Impact of human behavior on Insecticide Treated Nets(ITNs) control strategies to prevent the spread of vector borne diseases

Manalebish D. Asfaw , Bruno Buonomo and Semu M. Kassa

Addis Ababa University,Ethiopia
NORDITA-Unifying the epidemiological and evolutionary dynamics of pathogens
May 29 - June 22
Stockholm, Sweden

June 7,2023

Outline

Motivation

- 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).
- 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.
- 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).

Advantage of 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).

Disadvantages of ITNs

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).
- 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

- 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 talk, we introduce a behavioral change model (BCM) to assess the impact of human behavior on ITNs use. BCMs are major tools in behavioural epidemiology (Manifredi and d'Onofrio 2013)
- the key issue is the investigation of the interplay between human behavior and the spread of infectious diseases.
- contact rate depend on a goodwill index $w(t)$ interpreted as willingness to use ITNs. It is analogus to information index (d'Onofrio and Manifredi 2016)

- However,BCM 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. 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.

The malaria model with ITNs use considered in Augusto et al. (2013) by following the imitation-game approach as described in Buonomo et al.(2018a)
we denote the fraction of population willing to use ITNs and against it by $w(t)$ and $a(t)$ respectively.
The imitation game follows a double contagion of ideas process (Wang et al 2016) includes Public health(PH) agencies influencing perceptions regarding ITNs and disease consequences. the game produce a dynamic equation for $w(t)$.

Host-vector model with ITN usage

The basic model from Agusto et al (2013)
The human population N_h, and vector populations N_v therefore, for $t \geq 0$,

$$N_h(t) = S_h(t) + I_h(t), \qquad N_v(t) = S_v(t) + I_v(t),$$

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

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

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.

The infection rate per susceptible human and per susceptible vector are given, respectively, by

$$\lambda_h(b) = p_1\beta(b)\frac{I_v}{N_h}, \quad \text{and} \quad \lambda_v(b) = p_2\beta(b)\frac{I_h}{N_h}, \tag{2}$$

where p_1 and p_2 are probability of disease transmission from mosquito to human and from human to mosquito respectively.
$\beta(b)$ represents the human–mosquito contact rate and the parameter $b \in [0, 1]$ is the proportion of ITNs usage.
Using bed nets reduces the probability for humans to be bitten. Moreover, the nets are treated with insecticide. Two main assumptions are made:

- ITNs usage reduces the human–mosquito contact rate $\beta(b) = \beta_{\text{max}} - b\left(\beta_{\text{max}} - \beta_{\text{min}}\right)$, where β_{max} and β_{min} are the maximum and the minimum contact rate, respectively.
- ITNs usage increases the mosquito death rate η.
  $\eta(b) = \eta + \eta_{bn}b$, where η_{bn} is a non negative constant and $\eta_{bn}\, b$ represents the death rate due to insecticide on ITNs.

Imitation dynamics

Let us denote the measere of wilingness to use ITN and against by $W(t)$ 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, and therefore $w(t) + a(t) = 1$ for all t. The *imitation* game is a double *contagion of ideas* process (Wang2016):

$$\begin{aligned} \dot{w} &= -\alpha a w + \theta w a \\ \dot{a} &= \alpha a w - \theta w a. \end{aligned} \tag{3}$$

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.

The action of Public Health (PH) authorities 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:

$$\begin{aligned} \dot{w} &= -\alpha a w + \theta w a + \gamma(t) a \\ \dot{a} &= \alpha a w - \theta w a - \gamma(t) a, \end{aligned} \tag{4}$$

where $\gamma(t)$ is a positive function that, captures the effectiveness of actions of PH agencies (as information, education, distribution of ITNs, etc.) in influencing the perceptions regarding both ITNs and disease consequences. Since $a = 1 - w$, one can write down the following extension of an imitation-game equation:

$$\dot{w} = w(1-w)(\theta - \alpha) + \gamma(t)(1-w), \tag{5}$$

We will analyze the model in the simplest case where $\gamma(t) = \gamma > 0$,

Host vector model with ITN usage

We now couple the equation with the model to obtain:

$$\begin{aligned}
\dot{S}_h &= \Lambda_h - \lambda_h(w)S_h - \mu S_h + \delta I_h \\
\dot{I}_h &= \lambda_h(w)S_h - (\alpha_d + \mu + \delta)I_h \\
\dot{S}_v &= \Lambda_v - \lambda_v(w)S_v - \eta S_v \\
\dot{I}_v &= \lambda_v(w)S_v - \eta I_v, \\
\dot{w} &= (\theta - \alpha)w(1-w) + \gamma(1-w).
\end{aligned} \tag{6}$$

