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Disease dynamics in predator-prey population with disease in the prey

****Emmanuel O. Oghre and Dennis I. Egherha***

Department of Mathematics
University of Benin, Benin City. Nigeria

Abstract

Population and epidemiological models are constructed based on plausible explicit and implicit biological assumptions. While species spreads disease, it also competes with other species for space or food or is predated by other species. We have presented a predator prey system with disease in the prey putting the prey into three classes viz the susceptible, the infected but not yet infectious class and the infectious class. Both the sound and infected but not yet infectious prey and infectious prey are bounded above for all time and the total population at infinity is asymptotically constant. The threshold parameter $R_0 \equiv \frac{a}{\mu}$ ***controls the dynamics of the system.***

1.0 Introduction

While population ecology studies populations and how they change over time due to changes as a result of the various interactions between them and their resources, epidemiology aims at devising vaccination policies to fight infectious diseases. The two fields however are merging in recent years with the introduction of population growth in epidemic models. [5, 10]. The main object of modeling in this new area is to answer the role of infectious disease in regulating natural populations, that is, decreasing their sizes and either reducing their natural fluctuations or causing destabilization of equilibrium positions, into oscillations of the population states.

Basically in epidemiology, population can be classified into two categories: susceptible and infected. However when an individual is infected, he stays for some definite period after leaving the susceptible class and joining the intermediate class. This intermediate period is termed the incubation period. The incubation period is defined as the time from exposure to onset of disease. That is, it corresponds to the time from infection with a micro organism to symptom development [2]. While species spreads disease, it also competes with other species for space or food or is predated by other species. Therefore it is of biological significance to consider the effect of interacting species when we study the dynamical behavior of epidemiological models so an appropriate model is essential to study the effect of disease on interacting species.

The Lotka – Volterra model is the simplest way to model predator – prey interactions while the Kermack-Mckendrick model has been used to study Susceptible - Infective – Removal (SIR) epidemiological model [1]. Based on these two pioneering works several models have been developed in these areas [3, 8, 9]. Freedman [4] presented a study on predator prey system in which some members of the prey population and all predators are subject to infection by parasite. Mukherjee [11] studied a stochastic prey predator model with disease in the prey whereby he presented the stability analysis of the model. Haque et al [7] in a study of an ecoepidemiological prey predator model with standard disease incidence analysed the local and global stability of the system of equation and found the threshold property below which the infection disappears. They found out that a sufficiently strong disease in the prey may avoid predator extinction and its presence can distabilise an otherwise stable predator prey configuration. Dhar and Sharma [2] introduced the role of the incubation period in a disease model which created a new class of infected but not yet infectious individuals. In this work we consider a predator prey model, in which a disease that can be transmitted by contact spread among the prey population taking into consideration the infected but not yet infectious individuals.

2.0 Model Formulation

We consider a predator – prey population model in which a disease that can be transmitted by contact spreads among the prey. We assume that the disease places new recruits from the susceptible class into infected but not yet infectious class (exposed) for a period of incubation. This set of new recruits we denote by $\varphi(t)$. As usual $S(t)$, $I(t)$ and $P(t)$ represent

*Corresponding authors: Emmanuel O. Oghre, E-mail: eoghre@yahoo.com , Tel. +234 (0) 8033551266

the susceptible prey, the infectious prey capable of transmitting the disease and the predators class

We assume the disease that can be transmitted among the prey by contact. The contact rate is taken in the form of bilinear mass action incidence law. The way an individual leaves the susceptible class is by becoming infected at the rate **a or** by being consumed by predators at the rate **c**. Each infected but not yet infectious φ(t) individual generates $I(t)$ new individual per unit time while $S(t)\ I(t)$ individuals generate the infected but not yet infectious class φ. The ways an individual in the infected but not yet infectious class φ can leave the class is by being infectious at the rate β individuals per unit time or by being consumed at the rate **c**, which is the same as the rate the predators consume the susceptible. This is true because at this rate, the symptom has not developed, hence will not hinder its efficiency to avoid predators. Also we assume some of the individuals in the infectious class will die naturally, or die due to the infection at the rate μ **,** while others are being consumed by predator at the rate m. We also assume that the contact rate between the infected and not yet infectious class and the susceptible class cannot spread the disease. We define K as the carrying capacity of the prey and the predator's net gains for consuming the susceptible class, infected but not yet infectious class is θ_1, while θ_2 is the net gain for consuming the infectious class and that $\theta_1 > \theta_2$.

