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Dynamics Model of Bed Bug Infestation in Residential Community

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Abstract: The bed bug, *Cimex lectularius* L. (Hemiptera: Cimicidae), has experienced an extraordinary resurgence in recent years, the reasons for which remain poorly understood. Several studies have focused on determining the susceptibility of bed bug populations to insecticides. However, behavioral responses of bed bug to insecticide residues could influence their efficacy. Understanding the population dynamic and patterns of dispersal may prove critical to the development of effective control strategies. In this study, the two mathematical models for the dynamics of bed bug infestation in a residential community were developed and in which houses are considered to be patches in one of three states: susceptible, infested, or treated. The basic reproduction number and equilibria of the two models are discussed. The heterogeneous houses model was used to assess the effect of different control strategies on house infestation. The empirical results show that spraying only bad houses is more beneficial than spraying the whole community while using the same treatment rate.

Key words: Bed bug, dynamics model, basic reproduction number, residential community

INTRODUCTION

The bed bug, *Cimex lectularius* L. (Hemiptera: Cimicidae), a wingless hematophagous insect thought to have evolved from the ectoparasites of cave-dwelling mammals, have been associated with humans for at least the past 3,500 years (Usinger, 1966; Panagiotakopulu and Buckland, 1999). The name of the “bed bug” is derived from the preferred habitat of *Cimex lectularius*: Warm houses and especially nearby or inside of beds and bedding or other sleep areas. They usually feed on their hosts without being noticed (Goddard, 2009). Although, considered common in the years before World War 2, after the extensive application of organochlorine, organophosphate and carbamate insecticides (Usinger, 1966; Pereira *et al.*, 2009), reports of infestations became extremely sparse in industrialized countries after the 1950s. Recently, a global resurgence of both *C. lectularius* and *Cimex hemipterus* (F.) has occurred (Doggett *et al.*, 2011). They can now be found in hotels, apartments, college dormitories, health care facilities, public transportation systems, in middle-and upper-class single-family residences (Fong *et al.*, 2013). The reasons for the rapid increase and spread of bed bugs are not clear but among the possible factors are the reduction in the use of broad-spectrum residual pesticides, increased exchange of second-hand goods and furniture,

increased international traffic and the evolution of insecticide resistance internationally (Romero *et al.*, 2007; Booth *et al.*, 2012).

A number of adverse health effects may result from bed bug bites, including skin rashes, psychological effects and allergic symptoms (James *et al.*, 2011). Although, bed bug is not known to vector disease (Goddard, 2003), it is nonetheless a significant reemerging public health pest because it affects the human host in many other ways, including swelling and welts resulting in pruritus and secondary infection, the elicitation of immune responses that cause discomfort and psychological stress and social ostracism (Hwang *et al.*, 2005). The economic impact of bed bug infestations is dramatic. Annually, infestations result in millions of dollars in damages through both treatments and lawsuits within the hospitality industry and other residential settings (How and Lee, 2010). In recent years, the application of high-resolution molecular markers has provided important new insight into the population genetic structure and infestation dynamics of many insect pest species of public health concern (Conn and Mirabello, 2007; Szalanski *et al.*, 2011).

Despite the availability of powerful genetic tools to shed light on the biology and management of insect pests, the infestation dynamics of those primarily commensal within human dwellings remains largely

unaddressed. In this study, we studied and developed two mathematical models for the dynamics of bed bug infestation in a residential community. In the models, houses may be in one of three states: Susceptible, infested, or treated. We consider the dynamic properties of two models and assess the effect of different control strategies on house infestation.

BED BUG INFESTATION DYNAMICS MODEL

We study house infestation dynamics following a Kermack- McKendrick model (Kermack and McKendrick, 1991), we study two bed bug infestation models: basic model for a homogeneous house and model for a heterogeneous house. Each house in the residential community is considered as a patch which may be in only one of three states: susceptible, infested or treated. In the two frameworks, within house bed bug population dynamics is ignored. We assume that an infested house have some potential to spread the infestation to other houses in the community. We also consider that a house’s bed bug population has some probability per unit of time of becoming extinct and that houses may be treated (for example, with insecticide). Insecticide is assumed to provide protection against re-infestation for some period of time.

