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Implication of Predator Interaction of the Spread of Hantavirus Infection

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Abstract Hantaviruses are etiological agents of zoonotic diseases and certain other diseases, which pose a serious threat to human health. When rodent and predator populations share in an ecology, the competitive force of the populations can lead to a reduction or elimination of a hantavirus outbreak. The effect of the predator eliminating rodents and predator populations that tends to reduce or eliminate hantavirus infection is investigated. The existence of several equilibrium points of the model is identified and local and global stabilities of the model at these equilibrium points are analysed in detail. Numerical simulations are carried out to illustrate our model results.

Keywords Hantavirus; predator-prey model; Lyapunov function; competition dynamics

Mathematics Subject Classification

1 Introduction

Hantaviruses are etiological agents carried by rodents and are transmitted via air, soil, surfaces and water. Rodents cause a serious hygienic risk as transmitters of numerous diseases to humans and livestock; for instance: hantavirus pulmonary syndrome (HPS), hantavirus renal syndrome (HFRS) and leptospirosis [1]. It is maintained persistently in infected rodents which consequently serves as a natural reservoir of the virus in a real ecology.

In 2014, one of the major emerging diseases in Brazil, which led to high mortality rate, was hantavirus cardiopulmonary syndrome (HCPS, also called HPS) [2]. [3] introduced a basic mathematical model for hantavirus infection based on ordinary nonlinear differential equations. The spatio-temporal patterns of the spread of hantavirus infection were studied using this model. Several researchers have made contribution in relation to the spread of hantavirus infection using mathematical models [4–11].

In a natural ecology, rodents share the resource and environment with other animal species. This results in inter and intra-species competition for resources [12]. As stated by [13], rodents undergo two types of competition: intraspecific, among their own species, and interspecific, with aliens.

Predators can limit the size of populations and sometimes even eliminate a species from a community [14]. [12] studied the effect of biodiversity on the prevalence of the infection by using a model in which a single alien population competes with the host i.e. rodents. They showed that an existence of the second species has an important consequence for the prevalence of the infectious agent in the host. When the two rodent species exist in the ecosystem, the competitive pressure of the second species may lead to a reduction or complete elimination of the prevalence of the infection. [15] mentioned evidences where diseases decrease with an increasing number of predators. Among the evidences were correlation between bank vole population size and human cases of *nephropathia epidemica* (caused by the Puumala form of hantavirus) in Sweden, Belgium, and Finland, respectively. They produced a way to illustrate how predator removal can alter disease levels in an SI (susceptible-infected) model. The problem we aim to study is the effect of 3 species in the ecology - hantavirus carrying rodents, a first predator and a second predator which eats both the rodent and the first predator.

The topic of predator-prey model has been investigated extensively from the mathematical point of view by [16] and [17]. [16] studied the chaotic dynamics of a three species prey-predator competition model with bionomic harvesting due to delayed environmental noise as external driving force. They considered a biological economic model based on prey-predator interactions to study the dynamical behavior of a fishery resource system consisting of one prey and two predators surviving on the same prey. Equilibrium points with local and global stabilities were investigated. They also derived the biological and bionomical equilibriums of the system. They have analysed the population intensities of fluctuations i.e., variances around the positive equilibrium due to noise with incorporation of a constant delay leading to chaos. We will adopt the tools and techniques used in their paper for our study. A mathematical model which consists of non-linear simultaneous differential equations has been developed by [17] to investigate a chaotic dynamics of a three species prey-predator competition model with noise in ecology. They discussed the model of one prey and two predators system with the effect of stochastic perturbation. The predator survives on the prey and the predator interact survives on both prey and predator. In their model, noise just contributes even more fluctuations of intensities to the system that causes chaos. The results of the numerical simulations showed that a trajectories of the system oscillate randomly with remarkable variance of amplitudes with the increasing value of the strength of noises initially but ultimately fluctuating. Therefore, they concluded that due to inclusion of stochastic perturbation which creates a significant change of intensity in their considered dynamical system for a small change of sensitive parameters which causes large environmental fluctuations.

2 Formulation of Mathematical Equations

The model of [3] of no movements in a single population of rodents is given by

$$\frac{dr_s}{dt} = br - cr_s - \frac{r_s r}{k} - ar_s r_i$$
$$\frac{dr_i}{dt} = -cr_i - \frac{r_i r}{k} + ar_s r_i.$$

Here the susceptible rodent is denoted by r_s, the infected rodent is denoted by r_i and the total population of rodents is $r(t) = r_s(t) + r_i(t)$. The parameters b, $c$and$a$ are the birth rate, the natural death rate and the aggression parameter (i.e. the transmission rate), respectively. $k$is the most important parameter compared with the other parameters and is called as the environmental parameter. If the value of $k$is too high, it means higher availability of water, food, shelter and other resources for the rodents to thrive. [3] stated that the infected rodent, r_i tends to zero and disease free when $k < k_c$ and the infection thrives when $k > k_c$ where $k_c = \frac{b}{a(b-c)}$. Refer to [10] for additional discussion on the basic AK model.

In the model of [12], the host and alien populations are considered, and the ecosystem is described by first order nonlinear differential equations

$$\frac{dr}{dt} = (b-c)\, r - \frac{r}{k}(r + qz)$$
$$\frac{dz}{dt} = (\beta - \gamma)\, z - \frac{z}{\kappa}(z + \varepsilon r),$$

where r, z are the host and alien populations, respectively. All coefficients are positive constants. A limitation process in the rodent population growth is denoted by $-\frac{r^2}{k}$. The environmental parameter of host in the absence of the population of alien ($z = 0$) is denoted by$k$ and it depends on time. q and κ are the influence and carrying capacity of alien population, respectively. The parametersβ, γ and ε are the corresponding parameters (to b, c, q) for the alien population to get resources from the other species. Note that this study assumes there is only a single alien and it is not a predator of rodents.

The results for the model of [12] showed the critical value of the environmental parameter

$$k_c = \frac{b}{a(b-c)} + \frac{qz}{b-c}$$

that separates three distinctive regimes. If $k < k_c$, the competitor population is greater than the minimum necessary to force the infected subpopulation to extinction. If $k > k_c$, the system has a positive prevalence of infection and the point $k = k_c$ constitutes a critical point of the system, separating two behaviors that qualitatively differ in the stability of the equilibrium of the infected population. In the first case when $q < 1$ and $\varepsilon < 1$, the intensity of the interacting competition is not very high between both population (i.e. rodent and alien) and the coexistence is stable. In the second case when $q > 1$ and $\varepsilon > 1$, the competition is strong and bistability occurs: the final state depends on the initial conditions. In the last case when $q > 1$ and $\varepsilon < 1$ (or $q < 1$ and $\varepsilon > 1$), only the strong competitor survives. Refer to [11] for additional discussion on the Peixoto and Abramson model.

3 One Rodent Two Predators Model

We assume that our system consists of rodents (both infected and susceptible; deemed the host species), a first predator of rodents and a second predator which is a predator of both the first predator and the rodents.

Das *at el.* [17] have proposed a mathematical model for a three species multi-system as

given by the following first order nonlinear differential equations as

$$\left.\begin{aligned}\frac{dr}{dt} &= a_1 r - r\left(\alpha_{11} r + \alpha_{12} z_1 + \alpha_{13} z_2\right)\\ \frac{dz_1}{dt} &= -z_1\left(\alpha_{22} z_1 + \alpha_{23} z_2\right) + z_1\left(a_2 + \alpha_{21} r\right)\\ \frac{dz_2}{dt} &= -\alpha_{33} z_2^2 + z_2\left(a_3 + \alpha_{31} r + \alpha_{32} z_1\right)\end{aligned}\right\} \tag{1}$$

where r, z_1 and z_2 are populations of prey, a first predator and a second predator species respectively. a_1, a_2, a_3 are the natural growth rates of the three species and α_{11}, α_{22}, α_{33} are the rate of decrease of the species due to its insufficient resources. For the prey population α_{13} is the rate of the prey due to inhibition by the second predator. For the first predator population α_{21} is the rate of increase of the first predator due to its successful attacks on the prey and α_{23} is the rate of decrease of the predator due to inhibition by the predator. For the second predator population α_{31} is the increase of the second predator due to its successful attacks on the prey and α_{32} is the rate of increase of the second predator due to its successful attacks on the first predator. All parameters are assumed to be positive constants.

