Mathematical Analysis of the Control of the Spread of Infectious Disease in a Prey-Predator Ecosystem

E.A. Bakare[1,2],Y.A Adekunle[3], A.Nwagwo[4]

[1] *Department of Computer and Info Sc*

Lead City University, Ibadan, Oyo State, Nigeria.

[2] *Department of Mathematics*

University of Ibadan, Ibadan, Oyo State, Nigeria.

[3] *Department of Computer and Mathematics*

Babcock University, Ilishan-Remo, Ogun State, Nigeria.

[4] *Department of Mathematics*

Yaba College of technology, Lagos State, Nigeria.

***Abstract*-We present a model for the mathematical analysis of the control of the spread of an infectious disease in a predator-prey ecosystem. In this work, we present a compartmental mathematical model expressed by a systems of differential equations based on the dynamics of the Infection. We discuss the Disease Free Equilibrium(DFE) and the Endemic Disease Equilibrium(EDE). In this study, we realized that to eradicate or reduce the intensity of disease spread in the prey-predator ecosystem ,we apply vaccination strategies with herd immunity.**

Keywords: Compartmental mathematical model, herd immunity, endemic disease equilibrium, disease free equilibrium, vaccination strategies.

I. INTRODUCTION

When species interact, the population dynamics of each species is affected. In general there is a whole web of interacting species, called a trophic web, which makes for structurally complex communities. We consider here, systems involving two or more species, concentrating particularly on two species systems. There are three main types of interaction.(i) If the growth rate of one population is decreased and the other increased the populations are in a predator-prey situation.(ii) If the growth rate of each population is decreased, then it is competition.(iii) If each population's growth rate is enhanced, then it is called mutualism or symbiosis[8].Some mathematical models have been developed in this area. In 1926, Volterra [13] first proposed a simple model for the predation of one species by another to explain the oscillatory levels of certain fish catches in the Adriatic. This model was based on four assumptions. Firstly, the prey grows unboundedly in a Malthusian way in the absence of any predation. Secondly, the effect of the predation is to reduce the prey's per capita growth rate by a term proportional to the prey and the predator populations.

Thirdly, in the absence of any prey for sustenance the predator's death rate results in exponential decay. Fourthly, the prey's contribution to the predator's growth is proportional to the available prey as well as the size of the predator population. The model is $\frac{dN}{dt} = N(a - bp)$ and $\frac{dP}{dt} = P(cN - d)$ where N is the prey population and P is the predator Population. This model also called Lokta-Volterra model was analyzed. Murray [8] modified the Lokta-Volterra model by changing of the assumptions made by Volterra. In the model he obtained:

$$\frac{dN_1}{dt} = r_1 N_1 \left[1 - \frac{N_1}{K_1} - b_{12}\frac{N_2}{K_2}\right]$$

$$\frac{dN_2}{dt} = r_2 N_2 \left[1 - \frac{N_2}{K_2} - b_{21}\frac{N_1}{K_2}\right]$$ Where

$r_1, k_1, r_2, k_2, b_{12}, b_{21}$ are all positive constants. This model was analyzed and the conditions for stability established. Bedington et al [2] presented some results on the dynamic complexity of coupled predator-prey systems. Dunbar [3,4] studied in detail a modified Lokta-Volterra system with logistic growth of the prey and with both predator and prey dispersing by diffusion."Predator-Prey model are arguably the building blocks of the bio and