Based on these assumptions, we have the following differential equations respectively for the susceptible class, infected but not yet infectious class, the infected class and the predator class

$$\left.\begin{aligned} \frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right) - cSP - a\,SI \\ \frac{d\phi}{dt} &= aSI - \beta\phi - c\phi\,P \\ \frac{dI}{dt} &= \beta\phi - mIP - \mu I \\ \frac{dP}{dt} &= \theta_1 SP + \theta_1\phi P + \theta_2 IP - \delta P \end{aligned}\right\} \tag{1}$$

The initial conditions are given as

$$S(0) \geq 0\ ,\ \phi\ (0) \geq 0\ ,\ I(0) \geq 0\ ,\ P(0) \geq 0 \tag{2a}$$

$$r > 0,\ a > 0,\ \mu > 0,\ \beta > 0,\ \theta_1 > 0,\ \theta_2 > 0,\ c > 0,\ \delta > 0,\ m > 0\ . \tag{2b}$$

3.0 System Boundedness.

Consider the first equation of system (1),

$$\frac{dS}{dt} = rS\left(1-\frac{S}{K}\right) - cSP - aSI$$

$$\frac{dS}{dt} \leq rS\left(1-\frac{S}{K}\right) \tag{3}$$

Consider $0 < t_0 \leq t$,

$$S(t_0) \leq K \Rightarrow S(t) \leq K$$

To show boundedness we define a function

$$W = S + \phi + I + \omega P \tag{4}$$

where $$\omega = \min\left\{\frac{c}{\theta_1}\ ,\ \frac{m}{\theta_2}\right\} \tag{5}$$

The time derivative of equation (4) along a solution of system (1) is

$$\frac{dW}{dt} = \frac{dS}{dt} + \frac{d\phi}{dt} + \frac{dI}{dt} + \omega\frac{dP}{dt} \tag{6}$$

Using equations (1), equation (6) becomes

$$\frac{dW}{dt} = rS\left(1-\frac{S}{K}\right) - cSP - aSI + aSI - \beta\phi - c\phi P + \beta\phi - mIP - \mu I$$

$$+ \theta_1 \omega SP + \theta_1 \omega \phi P + \theta_2 \omega IP - \delta \omega P$$

$$\frac{dW}{dt} = rS\left(1 - \frac{S}{K}\right) - (c - \omega\theta_1)SP - (c - \omega\theta_1)\phi P - (m - \omega\theta_2)IP - \mu I - \omega\delta P \quad (7)$$

Using equation (5), (7) reduces to

$$\frac{dW}{dt} = rS\left(1 - \frac{S}{K}\right) - \mu I - \omega\delta P \quad (8)$$

Multiplying both sides of equation (3.4) by $\eta > 0$ gives

$$\eta W = \eta S + \eta\phi + \eta I + \eta\omega P \quad (9)$$

Adding equation (8) and (9), we then have,

$$\frac{dW}{dt} + \eta W = rS\left(1 - \frac{S}{K}\right) - \mu I - \omega\delta P + \eta S + \eta\phi + \eta I + \eta\omega P$$

$$\frac{dW}{dt} + \eta W = rS\left(1 - \frac{S}{K}\right) - (\mu - \eta)I - (\omega\delta - \eta\omega)P + \eta S + \eta\phi \quad (10)$$

If we take $\eta \leq \min(\mu, \delta)$, equation (10) reduces to

$$\frac{dW}{dt} + \eta W \leq rS\left(1 - \frac{S}{K}\right) + \eta S + \eta\phi \quad (11)$$

We see that the right hand side of the above inequality is bounded. We can then find $l > 0$ such that

$$\frac{dW}{dt} + \eta W \leq l \quad (12)$$

So that

$$\frac{dW}{l - \eta W} \leq dt \quad (13)$$

Integrating equation (14) with respect to time t in the interval $(0, t_0)$ where $(0 < t_0 \leq t)$

$$\int_0^{t_0} \frac{dW}{(l - \eta W)} \leq \int_0^{t_0} dt \quad (14)$$

to give us

$$W(t_0) \leq -\frac{1}{\eta}(1 - \eta W(0))e^{-\eta t_0} + \frac{l}{\eta} \quad (15)$$