A mathematical model based on the two differential equations for the analysis of the spread of hantavirus infection that was introduced by [12] as a model for competition dynamics of two species is

$$\left.\begin{aligned}\frac{dr}{dt} &= (b-c)\, r - \frac{r}{k}\left(r + qz\right)\\ \frac{dz}{dt} &= (\beta - \gamma)\, z - \frac{z}{\kappa}\left(z + \varepsilon r\right)\end{aligned}\right\} \tag{2}$$

where r, z are the populations of the host and alien, respectively. For the host population, b is birth rate, c is the natural death rate, k is the environmental parameter in the absence of an alien population ($z = 0$) and q is the influence of the alien population. For the alien population, β, γ and ε are the corresponding parameters to get resources from the other species and κ is a environmental parameter.

From the Peixoto and Abramson model (model (2)), the host population is maintained and then the alien population is modified by changing the populations of first predator and second predator similar to the model of [17] (model (1)). Consequently, the result is the one rodent two predators model as follows

$$\begin{aligned}\frac{dr}{dt} &= (b-c)\, r - \frac{r}{k}\left(r + qz\right)\\ \frac{dz_1}{dt} &= -\left(\beta - \gamma\right) z_1 z + z_1\left(a_2 + \varepsilon r\right)\\ \tfrac{dz_2}{dt} &= -\left(\beta - \gamma\right) z_2^2 + z_2\left(a_3 + \varepsilon\left(r + z_1\right)\right)\end{aligned}$$

where r, z_1, z_2 are populations of rodent, first predator and second predator, respectively while $r(t) = r_s(t) + r_i(t)$ and $z(t) = z_1(t) + z_2(t)$ are the total population of rodents and predators, respectively For the rodents population, b and c are the birth rate and the natural death rate, respectively. The parameter k is the environmental parameter in the absence of the predators population ($z_1 = z_2 = 0$) and the value q is the influence of the predators population (z_1 and z_2). For the predators populations (z_1 and z_2): β and γ are corresponding parameters

for the predators population to get resources from the other species where $(\beta - \gamma)$ is the death rate of predator with an assumption that the death rate is always positive if $\beta > \gamma$ for all the time and ε is the rate of increase of the predators population due to its successful attacks on the other species. The value of the parameter ε chosen is the same for the first and second predators population to ensure the rate of increase of both populations have the same successful attack (i.e., kill and eat) on rodent in the ecosystem. a_2 and a_3 are the natural growth rates of the predators population.

Suppose an internal classification of the rodent model is used where r_s is the susceptible rodent, r_i is the infected rodent and that r is the total rodent population

$$r(t) = r_s(t) + r_i(t).$$

Thus the one rodent two predators becomes

$$\left.\begin{aligned} \frac{dr_s}{dt} &= br - cr_s - \frac{r_s}{k}(r + qz) - ar_s r_i \\ \frac{dr_i}{dt} &= -cr_i - \frac{r_i}{k}(r + qz) + ar_s r_i \\ \frac{dz_1}{dt} &= -(\beta - \gamma) z_1 z + z_1(a_2 + \varepsilon r) \\ \frac{dz_2}{dt} &= -(\beta - \gamma) z_2^2 + z_2(a_3 + \varepsilon(r + z_1)) \end{aligned}\right\} \tag{3}$$

where r_s, r_i, z_1, z_2 represent the population of susceptible rodent, infected rodent, first predator and second predator, respectively and the parameter a is the transmission rate of the infection.

4 Model Analysis

In this analysis, we follow the works of [19], [20] and [21]. The local stability analysis of the equilibrium is more straightforward and can be done based upon the standard linearization technique and using the Jacobian matrix. To compute the equilibrium points, we solve the model (3) by assuming

$$\frac{dr_s}{dt} = 0,\ \frac{dr_i}{dt} = 0,\ \frac{dz_1}{dt} = 0 \text{ and } \frac{dz_2}{dt} = 0.$$

So, the positive equilibrium points of the model (3), namely $E_0\left(0, 0, 0, \frac{a_3}{\beta - \gamma}\right)$, disease-free equilibrium, $E_1(r_s^*, 0, 0, z_2^*)$ where

$$r_s^* = \frac{k(b - c)(\beta - \gamma) - qa_2}{\beta - \gamma},\ z_2^* = \frac{a_3}{\beta - \gamma},\ E_2(r_s^*, r_i^*, 0, z_2^*)$$

with

$$r_i^* = \frac{\beta - \gamma - a_3}{\varepsilon} - r_s^*,\ z_2^* = ar_s^* - kc - \frac{\beta - \gamma - a_3}{\varepsilon}$$

and interior equilibrium, $E_3(r_s^*, r_i^*, z_1^*, z_2^*)$ where

$$r_i^* = k(b - c) - q(z_1^* + z_2^*) - r_s^*,\ z_1^* = \frac{\varepsilon k(b - c) - (q\varepsilon + (\beta - \gamma)) z_2^* + a_2}{q\varepsilon + (\beta - \gamma)}$$

$$z_2^* = \frac{\varepsilon k(b - c) - (q\varepsilon + (\beta - \gamma)) z_2^* + a_2}{(q\varepsilon + (\beta - \gamma))[\varepsilon(1 - q) + (q\varepsilon + (\beta - \gamma))]}$$

Linearization of model (3) about the interior equilibrium, $E_3\left(r_s^*,\, r_i^*,\, z_1^*, z_2^*\right)$ gives the Jacobian matrix

$$J\left(r_s^*, r_i^*, z_1^*, z_2^*\right) = \begin{bmatrix} b-c-\frac{1}{k}\left(2r_s^*+r_i^*+qz^*\right)-ar_i^* & b-\frac{r_s^*}{k}-ar_s^* \\ -\dfrac{r_i^*}{k}+ar_i^* & -c-\frac{1}{k}\left(r_s^*+2r_i^*+qz^*\right)+ar_s^* \\ \varepsilon\, z_1^* & \varepsilon\, z_1^* \\ \varepsilon\, z_2^* & \varepsilon\, z_2^* \end{bmatrix}$$

$$\begin{bmatrix} -\dfrac{q}{k}r_s* & -\dfrac{q}{k}r_s* \\ -\dfrac{q}{k}r_i* & -\dfrac{q}{k}r_i* \\ -\left(\beta-\gamma\right)\left(2z_1*+z_2*\right)+\left(a_2+\varepsilon r*\right) & -\left(\beta-\gamma\right)z_1^* \\ \varepsilon\, z_2^* & -2\left(\beta-\gamma\right)z_2^*+\left(a_3+\varepsilon\left(r^*+z_1^*\right)\right) \end{bmatrix}$$

Now, the Jacobian matrix of model (3) at equilibrium $E_0\left(0,\, 0,\, 0,\, \dfrac{a_3}{\beta-\gamma}\right)$ is given by

$$J\left(0,\, 0,\, 0,\, \frac{a_3}{\beta-\gamma}\right) = \begin{bmatrix} b-c & b & 0 & 0 \\ 0 & -c-\dfrac{qa_3}{k\left(\beta-\gamma\right)} & 0 & 0 \\ \dfrac{a_3\varepsilon}{\beta-\gamma} & \dfrac{a_3\varepsilon}{\beta-\gamma} & \dfrac{a_3\varepsilon}{\beta-\gamma} & -2+a_3 \end{bmatrix}.$$

The characteristic equation of the equilibrium $E_0\left(0,\, 0,\, 0,\, \dfrac{a_3}{\beta-\gamma}\right)$ of the model (3) is

$$\left(b-c-\lambda\right)\left(-c-\frac{qa_3}{k\left(\beta-\gamma\right)}-\lambda\right)\left(-a_3+a_2-\lambda\right)\left(-2+a_3-\lambda\right)=0.$$

Thus, eigenvalues of the characteristic equation of the equilibrium $E_0\left(0,\, 0,\, 0,\, \dfrac{a_3}{\beta-\gamma}\right)$ are $\lambda_1 = b-c$, $\lambda_2 = -c-\dfrac{qa_3}{k\left(\beta-\gamma\right)}$, $\lambda_3 = a_2-a_3$ and $\lambda_4 = a_3-2$. It is clear that λ_2 is negative when $\beta>\gamma$ and λ_1, λ_3, λ_4 are positive of which implies that E_0 is unstable. Then, λ_1, λ_2, λ_3, λ_4 will be negative if $b<c$, $\beta>\gamma$, $a_2<a_3$, $a_3<2$, respectively. Hence, the equilibrium E_0 of the model (3) is locally asymptotically stable.

