A Review of "Four Predator Prey Models with Infectious Diseases,"[1] by Litao Han and Zhien Ma, Xi'an Jiaotong University, and H.W. Hethcote, University of Iowa

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MATH 361, Technical Article Review, February 14, 2011

SUMMARY

1. The Introduction

The authors of this paper present an analysis of the influence of infectious diseases on predator prey ecological interactions by presenting scenarios involving two different cases. The first where the infectives (those able to become diseased) become susceptible to the disease again after recovery (SIS), and the second case, where infectives develop a permanent immunity to the infection after recovery (SIR).

For the four models considered, a global stability is achieved in which the natural predator prey equilibrium is not destroyed. The models presented in this article differ from previous predator prey models with infectious diseases because they are based on a predator prey model with a stable equilibrium and present the possibility of the disease persisting in the predator population by being passed on through the feeding cycle.

2. Incidence Forms and the Predator Prey Model

Section 2 presents the incidence forms for the SIS and SIR models. The total number of prey (N_1) for the SIS model (no immunity after recovery) is given by

$$N_1 = S_1 + I_1 \tag{1}$$

Where S_1 is the number of prey susceptible to infection, and I_1 is the number of infected prey. Similarly, predator population for SIS models is

$$N_2 = S_2 + I_2 \tag{2}$$

Population in SIR models are presented in the same fashion except that the recovered population (R_1) must also be accounted for. The equations then become:

$$N_1 = S_1 + I_1 + R_1 \tag{3}$$
$$N_2 = S_2 + I_2 + R_2 \tag{4}$$

The average number of contacts a preyed upon animal has with an infected animal per unit time (β_1) is taken into account, so that the average number of contacts that occur between infected animals and susceptible animals can be given by:

$$\frac{\beta_1 S_1}{N_1}$$

Therefore, the standard incidence,

$$\frac{\beta_1 S_1}{N_1} I_1 \tag{5}$$

is the number of new cases per unit time due to I_1 infectives.

In addition to this, the number of new cases per unit time can also be modeled by the simple mass action law,

$$\eta_1 I_1 S_1 \tag{6}$$

where η_1 is a mass action coefficient.

When compared to Equation (5) it can be seen that this model assumes that the contact rate will increase linearly with respect to the population size as shown by

$$\beta_1 = \eta_1 N_1$$

In human diseases however, the contact rate does not appear to increase linearly with population size, so the standard incidence is a better approximation. In this paper however, the authors examine models using both types of incidence.

The predator-prey model used by the authors is a modification of the Lotka-Volterra equations with density dependent, logistic growth of the prey:

$$\begin{aligned} \dot{N}_1 &= r_1 \left(1 - \frac{N_1}{K_1}\right) N_1 - aN_1N_2 \\ &= \left[r_1 \left(1 - \frac{N_1}{K_1}\right) - aN_2\right] N_1, \end{aligned} \tag{7}$$
$$\dot{N}_2 = kaN_1N_2 - d_2N_2 = (kaN_1 - d_2)N_2$$

3. Predator Prey SIS Model With Standard Incidence

Section 3 presents a predator prey model with an SIS infection and standard incidence. Equation (7) is combined with Equations (1) and (2), and the standard incidence model, (5). It is then reduced to a set of four differential equations, that contain solutions for all positive times in the domain D,

$$D = \{(I_1, N_1 m I_2, N_2) \mid 0 \le I_1 \le N_1 \le K_1, 0 \le I_2 \le N_2\}$$

Good job

These kinds of insights are vital to a good mathematical modeling project.

In which K_1 is the prey carrying capacity. Three epidemiological threshold values are obtained, along with six equilibrium points within D. Theorem 4 shows the six solution paths of the system in D, and the six cases are interpreted into real life biological examples in Corollary 5. After these explanations of the meaning of these situations, the cases are proven.

4. Predator Prey SIS Model With Mass Action Incidence

In Section 4, the predator-prey SIS model is repeated in much the same way, but with the mass action incidence model, (6), instead of the standard incidence model. Like Section 3, the model exists in D, and has similar threshold values and equilibria in the region. The proof was omitted for this section, due to its similarity to Section 3.

5. Predator Prey SIR Model With Standard Incidence

Section 5 examines the SIR model, in which infected animals gain immunity after recovery, using Equations (3) and (4), and the standard incidence model.

The six possible cases for this section require a much more lengthy proof, involving the Jacobian of the limit equations and expressing the trace and determinant for the functions to prove that the equilibria are always locally asymptotically stable.

6. Predator Prey SIR Model With Mass Action Incidence

Section 6 analyzes an SIR model identical to that of the previous section, but as in Section 4, employs the mass action incidence model instead of the standard incidence model. The thresholds and some equilibria are different than the model in Section 5, but the theorem and proof are identical, except for minor changes, so they are omitted.

REVIEW

Our group found the paper to be very informative. The introduction provided accessible information about previous models, as well as clearly presenting the basic premises behind the four models in this paper. The four models were a good choice, as all were closely related, yet distinct enough to each have their own applications. The authors did an excellent job of providing citations from other's work, and explaining their additions to past models. The work is significant as it is the first to consider cases where predators could catch the disease.

On the down side however, some of the equations could have been explained better. On several occasions, constants controlling the birth-rate, or death-rate just appeared in the equations without even being mentioned, let alone explained. The sub-scripted variables and constants were sometimes confusing to us, but overall the paper was fairly clear, and showed quality work.

This will be your main goal in your project.

INTEREST

We found the subject of predator prey models to be interesting, as they present ways in which communities of animals interact and their populations grow, shrink, and settle into equilibria. This particular model added in the variable of an infectious disease which presents a new set of equations governing survival. Since epidemics have been an influential (no pun intended) part of human history, it is worth studying models such as these. The applications of these models extend to areas outside of mathematics such as biology, medicine, sociology, and public policy.

Our group chose to review this article, because we are interested in further investigation of unique predator prey and population models. As a group project, we might possibly examine the population models of various other specialized cases. The population model of a cannibalistic insect society, in which the food supply is increased as death occurs, for example. The ideas of infectious diseases and population growth could also be applied to a zombie apocalypse, with very interesting mathematical results.

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[1] Litao Han, Zhien Ma, H. W. Hethcote, Four Predator Prey Models with Infectious Diseases, *Mathematical and Computer Modelling*, **34**, 849-858, (2001).