ecosystems as biomasses are grown out of their resources to sustain their struggle for their very existence. Depending on their specific settings of applications, they can take the forms of resource-consumer, plant-herbivore, parasitic-host, tumor cells (virus)-immune system, susceptible-infectious interactions, etc. They deal with the general loss-win interactions and hence may have applications outside of ecosystems. When seemingly competitive interactions are carefully examined, they are often in fact some facts of predator-prey interaction in disguise"[5]. Another approach to modeling the interaction between prey and predators was developed to account as well for organisms (such as bacteria) taking up nutrients and this is called Jacob-Mond Model. This model was discovered independently in the several diverse applications. It is akin to the Haldane-Briggs Model and Michaelis-Menten Model in Biochemistry the Jacob-Mond Model in microbial ecology and the Beaverton-Holt model in fisheries. It serves as one of the important building blocks in studies of complex biochemical reactions and in ecology [12]. B.Dubey and R.K Upadhay, in their paper, a mathematical model is proposed and analyzed to study the dynamics of one-prey two-predator system with ratio-dependent predators' growth rate. Criteria for local stability, instability and global stability of the nonnegative equilibria are obtained. The permanent co-existence of the three species is also discussed. Finally, computer simulations are performed to investigate the dynamics of the system. S.Pathak et al in his work, we discovered that over the past hundred years, mathematics has been used to understand and predict the spread of disease, relating important public-health questions to basic transmission parameters. From prehistory to the present day, diseases have been a source of fear and superstition. A comprehensive picture of disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations to statistical analysis. Although mathematics has been so far done quite well in dealing with epidemiology but there is no denying that there are certain factors which still lack proper mathematization.

Almost all mathematical models of disease start from the base premise: that the population can be subdivided into a set of distinct classes dependent upon their experience with respect to the disease. One line of investigation classifies individuals as one susceptible, infectious or recovered. Such a model is termed as an SIR model. Disease transmission is a dynamical process driven by the interaction between the susceptible and the infective. Many models of epidemiology are based on the so called "mass action" assumption for transmission. In this work, we have considered the case of the mathematical modeling of the spread of disease (infection) in Predator-Prey ecosystems. This paper is organized as follows. In the next section, we present the model assumptions. In the third section, we present the model equations and described various parameters and terms in the model. In the fourth section, we carry out the qualitative analysis of the model. Stability criteria's for the disease free equilibrium and the endemic equilibrium are derived. Basic reproductive number were also discussed. The fifth section presents an illustrative example for the model. In the sixth section, we present different computer simulations of the system. In the last section, the biological significance of our analytical and numerical findings are discussed.

II.MODEL ASSUMPTIONS

The following examines the evolution of a predator-prey system, after an infectious disease has been introduced into the colony. We assume the following:

(a) The disease is benign to the prey; that is, the prey are carriers. The relative birth rate for Infected prey remains the same as that of the healthy susceptible prey.

(b) The disease is debilitating and ultimately fatal for the predators. Once a predator is Infected, it can be assumed to be dead. We will therefore consider only one population of predators, those that are susceptible.

(c) The disease is spread among the prey by contact, and the rate of infection is proportional to the infected and the susceptible population.

(d) The predators make no distinction between susceptible and infected members of the Prey population.

(e) The predator contract the disease by consuming the prey. The rate of predator infection is proportional to the product of infected prey and susceptible predators.

(f) The model is applied to study the effect of vaccination strategies on the disease.

III. THE MODEL EQUATION

The model we analyzed in this paper is considered under the framework of the following nonlinear ordinary differential equations:

$$\frac{dR_2}{dt} = -a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i}(1-\theta)$$

$$\frac{dR_{1s}}{dt} = a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i}(1-\theta) + d_2R_{1,i}$$

$$\frac{dR_{1i}}{dt} = a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i}(1-\theta) - d_2R_{1,i}$$

(3.1)

At this points we will observe a qualitative change when a smooth small change is made to the parameter values(bifurcation parameters).

TABLE1
Description of variables for transmission model

Variable	descriptions
R_{1i}	Number of Infected Prey at time t
$R_{1,s}$	Number of Susceptible Prey at time t
R_2	Number of healthy Susceptible Predators at time t

TABLE 2
Description of constants for transmission model

Constant	descriptions
a_1	Natural death of the Healthy Susceptible Predator
a_2	per capita birth rate of Susceptible Prey (per time) and Infected Prey
b_1	Number of contact between Healthy Susceptible Prey and Healthy Predator.
b_2	Number of contact between HealthySusceptiblePredator and infected prey.
c_1	Number of contact between Healthy Susceptible Predator and Infected Prey
d_2	rate at which infected Prey (carriers) are removed.
c_2	Number of contact between Healthy Susceptible Prey and Infected Prey
θ	Proportion of those successively vaccinated at birth.