The following theorem, stated and proven by [7], demonstrates the local stability of the positive equilibrium $E_0\left(0,\, 0,\, 0,\, \dfrac{a_3}{\beta-\gamma}\right)$ of the model (3).

Theorem 1.1 *The equilibrium E_0 of model (3) is locally asymptotically stable for $b<c$, $a_2<a_3$ and $a_3<2$ and unstable when $b>c$, $a_2>a_3$ and $a_3>2$.*

Evaluated at the disease-free equilibrium $E_1(r_s*,\ 0,\ 0,\ z_2*)$ has

$$J(r_s^*,\ 0,\ 0,\ z_2^*) = \begin{bmatrix} b-c-\frac{1}{k}(2r_s^*+qz_2^*) & b-\frac{r_s^*}{k}-ar_s^* & -\frac{q}{k}r_s^* & -\frac{q}{k}r_s^* \\ 0 & -c-\frac{1}{k}(r_s^*+qz_2^*)+ar_s^* & 0 & 0 \\ 0 & 0 & -(\beta-\gamma)z_{2^*}+(a_2+\varepsilon r_s^*) & 0 \\ \varepsilon z_2^* & \varepsilon z_2^* & \varepsilon z_2^* & -2(\beta-\gamma)z_2^*+(a_3+\varepsilon r_s^*) \end{bmatrix}.$$

where $r_s^* = \dfrac{k(b-c)(\beta-\gamma)-qa_2}{\beta-\gamma}$, $z_2^* = \dfrac{a_3}{\beta-\gamma}$.

We now study the coexistent steady value E_1. The corresponding characteristic equation of the above Jacobian matrix is

$$\left(b-c-\frac{1}{k}(2r_s*+qz_2^*)-\lambda\right)\left(-c-\frac{1}{k}(r_s^*+qz_1^*)+ar_s^*-\lambda\right) (-(\beta-\gamma)z_2^*+(a_2+\varepsilon r_s^*)-\lambda)(-2(\beta-\gamma)z_2^*+(a_3+\varepsilon r_s^*)-\lambda)=0$$

where

$$r_s^* = \frac{k(b-c)(\beta-\gamma)-qa_2}{\beta-\gamma},\ z_2^* = \frac{a_3}{\beta-\gamma}.$$

Then, we rewrite the similar form for characteristic equation is

$$\lambda^4 + A\lambda^3 + B\lambda^2 + C\lambda + D = 0$$

where

$$\begin{aligned}
&A = -(a_{11}+a_{22}+a_{33}+a_{44}),\\
&B = a_{11}a_{22}+a_{11}a_{33}+a_{11}a_{44}+a_{22}a_{33}+a_{22}a_{44}+a_{33}a_{44},\\
&C = -a_{11}a_{22}a_{33}-a_{11}a_{22}a_{44}-a_{11}a_{33}a_{44}-a_{22}a_{33}a_{44},\\
&D = a_{11}a_{22}a_{33}a_{44},\\
&a_{11} = b-c-\frac{1}{k}(2r_s^*+qz_2^*),\quad a_{12} = b-\frac{r_s^*}{k}-ar_s^*,\quad a_{13} = -\frac{q}{k}r_s^*,\quad a_{14} = -\frac{q}{k}r_s^*,\\
&a_{21} = 0,\quad a_{22} = -c-\frac{1}{k}(r_s^*+qz_2*)+ar_s^*,\quad a_{23} = 0,\quad a_{24} = 0,\quad a_{31} = 0,\quad a_{32} = 0,\\
&a_{33} = -(\beta-\gamma)z_2^*+(a_2+\varepsilon r_s^*),\quad a_{34} = 0,\quad a_{41} = \varepsilon z_2^*,\quad a_{42} = \varepsilon z_2^*,\quad a_{43} = \varepsilon z_2^*,\\
&a_{44} = -2(\beta-\gamma)z_2^*+(a_3+\varepsilon r_s^*).
\end{aligned}$$

Then, the Routh Hurwtiz criterion implies that the equilibrium, $E_1(r_s^*,\ 0,\ 0,\ z_2^*)$ of model (1) is locally asymptotically stable if $A>0$, $B>0$, $C>0$, $D>0$ and $ABC > C^2 + A^2D$.

In this theorem, we follow the approach based on [18] to demonstrate the global stability of the positive equilibrium $E_1(r_s^*,\ 0,\ 0,\ z_2^*)$ of the model (3).

Theorem 1.2 *The disease-free equilibrium,* $E_1(r_s^*, 0, 0, z_2^*)$ *is globally asymptotically stable.*

Proof Let us build the following Lyapunov function

$$H(r_s^*, 0, 0, z_2^*) = \rho_1 \left(r_s - r_s^* - r_s^* \ln \frac{r_s}{r_s^*} \right) + r_i + z_1 + \rho_2 \left(z_2 - z_2^* - z_2^* \ln \frac{z_2}{z_2^*} \right)$$

where ρ_1 and ρ_2 are positive constants to be chosen later.

Differentiating $H$with respect to t along the solutions of model (3), we get

$$\begin{aligned}
\frac{dH}{dt} &= \rho_1 \left(1 - \frac{r_s^*}{r_s}\right) \frac{dr_s}{dt} + \frac{dr_i}{dt} + \frac{dz_1}{dt} + \rho_2 \left(1 - \frac{z_2^*}{z_2}\right) \frac{dz_2}{dt} \\
&= \rho_1 \left(1 - \frac{r_s*}{r_s}\right) \left(br - cr_s - \frac{r_s(r + qz)}{k} - ar_s r_i \right) + \left(-cr_i - \frac{r_i(r + qz)}{k} + ar_s r_i \right) \\
&\quad + \left(-(\beta - \gamma) z_1 z + z_1 (a_2 + \varepsilon r) \right) + \rho_2 \left(1 - \frac{z_2^*}{z_2}\right) \left(-(\beta - \gamma) z_2^2 + z_2 (a_3 + \varepsilon (r + z_1)) \right)
\end{aligned}$$

Substitutive $r = r_s + r_i$ and $z = z_1 + z_2$ into dH/dt, then

$$\begin{aligned}
\frac{dH}{dt} &= \rho_1 (r_s - r_s^*) \left(b \left(1 + \frac{r_i}{r_s}\right) - c - \frac{1}{k} (r_s + r_i + q(z_1 + z_2)) - ar_i \right) \\
&\quad + \left(-cr_i - \frac{r_i (r_s + r_i + q(z_1 + z_2))}{k} + ar_s r_i \right) \\
&\quad + \left(-(\beta - \gamma) z_1 (z_1 + z_2) + z_1 (a_2 + \varepsilon (r_s + r_i)) \right) \\
&\quad + \rho_2 (z_2 - z_2*) \left(-(\beta - \gamma) z_2 + (a_3 + \varepsilon (r_s + r_i + z_1)) \right)
\end{aligned}$$

Then, dH/dt can further be written as

$$\begin{aligned}
\frac{dH}{dt} &= \rho_1 (r_s - r_s^*) \left(-b \left(\frac{r_i}{r_s} - \frac{r_i*}{r_s^*} \right) \right. \\
&\quad \left. -\frac{1}{k} \left((r_s - r_s^*) + (r_i - r_i^*) + q \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] \right) - a (r_i - r_i^*) \right) \\
&\quad + \left(-\frac{1}{k} \left((r_s - r_s^*) + (r_i - r_i^*) + q \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] \right) + a (r_s - r_s^*) \right) \\
&\quad + \left(-(\beta - \gamma) \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] + \varepsilon \left[(r_s - r_s^*) + (r_i - r_i^*) \right] \right) \\
&\quad + \rho_2 (z_2 - z_2^*) \left(-(\beta - \gamma) (z_2 - z_2^*) + \varepsilon \left((r_s - r_s^*) + (r_i - r_i^*) + (z_1 - z_1^*) \right) \right).
\end{aligned}$$