Basic model for homogeneous houses: In this scenario, there is a single community composed of houses of the same quality. The rate of house infestation depends on the number of infested houses as well as the proportion of treated houses in the residential community. More specifically, we assume that infested houses spread the infestation at the rate β . Under the homogeneous assumption, only the fraction S/N of the houses is susceptible to infestation. Infested houses become extinct at the rate γ . Infested houses are treated at the rate τ and the protection conferred is lost at the rate α . A transfer diagram of compartmental model is shown as Fig. 1. where, S, I and T represent the number of susceptible, infested and treated houses, respectively, basic model (called Model 1) has the following form:

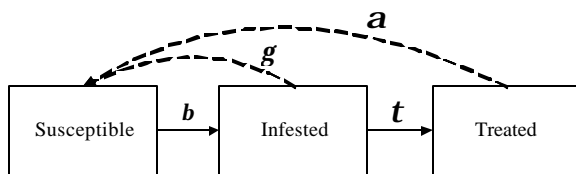


Fig. 1: Compartment model of houses with Susceptible (S), Infested (I) and Treated (T) in a community

$$\frac{dS}{dt} = -\frac{\beta}{N}SI + \gamma I + \alpha T \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - (\gamma + \tau)I \tag{2}$$

$$\frac{dT}{dt} = \tau I - \alpha T \tag{3}$$

where, $\beta > 0$, $\tau > 0$, $\alpha \geq 0$ and $\gamma \geq 0$. The total population size satisfies $N = S(t) + I(t) + T(t)$. The initial conditions satisfy $S(0) > 0$, $I(0) > 0$, $T(0) \geq 0$ and $S(0) + I(0) = N$, where N is the constant total population size, $dN/dt = 0$.

The dynamics of Model 1 like communicable disease models (Hethcote, 2000), they are determined by the basic reproduction number, the basic reproduction number is the number of secondary infections caused by one infected individual in an entirely susceptible population (Hethcote, 2000). We computed the basic reproductive numbers using the next generation operator method (Brauer and Castillo-Chavez, 2012). Basic reproduction number computed for Model 1 is:

$$R_0 = \frac{\beta}{\gamma + \tau} \tag{4}$$

Which has a straight forward interpretation as the number of secondary cases produced by one infested house in a population of susceptible houses. In this case, houses are infested at rate β while:

$$\frac{1}{\gamma + \tau}$$

is the mean infestation period.

According to above Eq. 1-3 of model 1, the following two equilibrium solutions are obtained: the infestation free equilibrium $E_1 = (N, 0, 0)$ and the endemic equilibrium:

$$E_2 = \left(\frac{N(\gamma + \tau)}{\beta}, \frac{\alpha N(\beta - \gamma - \tau)}{\beta(\alpha + \tau)}, \frac{\tau N(\beta - \gamma - \tau)}{\beta(\alpha + \tau)} \right)$$

Considering the initial condition:

$$R_0 = \frac{\beta}{\gamma + \tau}$$

then:

$$E_2 = \left(\frac{N}{R_0}, \frac{\alpha N}{\alpha + \tau} \left(1 - \frac{1}{R_0}\right), \frac{\tau N}{\alpha + \tau} \left(1 - \frac{1}{R_0}\right) \right) \tag{5}$$

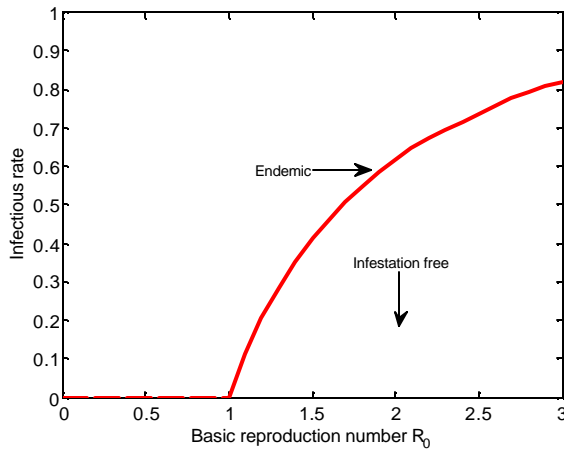


Fig. 2: Bifurcation diagram of basic reproduction number vs. equilibrium value

This expression shows explicitly that endemic equilibrium exists only for $R_0 > 1$. The infestation free equilibrium is stable for $R_0 < 1$ and it is unstable for $R_0 > 1$.

Bifurcation diagram of basic reproduction number vs. equilibrium value of $I(t)$ shows as Fig. 2.

The basic reproduction number R_0 , extinction rate γ and recovered rate after treated α determine the dynamics of model 1. The dynamics are summarized in the following theorem.