where

$$\lambda_h(w) = p_1\beta(w)\frac{I_v}{N_h}; \qquad \lambda_v(w) = p_2\beta(w)\frac{I_h}{N_h}, \tag{7}$$

and

$$\beta(w) = \beta_{\max} - w\left(\beta_{\max} - \beta_{\min}\right). \tag{8}$$

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

Equilibria and stability

Theorem

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 coverage of the use of ITNs, the model admit two disease-free equilibria a *disease-free and full–ITNs utilization equilibrium*,

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

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

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

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

Local stability of the two DFE

Now, let us introduce the following quantity:

$$\mathcal{R}_0 = \frac{p_1 p_2 \beta_*^2 \mu \Lambda_v}{\alpha_o \eta^2 \Lambda_h}, \tag{9}$$

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

Theorem

If the internal influence parameters α and θ satisfy the condition that

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

Global stability

Epidemiologically this implies the disease transmission can be controlled in the community when $\mathcal{R}_o < 1$ provided the initial values of the sub-populations of the model system are in the neighborhood of the stable DFE $\mathcal{E}_o$ (that is, either $\mathcal{E}_o = \mathcal{E}_{o1}$ or $\mathcal{E}_o = \mathcal{E}_{o2}$, 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 $\mathcal{R}_o < 1$.

Theorem

If $\mathcal{R}_o \leq 1$. then the disease free equilibrium $\mathcal{E}_o$ is globally asymptotically stable.

(Kamgang-Sallet Stability Theorem) the five hypothesis on (Kamgang and Sallet 2008) satisfied.

Endemic equilibrium

Let us denote endemic equilibrium by

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

Let us set

$$\begin{aligned} A &= p_2\beta_* + \eta, \\ B &= (p_2\beta_* + \eta)\alpha_o + \eta\alpha_o\left[1 - \left(\frac{\mu + \alpha_d}{\mu}\right)\mathcal{R}_o\right], \\ C &= \alpha_o^2\eta[1 - \mathcal{R}_o]. \end{aligned} \tag{10}$$

in the quadratic equation

$$A\lambda_h^2 + B\lambda_h + C = 0, \tag{11}$$

Stability and Bifurcation analysis

Theorem

If $\mathcal{R}_o > 1$ then the model admits a unique endemic equilibrium If $A > 0$ and no endemic equilibrium if $A = 0$

using the General Center Manifold Theorem ,

Theorem

The model system exhibits a forward bifurcation at the DFE $\mathcal{E}_o$ for $\mathcal{R}_o = 1$.

Sensitivity analysis

The objective of sensitivity analysis is to identify critical parameters which significantly affect the model system. (using LHS/PRCCs analysis Marino et al. 2008; Wu et al. 2013)