We expand dH/dt about $E_1(r_s^*, 0, 0, z_2*)$ and obtain

$$\begin{aligned}
&\frac{dH}{dt} = -\frac{p_1}{k} (r_s - r_s^*)^2 - \frac{1}{k} (r_i - r_i^*)^2 - (\beta - \gamma) (z_1 - z_1^*)^2 - \rho_2 (\beta - \gamma) (z_2 - z_2^*)^2 \\
&- \rho_1 (r_s - r_s^*) (r_i - r_i^*) \left(\frac{1}{k} + a \right) - (r_s - r_s^*) (z_1 - z_1^*) \left(\frac{q\rho_1}{k} - \varepsilon \right) - (r_i - r_i^*) (z_1 - z_1^*) \left(\frac{q}{k} - \varepsilon \right) \\
&- (r_s - r_s*) (z_2 - z_2^*) \left(\frac{q\rho_1}{k} - \varepsilon\rho_2 \right) - (r_i - r_i^*) (z_2 - z_2^*) \left(\frac{q}{k} - \varepsilon\rho_2 \right) \\
&- (z_1 - z_1^*) (z_2 - z_2^*) ((\beta - \gamma) - \varepsilon\rho_2) - \rho_1 b \frac{(r_s - r_s^*)}{r_s r_s^*} (r_i r_s * - r_s r_i^*) < 0.
\end{aligned}$$

We obtain $dH/dt < 0$, and hence the disease-free equilibrium point $E_1\left(r_s^*, 0, 0, z_2^*\right)$ is globally asymptotically stable. We complete proof of Theorem 1.2. □

For $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$, the Jacobian matrix is given by

$$J\left(r_s^*, r_i*, 0, z_2^*\right) = \begin{bmatrix} b-c-\dfrac{1}{k}\left(2r_s^*+r_i^*+qz_2^*\right)-ar_i^* & b-\dfrac{r_s^*}{k}-ar_s^* & -\dfrac{q}{k}r_s^* & -\dfrac{q}{k}r_s^* \\ -\dfrac{r_i^*}{k}+ar_i^* & -c-\dfrac{1}{k}\left(r_s^*+2r_i^*+qz_2^*\right)+ar_s^* & -\dfrac{q}{k}r_i^* & -\dfrac{q}{k}r_i^* \\ 0 & 0 & -\left(\beta-\gamma\right)z_2^*+\left(a_2+\varepsilon\left(r_s^*+r_i^*\right)\right) & 0 \\ \varepsilon\, z_2^* & \varepsilon\, z_2^* & \varepsilon\, z_2^* & -2\left(\beta-\gamma\right)z_2^*+\left(a_3+\varepsilon\left(r_s^*+r_i^*\right)\right) \end{bmatrix}$$

where

$$r_i^* = \frac{\beta-\gamma-a_3}{\varepsilon} - r_s^*,\ z_2^* = ar_s^* - kc - \frac{\beta-\gamma-a_3}{\varepsilon}$$

We rewrite the characteristic equation at the equilibrium, $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ as

$$\lambda^4 + A\lambda^3 + B\lambda^2 + C\lambda + D = 0$$

where

$$\begin{aligned}
&A = -\left(a_{11}+a_{22}+a_{33}+a_{44}\right),\\
&B = a_{11}a_{22}+a_{11}a_{33}+a_{11}a_{44}+a_{22}a_{33}+a_{22}a_{44}+a_{33}a_{44}-a_{24}a_{42},\\
&C = a_{11}a_{24}a_{42}+a_{24}a_{33}a_{42}-a_{11}a_{22}a_{33}-a_{11}a_{22}a_{44}-a_{11}a_{33}a_{44}-a_{22}a_{33}a_{44},\\
&D = a_{11}a_{22}a_{33}a_{44}-a_{11}a_{24}a_{33}a_{42},\\
&a_{11} = b-c-\frac{1}{k}\left(2r_s^*+r_i^*+qz_2^*\right)-ar_i^*,\ a_{12} = b-\frac{r_s^*}{k}-ar_s^*,\ a_{13} = -\frac{q}{k}r_s^*,\\
&a_{14} = -\frac{q}{k}r_s^*,\ a_{21} = -\frac{r_i^*}{k}+ar_i^*,\ a_{22} = -c-\frac{1}{k}\left(r_s^*+2r_i^*+qz_2^*\right)+ar_s^*,\\
&a_{23} = -\frac{q}{k}r_i^*,\ a_{24} = -\frac{q}{k}r_i^*,\ a_{31} = 0,\ a_{32} = 0,\ a_{33} = -\left(\beta-\gamma\right)z_2^*+\left(a_2+\varepsilon\left(r_s^*+r_i^*\right)\right),\\
&a_{34} = 0,\ a_{41} = \varepsilon\, z_2^*,\ a_{42} = \varepsilon\, z_2^*,\ a_{43} = \varepsilon\, z_2^*,\ a_{44} = -2\left(\beta-\gamma\right)z_2^*+\left(a_3+\varepsilon\left(r^*+z_1^*\right)\right).
\end{aligned}$$

From Routh Hurwtiz stability criteria, the implies that the equilibrium, $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ of model (3) is locally asymptotically stable if $A>0$, $B>0$, $C>0$, $D>0$ and $ABC > C^2+A^2D$. □

Theorem 1.3 *The equilibrium point $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ is globally asymptotically stable.*

Proof Since the equilibrium point $E_2(r_s^*, r_i^*, 0, z_2^*)$ is globally asymptotically stable then we define a Lyapunov function as follows:

$$L(r_s, r_i, 0, z_2) = \omega_1 \left(r_s - r_s^* - r_s^* \ln \frac{r_s}{r_s^*} \right) + \omega_2 \left(r_i - r_i^* - r_i^* \ln \frac{r_i}{r_i^*} \right) + z_1 + \omega_3 \left(z_2 - z_2^* - z_2^* \ln \frac{z_2}{z_2^*} \right)$$

where ω_1, ω_2 and ω_3 are positive constants to be chosen later.

Differentiating L with respect to t along the solutions of model (3), we get

$$\begin{aligned} \frac{dL}{dt} &= \omega_1 \left(1 - \frac{r_s^*}{r_s} \right) \left(br - cr_s - \frac{r_s (r + qz)}{k} - ar_s r_i \right) \\ &\quad + \omega_2 \left(1 - \frac{r_i^*}{r_i} \right) \left(-cr_i - \frac{r_i (r + qz)}{k} + ar_s r_i \right) \\ &\quad + \left(-(\beta - \gamma) z_1 z + z_1 (a_2 + \varepsilon r) \right) + v_4 \left(1 - \frac{z_2^*}{z_2} \right) \left(-(\beta - \gamma) z_2^2 + z_2 (a_3 + \varepsilon (r + z_1)) \right) \end{aligned}$$

Substitutive $r = r_s + r_i$ and $z = z_1 + z_2$intodG/dt, then

$$\begin{aligned} \frac{dL}{dt} &= \omega_1 (r_s - r_s^*) \left(b \left(1 + \frac{r_i}{r_s} \right) - c - \frac{1}{k} (r_s + r_i + q (z_1 + z_2)) - ar_i \right) \\ &\quad + \omega_2 (r_i - r_i^*) \left(-c - \frac{1}{k} (r_s + r_i + q (z_1 + z_2)) + ar_s \right) \\ &\quad + \left(-(\beta - \gamma) z_1 (z_1 + z_2) + z_1 (a_2 + \varepsilon r) \right) \\ &\quad + \omega_3 (z_2 - z_2^*) \left(-(\beta - \gamma) z_2 + (a_3 + \varepsilon (r_s + r_i + z_1)) \right) \end{aligned}$$

Then, dL/dt can further be written as

$$\begin{aligned} \frac{dL}{dt} &= \omega_1 (r_s - r_s^*) \left(-b \left(\frac{r_i}{r_s} - \frac{r_i^*}{r_s^*} \right) \right. \\ &\quad \left. - \frac{1}{k} \left((r_s - r_s^*) + (r_i - r_i^*) + q \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] \right) - a (r_i - r_i^*) \right) \\ &\quad + \omega_2 (r_i - r_i^*) \left(-\frac{1}{k} \left((r_s - r_s^*) + (r_i - r_i^*) + q \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] \right) + a (r_s - r_s^*) \right) \\ &\quad + \left(-(\beta - \gamma) \left[(z_1 - z_1^*) + (z_2 - z_2^*) \right] + \varepsilon \left[(r_s - r_s^*) + (r_i - r_i^*) \right] \right) \\ &\quad + \omega_3 (z_2 - z_2^*) \left(-(\beta - \gamma) (z_2 - z_2^*) + \varepsilon \left((r_s - r_s^*) + (r_i - r_i^*) + (z_1 - z_1^*) \right) \right) \end{aligned}$$