Theorem 1: Let $S(t)$, $I(t)$ and $T(t)$ be a solution to model 1:

- If $R_0 \leq 1$, then $\lim_{t \rightarrow \infty} I(t) = 0$ (infestation free equilibrium)
- If $R_0 > 1$, then:

$$\lim_{t \rightarrow \infty} (S(t), I(t), T(t)) = \left(\frac{N}{R_0}, \frac{\alpha N}{\alpha + \tau} \left(1 - \frac{1}{R_0}\right), \frac{\tau N}{\alpha + \tau} \left(1 - \frac{1}{R_0}\right) \right)$$

(endemic equilibrium)

- Assume $\alpha = 0$. If:

$$\frac{R_0 S(0)}{N} > 1$$

then there is an initial increase in the number of infected cases $I(t)$ (epidemic), but if:

$$\frac{R_0 S(0)}{N} \leq 1$$

then $I(t)$ decreases monotonically to zero (disease-free equilibrium)

The quantity $R_0 S(0)/N$ is referred to as the initial replacement number, the average number of secondary infections produced by an infected individual during the period of infectiousness at the outset of the epidemic (Hethcote, 2000). Since, the infectious fraction changes during the course of the epidemic, the replacement number is generally defined as $R_0 S(t)/N$ (Hethcote, 2000). In case iii) of theorem 1, the bed bug infestation eventually disappears from the population but if the initial replacement number is greater than one, the population experiences an outbreak.

Model for heterogeneous houses: In this scenario, there is a community of good and bad houses which are randomly distributed. Mud and thatch roof houses, more ancient houses and filled with old or secondhand furniture are considered “bad” houses as they may sustain high levels of bed bug population infestation (Gurevitz *et al.*, 2011). New or improved houses do not provide well enough conditions for the bed bug population to survive and are labeled as “good” houses. We consider that bad houses act as sources while good houses act as sinks.

When houses are randomly distributed, each house has the same probability to contact good or bad houses. A straightforward modification of model 1 (called model 2) shows as following:

$$\frac{dS_g}{dt} = -\frac{\beta_g}{N} S_g I_g - \frac{\beta_b}{N} S_g I_b + \gamma_g I_g + \alpha_g T_g \tag{6}$$

$$\frac{dI_g}{dt} = \frac{\beta_g}{N} S_g I_g + \frac{\beta_b}{N} S_g I_b - (\gamma_g + \tau_g) I_g \tag{7}$$

$$\frac{dT_g}{dt} = \tau_g I_g - \alpha_g T_g \tag{8}$$

$$\frac{dS_b}{dt} = -\frac{\beta_b}{N} S_b I_b - \frac{\beta_g}{N} S_b I_g + \gamma_b I_b + \alpha_b T_b \tag{9}$$

$$\frac{dI_b}{dt} = \frac{\beta_b}{N} S_b I_b + \frac{\beta_g}{N} S_b I_g - (\gamma_b + \tau_b) I_b \tag{10}$$

$$\frac{dT_b}{dt} = \tau_b I_b - \alpha_b T_b \tag{11}$$

where, the variables denote the total number of houses in the corresponding state and the subindices g and b are used to label good and bad houses, respectively. The total number of good and bad houses is given by:

$$N_g = S_g(t) + I_g(t) + T_g(t) \text{ and } N_b = S_b(t) + I_b(t) + T_b(t)$$

while the total number of houses is $N = N_g + N_b$. Again, we assume both N_g and N_b are constant, namely, $dN_g/dt = 0$ and $dN_b/dt = 0$.

In Eq. 6-8 of model 2, the infested good houses spread the infestation at the rate β_g . Infested good houses become extinct at the rate γ_g . Infested good houses are treated at the rate τ_g and the protection conferred is lost at the rate α_g . A similar description follows for bad houses.

The reproductive number R_0 of model 2 will be greater than one for a population of bad houses and less than one for a population of good houses. Because the bed bug population is substantially higher in bad houses, it assumed that $\beta_b > \beta_g$ and that $\gamma_b > \gamma_g$.

For model 2, the basic reproduction number is defined as follows:

$$R_0^{(2)} = \frac{N_b}{N} R_{0b} + \frac{N_g}{N} R_{0g} \tag{12}$$

Where:

$$R_{0b} = \frac{\beta_b}{\gamma_b + \tau_b}$$

and:

$$R_{0g} = \frac{\beta_g}{\gamma_g + \tau_g}$$

are the basic reproductive numbers of model 1 for the cases of only bad and only good houses.

Similar to model 1, the 2 has the same dynamic properties with Theorem 1.

NUMERICAL SIMULATIONS

Experiment settings: We explored the effect of different control strategies for the different scenarios under study using Model 1 and 2. In all cases, we considered a sample size of 1000 houses.

The initial parameter values used in the simulation are shown in Table 1.