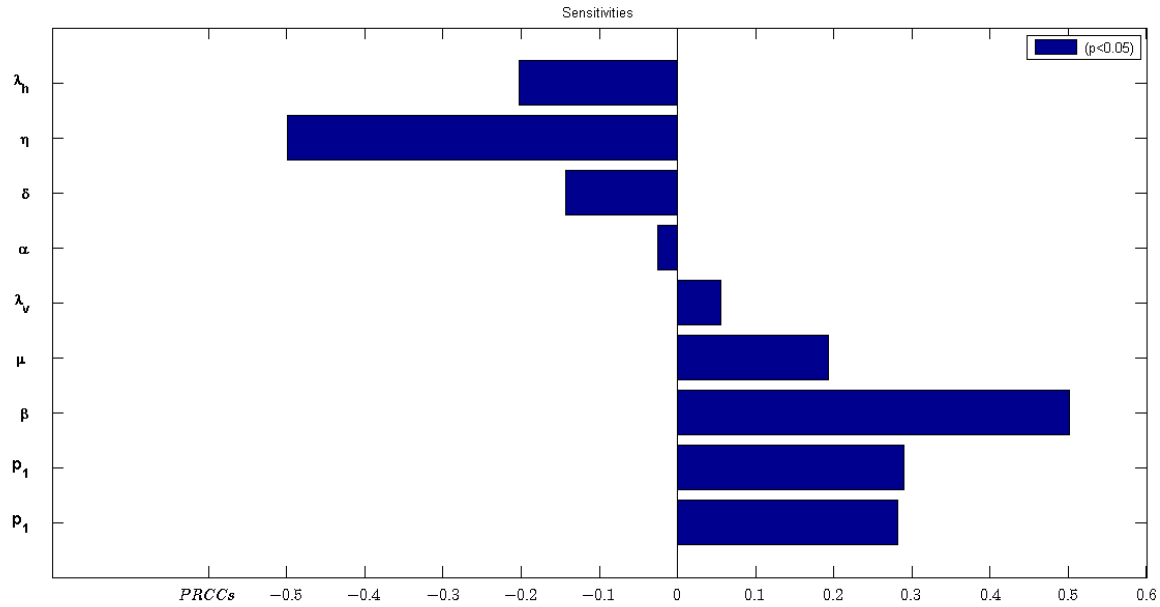


Figure: Sensitivity analysis for model

From fig 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 $\mathcal{R}_o$ respectively.

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

Table: Parameter PRCC Significance and unadjusted P-values

The parameters with large Partial Rank Correlation Coefficient (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.