IV. ANALYSIS OF THE MODEL

$$\frac{dR_2}{dt} = -a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i}(1-\theta)$$

$$\frac{dR_{1s}}{dt} = a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i}(1-\theta) + d_2R_{1,i}$$

$$\frac{dR_{1i}}{dt} = a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i}(1-\theta) - d_2R_{1,i}$$

From (3.1)

The equilibria are obtained by setting the right-hand side of system (3.1) equal to zero, giving the following:

Solution

$$-a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i}(1-\theta) = 0$$

$$a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i}(1-\theta) + d_2R_{1,i} = 0$$

$$a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i}(1-\theta) - d_2R_{1,i} = 0$$

(4.1)

The system in (3.1) has two equilibrium solutions
A disease-free equilibrium at (0, 0, 0) $=(R_2^*, R_{1,s}^*, R_{1,i}^*)$
An endemic equilibrium at $(\frac{a_2}{b_2}, \frac{d_2}{c_2(1-\theta)}, \frac{-a_1c_2(1-\theta)+b_1d_2}{c_1c_2(1-\theta)^2}) = (R_2^*, R_{1,s}^*, R_{1,i}^*)$

We determine the stability of the equilibrium points by computing the Jacobian Matrix of the system (3.1) at each equilibrium point

$$J(R_2, R_{1,s}, R_{1,i}) = \begin{pmatrix} \frac{\partial f}{\partial R_2} & \frac{\partial f}{\partial R_{1,s}} & \frac{\partial f}{\partial R_{1,i}} \\ \frac{\partial g}{\partial R_2} & \frac{\partial g}{\partial R_{1,s}} & \frac{\partial g}{\partial R_{1,i}} \\ \frac{\partial h}{\partial R_2} & \frac{\partial h}{\partial R_{1,s}} & \frac{\partial h}{\partial R_{1,i}} \end{pmatrix}$$

A. The stability of the disease free state

The Jacobian of equation (1.1) at the equilibrium point is

$J(R_2, R_{1,s}, R_{1,i}) =$

$$\begin{pmatrix} -a_1 + b_1R_{1,s} - c_1R_{1,i}(1-\theta) & b_1R_2 & -c_1R_2(1-\theta) \\ -b_2R_{1,s} & a_2 - b_2R_2 - c_2R_{1,i}(1-\theta) & -c_2R_{1,s}(1-\theta) + d_2 \\ -b_2R_{1,i} & c_2R_{1,i}(1-\theta) & a_2 - b_2R_2 + c_2R_{1,s}(1-\theta) - d_2 \end{pmatrix}$$

In the absence of Infection , the Jacobian of (1.1) at

the disease-free equilibrium V_0= (0, 0, 0) is

$$J(0,0,0) = \begin{pmatrix} -a_1 & 0 & 0 \\ 0 & a_2 & d_2 \\ 0 & 0 & a_2 - d_2 \end{pmatrix}$$

$$\text{J-}\lambda I = \begin{vmatrix} -a_1 - \lambda & 0 & 0 \\ 0 & a_2 & d_2 \\ 0 & 0 & (a_2 - d_2) - \lambda \end{vmatrix}$$

$$-a_1 - \lambda[(a_2 - \lambda)(a_2 - d_2) - \lambda)] = 0$$

$$[\,(-a_1 - \lambda_1)(a_2 - \lambda_2)(a_2 - d_2) - \lambda_3)] = 0$$

$$\lambda_1 = -a_1,\ \lambda_2 = a_2\ ,\ \lambda_3 = a_2 - d_2$$

Theorem1: The disease-free equilibrium V_0 is locally stable if $R_\theta < 1$ and Unstable if $R_\theta > 1$ where

$$R_\theta = (1-\theta)\frac{c_2}{d_2}$$

Proof : λ_1 is negative, it remains to prove that λ_2 and λ_3, the roots of the quadratic part of the characteristic polynomial of J are both positive. We know that using Routh-Hurwitz Conditions (theorem) it is the case when $\lambda_2 + \lambda_3 > 0$ and $\lambda_2 \lambda_3 > 0$

As $\lambda_2 + \lambda_3 = (a_2 + a_2 - d_2) > 0$ is true, ($if\ a_2 > 0\ and\ d_2 > 0$)

We are done with $\lambda_2 \lambda_3 = a_2(a_2 - d_2) > 0$ (if $a_2 > 0\ and\ d_2 > 0$) ■

Note:

. R_θ is the effective reproduction number in the presence of vaccination.

