha - \theta \leq 1$*, then we have*
   (i) *the DFE $\mathscr{E}_{o1}$ is unstable, and*
   (ii) *the DFE $\mathscr{E}_{o2}$ is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$;*
2. $-1 \leq \alpha - \theta < 0$*, then there is a unique DFE $\mathscr{E}_{o1}$ which is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable otherwise.*

Proof. Evaluating the Jacobian at $\mathscr{E}_{o1}$ we get

$$J(\mathscr{E}_{o1}) = \begin{pmatrix} -\mu & \delta & 0 & -p_1\beta_{\min} & 0 \\ 0 & -\alpha_o & 0 & p_1\beta_{\min} & 0 \\ 0 & -\dfrac{p_2\beta_{\min}\mu\Lambda_v}{\eta\Lambda_h} & -\eta & 0 & 0 \\ 0 & \dfrac{p_2\beta_{\min}\mu\Lambda_v}{\eta\Lambda_h} & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & -(\gamma+\theta-\alpha) \end{pmatrix}.$$

If the internal influence parameters satisfy the condition $0 < \gamma < \alpha - \theta < 1$, then $\gamma + \theta - \alpha < 0$. Since this implies that $-(\gamma+\theta-\alpha) > 0$ the DFE $\mathscr{E}_{o1}$ is always unstable.

Hence, the equilibrium $\mathscr{E}_{o2}$ emerges, and its corresponding Jacobian is

$$J(\mathscr{E}_{o2}) = \begin{pmatrix} -\mu & \delta & 0 & -p_1\beta_* & 0 \\ 0 & -\alpha_o & 0 & p_1\beta_* & 0 \\ 0 & -\dfrac{p_2\beta_*\mu\Lambda_v}{\eta\Lambda_h} & -\eta & 0 & 0 \\ 0 & \dfrac{p_2\beta_*\mu\Lambda_v}{\eta\Lambda_h} & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & \gamma+\theta-\alpha \end{pmatrix},$$

where $\beta_* = \beta_{\max} - \frac{\gamma}{\alpha-\theta}(\beta_{\max} - \beta_{\min})$.

The eigenvalues of this matrix are $\lambda_1 = -\mu$, $\lambda_2 = -\eta$, $\lambda_3 = (\gamma+\theta-\alpha) < 0$, and the roots of the quadratic equation,

$$\lambda^2 + (\alpha_o + \eta)\lambda + \eta\alpha_o - \frac{p_1 p_2 \beta_*^2 \mu\Lambda_v}{\eta\Lambda_h} = 0,$$

or in terms of $\mathscr{R}_0$ it is,

$$\lambda^2 + (\alpha_o + \eta)\lambda + \eta\alpha_o[1 - \mathscr{R}_0] = 0.$$

Therefore, all the eigenvalues of $J(\mathscr{E}_{o2})$ are negative if $\mathscr{R}_0 < 1$, whereas there is at least one positive eigenvalue if $\mathscr{R}_0 > 1$.

On the other hand, when $\alpha - \theta < 0$, then $-(\gamma+\theta-\alpha) < 0$ and all the remaining eigenvalues of $J(\mathscr{E}_{o1})$ are negative for $\mathscr{R}_0 < 1$ as shown above. Hence, the theorem is proved. □

Epidemiologically Theorem **3.1** implies that the disease transmission can be controlled in the community when $\mathscr{R}_0 < 1$ provided the initial values of the sub-populations of the model system (9) are in the neighborhood of the stable DFE $\mathscr{E}_o$ (that is, either $\mathscr{E}_o = \mathscr{E}_{o1}$ or $\mathscr{E}_o = \mathscr{E}_{o2}$, and note that both of them do not appear to be stable at the same time). However, to ensure that the disease elimination is independent of the choice of the initial sizes of the sub-populations, it is necessary to show that the DFE is globally-asymptotically stable (GAS) for $\mathscr{R}_0 < 1$.

Theorem 3.2. *If $\mathscr{R}_0 \leq 1$, then the locally stable disease free equilibrium $\mathscr{E}_o$ is also globally asymptotically stable.*

Proof: We use Kamgang-Sallet Stability Theorem (Kamgang and Sallet 2008) to prove this assertion. We shall check the five hypotheses on which this theorem is based on. Let $x_1 = (S_h, S_v)$ and $x_2 = (I_h, I_v)$. Then, from (9) we have

$$\dot{x_1} = A_1(x)(x_1 - x_1^*) + A_{12}(x)x_2 \tag{15}$$

$$\dot{x_2} = A_2(x)x_2, \tag{16}$$

where $x = (x_1, x_2) \in \mathbb{R}^2 \times \mathbb{R}^2$ and $x_1^* = \left(\frac{\Lambda_h}{\mu}, \frac{\Lambda_v}{\eta}\right)$. Since the equation corresponding to w does not contribute directly to the infection, it is omitted from consideration here. Then, we shall check if the sufficient conditions of Kamgang-Sallet Theorem (say, the hypothesis (H1)–(H5) in Kamgang and Sallet (2008)) are satisfied as follows.

(1) Since all the state variables are from the positively invariant set, they are all non negative as shown in section (2.3). Hence the first condition H_1 is satisfied.

