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# MATHEMATICAL MODELING ON FLIES CONTROL TO PREVENT THE SPREAD OF CARRIER DEPENDENT INFECTIOUS DISEASES

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***Abstract***

*The paper presents a mathematical model to control flies population using biocontrol agent parasitic wasps. Parasitic wasp will control the growth of larvae of flies and thus adult fly population. Mathematical analysis has been done by using stability theory of differential equation.*

***Keywords:*** *Typhoid, Mathematical model, carrier, flies control, modeling*



## 1. Introduction

Many tiny, parasitic wasps attack immature stages of flies. The wasps insert their eggs into the immature stages of several species of flies [1]. The white, legless wasp larvae feed inside the host and eventually kill it. The wasp completes its development, emerges as an adult and continues the process by searching out more hosts [7]. These small wasps only attack flies, they neither sting nor bite other insects, animals, or humans [3].

### 1.1 Model Formulation

In this section, we formulate and analyze an eco-epidemiological model, which combines the two basic models; one describing the predator-prey dynamics of immature stage of flies i.e., pupae stage and parasitic wasps, and the other describing the disease transmission dynamics in human population through direct contact between susceptible and the infected individuals and indirect transmission through adult flies population [4].

In the model formulation, it is assumed that the food-borne diseases, like typhoid fever spread due to direct contact between susceptible and the infected individuals, also indirect transmission through carriers, like flies population [8]. In this study, we assume that the one of the main mode of transmission of the disease is through indirect transmission. However,

other modes of transmission has been observed in the realistic scenario. Therefore, we assume that rest of modes of transmission of the diseases other than carrier are considered under the assumption of the direct contact between the susceptible and infected individuals [2].

It is well known fact that flies transport the bacteria of various infectious diseases, like typhoid, dysentery, etc., from the environment to the edibles of human population [6]. In this way the edibles of humans become contaminated with the bacteria of infectious diseases. Further, When humans consume this contaminated food, they contract diseases, like typhoid, dysentery, etc [5]. The control of the carrier population in a region, where human population reside is one of the effective method to control the spread of the carrier dependent infectious diseases.

Control measures, like chemical control includes the application of insecticides to target the immature stages as well as adult carrier population. Chemical control also include the use of larvicidal sprays, adulticidal baits, adulticidal space sprays and adulticidal surface treatments. These tools may leads to success for the carrier control. However, it has been observed that resistance to these chemicals has been developed in carriers. Therefore, the chemical approaches to control carrier population may not yield estimated results in the long run. On the other hand, biological control agents are more effective to control the growth of carrier population [4]. They almost deny the possibility of developing resistance to chemicals in carrier population.

In a region under consideration, we categories total human population into two epidemiological classes; susceptible individuals S(t) and infected individuals I(t) at a time $t > 0$. We propose the control strategy for carrier population in a region by targeting the immature stage of carrier, i.e., pupae stage P(t) at a time $t > 0$. As carriers grow to adult stage through egg, larva, pupa. Therefore, control of any stage will leads to prevention of adult carrier. The adult carrier population C(t), thus transport the bacteria from the environment to edibles of human population. The natural organisms parasitic wasps W(t) is well known to control the pupae of carrier. The wasps insert their eggs into the pupae of flies. The white, legless wasp larvae feed inside the host and eventually kill it. The wasp completes its development inside the pupae of flies. In this way, the wasps emerges as an adult and continue the process by searching out new hosts. These small wasps only target pupae of flies. They neither sting nor bite other insects, animals, or humans. Therefore, its use is

preferential over the chemical based approaches and it does not harm any nontarget population like, human population.

