Mathematical Model for Vector-Parasite Interactions in Lymphatic Filariasis Transmission

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Abstract

Interaction between vector mosquitoes and filarial parasites is a key concern in Lymphatic Filariasis transmission. Model for describing this interaction must strongly be incorporated with heterogeneity in the transmission via parameters in model equations. Various dynamics occur in parasite uptake by mosquitoes, parasite development and survival. Hosting filarial parasites by vector mosquitoes and parasite induced vector mortality usually resemble a situation similar to predator-prey interaction. This paves the way for more mathematical analysis through well-known model Lotka-Volterra, where phase space analysis suggests possible dynamics in vector-parasite interactions. Simulation trials can be executed to validate parameter estimations and attributed variations. Based on the corresponding interpretations, one can decide what would be the ideal set of parameters suited for an epidemiological situation of Lymphatic Filariasis in an endemic area.

1 Introduction

In Lymphatic Filariasis (LF), humans harbor adult parasites and produced microfilariae (Mf) are developed into L1, L2 and L3 larval stages sequentially within mosquito body. Model presented here mainly incorporates possible variations in vector-parasite interactions. Parasite development and release are also modeled separately.

2 Mathematical formulation

Number of L1 parasites in vector mosquitoes at a particular time of a day (P) and number of infected mosquitoes containing any filarial parasite stage at that time (M) are the variables considered here. Higher removal rate of P is evident for higher P due to competition, mortality and development into subsequent stages. Therefore, P can be modeled as given in (1), where a is a positive parameter.

$$\frac{dP}{dt} = -aP \qquad (1)$$

The term PM provides a measure to availability of vector mosquitoes and parasites to be engorged. Then a modified equation can be obtained as (2), where b is a positive parameter.

$$\frac{dP}{dt} = -aP + bPM \qquad (2)$$

If M increases due to more mosquito occupation, then the increasing rate can be assumed to be proportional to M. If M decreases, it may be a result of mosquito control or loss of infection. These facts can be modeled by (3), where r should be positive for increasing rate and negative for decreasing rate of M.

$$\frac{dM}{dt} = rM \qquad (3)$$

According to experimental findings, higher mortality rate is observable in mosquitoes fed on individuals with Mf and the mortality rate increases as Mf load increases which can be modeled by (4), where s is a positive parameter.

$$\frac{dM}{dt} = rM - sPM \qquad (4)$$

Now, the following system is the preliminary model for vector-parasite interaction.

$$\frac{dP}{dt} = -aP + bPM \qquad (5)$$

$$\frac{dM}{dt} = rM - sPM$$

Parameter values can be determined by experimental or reliable observational data on gain or loss rates regarding parasites and mosquitoes [1].

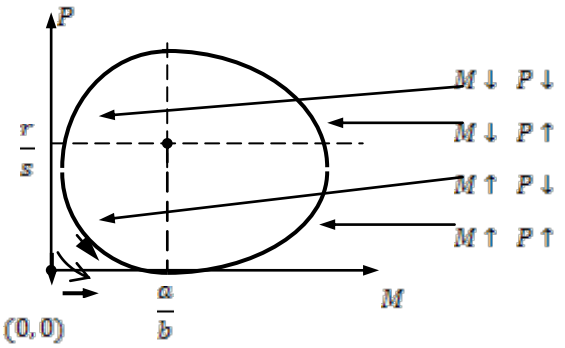


Figure 1: Trajectory directions around (0, 0) and Increasing (↑) and decreasing (↓) behavior of P and M

System (5) resembles the Lotka-Volterra system when $r > 0$. Then equilibrium levels of (5) are $M = 0, P = 0$ and $M = a/b, P = r/s$. Next, the trajectory directions around equilibrium levels can be obtained using $\frac{dP}{dM} = \frac{dP/dt}{dM/dt} = \frac{(-a+bM)P}{(r-sP)M}$. Unstable nature around $(0, 0)$ level is clear and more mathematical analysis guarantees closed trajectories, which results periodic behavior in both M and P (Fig. 1) [2]. For the case of $r < 0$, all the solution trajectories are directed towards $(0, 0)$ claiming both P and M will have a potential of dying off.

L1 parasites per infected mosquito (P/M) can be determined by (5). This $\alpha = P/M$ taken at the end of the simulation period 18.00hr - 06.00hr leads to formulate an equation for L3 yield as (6).

$$\frac{dL}{dt} = \alpha - \beta L \qquad (6)$$

Here, L is the number of L3 parasites per mosquito and β is the death rate of L3 parasites. Time variable t is measured in days. β can be roughly quantified as $0.12\ \text{day}^{-1}$ according to the lifespan estimations.

Equation (6) gives $L = \frac{\alpha}{\beta}\left(1 - e^{-\beta t}\right)$. For a domestic setting with $\overline{M}$ mean number of infected mosquitoes, the mean L3 yield can be given by $L_d = \frac{\alpha}{\beta}\left(1 - e^{-\beta t}\right)\overline{M}$. To incorporate L3 loss other than to natural deaths, revised L_d can be taken as $\frac{\alpha}{\beta}\left(1 - e^{-\beta t}\right)(1-h)\overline{M}$ using stochastic variable h $(0 \le h \le 1)$. In literature, there are experimental findings where at least bounds can be made for the above stochastic variation.

3 Simulation results

4th order Runge-Kutta method was used to simulate the solutions. In Fig. 2, simulations were executed for a time period of 12 hours (18.00hr to 06.00hr), where the solid curve segments represent various possibilities presented in Fig. 1.