(2) We express the subsystem $\dot{x_1} = A_1(x_1^*, 0)(x_1 - x_1^*)$ as

$$\begin{cases} \dot{S_h} = \Lambda_h - \mu S_h \\ \dot{S_h} = \Lambda_v - \eta S_v \end{cases}$$

This is a linear system which is globally asymptotically stable at the equilibrium $\left(\frac{\Lambda_h}{\mu}, \frac{\Lambda_v}{\eta}\right)$ corresponding to the disease free equilibria. The solution of the sub system will always converge to x_1^* for any initial condition. Hence, the second condition H_2 is satisfied.

(3) The matrix $A_2(x)$ is the Jacobian of system (16) and it is given by

$$A_2(x) = \begin{pmatrix} -\dfrac{p_1\beta(w)I_vS_h}{N_h^2} - \alpha_o & \dfrac{p_1\beta(w)S_h}{N_h} \\ \dfrac{p_2\beta(w)S_vS_h}{N_h^2} & -\eta \end{pmatrix}$$

The matrix $A_2(x)$ is Metzler and irreducible for any given $x \in \Omega$. Therefore, the third condition H_3 is also satisfied.

(4) There exists an upper bound matrix $\overline{A}_2$ for the set $\mathfrak{M} = \{A_2(x) : x \in \Omega\}$. For instance, taking $S_h = N_h$ and $S_v = N_v$,

$$\overline{A}_2(x) = \begin{pmatrix} -\alpha_o & p_1\beta(w) \\ \dfrac{p_2\beta(w)\Lambda_v\mu}{\Lambda_h\eta} & -\eta \end{pmatrix}$$

is the upper bound for $\mathfrak{M}$. Thus, the fourth condition H_4 is satisfied.

(5) For $\mathscr{R}_0 \leq 1$, $\alpha(\overline{A}_2) = \max\{Re(\lambda) : \lambda \text{ is an eigenvalue of } \overline{A}_2\} \leq 0$, which justifies that the fifth (and last) condition H_5 is also satisfied.

Therefore, by Kamgang-Sallet Stability Theorem the disease free equilibrium $\mathscr{E}_o$ is globally asymptotically stable for $\mathscr{R}_0 \leq 1$. □

Next we determine and analyze endemic equilibria for the model system (9) if it has any.

3.1. Endemic equilibrium. Denote the generic endemic equilibrium by

$$\mathscr{E}^* = (S_h^*, I_h^*, S_v^*, I_v^*, w^*).$$

Let us set

$$\begin{aligned} A &= p_2\beta_* + \eta, \\ B &= (p_2\beta_* + \eta)\alpha_o + \eta\alpha_o\left[1 - \left(\frac{\mu+\alpha_d}{\mu}\right)\mathscr{R}_o\right], \\ C &= \alpha_o^2\eta[1 - \mathscr{R}_o]. \end{aligned} \tag{17}$$

where R_0 and β_* are given in (13) and (14), respectively. Then the following theorem holds:

Theorem 3.3. *If $R_0 > 1$, then model (9) admits an unique endemic equilibrium if $A > 0$ and no endemic equilibrium if $A = 0$.*

Proof. By equating the system (9) to zero and solving we get each of the components of $\mathscr{E}^*$ to be

$$S_h^* = \frac{\Lambda_h}{\mu + \lambda_h^*}\left[1 + \frac{\delta}{\alpha_d}\left(1 - \frac{\alpha_o\mu + \mu\lambda_h^*}{\alpha_o\mu + (\alpha_d + \mu)\lambda_h^*}\right)\right] \tag{18}$$

$$I_h^* = \frac{\Lambda_h}{\alpha_d}\left[1 - \frac{\alpha_o\mu + \mu\lambda_h^*}{\alpha_o\mu + (\alpha_d + \mu)\lambda_h^*}\right] \tag{19}$$

$$S_v^* = \frac{\Lambda_v(\lambda_h^* + \alpha_o)}{(p_2\beta_* + \eta)\lambda_h^* + \eta\alpha_o} \tag{20}$$

$$I_v^* = \frac{\Lambda_v}{\eta}\left[\frac{p_2\beta_*\lambda_h^*}{(p_2\beta_* + \eta)\lambda_h^* + \eta\alpha_o}\right] \tag{21}$$

$$w^* = \begin{cases} 1, & \text{if } \gamma - (\alpha - \theta) > 0 \text{ or } \alpha - \theta < 0; \\ \frac{\gamma}{\alpha - \theta}, & \text{if } \alpha - \theta > 0. \end{cases} \tag{22}$$

$$\lambda_v^* = p_2\beta_*\frac{\lambda_h^*}{\alpha_o + \lambda_h^*} \tag{23}$$

where λ_h^* is a real positive solution of the quadratic equation

$$A\lambda_h^2 + B\lambda_h + C = 0, \tag{24}$$

with the coefficients A, B, C described in equation (17). In this equation, since all the parameter values are assumed to be nonnegative, we always have $A \geq 0$.

Now to check the assertions of the theorem, we shall consider the following cases.

(i) For $A > 0$ and $\mathscr{R}_0 > 1$, we have $C < 0$. Then, regardless of whether the value of B is positive or negative there will be no other sign change in the coefficients. Hence equation

(24) has a unique positive solution which implies that model system (9) has a unique endemic equilibrium.