Optimal strategy for action by PHS

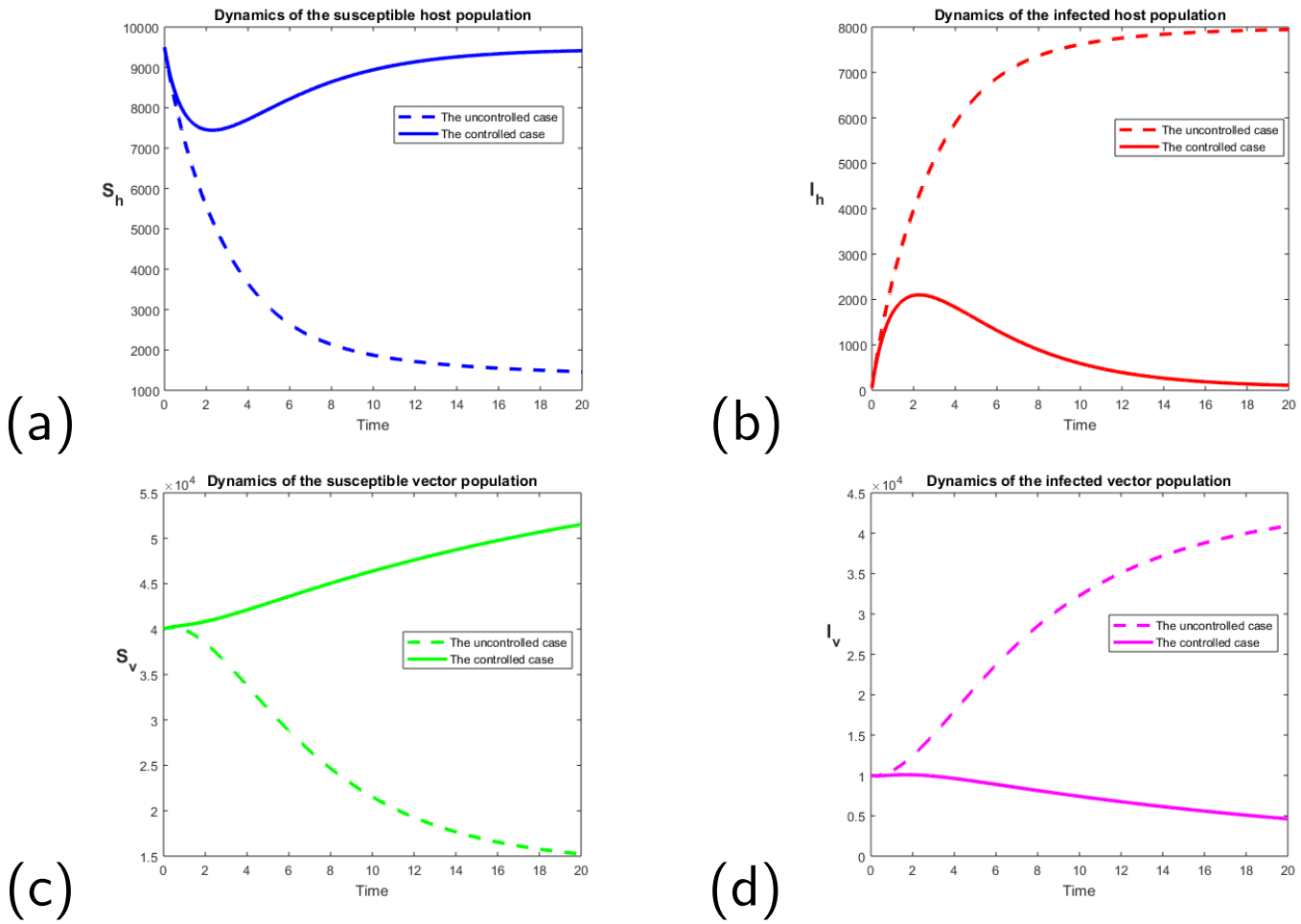


Figure: Graphs for the dynamics of (a) Susceptible humans, (b) Infected humans, (c) Susceptible vector, and (b) Infected vector. The continues lines represent the controlled case and the dotted lines represent the uncontrolled case. In this case parameter values $p_1 = 0.525$, $p_2 = 0.305$, $\alpha = 0.5$ and $\theta = 0.1$ are used.

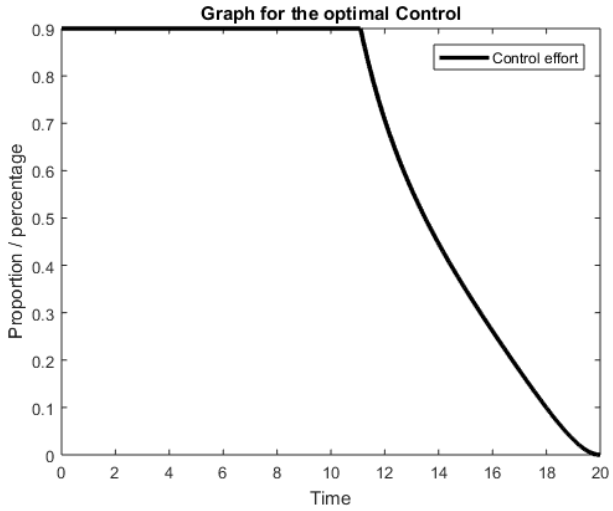


Figure: Trajectory for the Optimal Control, with the para.values as in cap of Fig 2

In this graph, 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.

When we take different combinations of the parameter values for α and θ, the infection level changes accordingly.