.If $\theta = 0$, we have the basic reproduction number $R_0 = \frac{c_2}{d_2}$

V.OPTIMAL VACCINATION STRATEGIES: CONTROLLING the SPREAD of the DISEASE in the ECOSYSTEM by HERD IMMUNITY

A. Herd Immunity

Many Infectious diseases can be controlled when there is availability of effective and cheap vaccination. Although, it is totally impossible to vaccinate everyone in the system against the disease.

We need to determine the percentage of a population that needs to be vaccinated in order to eliminate the disease from the population.

A population of people(animals) is said to have HERD IMMUNITY to a disease when enough people(animals) are immune to the disease so that if it is introduced into the population, it will not spread throughout the population. To have herd immunity, an infected person must infect less than one uninfected person during the time that the person is infectious.

Herd immunity can also be referred to has the level of immunity in a population which prevents epidemics, even if some transmission may still occur. For instance, when a cohort of 1000000 newborns benefit a 90% vaccine coverage, it yields 900,000 vaccinated and 100,000 unvaccinated. If vaccine efficacy is only 95%, it gives 855000 immune and 45000 vaccinated but non-immune. Thus, it sums up to 855000 immune and 145000 susceptible and the corresponding herd immunity is 85.5%.

It is well-known that the higher R_0 is for a disease, the higher the proportion of the population will have to be vaccine to achieve her immunity. Although, this statement could seem very theoretical it was almost the perspective followed by WHO's technical working group, when devising strategies to control a full range of diseases; for instance, this procedure has succeeded during the worldwide campaign for small pox eradication in the 1960s

B. The criteria for the Control

Let θ be the proportion immune after a vaccination campaign. To reach the critical proportion θ_c, we need the control condition (To have herd immunity, an infected person(animal) must infect less than one uninfected person during the time that the person(animal) is infectious. Thus, we must have $R_0 S < 1$)

Consequently herd immunity is achieved when $R_0(1-\theta_c) < 1$ it means that $R_0(1-\theta_c) = 1 \iff 1\text{-}\theta_c = \frac{1}{R_0} \iff \theta_c = 1 - \frac{1}{R_0}$ then $R_0 - R_0\theta_c < 1\ such\ that\ -R_0\theta_c < 1 - R_0$

$\therefore \theta_c > \frac{R_0 - 1}{R_0} = 1 - \frac{1}{R_0}$

For Instance, in most sub-Saharan Africa Countries, the basic reproductive number for (e.g. measles is approximately 18) so θ_c= 0.94.

Under the schedule of a unique dose, the ,minimal coverage to control measles is such that everyone does not need to be immune through vaccination to control measles. We need high herd immunity to succeed control, it may require everyone receiving a 95% efficacious vaccine as Coverage × efficacy = 0.94 □ coverage = 0.99 .We conclude that it is quite impossible via a single opportunity schedule. Similarly, for disease like chickenpox the basic reproduction number R_0 is approximately 11.3,so θ_c= 0.91.

Under the schedule of a unique dose, the minimal coverage to control chickenpox is such that everyone does need to be immune through vaccination to control chickenpox(varicella). We need high herd immunity to succeed control. It may require everyone receiving a 95% efficacious vaccine as Coverage × efficacy = 0.91 □ coverage = 0.96.

We conclude that this coverage is also quite impossible via a single opportunity schedule.

VI. NUMERICAL SIMULATION

Numerical simulations are carried out to illustrate some of the theoretical results in this paper .We take parameters of the system as a1= 1.0, a2 = 1.0, b1 = 1.0, b2 = 1.0, c1 = 1.0, c2 = 0.5, d1 = 1.0, θ = 0.94 . We take parameters of the system also as $R_2(0)$= 0.9, $R_{1,s}(0)$= 1.90,$R_{1,i}(0)$=0.80 at time t = 0,over the time interval [0,20].