Based on the above assumptions, the population dynamics of the carrierdependent infectious diseases and their control are governed by the following system of non-linear differential equations:

$$\frac{dS}{dt} = A - \beta SI - \lambda SC - \mu S + \nu I,$$
$$\frac{dI}{dt} = \beta SI + \lambda SC - (\alpha + \mu + \nu)I,$$
$$\frac{dP}{dt} = gC - \mu_1 P - \alpha_1 P^2 - \gamma P - \phi PW, \quad (1)$$
$$\frac{dC}{dt} = \gamma P - \theta_0 C,$$
$$\frac{dW}{dt} = \kappa\phi PW - \theta_1 W,$$

In the model system (1), is analyzed with the following initial conditions

$S(0) > 0$, $I(0) > 0$, $P(0) \geq 0$, $C(0) \geq 0$, $W(0) \geq 0$.

Here, we simplify the biological cycle of carrier population, like flies. The flies grow to adult through eggs, larvae and pupae, P with birth into pupae stage and natural death rate in each stage. From the perspective of epidemiology, adult fly will transport the bacteria from the environment and transport the humans' edibles, leads to the transmission of disease in humans. Thus, pupae control is an important measure for adult fly control. For the immature fly, i.e., pupae, the natural death rate in human population and the maturation rate are μ and γ, respectively. Further, the crowding in pupae of fly cannot be ignored in small garbage area. Thus, we use α1 to denote the density dependent development mortality of pupae. To account for the predation of immature flies by predators, i.e., parasitic wasps W has been considered and we assume the bilinear response form with a constant rate ϕ, similar to that in Lotka-Volterra model.

For the sake of simplicity, we write $N(t) = S(t) + I(t)$. Now, it is easy to see that the model system (1) is equivalent to the following model system:

$$\frac{dN}{dt} = A - \mu N + \alpha I,$$
$$\frac{dI}{dt} = (\beta I + \lambda C)(N - I) - (\alpha + \mu + \nu)I,$$
$$\frac{dP}{dt} = gC - \mu_1 P - \alpha_1 P^2 - \gamma P - \phi PW, \quad (2)$$
$$\frac{dC}{dt} = \gamma P - \theta_0 C,$$

$\frac{dW}{dt} = \kappa\phi PW - \theta_1 W,$

**Parameters definition**

A = constant birth or immigration rate,

β = direct contact rate,

λ = indirect transmission of the bacteria through flies,

μ = natural death rate in humans,

α = disease induced mortality,

ν = recovery rate of infected humans,

g = egg laying rate of flies,

μ1 = natural death rate of pupae or death rate of pupae by other factors, like climatic factors,

α1 = coefficient of intraspecific competition,

γ = maturation rate (the rate at which pupae becomes adult),

ϕ = depletion coefficient due to attack of parasitic wasps at fly's pupae,

k = the conversion efficiency of wasps,

θ0 = death rate of adult fly population,

θ1 = death rate of wasps.

Our first task is to show that the model system (2) is biologically meaningful.

**2. Analysis of the systems' equilibria**

**2.1** Equilibria Feasibility

We begin the model analysis by discussing the feasibility conditions of four nonnegative equilibria of the model system (2), which are listed as follows:

**(i)** Disease free equilibrium E1 ( A /μ , 0, 0, 0, 0 ) is always feasible,

**(ii)** Endemic equilibrium in absence of carrier E2 (N2, I2, 0, 0, 0) is feasible if,

$$R_p = \frac{\beta A}{\mu(\nu+\alpha+d)} > 1, \quad (3)$$

**(iii)** The disease persistent equilibrium with carrier population in absence of wasps $E_3$ (N3, I3, P3, C3, 0) is feasible if

$$R_p = \frac{g\gamma}{\theta_0\{\mu_1+\gamma\}} > 1, \quad (4)$$

**(iv)** The coexistence equilibrium E ∗ (N∗ , I∗ , P∗ , C∗ , W∗ ) is feasible if

$$R_w = \frac{g\gamma}{\theta_0\left\{\mu_1+\gamma+\frac{\alpha_1\theta_1}{k\phi}\right\}} > 1 \quad (5)$$

At the disease free equilibrium E1 ( A /d , 0, 0, 0, 0 ) , no infected humans and carrier population are present, thus no control effort is needed. Therefore, total human population remains susceptible which equals to $Ad^{-1}$ .