(ii) For $A = 0$ and $\mathscr{R}_0 > 1$, since $B = A\alpha_o + \eta\alpha_o\left[1 - \left(\frac{\mu+\alpha_d}{\mu}\right)\mathscr{R}_o\right] < 0$ and $C < 0$, then the value of λ_h^* becomes negative. Therefore, the model system (9) has no endemic equilibrium. □

3.2. Bifurcation analysis. In this section we use the general center manifold theorem to investigate the occurrence of a backward or forward bifurcation at the disease free equilibrium points. The normal form representing the dynamics of the system on the center manifold is given by

$$\dot{u} = yu^2 + z\mu u \tag{25}$$

where

$$y = \frac{\mathbf{v}}{2} \cdot D_{xx} f(\mathscr{E}_o, 0)\mathbf{w}^2 \equiv \frac{1}{2} \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathscr{E}_o, 0) \tag{26}$$

and

$$z = \mathbf{v} \cdot D_{x\xi} f(\mathscr{E}_o, 0)\mathbf{w} \equiv \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \xi}(\mathscr{E}_o, 0), \tag{27}$$

where ξ is a bifurcation parameter, with f_i's denoting the right-hand-side of the system (9), x denoting the state vector, $\mathscr{E}_o$ the disease free equilibrium and $\mathbf{v}$ and $\mathbf{w}$ are the left- and right-eigenvectors respectively, corresponding to the null eigenvalue of the Jacobian matrix of system (9) evaluated at the critical points.

Let us take

$$\xi = p_2$$

as bifurcation parameter. Then, from (13) it follows that $\mathscr{R}_0 = 1$ is equivalent to

$$\xi^* = p_2^* = \frac{\eta^2\Lambda_h\alpha_o}{p_1\beta_*^2\Lambda_v\mu} = \frac{\eta\alpha_o}{p_1\beta_*^2}\frac{\tilde{N}_h}{\tilde{N}_v}, \tag{28}$$

where $\mathscr{R}_0 = \mathscr{R}_0(w) = \dfrac{p_1 p_2 \left[\beta_*(w)\right]^2 \Lambda_v\mu}{\eta^2\Lambda_h\alpha_o}$, is a threshold, whose exact value is dependent on the choice of the variable w, with either $w = w^* = \frac{\gamma}{\alpha-\theta}$ or $w = 1$ at a disease free equilibrium.

Here, $\mathscr{R}_0 < 1$ corresponds to the inequality $\xi < \dfrac{\eta\alpha_o}{p_1\beta_*^2}\dfrac{\tilde{N}_h}{\tilde{N}_v}$, where $\beta_* = \beta_{\max} - \frac{\gamma}{\alpha-\theta}(\beta_{max} - \beta_{min})$ for $w^* = \frac{\gamma}{\alpha-\theta}$ or $\beta_* = \beta_{\min}$ for $w^* = 1$. Then, we have the following theorem.

Theorem 3.4. *The model system (9) exhibits a forward bifurcation at the DFE $\mathscr{E}_o$, for $\mathscr{R}_0 = 1$.*

Proof: Let us begin with the Jacobian matrix of system (9) at the DFE $\mathscr{E}_{o2}$ and when $\xi^* = p_2^*$.

$$J(\mathscr{E}_{o2};\xi^*) = \begin{bmatrix} -\mu & \delta & 0 & -p_1\beta_* & 0 \\ 0 & -\alpha_o & 0 & p_1\beta_* & 0 \\ 0 & -\xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & -\eta & 0 & 0 \\ 0 & \xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & \gamma+\theta-\alpha \end{bmatrix}.$$

which has eigenvalues $\lambda_1 = 0, \lambda_2 = -\mu, \lambda_3 = -\eta, \lambda_4 = \gamma+\theta-\alpha, \lambda_5 = -(\alpha_o+\eta)$.

This matrix admits a simple zero eigenvalue and all the other eigenvalues are negative real numbers (as $\gamma < \alpha - \theta$ in this case).
Hence when $\mathscr{R}_0 = 1$, the disease free equilibrium $\mathscr{E}_{o2}$ is a non hyperbolic equilibrium.

Let $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5)$ and $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5)^T$ be left- and right-eigenvectors associated with the zero eigenvalue of $J(\mathscr{E}_{o2}, \xi^*)$ respectively, such that $\mathbf{v}\cdot\mathbf{w} = 1$. Then,

$$0 = (v_1, v_2, v_3, v_4, v_5) \begin{bmatrix} -\mu & \delta & 0 & -p_1\beta_* & 0 \\ 0 & -\alpha_o & 0 & p_1\beta_* & 0 \\ 0 & -\xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & -\eta & 0 & 0 \\ 0 & \xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & \gamma+\theta-\alpha \end{bmatrix}$$

$$\Rightarrow \begin{cases} \mu v_1 = 0 \qquad \text{or } v_1 = 0 \\ -\alpha_o v_2 - \xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h}(v_3 - v_4) = 0 \\ \eta v_3 = 0 \qquad \text{or } v_3 = 0 \\ -p_1\beta_*(v_1 - v_2) - \eta v_4 = 0 \\ (\gamma+\theta-\alpha)v_5 = 0 \qquad \text{or } v_5 = 0 \end{cases}$$

Since at $\xi = \xi^*$ we have that $p_1\beta_* = \dfrac{\eta\alpha_o}{\xi^*\beta_*}\dfrac{\tilde{N}_h}{\tilde{N}_v}$, the left eigenvector becomes,