Solution to the model 1: In the infestation Model 1, when $S(0) = 900$, $I(0) = 100$, $\beta = 0.4$, $\gamma = 0.3$ and $\tau = 0.2$, the solution to the Model 1 shows as Fig. 3.

When $S(0) = 900$, $I(0) = 100$, $\beta = 0.8$, $\gamma = \tau = 0.1$, the solution to the Model 1 is shows as Fig. 3.

As shows in Fig. 3 and 4, when $R_0 = 0.8 < 1$, the bed bug infestation model exits the infestation free equilibrium. on the contrary, when $R_0 = 4 > 1$, the bed bug

Table 1: Parameters for simulations of SIT models

Parameter	Definition	Value
β	Colonization rate	0.4
τ	Treatment rate	0.2
α	Removal rate from treatment	0.5
γ	Extinction rate	0.3
β_g	Colonization rate from the good houses	0.2
γ_g	Extinction rate of the good houses	0.4
α_g	Rate to become susceptible after being treated for good houses	0.5
τ_g	Treatment rate of the good houses	[0, 0.05]
β_b	Colonization rate from the bad houses	0.5
γ_b	Extinction rate of the bad houses	0.2
α_b	Rate to become susceptible after being treated for bad houses	0.5
τ_b	Treatment Rate of the Bad Houses	[0, 0.1]
R_{0g}	Basic reproductive number of good houses with no treatment	0.5
R_{0b}	Basic reproductive number of bad houses with no treatment	2.5

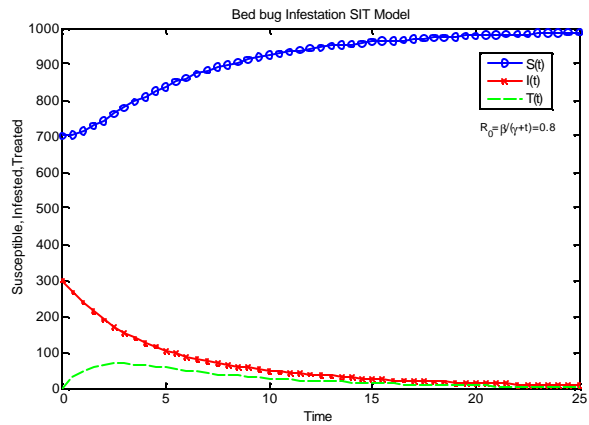


Fig. 3: Solution to the SIT model when $\beta = 0.4$, $\gamma = 0.3$, $\tau = 0.2$ and $R_0 = 0.8$

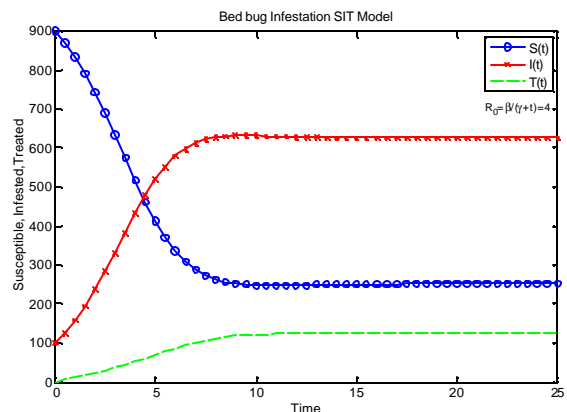


Fig. 4: Solution to the SIT model when $\beta = 0.8$, $\gamma = \tau = 0.1$ and $R_0 = 4$

infestation model exits the endemic equilibrium. The results of two above numerical simulation results further verify the Theorem 1.

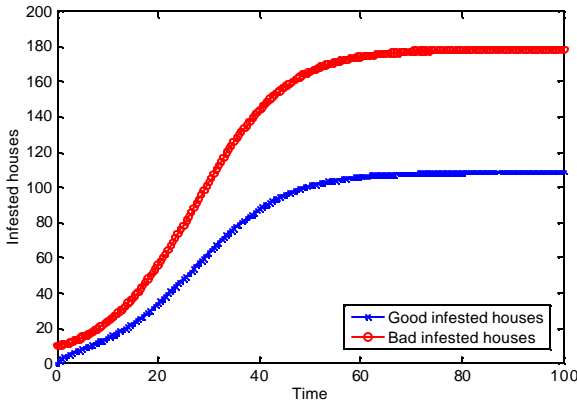


Fig. 5: Infested houses without treatment of $\tau_b = \tau_g = 0$, $R_{0b} = 2.5$ and $R_{0g} = 0.5$