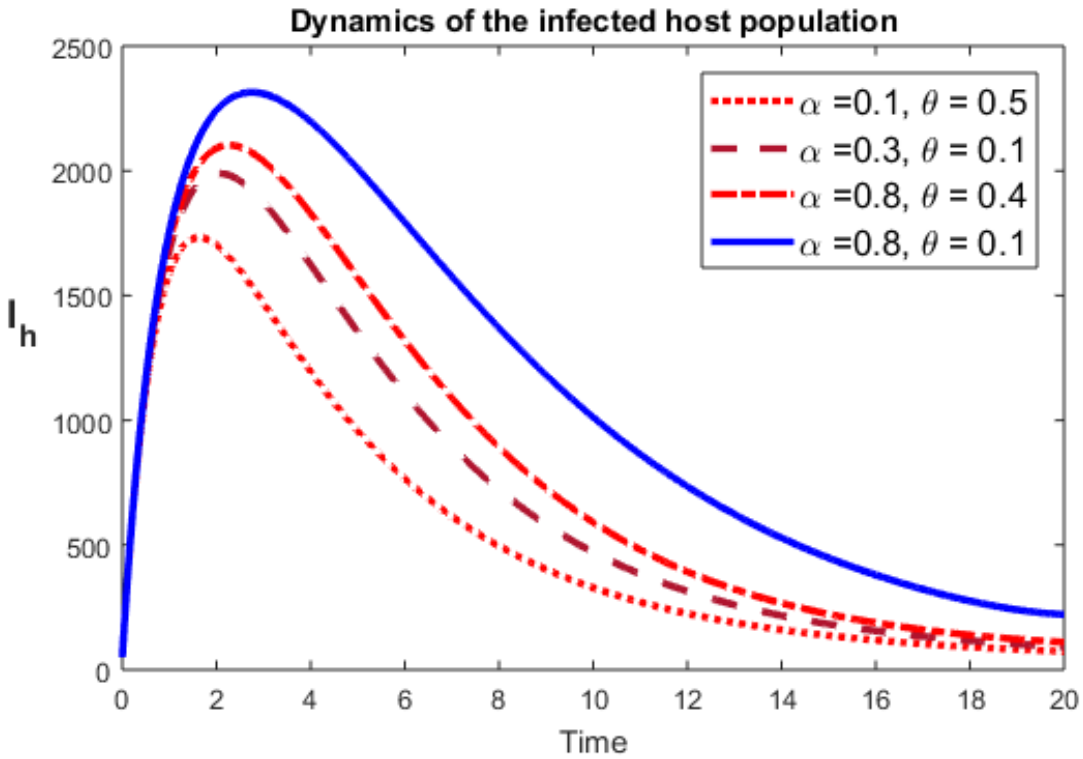
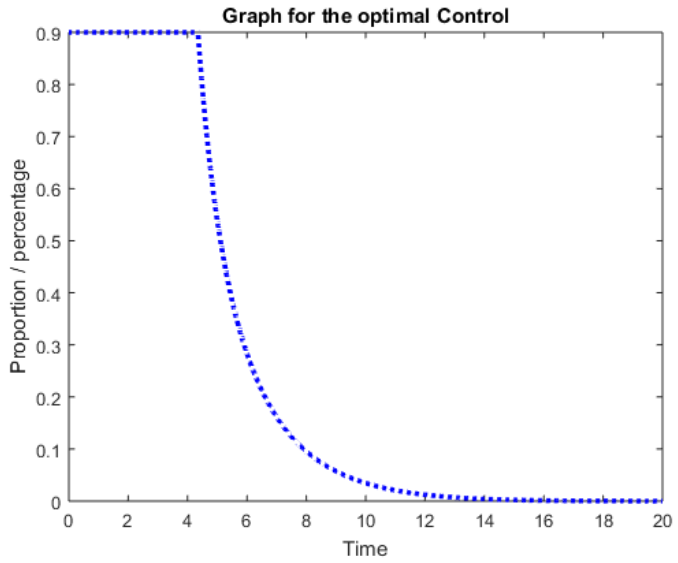
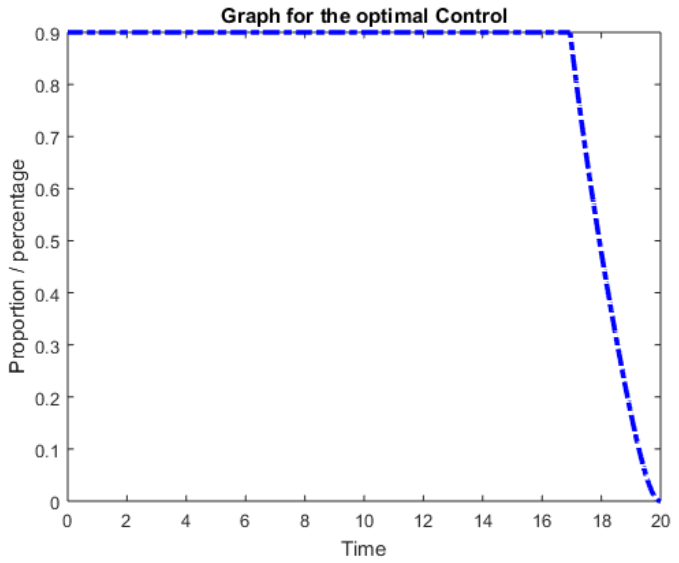


Figure: Trajectories for the infected humans, for various combinations of the parameters α and θ, with the remaining parameter values as in the caption of Figure 2.



(a)



(b)

Figure: 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. 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.

Conclusion

In this work

- 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 behaviour. The behaviour 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 $\mathcal{R}_o < 1$, 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 asserted that the model system does not undergo a backward bifurcation when the threshold value pass the point $\mathcal{R}_o = 1$.

In addition, the sensitivity analysis shows that the value of $\mathcal{R}_o$ 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.

Acknowledgment

- ISP International Science Program based at Uppsala university
- Nordita
- Prof. Tom Britton and Epi group in Stockholm
- Addis Ababa University Department of Mathematics

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- Manalebish Debalike Asfaw;Bruno Buonomo ;Semu Mitiku Kassa. Impact of Human Behavior on ITNS Control Strategies to Prevent the Spread of Vector Borne Diseases,*AAPP Atti della Accademia Peloritana dei Pericolanti Classe di Scienze Fisiche, Matematiche e Naturali* **2018** ISSN 1825-1242 Vol. 96, No. S3, A2 DOI: 10.1478/AAPP.96S3A2

Thank you for attention