$\mathbf{v} = (v_1, v_2, v_3, v_4, v_5) = \left(0, v_2, 0, \frac{\alpha_o}{\xi^* \beta_*} \frac{\tilde{N}_h}{\tilde{N}_v} v_2, 0\right)$ for any real number v_2, and similarly

$$0 = \begin{bmatrix} -\mu & \delta & 0 & -p_1\beta_* & 0 \\ 0 & -\alpha_o & 0 & p_1\beta_* & 0 \\ 0 & -\xi\beta_* \frac{\tilde{N}_v}{\tilde{N}_h} & -\eta & 0 & 0 \\ 0 & \xi\beta_* \frac{\tilde{N}_v}{\tilde{N}_h} & 0 & -\eta & 0 \\ 0 & 0 & 0 & 0 & \gamma+\theta-\alpha \end{bmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \end{pmatrix}$$

$$\Rightarrow \begin{cases} -\mu w_1 + \delta w_2 - p_1\beta_* w_4 = 0 \\ -\alpha_o w_2 + p_1\beta_* w_4 = 0 \\ -\xi^*\beta_* \frac{\tilde{N}_v}{\tilde{N}_h} w_2 - \eta w_3 = 0 \\ \xi^*\beta_* \frac{\tilde{N}_v}{\tilde{N}_h} w_2 - \eta w_4 = 0 \\ (\gamma+\theta-\alpha) w_5 = 0 \qquad \text{or } w_5 = 0 \end{cases}$$

Solving these equations yields $\begin{bmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \end{bmatrix} = w_2 \begin{bmatrix} -\frac{(\alpha_d+\mu)}{\mu} \\ 1 \\ -\frac{\xi^*\beta_*}{\eta} \frac{\tilde{N}_v}{\tilde{N}_h} \\ \frac{\xi^*\beta_*}{\eta} \frac{\tilde{N}_v}{\tilde{N}_h} \\ 0 \end{bmatrix}$

Now, taking $w_2 = 1$ we can calculate the value of v_2 so that $\mathbf{v} \cdot \mathbf{w} = 1$. To this end,

$$\begin{aligned} \left(0, v_2, 0, \frac{\alpha_o}{\xi^*\beta_*} \frac{\tilde{N}_h}{\tilde{N}_v} v_2, 0\right) \cdot \begin{bmatrix} -\frac{(\alpha_d+\mu)}{\mu} \\ 1 \\ -\frac{\xi^*\beta_*}{\eta} \frac{\tilde{N}_v}{\tilde{N}_h} \\ \frac{\xi^*\beta_*}{\eta} \frac{\tilde{N}_v}{\tilde{N}_h} \\ 0 \end{bmatrix} &= 1 \\ \Rightarrow v_2 + \frac{\xi^*\beta_*}{\eta} \frac{\tilde{N}_v}{\tilde{N}_h} \frac{\alpha_o}{\xi^*\beta_*} \frac{\tilde{N}_h}{\tilde{N}_v} v_2 &= 1 \\ \Rightarrow v_2 &= \frac{\eta}{\eta+\alpha_o} \end{aligned}$$

Therefore, the left- and right-eigenvectors of $J(\mathscr{E}_{o2},\xi^*)$ associated with eigenvalue 0 are respectively,

$$\begin{aligned}\mathbf{v} &= \left(0,\frac{\eta}{\eta+\alpha_o},0,\frac{\eta\alpha_o}{(\eta+\alpha_o)\xi^*\beta_*}\frac{\tilde{N}_h}{\tilde{N}_v},0\right)\\ \mathbf{w} &= \left(-\frac{(\alpha_d+\mu)}{\mu},1,-\frac{\xi^*\beta_*\tilde{N}_v}{\eta\tilde{N}_h},\frac{\xi^*\beta_*\tilde{N}_v}{\eta\tilde{N}_h},0\right)^T\end{aligned}$$

Next considering only nonzero components of these vectors it follows that

$$\begin{aligned}y=&\frac{1}{2}v_2\left[w_4w_2\frac{\partial^2 f_2}{\partial I_v\partial I_h}(\mathscr{E}_{o2},\xi^*)+w_2w_4\frac{\partial^2 f_2}{\partial I_h\partial I_v}(\mathscr{E}_{o2},\xi^*)\right]\\ &+\frac{1}{2}v_4\left[w_2w_1\frac{\partial^2 f_4}{\partial I_h\partial S_h}(\mathscr{E}_{o2},\xi^*)+w_2w_2\frac{\partial^2 f_4}{\partial I_h\partial I_h}(\mathscr{E}_{o2},\xi^*)\right]\end{aligned}$$

Which yields when simplified

$$y = \frac{\alpha_o\eta}{2\mu p_1\tilde{N}_h(\eta+\alpha_o)}\left(\frac{\alpha_d}{\mu}-\frac{\xi}{p_1}-2\right)<0 \tag{29}$$

And doing the same for z we get

$$\begin{aligned}z &= v_4w_2\frac{\partial^2 f_4}{\partial I_h\partial\xi}(\mathscr{E}_{o2},\xi^*)\\ \Rightarrow\quad z &= \frac{\eta\alpha_o}{\xi(\eta+\alpha_o)}>0\end{aligned} \tag{30}$$

Since $y<0$ and $z>0$, then the system (9) exhibits a forward bifurcation at ξ^*.