We expand dL/dt about $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ and obtain

$$\begin{aligned}\frac{dL}{dt} = &-\frac{\omega_1}{k}\left(r_s - r_s^*\right)^2 - \frac{\omega_2}{k}\left(r_i - r_i^*\right)^2 - \left(\beta - \gamma\right)\left(z_1 - z_1^*\right)^2 - \omega_3\left(\beta - \gamma\right)\left(z_2 - z_2^*\right)^2 \\ &- \left(r_s - r_s^*\right)\left(r_i - r_i^*\right)\left(\frac{1}{k}\left(\omega_1 + \omega_2\right) + a\left(\omega_1 - \omega_2\right)\right) \\ &- \left(r_s - r_s^*\right)\left(z_1 - z_1^*\right)\left(\frac{q\omega_1}{k} - \varepsilon\right) - \left(r_i - r_i^*\right)\left(z_1 - z_1^*\right)\left(\frac{q\omega_2}{k} - \varepsilon\right) \\ &- \left(r_s - r_s^*\right)\left(z_2 - z_2^*\right)\left(\frac{q\omega_1}{k} - \varepsilon\omega_3\right) - \left(r_i - r_i^*\right)\left(z_2 - z_2^*\right)\left(\frac{q\omega_2}{k} - \varepsilon\omega_3\right) \\ &- \left(z_1 - z_1^*\right)\left(z_2 - z_2^*\right)\left(\left(\beta - \gamma\right) - \varepsilon\omega_3\right) - \omega_1 b\frac{\left(r_s - r_s^*\right)}{r_s r_s^*}\left(r_i r_s^* - r_s r_i^*\right) < 0\end{aligned}$$

We get $dL/dt < 0$, and hence the equilibrium point $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ is globally asymptotically stable. This complete proof of Theorem 1.3. □

Theorem 1.4 *The interior equilibrium point $E_3\left(r_s^*,\, r_i^*,\, z_1^*, z_2^*\right)$ is globally asymptotically stable.*

Proof Since the interior equilibrium point $E_3\left(r_s^*,\, r_i^*,\, z_1^*, z_2^*\right)$ is globally asymptotically stable then we define a Lyapunov function as follows:

$$\begin{aligned}G\left(r_s,\, r_i,\, \mathrm{z}_1, z_2\right) = &\, v_1\left(r_s - r_s^* - r_s^*\ln\frac{r_s}{r_s^*}\right) + v_2\left(r_i - r_i^* - r_i^*\ln\frac{r_i}{r_i^*}\right) \\ &+ v_3\left(z_1 - z_1^* - z_1^*\ln\frac{z_1}{z_1^*}\right) + v_4\left(z_2 - z_2^* - z_2^*\ln\frac{z_2}{z_2^*}\right)\end{aligned}$$

where v_1, v_2, v_3 and v_4 are positive constants to be chosen later.

Differentiating G with respect to t along the solutions of model (3), we get

$$\begin{aligned}\frac{dG}{dt} = &\, v_1\left(1 - \frac{r_s^*}{r_s}\right)\left(br - cr_s - \frac{r_s\left(r + qz\right)}{k} - ar_s r_i\right) \\ &+ v_2\left(1 - \frac{r_i^*}{r_i}\right)\left(-cr_i - \frac{r_i\left(r + qz\right)}{k} + ar_s r_i\right) \\ &+ v_3\left(1 - \frac{z_1^*}{z_1}\right)\left(-\left(\beta - \gamma\right)z_1 z + z_1\left(a_2 + \varepsilon r\right)\right) \\ &+ v_4\left(1 - \frac{z_2^*}{z_2}\right)\left(-\left(\beta - \gamma\right)z_2^2 + z_2\left(a_3 + \varepsilon\left(r + z_1\right)\right)\right)\end{aligned}$$

Substitutive $r = r_s + r_i$ and $z = z_1 + z_2$ into dG/dt, then

$$\begin{aligned}\frac{dG}{dt} = &\, v_1\left(r_s - r_s^*\right)\left(b\left(1 + \frac{r_i}{r_s}\right) - c - \frac{1}{k}\left(r_s + r_i + q\left(z_1 + z_2\right)\right) - ar_i\right) \\ &+ v_2\left(r_i - r_i^*\right)\left(-c - \frac{1}{k}\left(r_s + r_i + q\left(z_1 + z_2\right)\right) + ar_s\right) \\ &+ v_3\left(z_1 - z_1^*\right)\left(-\left(\beta - \gamma\right)\left(z_1 + z_2\right) + \left(a_2 + \varepsilon\left(r_s + r_i\right)\right)\right) \\ &+ v_4\left(z_2 - z_2^*\right)\left(-\left(\beta - \gamma\right)z_2 + \left(a_3 + \varepsilon\left(r_s + r_i + z_1\right)\right)\right)\end{aligned}$$

Using

$$
\begin{aligned}
c &= b\left(1+\frac{r_i^*}{r_s^*}\right)-\frac{1}{k}\left(r_s^*+r_i^*+q\left(z_1^*+z_2^*\right)\right)-ar_i^*,\\
c &= -\frac{1}{k}\left(r_s^*+r_i^*+q\left(z_1^*+z_2^*\right)\right)+ar_s^*,\\
a_2 &= (\beta-\gamma)\left(z_1^*+z_2^*\right)-\varepsilon\left(r_s^*+r_i^*\right),\\
a_3 &= (\beta-\gamma)\, z_2-\varepsilon\left(r_s^*+r_i^*+z_1^*\right),
\end{aligned}
$$

to write dG/dt, we obtain

$$
\begin{aligned}
\frac{dG}{dt} = {} & v_1\left(r_s-r_s^*\right)\left(-b\left(\frac{r_i}{r_s}-\frac{r_i^*}{r_s^*}\right)\right.\\
& \left.-\frac{1}{k}\left(\left(r_s-r_s^*\right)+\left(r_i-r_i^*\right)+q\left[\left(z_1-z_1^*\right)+\left(z_2-z_2^*\right)\right]\right)-a\left(r_i-r_i^*\right)\right)\\
& +v_2\left(r_i-r_i^*\right)\left(-\frac{1}{k}\left(\left(r_s-r_s^*\right)+\left(r_i-r_i^*\right)+q\left[\left(z_1-z_1^*\right)+\left(z_2-z_2^*\right)\right]\right)+a\left(r_s-r_s^*\right)\right)\\
& +v_3\left(z_1-z_1^*\right)\left(-(\beta-\gamma)\left[\left(z_1-z_1^*\right)+\left(z_2-z_2^*\right)\right]+\varepsilon\left[\left(r_s-r_s^*\right)+\left(r_i-r_i^*\right)\right]\right)\\
& +v_4\left(z_2-z_2^*\right)\left(-(\beta-\gamma)\left(z_2-z_2^*\right)+\varepsilon\left(\left(r_s-r_s^*\right)+\left(r_i-r_i^*\right)+\left(z_1-z_1^*\right)\right)\right)
\end{aligned}
$$

We expand dG/dt about $E_3\left(r_s^*,\, r_i^*,\, z_1^*, z_2^*\right)$ and obtain

$$
\begin{aligned}
\frac{dG}{dt} = {} & -\frac{v_1}{k}\left(r_s-r_s^*\right)^2-\frac{v_2}{k}\left(r_i-r_i^*\right)^2-v_3(\beta-\gamma)\left(z_1-z_1^*\right)^2-v_4(\beta-\gamma)\left(z_2-z_2^*\right)^2\\
& -\left(r_s-r_s^*\right)\left(r_i-r_i^*\right)\left(\frac{1}{k}\left(v_1+v_2\right)+a\left(v_1-v_2\right)\right)\\
& -\left(r_s-r_s^*\right)\left(z_1-z_1^*\right)\left(\frac{qv_1}{k}-\varepsilon v_3\right)-\left(r_i-r_i^*\right)\left(z_1-z_1^*\right)\left(\frac{qv_2}{k}-\varepsilon v_3\right)\\
& -\left(r_s-r_s^*\right)\left(z_2-z_2^*\right)\left(\frac{qv_1}{k}-\varepsilon v_4\right)-\left(r_i-r_i^*\right)\left(z_2-z_2^*\right)\left(\frac{qv_2}{k}-\varepsilon v_4\right)\\
& -\left(z_1-z_1^*\right)\left(z_2-z_2^*\right)\left((\beta-\gamma)\, v_3-\varepsilon v_4\right)\\
& -v_1 b\frac{\left(r_s-r_s^*\right)}{r_s r_s^*}\left(r_i r_s^*-r_s r_i^*\right)<0
\end{aligned}
$$

We obtain $dG/dt<0$, and hence the interior equilibrium point $E_3\left(r_s^*,\, r_i^*,\, z_1^*, z_2^*\right)$ is globally asymptotically stable. This complete proof of Theorem 1.4. □

There are three equilibrium of one rodent two predators model, namely $E_0\left(0,\, 0,\, 0,\, \mathrm{z}_2^*\right)$, $E_1\left(r_s^*,\, 0,\, 0,\, z_2^*\right)$ and $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$. The equilibrium $E_0\left(0,\, 0,\, 0,\, \mathrm{z}_2^*\right)$ exists in the absence of rodents and first predator populations and the equilibrium $E_1\left(r_s^*,\, 0,\, 0,\, z_2^*\right)$ exists when the susceptible rodent and second predator populations survive without the infection carried by infected rodent. Lastly, the equilibrium point $E_2\left(r_s^*,\, r_i^*,\, 0, z_2^*\right)$ exists when all population survive except the first predator population. For this situation, rodent not only shares with the environment, but they have to share with other rodents and second predator populations. It is clear that the disease will always persist in the environment.