From the inequality (15), we see that at time t = 0, the total population of the susceptible, infected not yet infectious, infectious and the predator is $W(0)$ where $W(t_0)$ is the total population at time $t_0 > 0$. At any time t, we have

$$W(t) = \left(W(0) - \frac{l}{\eta}\right)e^{-\eta t} + \frac{1}{\eta} \quad (16)$$

When $t \to \infty$, we have

$$\lim_{t \to \infty} W(t) \leq \frac{1}{\eta} \quad (17)$$

Since $\frac{l}{\eta}$ is a constant, then for a suitable M independent of the initial condition, we have

$$\lim_{t \to \infty} W(t) \leq \frac{1}{\eta} = M \quad (18)$$

The above shows that the total population at infinity is asymptotically constant.

4.0 Conditions For Epidemic Outbreaks

From the second and third equation of system (1), if follows that

$$\frac{d(\phi + I)}{dt} = asI - c\phi p - mIp - \mu I \quad (19)$$

$$\frac{d(\phi + I)}{dt} \leq (aS - \mu) I$$

$$\frac{d(\phi + I)}{dt} \leq (aS - \mu) I$$

This implies that

$$\frac{dI}{dt} \leq (aS - \mu) I \tag{20}$$

Using the proposition in [7],

Let $R_0 \equiv \frac{a}{\mu}$. Both the sound and infected but not yet infectious prey and infectious prey are bounded above for all time;

$$S(t) \leq \max \{K, S(0)\},$$

$$I(t) \leq k_0 \equiv k\left(\frac{a}{\mu} - 1\right)$$

If $R_0 < 1$, the infection disappears.

$$\lim_{t \to \infty} I(t) = 0 \tag{21}$$

Thus in a predator-prey ecosystem in which the prey is affected by a disease, there is an upper limit on the number of the infected prey. The infection can propagate only if $R_0 > 1$, a threshold phenomenon closely related to the "basic reproductive ratio'' R_0 of the classical epidemic theory.

5.0 Conclusion

In this work we have presented a predator prey system with disease in the prey putting the prey into three classes, the susceptible, the infected but not yet infectious class and the infectious class. Intra-specific competition of the infected prey and predator are also incorporated in the model. Both the sound and infected but not yet infectious prey and infectious prey are bounded above for all time and the total population at infinity is asymptotically constant. The threshold parameter $R_0 \equiv \frac{a}{\mu}$ controls the dynamics of the system. The infection can propagate only if $R_0 > 1$

References

[1] Chattopadhyay J., Arino O. (1999). A Predator-Prey Model with Disease in The Prey. *Nonlinear Analysis* ***36****, 747- 766*

[2] Dhar J., Sharma A. K. (2009). The Role of the Incubation Period in A Disease. *Applied Mathematics E-Note., 9 146-153*

[3] Eshel G. (2008). The Lotka –Volterra Predator-Prey Model. A Lecture note on Mathematical Ecology.

[4] Freedman H. I. (1990). A model of Predator-Prey dynamics as modified by the action of a parasite *Math. Biosci ,99:143-155.*

[5] Gao, L. Q. Hethcote, H. W. (1992). Disease transmission models with density dependent demographs. *Journal of Mathematical Biology. 30:* 717 - 731

[6] Hadeler K. P., Freedman H. I. (1989). Predator-Prey Populations with Parasitic Infection. *Mathematical Biology,* ***27****, 609- 631.*

[7] Haque, M., Zhen J., Venturino E. (2008). An Ecoepidemiological Predator- Prey Model with Standard Disease incidence. *Mathematical Methods in the Applied Sciences. 1071*

[8] Hethcote, H. W. *(2000) The mathematics of Infectious Diseases. SIAM Review; 42: 599-653*

[9] Kuang Y. (2002). Basic Properties of Mathematical Population Models. *Mathematical Methods in the Applied Sciences, 1071.*

[10] Mena-Loca, J. and Hethcote, H. W.(1992) *Dynamic models of infectious diseases as regulator of population sizes. Journal of Mathematical Biology. 30;* 693-716

[11] Mukherjee D. (2003). Stability Analysis of a stochastic model for Predator- Prey System with Disease in the Prey. *Nonlinear Analysis: Modeling and control* ***8****, p 83-92.*