On the other hand since the equilibrium $\mathscr{E}_{o1}$ occurs in the case where $w^*=1$, and it is stable only when $\theta-\alpha>0$, we will have $-(\gamma+\theta-\alpha)<0$ and

$$J(\mathscr{E}_{o1};\xi^*)=\begin{bmatrix}-\mu & \delta & 0 & -p_1\beta_* & 0\\ 0 & -\alpha_o & 0 & p_1\beta_* & 0\\ 0 & -\xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & -\eta & 0 & 0\\ 0 & \xi\beta_*\dfrac{\tilde{N}_v}{\tilde{N}_h} & 0 & -\eta & 0\\ 0 & 0 & 0 & 0 & -(\gamma+\theta-\alpha)\end{bmatrix},$$

with $\beta_*=\beta_{\min}$. One can see that, every thing what has been shown for the case of $J(\mathscr{E}_{o2};\xi^*)$ repeats itself with the term $\gamma+\theta-\alpha$ multiplied by -1. Therefore, the system (9) exhibits a forward bifurcation at ξ^* also for the occurrence of the stable equilibrium $\mathscr{E}_{o1}$ as well. □

The above theorem asserts that the system does not undergo a backward bifurcation at $\mathscr{R}_0=1$.

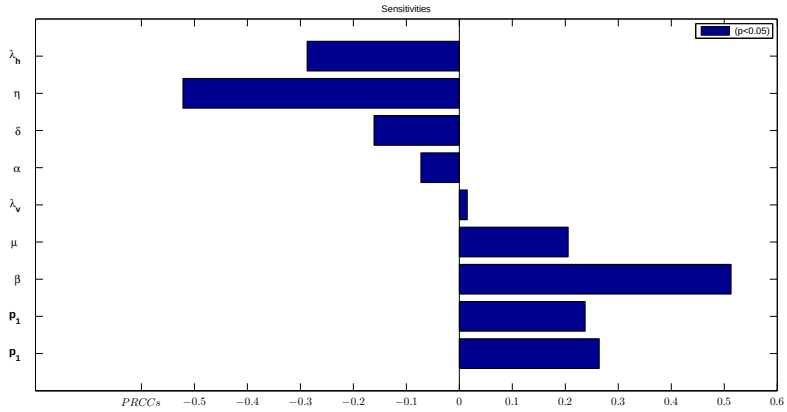

FIGURE 1. Sensitivity analysis for model (9).

4. Sensitivity analysis

The objective of sensitivity analysis is to identify critical parameters which significantly affect the model system. We examine the partial derivative of the threshold value $\mathscr{R}_0$ with respect to the input parameters performing multiple simulations varying the parameters around normal values. The uncertainty and sensitivity analysis is done by using LHS/PRCCs analysis (Stein 1987; Marino *et al.* 2008; Wu *et al.* 2013) with 1000 samples for various input parameters.

From Figure 1 we can see that the parameters β and η are highly sensitive to influence the model *i.e.*, β is directly proportional and η is inversely proportional to $\mathscr{R}_0$ respectively.

TABLE 2. Parameter PRCC Significance and unadjusted P-values

Variable	PRCC	Pvalue	Keep
p_1	0.26409	0.000e+00	TRUE
p_2	0.2375	2.642e-14	TRUE
β	0.51295	0.000e+00	TRUE
μ	0.20541	5.638e-11	TRUE
α_d	-0.075268	1.771e-02	TRUE
δ	-0.16111	3.202e-07	TRUE
η	-0.52176	0.000e+00	TRUE

The parameters with large PRCC values (> 0.5 or < -0.5) as well as corresponding small p-values(< 0.05) are the most important. The closer the PRCC value is to $+1$ or -1 the more strongly the parameter influences the model. The negative sign for PRCC indicates inverse proportionality. The PRCC significance for each parameter is reported in Table 2.

Next we perform pairwise comparisons for the parameters to see if they are dependent on one another.

TABLE 3. Pairwise PRCC Comparisons (P-values $p < 0.05$)

	p_1	p_2	β	μ	α_d	δ	η
p_1		*0.528	4.48e-11	0.1671	1.443e-14	0	0
p_2			5.265e-13	0.453	1.652e-12	0	0
β				1.554e-15	0	0	0
μ					2.779e-10	2.22e-16	0
α_d						0.05272	0
δ							0
η							

From Table 3 we can see that p_1 and p_2 are strongly dependent on each other while α_d, δ and η are independently sensitive to the reproduction number $\mathscr{R}_0$.

5. Optimal strategy for action enacted by PHS

In this section, we present an optimal control problem that describes the optimal strategy for the efforts made by the Public Health (PH) organizations to increase the propensity to use ITNs. In the model described so far, the constant parameter γ is used in model system (9) to describe the external effort to convince people in the anti-ITNs group so that they can also use bed nets as a control mechanism. The information generated by an optimal health-promotion campaign aimed at encouraging people to use ITNs may not be constant in its intensity and strength; it may vary with time.

Therefore, the last term in equation (8) can be interpreted as the the actions enacted by PH agencies to increase the propensity to use ITNs which is represented as a feedback control:

$$F(t) = -\gamma(t)(w(t) - 1),$$

where the feedback is on the measure of difference between the actual value of $w(t)$ and the ideal 'trajectory' $w_{id}(t) = 1$ to be tracked by the state variable $w(t)$.