5 Numerical Experiments and Discussion of Results

In this paper, the one rodent two predators model is solved using Runge-Kutta fourth order scheme. The fixed parameters $a = 0.1$, $b = 1$, $c = 0.6$, $\beta = 1.0$, $\gamma = 0.5$ are used as they were used by [17]. The value $k_c^{AK} = 25$ represents the critical environmental condition for the basic Abramson and Kenkre model. Meanwhile the model fixed parameters used by [9] were used in the experiments, i.e., $a_2 = 3$, $a_3 = 4$. The value $k = 10$, which means the environmental condition is adverse, will eliminate the infection. Nevertheless, the value of $k = 150$ is used which implies that the environmental condition is favourable resulting that the infection will thrive. The value of environmental condition,$(k = 150)$, chosen is very high to ensure the competition for resources shared between susceptible and infected rodents are low when higher resources are available. Here we study what happens to the predators and rodent populations in different environmental parameter(k), influence of the predators population (q) and the rate of increase of the predators population (ε) in our model of one rodent two predators.

Figures 1, 2 and 3 show the rodent and predators populations for the case of adverse environmental conditions $(k = 10)$ when the one rodent two predators model is solved using the same initial values $(= 50)$ for r_s, r_i, z_1, z_2 and the different values for q and ε.

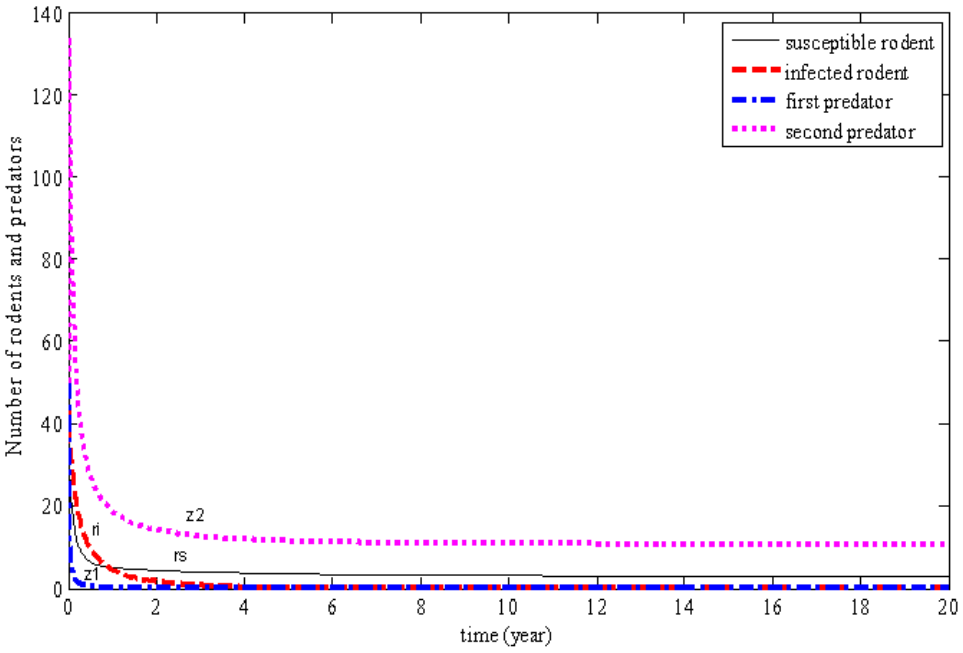

Figure 1: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50, z_2 = 50, q = 0.1$ and $z_1 = 50$, $z_2 = 50$, $q = 1.1$ and $\varepsilon = 0.5\,(q < 1\,\text{and}\,\varepsilon < 1)$

Figures 4, 5 and 6 show that phase-space trajectories of population amongst the rodent and predators populations is solved using the same initial values $(= 50)$ for r_s, r_i, z_1, z_2 and the different values for q and ε.

The populations of susceptible rodent, infected rodent, first predator and second predator are time plotted in Figure 1, 2 and 3. When resources are low and $(q < 1\,\text{and}\,\varepsilon < 1)$, the population of the infected rodent r_i and first predator z_1 become extinct (see Figure 1). The

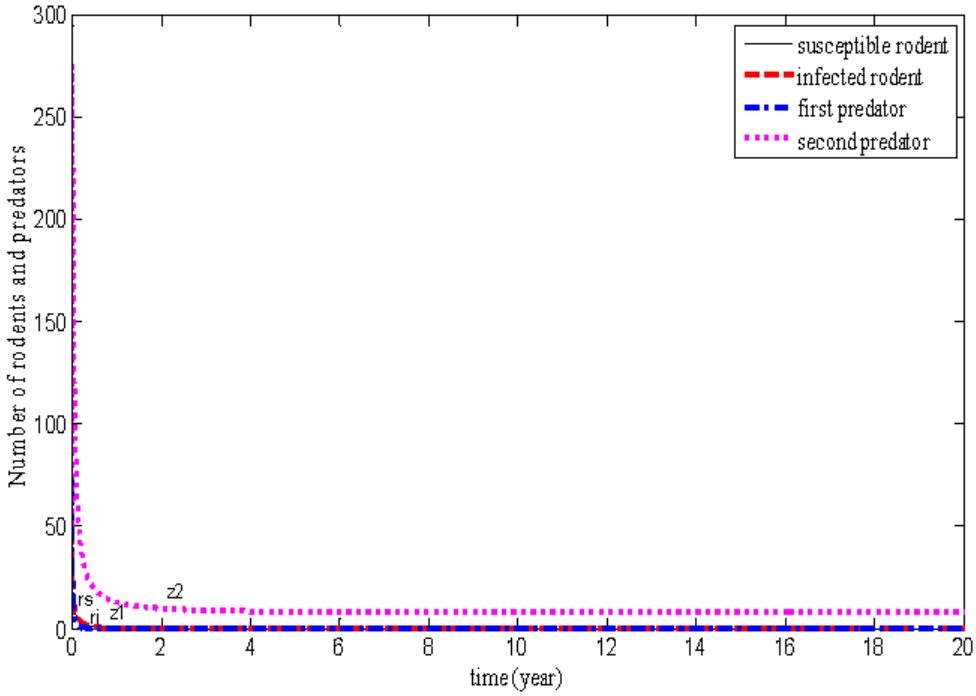

Figure 2: Values of r_s, r_i, z_1 and z_2 for One Rodent two Predators Model with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 1.1$ and $\varepsilon = 1.5 (q > 1 \text{ and } \varepsilon > 1)$