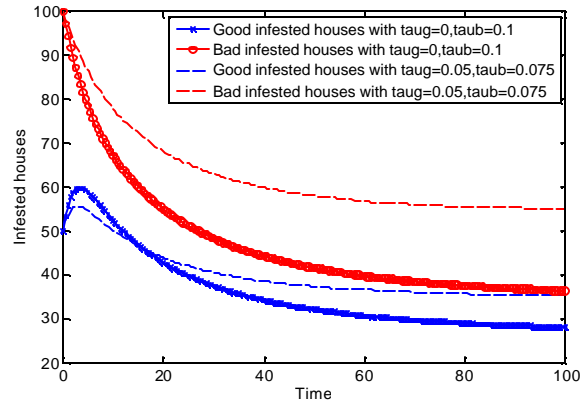


Fig. 7: Spray of only bad houses with $\tau_b = 0.1$ vs. equally spray with $\tau_b = 0.075$ and $\tau_g = 0.05$

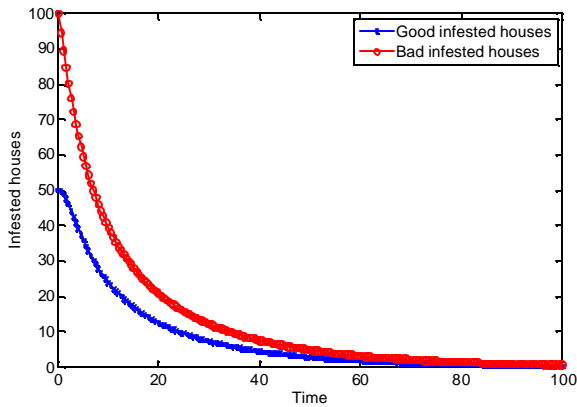


Fig. 6: Infested houses with a treatment of $\tau_b = 0.15$, $\tau_g = 0.15$, $R_{0b} = 1.43$ and $R_{0g} = 0.36$

Solution to the model 2: In model 2, the good and bad houses randomly distributed in the same community. Without treatment, house infestation reaches its equilibrium values monotonically. Figure 5 displays typical solutions, where $N_g = N_b = 0.5N$, $I_g(0) = 0$, $I_b(0) = 0.02N_b$. As expected, the deterministic solution presents an initial phase of exponential growth.

In this numerical simulated experiment, the basic reproduction number of model 2 is $R^{(2)}_0 = 0.5 R_{0b} + 0.5 R_{0g} = 1.5$.

The effect of treatment at a constant rate is shown in Fig. 6, where $N_g = N_b = 0.5N$, $I_g(0) = 0.1N_g$, $I_b(0) = 0.2N_b$.

In this numerical experiment, the basic reproduction number $R^{(2)}_0 = 0.5 R_{0b} + 0.5 R_{0g} = 0.895$.

Efficacy of insecticide spray: One question that may be asked is focusing on the bad houses in case of spray or the whole community including the good houses while

keeping the percentage of pesticide used the same. An analysis is done on both the bad and good randomly distributed houses. We first spray only the bad houses, then the whole community with the same amount of pesticide to see which way is more efficient. Where $I_g(0) = 0.1N_g$, $I_b(0) = 0.2N_b$, $N_g = N_b = 0.5N$. The simulation results show as Fig. 7.

As shows in Fig. 7, in the first case, we used a constant rate $\tau_b = 0.1$ and $\tau_g = 0$ while in the second case, for $I_g(0) = 0.5 I_b(0)$, we used $\tau_b = 0.075$ and $\tau_g = 0.05$ for the use of the same amount of insecticide in both cases. Simulations show that concentrating the treatment on only the bad houses is more efficient.

CONCLUSION

The transmission dynamics of bed bug is complex and varies on geographic location and economic factors (Bernardeschi *et al.*, 2013; Wang *et al.*, 2013). In this study we used a simple metapopulation approach to understand the dynamics of bed bug infestation in a residential community and proposed two SIT models. The basic reproduction number and equilibria of the two models were also discussed. The simulation results demonstrated the efficacy of the proposed models. We also used the heterogeneous houses model to assess the effect of different control strategies and the model predicted that spraying only the bad houses is more efficient than spraying the whole community. This result is consequence of the assumed reduced infestation potential of good houses respect to bad houses.

A further level of realism can be achieved by considering different level of infestation. One can be derived from this is to see the effect of infestation in the

community when very low percentage of pesticide are applied to low infested houses. Because of their short life span of the *T. infestans* (Booth *et al.*, 2012), one may also incorporate the life cycle in the model and look at the period of time that the pesticide take effect on the bed bug population in the community. However, seasonality is an important driver of population dynamics (Fong *et al.*, 2013) and we expect that it will play an even more significant role in models including seasonality in our future works.

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