Our goal is to obtain an optimal strategy for implementing health-promotion campaigns, by varying the value of the parameter γ with time within the state system (9), in such a way that the total cost associated to the disease as well as the cost of the control efforts are minimized. The costs associated to the disease is assumed to be linearly dependent on the total number of infected humans, whereas the cost of health-campaign efforts are related to the proportion of the total population that are addressed by the information as well as the cost of preparation for the campaign materials. Here we assumed the cost of the controls to be quadratic with respect to the proportion of the effort exerted by the PHS. Quadratic expressions of the controls are widely used in mathematical epidemiology literature (see e.g. Blayneh *et al.* (2010), Neilan *et al.* (2010), Prosper *et al.* (2011), Chamchod *et al.* (2014), Rachah and Torres (2016), and Zhao *et al.* (2016)). This assumption mimics the idea that costs might increase non-linearly at high intervention levels. For a more detailed discussion, see Buonomo *et al.* (2018b).

In summary, the objective function to be minimized is given by

$$J(\gamma) = \int_0^T \left(K_1 I_h(t) + K_2 w N_h(t) + \frac{K_3}{2} \gamma(t)^2 \right) dt, \tag{31}$$

where the control $\gamma(t)$ is a Lebesgue measurable function with $0 \leq \gamma(t) \leq \gamma_{\max} \leq 1$ for all $t \in [0,T]$. The upper bound for γ reflects the idea that there are practical limitations on the maximum rate at which information on ITNs may be spread by the PHS. In equation (31) the values K_1, K_2, and K_3 are positive constants that represent relative weight of each term in the marginal cost.

The optimal control problem (minimizing the objective functional (31), where the state and control variable satisfy the equation system in (9), (10) and (11)) can investigated by using the Pontryagin's maximum principle. To define the Hamiltonian, let $f(\mathbf{x},\gamma,t)$ represent the the integrand in (31), and $g(\mathbf{x},\gamma,t)$ represents the right-hand vector of system (9), where $\mathbf{x}$ represents the state variable vector. Then, the Hamiltonian of the optimal control problem is given by

$$H(\mathbf{x},\zeta,\gamma,t) = f(\mathbf{x},\gamma,t) + \sum_{i=1}^{5} \zeta_i(t) g_i(\mathbf{x},\gamma,t),$$

$\zeta_i, i = 1,\ldots,5$, are the co-state functions corresponding to the ith equation of system (9), whose value is obtained by solving the so called the *co-state system*:

$$\frac{\mathrm{d}\zeta_i}{\mathrm{d}t} = -\frac{\partial H}{\partial x_i}(\mathbf{x},\zeta,\gamma,t), \quad i = 1,\ldots,5.$$

Hence, the optimal control is obtained by minimizing the Hamiltonian pointwise for each $t \in [0,T]$. That means, we need to solve the system

1. *State equations:* equation systems (9), (10) and (11) together with the initial values $S_h(0) = S_0^o, I_h(0) = I_h^o, S_v(0) = S_v^o, I_v(0) = I_v^o$ and $w(0) = w^o$.
2. *Adjoint (Co-state) system:* it is given by

$$\begin{aligned}
\frac{\mathrm{d}\zeta_1}{\mathrm{d}t} &= -K_2 w + (\zeta_1 - \zeta_2)\left[\lambda_h(w) + S_h p_1 (w\tilde{\beta} - \beta_{\max})\frac{I_v}{N_h^2}\right] \\
&\quad + (\zeta_3 - \zeta_4) S_v p_2 (w\tilde{\beta} - \beta_{\max})\frac{I_h}{N_h^2} + \mu\zeta_1 \\
\frac{\mathrm{d}\zeta_2}{\mathrm{d}t} &= -(K_1 + K_2 w) + (\zeta_1 - \zeta_2) S_h p_1 (w\tilde{\beta} - \beta_{\max})\frac{I_v}{N_h^2} \\
&\quad + (\zeta_4 - \zeta_3) S_v p_2 (w\tilde{\beta} - \beta_{\max})\frac{I_h}{N_h^2} + (\zeta_4 - \zeta_3)\delta + \zeta_1(\alpha_d + \mu) \\
\frac{\mathrm{d}\zeta_3}{\mathrm{d}t} &= (\zeta_3 - \zeta_4)\lambda_v(w) + \zeta_3\eta \\
\frac{\mathrm{d}\zeta_4}{\mathrm{d}t} &= (\zeta_2 - \zeta_1) p_2 \frac{S_v}{N_h}(w\tilde{\beta} - \beta_{\max})\frac{I_h}{N_h^2} + \zeta_4\eta \\
\frac{\mathrm{d}\zeta_5}{\mathrm{d}t} &= -K_2 N_h + (\zeta_2 - \zeta_1) S_h p_1 \tilde{\beta}\frac{I_v}{N_h} + (\zeta_4 - \zeta_3) S_v p_2 \tilde{\beta}\frac{I_h}{N_h} \\
&\quad + \zeta_5\left[(\theta - \alpha)(2w - 1) + \gamma\right],
\end{aligned} \tag{32}$$

together with the *transversality conditions* $\zeta_i(T) = 0$ for all $i = 1,2,3,4,5$.