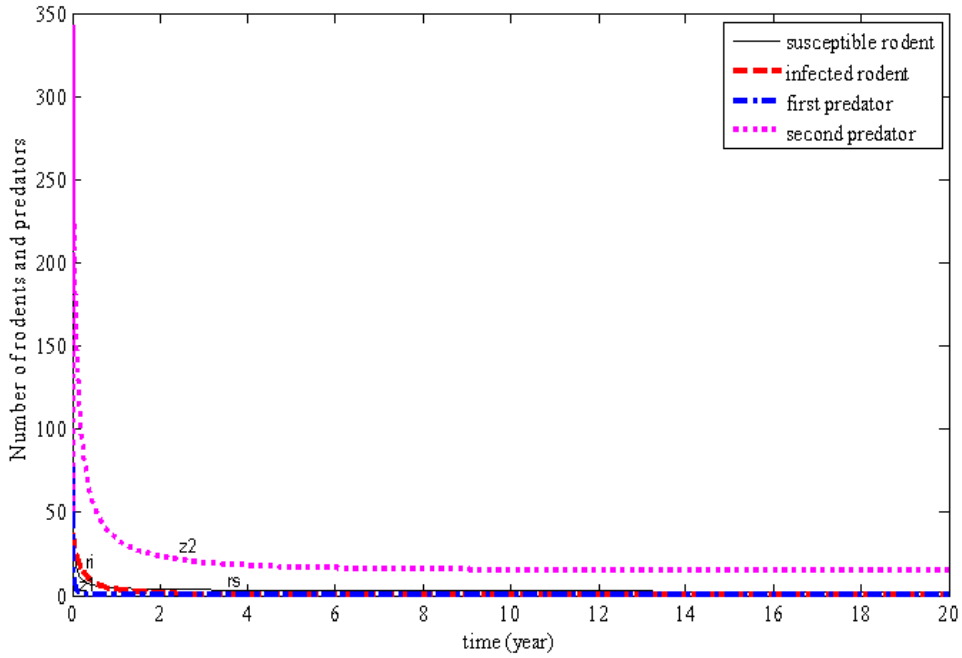

Figure 3: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 1.5 \, (q < 1 \text{ and } \varepsilon > 1)$.

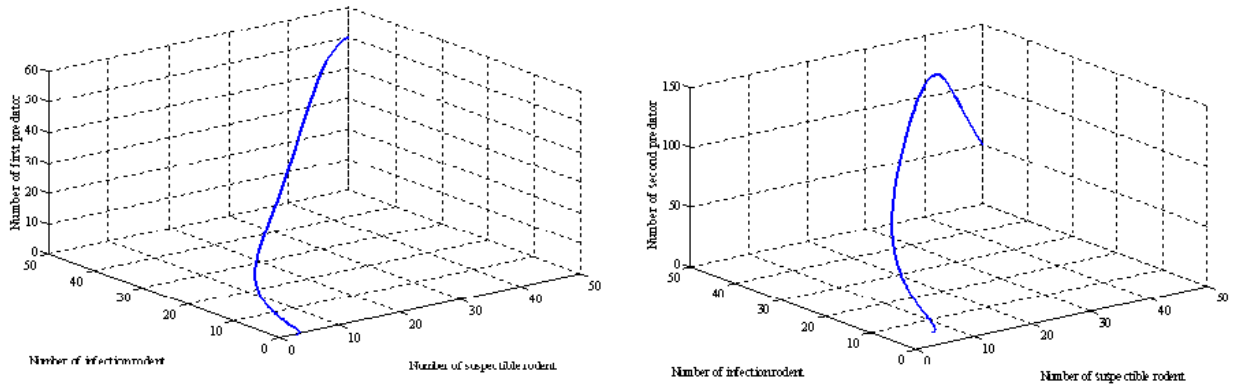

Figure 4: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 0.5\,(q < 1\,\text{and}\,\varepsilon < 1)$

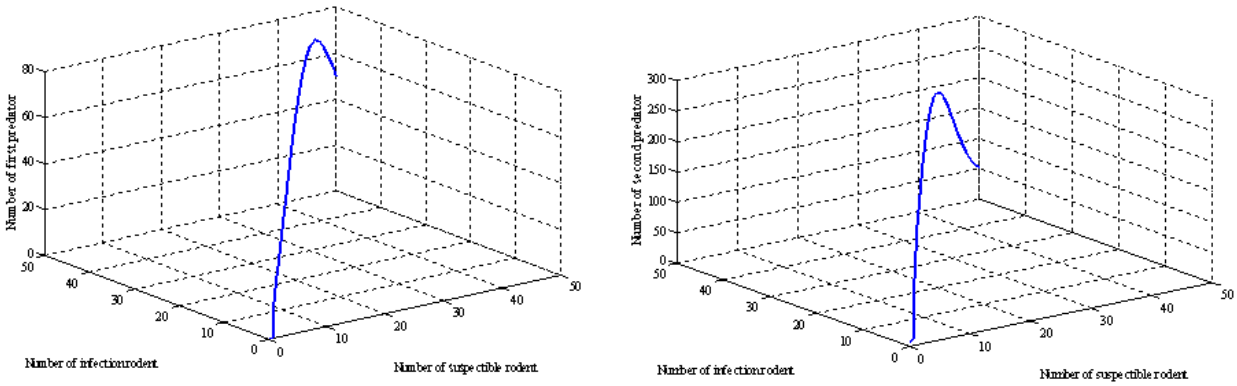

Figure 5: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 1.1$ and $\varepsilon = 1.5\,(q > 1\,\text{and}\,\varepsilon > 1)$

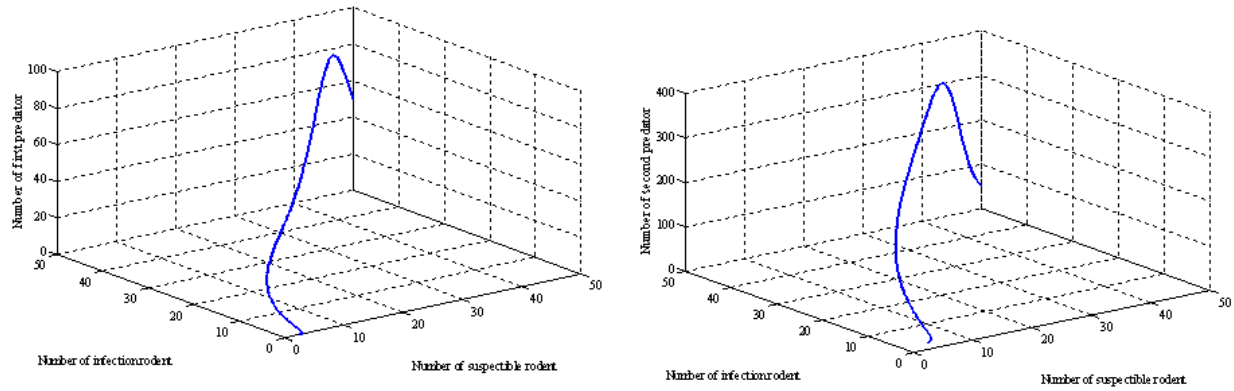

Figure 6: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 1.5\,(q < 1\,\text{and}\,\varepsilon > 1)$

frequency of fighting increases when the environment parameter is low causing a decline of the rodent and predator populations in the ecosystem. This scenario shows that the dynamics of infection propagate does not occur among the population of susceptible rodent and second predator. The second predator z_2 increase rapidly within the first year and reaches a certain maximum previous to fall and stabilizing at a steady value of 10.9. The susceptible rodent r_s will finally stabilize at a steady value of 2.9 and the two species (i.e. susceptible rodent and second predator) can coexist in the ecosystem. The steady value for second predator population $z_2$is higher than the value of susceptible rodent r_s. The reason is that the second predator z_2 consumes infected rodent r_i, first predator z_1 and a small portion of the susceptible rodent r_s. In this situation, the competition is not so high between rodent and predator populations and is free of infection in the ecosystem when $k < k_c$ and $(q < 1 \text{ and } \varepsilon < 1)$. The model (3) reaches the equilibrium point (2.9, 0, 0, 10.9) when we fix parameter as $a = 0.1$, $b = 1$, $c = 0.6$, $\beta = 1.0$, $\gamma = 0.5$, $q = 0.1$ and $\varepsilon = 0.5$. Thus, our numerical experiment results show that the equilibrium (2.9, 0, 0, 10.9) is global asymptotically stable. The stability of the model (3) is shown in Figures 1 and 4.

In Figure 2, it is observed that the second predator z_2 increases rapidly within the first year. In this situation, an increase in the population of second predator z_2 will lead the population of rodents and first predator to decrease and the surplus of second predatorz_2 will survive due to the high natural growth rate of z_2. For a long time, there is no food for second predator population anymore, so may be the second predator population goes extinct. When we choose $q = 1.1$ and $\varepsilon = 1.5$ and other parameters are the same in Figure 1, it is easy to see that the model (3) has a positive equilibrium point (0, 0, 0, 8.0) and shows that (0, 0, 0, 8.0) is global asymptotically stable where the infection dies away. The stability is shown in Figures 2 and 5.