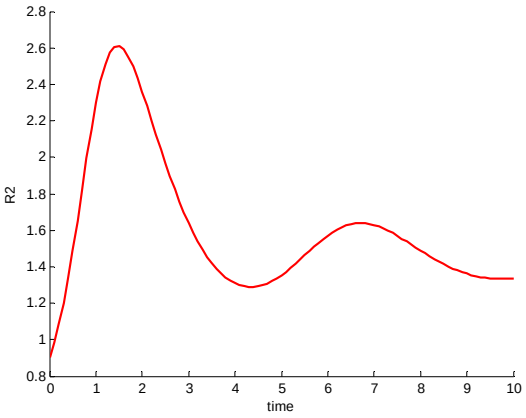

Fig 5a: Describes the rate of change of population of R2 with respect to time

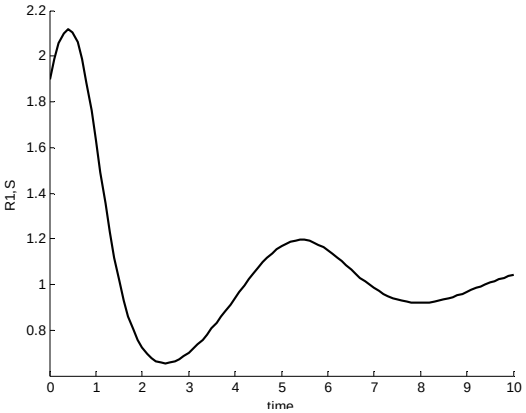

Fig 5b:Describes the rate of change of population of R1,S with respect to time

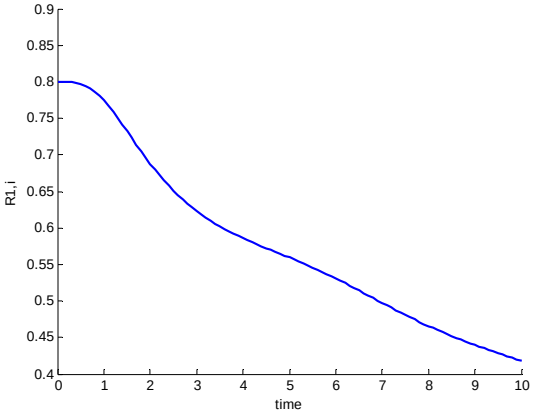

Fig 5c: Describes the rate of change of population of R1,i with respect to time

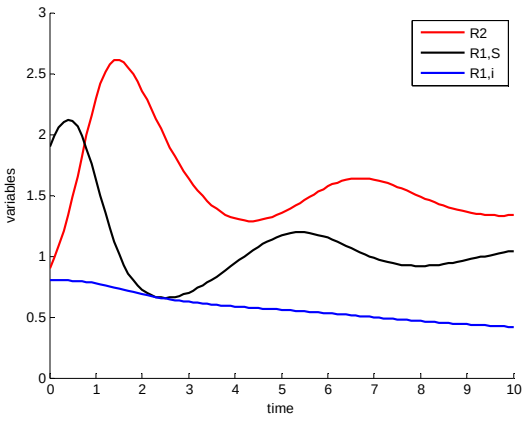

Fig 5d: Describes the rate of change of population of R1,S, R1,I and R2 with respect to time

VII. CONCLUSIONS

A deterministic model for the transmission dynamics of a disease in a Prey-Predator ecosystem is designed and rigorously analysed. The study, which allows for the assessment of an intervention strategy based on vaccination of the Predator and population.The basic reproduction number, R_0 is a key concept in epidemiology, and is arguably one of the foremost and valuable ideas that mathematical thinking has brought to epidemic theory (Heesterbeek and Dietz 1996). Most importantly, R_0 often serves as a threshold parameter that predicts whether an infection will spread. It is very important to found that conceptual tool as R_0 can solve concrete problem as devising optimal vaccination strategies for all range of diseases. Determination of the threshold parameter R_0 theoretically is of important public health interest.

Although the simulation results above are based on a set of parameter values, for which uncertainties in parameter estimates may exist, the study suggests that a potential epidemic can be effectively controlled using basic public health control measures such as isolation of symptomatic individuals and infection-reduction measures (such as taking precautions against handling poultry products, wearing face-masks, minimizing contacts etc.).

Mathematically, our results stand upon local stability of the disease-free equilibrium point (DFE). We have observed and studied the local stability of endemic equilibrium again by linearization, Jacobian matrix and Routh-Hurwitz theorem. We hope to do further work on this much later.

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