3. *The Optimality condition:* It is the solution of minimizing the Hamiltonian with respect to the control $\gamma \in [0, \gamma_{\max}]$ that gives us

$$\gamma^* = \max\left\{0, \min\left\{\gamma_{\max}, \frac{1}{K_3}\zeta_5(w-1)\right\}\right\} \tag{33}$$

Since the cost functional (31) is convex with respect to the control variable and the model system (9) is regular, existence of the optimal solution is assured as described in Fleming and Rishel (2012).

We estimate the results by using fourth order Runge-Kutta method for solving the state equation system (9), and the adjoint (or co-state) equation system (32), together with the optimality equations (33). The process begins with an initial guess on the control variables. Then, the state equations are simultaneously solved forward in time starting from the initial conditions and the adjoint equations are solved backward in time starting from the transversality conditions. The control is updated by inserting the new values of the state and adjoint vectors into its characterization (equations (33)), and the process is repeated until convergence occurs (Hackbusch 1978; Lenhart and Workman 2007).

In our simulations we used the parameter values given in Table 1 and the coefficient values for the cost function to be, $K_1 = 100$, $K_2 = 0.25$ and $K_3 = 6000$. The initial values are taken for simulation purpose to be $S_h(0) = 9500, I_h(0) = 50, S_v(0) = 40000, I_v(0) = 10000$ and $w(0) = 0.005$.

After solving the optimal control system numerically, we have drawn the graphs of the solution trajectories both for the optimal control strategy as well as for the corresponding state variables. In Figure 2, the trajectories for the susceptible and the infected human (host) populations (a) and (b) and the corresponding susceptible and infected mosquito populations in (c) and (d) before and after applying control efforts by the public health agencies. As can be seen from these plots, the controls reduce the infection in both human and vector population, but the effect on the human population is more significant. The time profiles in Figure 2 correspond to control profile given in Figure 3. It can be seen in this graph that the control effort must be applied at its full intensity for more than half of the planning period and can be dropped slowly to zero afterwards. Since it is assumed in the simulated model that the persuasion power of the anti-ITNs group is higher than those of the pro-ITNs use group (*i.e.*, since $\alpha - \theta = 0.5 - 0.1 > 0$ is taken in the simulation), the idea of applying the control efforts at its upper level in the initial period is clear from intuition. Unless this is done the remaining population will migrate to the anti-ITNs group with a constant positive rate, which will further imply the increase in the infection because of luck of appropriate protections for the population.

The persuasion rates may not remain constant across the whole population. It may possibly vary for example, from place to place due to the environmental factors as well as from one social group to the other social groups due to their level of awareness. The simulations in Figure 4 shows that when we take different combinations of the parameter values for α and θ, the infection level changes accordingly. It can be observed from this simulation that as the difference $\alpha - \theta$ increases, reducing the burden of the disease become more challenging and costly.

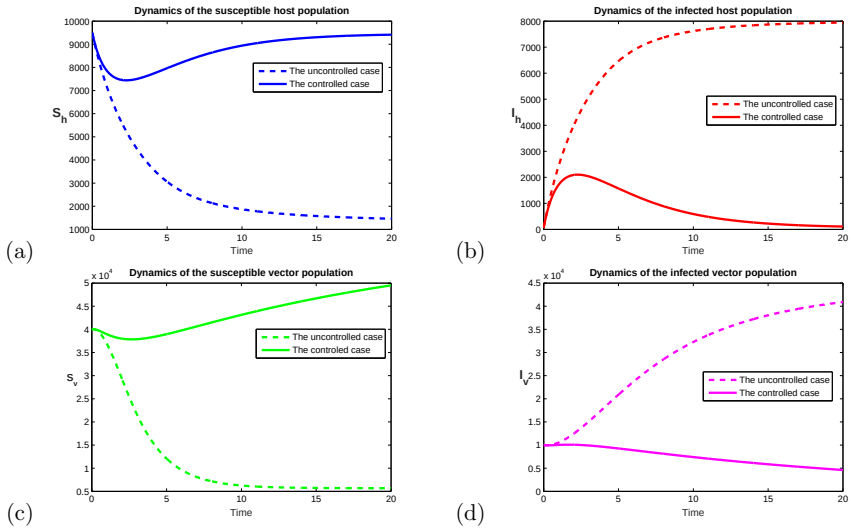

FIGURE 2. Dynamics of (a) Susceptible humans, (b) Infectious humans, (c) Susceptible vector, and (b) Infectious vector. The continues lines represent the controlled case and the dotted lines represent the uncontrolled case. In this case parameter values $p_1 = 0.525$, $p_2 = 0.305$, $\alpha = 0.5$ and $\theta = 0.1$ are used and the remaining parameters are as in Table 1.