At the endemic equilibrium E2, only human population survives in absence of carriers and parasitic wasps. This shows that disease spread in human population due to direct contact between the susceptible and infected individuals.

At the disease persistent equilibrium E3, disease spread in human population when the carriers are present and parasitic wasps are absent.

At the coexistence equilibrium $E^*$, all the components are present. This captures the spread of the disease as well as control effort.

Now, we discuss the feasibility conditions of all the possible equilibria of the model system (2) one by one. The feasibility of the all equilibria of the model system (2) may be obtained by the setting the derivatives zero present in the model system (2).

We first find the components of the disease free equilibrium $E_1$, using W = 0, P = 0 and C = 0, and then from the second equilibrium equation of the model system (2), we choose I = 0. Thus, from the first equilibrium equation, total human population settles to $Ad^{-1}$ .

Now substituting W = 0 in the third equilibrium equation of the model system (2), we discuss the feasibility of the equilibrium E2. From the fourth equilibrium equation, we have

$$C_2 = \frac{\gamma}{\theta_0} P_2 \tag{6}$$

Using equation (6) in the third equilibrium equation, we obtain either P2 = 0 or

$$P_2 = \frac{1}{\alpha}\left\{\frac{g\gamma}{\theta_0} - (\mu_1 + \gamma)\right\}, \tag{7}$$

Assuming P2 = 0, we have C2 = 0, from equation (6). Now, using C2 = 0 and the value of N2 from the first equilibrium equation, in the second equilibrium equation of the model system (2), we obtain

$$I_2 = \frac{\mu(\nu+\alpha+\mu)}{\beta(\alpha+\mu)}(R_0 - 1) \tag{8}$$

For the positive value of I2, the condition (3) must be satisfied, which is R0 > 1, where R0 denotes the basic reproduction number of the disease when carrier and wasps are absent. It is defined as the average number of secondary infection caused by an index case in wholly susceptible population in an individuals' entire infectious period.

Now, we discuss the feasibility of the equilibrium E3. From fifth equilibrium equation, we have W = 0. From the fourth equilibrium equation, we have

$$C_3 = \frac{\gamma}{\theta_0} P_3 \tag{9}$$

Using equation (9) in the third equilibrium equation and assuming P3/= 0, we obtain

$$P_3 = \frac{1}{\alpha_1}\left\{\frac{g\gamma}{\theta_0} - (\mu_1 + \gamma)\right\}, \tag{10}$$

It is easy to see that P3 > 0, provided condition (4) is satisfied, which is Rp > 1. The expression Rp is meaningful, since one fly vector can produce an average of g pupae per unit time, which will survive upto an adult stage with the probability of γ μ1+γ , and (θ0) −1 is the average lifespan of the flies. Thus, the adult fly can produce gγ θ0(μ1+γ) adult flies in its lifetime. Denoting this threshold by Rp = gγ θ0(μ1+γ) , which is the feasibility condition (4), where wasps is absent. Borrowing the idea of basic reproduction number from biology [], we say Rp the carrier reproduction number. Substituting (10) in equation (9), we can obtain C3. Now using (9) and the value of N3 from the first equilibrium equation in the second equilibrium equation, we get the following quadratic

$$a_1 I_3^2 + a_2 I_3 + a_3 = 0, \tag{11}$$

Where

$a_1 = \frac{\beta\,(\alpha+\mu)}{\mu}$

$a_2 = \frac{-\beta\, A}{\mu} + \frac{\lambda\,(\alpha+\mu)C_3}{\mu} + \nu + \alpha + \mu$

$a_3 = \frac{-\lambda\, AC_3}{\mu}$

From the above expression a1, a2 and a3, we assert that the equation (11) has unique positive solution. Therefore, the equilibrium E3 is feasible if the condition (4) is satisfied.