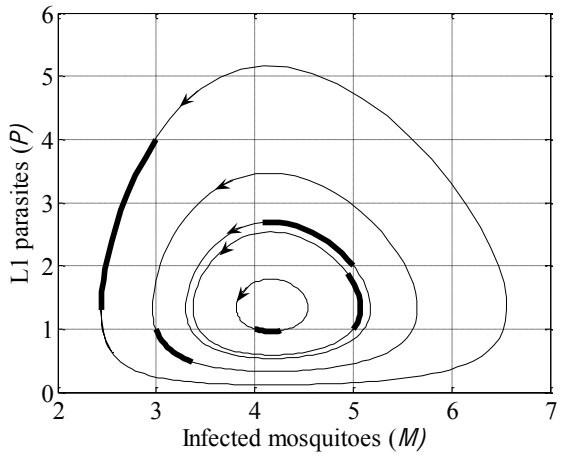


Figure 2: Behavior of M and P for different initial values ($a = 0.25$, $b = 0.06$, $r = 0.02$ and $s = 0.015$)

Impact of parameter a and b is illustrated in Fig. 3 (A). Decreasing trend of α when a increases is evident as more parasite loss yields lesser α. Meanwhile, α shows an increasing trend for increasing b since higher b values indicate higher potential of vector-parasite interactions. Fig. 3 (B) illustrates how α moves from decreasing to increasing trend as s and r vary.

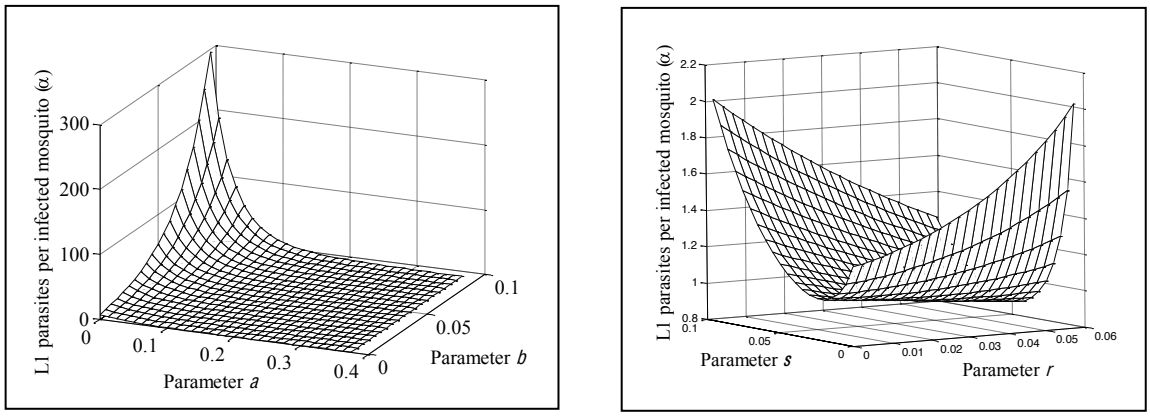
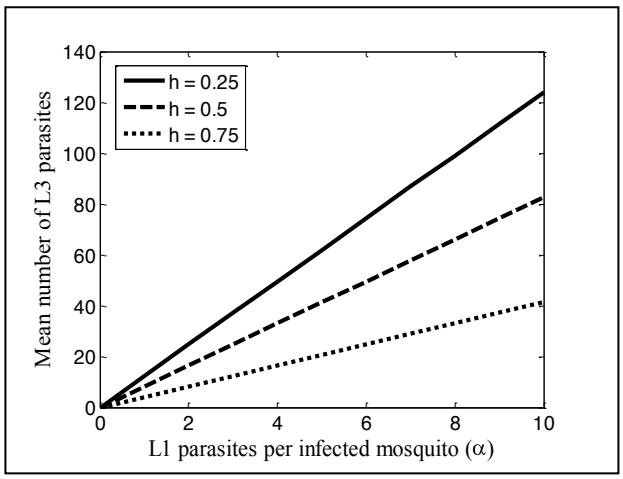


Figure 3: (A) Number of L1 parasites per infected mosquito (Fixed parameters: $r = 0.02$ and $s = 0.03$) (B) Number of L1 parasites per infected mosquito (Fixed parameters: $a = 0.25$ and $b = 0.075$).

Now, mean L3 yield can be simulated by revised L_d. Several results are depicted in Fig. 4 by fixing $\overline{M}$ at 3 and t at 9.



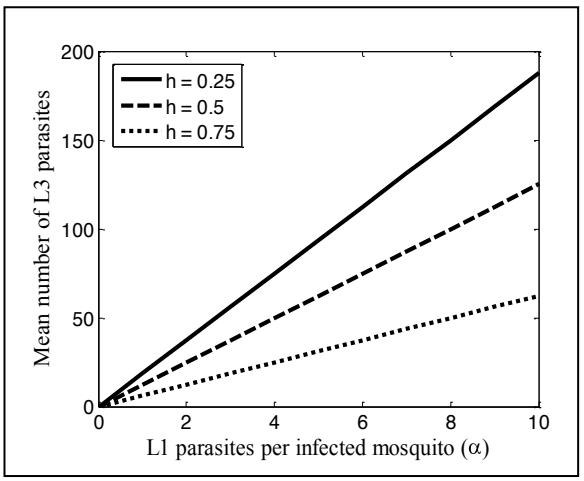


Figure 4: (A) Behavior of mean number of L3 parasites

4 Conclusion

Model equations presented here acquire main mechanisms in vector-parasite interactions. Simulation trials can be used to choose a suitable set of parameters to model different epidemiological settings.

References

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