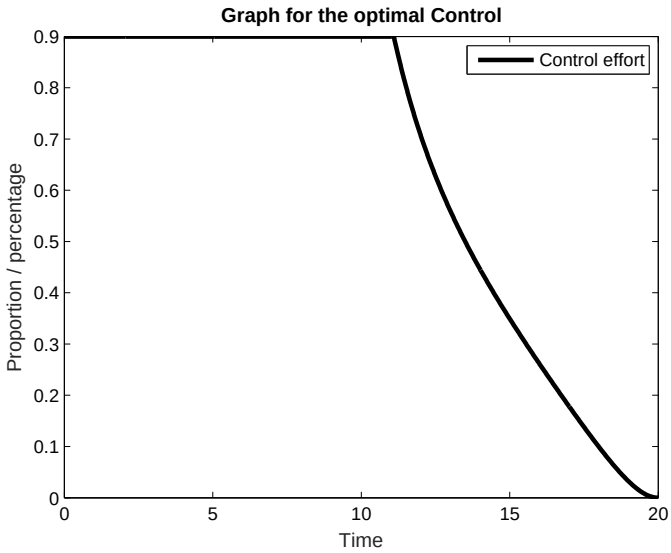

FIGURE 3. The optimal control time profile. The parameter values as in the caption of Figure 2.

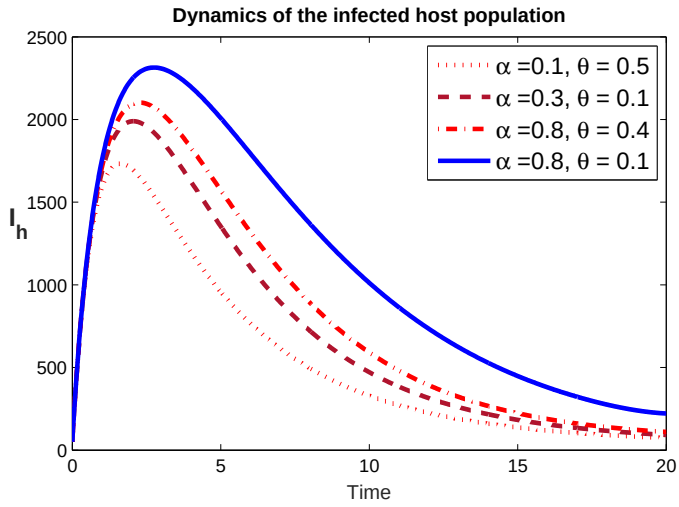

FIGURE 4. Time profiles of infectious human population, for various combinations of the parameters α and θ, with the remaining parameter values as in the caption of Figure 2.

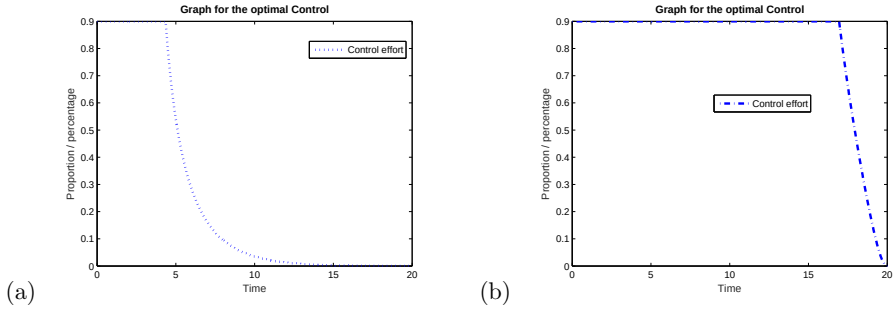

FIGURE 5. The values of the control $\gamma(t)$ when (a) $\alpha - \theta = -0.4$ and (b) $\alpha - \theta = 0.7$.

The control profile $\gamma(t)$ when the difference $\alpha - \theta$ is negative ($= -0.4$) and when it is large positive ($= 0.7$) are shown in Figure 5. The graph indicates that the optimal strategy in controlling the disease requires that an additional control effort must be implemented by the public health authorities at the initial stage even if the difference $\alpha - \theta$ is negative. That means, even if the persuasion power of the pro-ITNs group overpowers that of the anti-ITNs group, some additional effort should be made from the external body (the public health authorities) to arrive at the critical number of persuasive power for the pro-ITNs group produce the best result.

6. Conclusions

In this paper we formulated and analyzed a continuous time dynamical model for the spread of mosquito-borne human diseases with the use of ITNs as a preventive mechanism and when the decision of use of these ITNs depends on information dependent human behavior. The behavior change function is assumed to follow an imitation game dynamics. The mathematical analysis of the model shows that the disease free equilibrium is globally asymptotically stable for $\mathscr{R}_0 < 1$, and unstable otherwise, whether the persuasion power of the anti-ITNs use group is larger than that of the pro-ITNs use group or not. Moreover, using the center manifold theory it has been shown that a transcritical forward bifurcation occurs for $\mathscr{R}_0 = 1$. In addition, the sensitivity analysis shows that the value of $\mathscr{R}_o$ is more sensitive to the changes in the parameter values of the contact rate β and the death rate of the mosquito η. Hence working on changing this values as the additional control effort will increase the chance of eradicating the disease.

The optimal control analysis of the model and the simulations on the controlled system shows that if the persuasion power of the anti-ITNs use group is small as compared to that of the pro-ITNs use group, the effort of the PHS to control the spread of the disease is relatively simple and less costly. On the other hand if the relative persuasion power of the anti-ITNs group is larger the control effort becomes harder and costly. That means, the control effort as well as the burden of the disease varies with values of the parameters α and θ.

In this paper we assumed that the values of the parameters α and θ are constant. In practical terms, however, they vary with the prevalence or incidence of the disease as well as on the way information is spread in the population. Therefore, it will be interesting to consider this situation in the future work.

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