Finally, we will find the conditions for the feasibility of the coexistence equilibrium E ∗ . The component of the coexistence equilibrium for the model system (2) may be obtained by the following set of algebraic equations:

$$A - \mu N + \alpha I = 0, \tag{12}$$

$$(\beta I + \lambda C)(N - I) - (\alpha + \mu + \nu)I = 0, \tag{13}$$

$$gC - \mu_1 P - \alpha_1 P^2 - \gamma P - \phi PW = 0, \tag{14}$$

$$\gamma P - \theta_0 C = 0, \tag{15}$$

$$\kappa\phi PW - \theta_1 W = 0, \tag{16}$$

Now from equation (16), we get W = 0 or

$$P^* = \frac{\theta_1}{k\phi} \tag{17}$$

then from equation (15), we obtain

$$C^* = \frac{\gamma}{\theta_0} P^* \tag{18}$$

Using equations (17) and (18) in the equation (14), we get

$$W^* = \frac{\gamma}{\theta_0} P^* \tag{19}$$

Clearly, W∗ > 0 if the condition (5) is satisfied. Now, using the value of N∗ from the equation (12) in the equation (13), we obtain a quadratic equation in I ∗ as follows:

$$b_1 I^{*2} + b_2 I^* + b_3 = 0, \tag{20}$$

where

$b_1 = \frac{\beta\,(\alpha+\mu)}{\mu}$

$b_2 = \frac{-\beta\, A}{\mu} + \frac{\lambda\,(\alpha+\mu) I^*}{\mu} + \nu + \alpha + \mu$

$b_3 = \frac{-\lambda\, A I^*}{\mu}$

It is clear from the above expressions that the equation (20) has a unique positive solution. Further, using the value of I ∗ in the equation (12), we obtain the value of N∗ . Therefore, the coexistence equilibrium E ∗ is feasible provided condition (5) is satisfied.

**2.2** Stability analysis

In this section, we discuss the stability of the equilibria.

**Theorem 1** (i) The equilibrium E1 is locally asymptotically stable if R0 < 1 and Rp < 1.

(ii) The equilibrium E2 is locally asymptotically stable if R0 > 1 and Rp < 1.

(iii) The equilibrium $E_3$ is locally asymptotically stable if $R_w < 1$ and it is unstable if $R_w > 1$, i.e., when

E * exists.

(iv) The equilibrium E * is always locally asymptotically stable.

Proof.

The Jacobian matrix of the (2) is given by

a11 = −d, a21 = βI + λC, a22 = (βI + λC) − β(N − I) + ν + α + μ, Let ψ denote an eigenvalue of the Jacobian matrix J.

## 3 Conclusion

To control the spread of these diseases, it is advisable to keep away the edibles of humans so that humans will not contract the infection through flies.

## References

*Zhao X. Q., Jing Z., (1996). Global asymptotic behavior in some cooperative systems of functional-differential equations. Can. Appl. Math. Q., 4, 421444.*

*Loftin KM, Hopkins JD, Corder R. Biology and Control of Flies in Poultry Facilities. Agri Nat Res 2014;FSA7063-PD-8-2014RV.*

*Fly Predators, Available at: http://www.planetnatural.com/beneficial-insects-101/fly-predator/.*

*Das P, Mukherjee D, Sarkar AK. Study of carrier-dependent infectious diseasecholera J Biol Syst 2005;13:233-244.*

*Okamoto KW, Amarasekare P. The biological control of disease vectors. J Theo Biol 2012;309:47-57.*

*Kumari N, Sharma S. Does water disinfectant play a supportive role in the spread of infectious disease? A mathematical study. Nat Res Model 2016;29(2):259-288.*

*Code¸co CT. Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir. BMC Infect Dis 2001;1(1):1-14.*

*Gonzalez-Guzman J. An epidemiological model for direct and indirect transmission of Typhoid Fever, Math Biosci 1989;96:33-46.*