The results displayed in Figure 3 is quite similar with graphical pattern of the results displayed in Figure 1 which have the same value of r_i and $z_1$but with varying values of r_s, z_2 and t. From Figure 3, when $k < k_c$, $q < 1 \text{ and } \varepsilon > 1$, both r_i and z_1 go extinct. Thus, free of infection will occur in an ecosystem. The second predator population z_2 will maintain at 15.4 while the susceptible rodent r_s will finally stabilize at a steady value of 2.5. The steady value of second predator population is higher than in the case $q < 1 \text{ and } \varepsilon > 1$ compared with the other cases for the one rodent two predators. This could be due to the second predator population getting larger, causing heavy predation and resulting in infected rodent r_i and first predator z_1 going extinct. In addition, it is consuming a small portion of the susceptible rodent $r_s$and maintaining their breeding. Numerical simulation of the model (3) with $a = 0.1$, $b = 1$, $c = 0.6$, $\beta = 1.0$, $\gamma = 0.5$, $q = 0.1$, $\varepsilon = 1.5$ and $k(= 10) < k_c$. Figure 3 shows that the solution converge to the positive equilibrium value (2.5, 0, 0, 15.4). This equilibrium point of model (3) is globally asymptotically stable.

In all of the above cases, free of infection will occur in population and ecosystem when $k < k_c$, $(q < 1 \text{ and } \varepsilon < 1)$, $(q > 1 \text{ and } \varepsilon > 1)$ and $(q < 1 \text{ and } \varepsilon > 1)$. Thus, the infection can be eliminated by the presence of second predator in the ecosystem.

Figures 7, 8 and 9 show the rodent and predator populations for the case of favourable environmental conditions ($k = 150$) when the one rodent two predators model is solved using the same initial values (= 50) for r_s, r_i, z_1, z_2 and the different values for q and ε.

Figures 10, 11 and 12 show that phase-space trajectories of population amongst the rodent and predators populations is solved using the same initial values (= 50) for r_s, r_i, z_1, z_2 and the different values for q and ε.

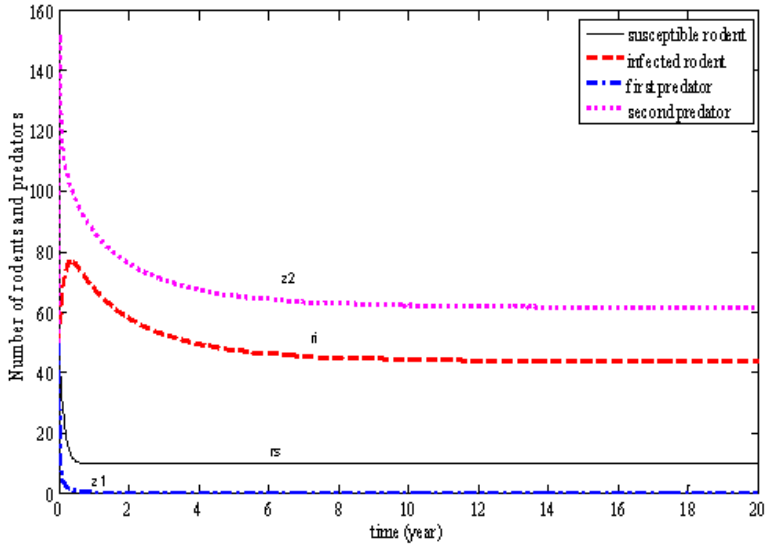

Figure 7: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 0.5\,(q < 1\,\text{and}\,\varepsilon < 1)\,\varepsilon = 1.5\,(q > 1\,\text{and}\,\varepsilon > 1)$

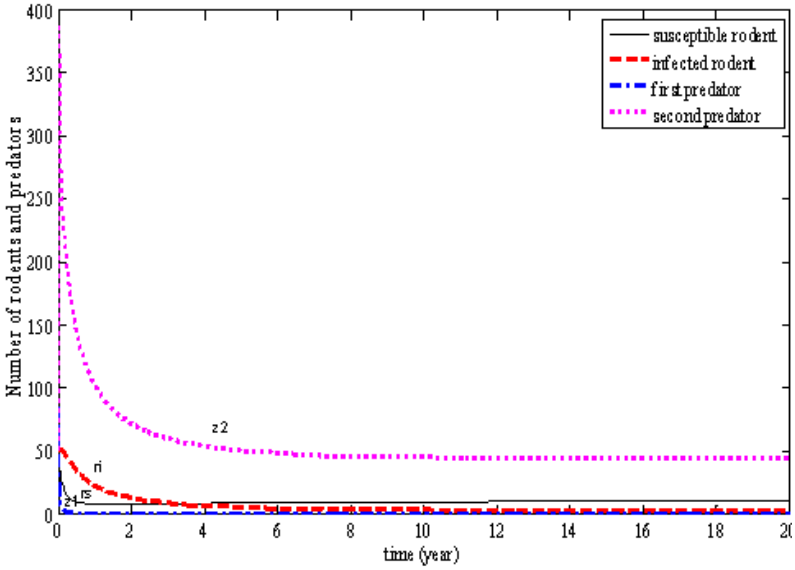

Figure 8: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 1.1$ and $\varepsilon = 1.5\,(q > 1\,\text{and}\,\varepsilon > 1)$

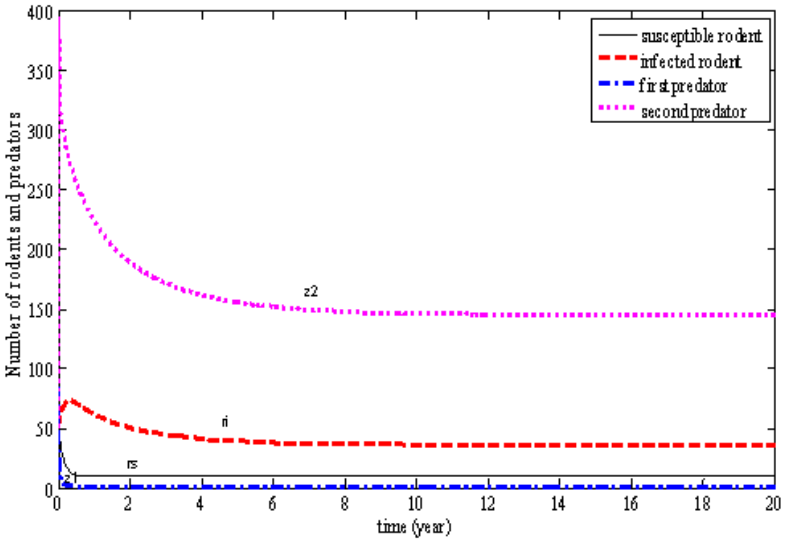

Figure 9: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$,$r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 1.5\,(q < 1\,\text{and}\,\varepsilon > 1)$

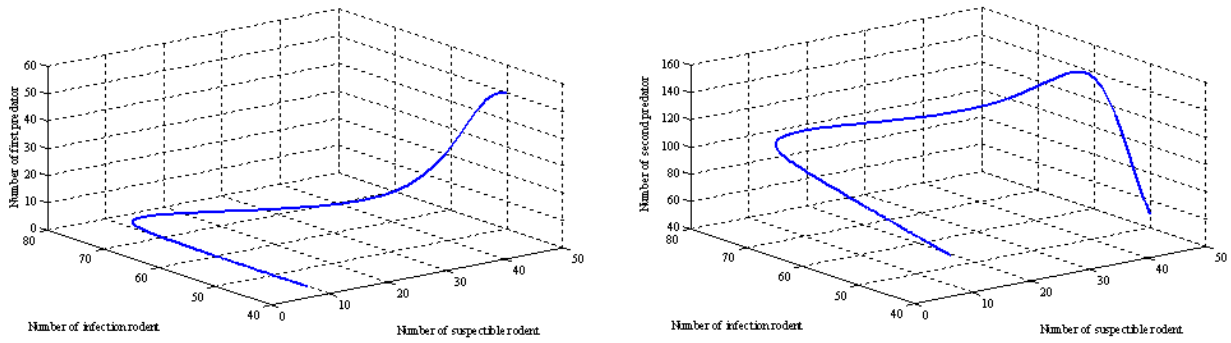

Figure 10: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 0.5\,(q < 1\,\text{and}\,\varepsilon < 1)$

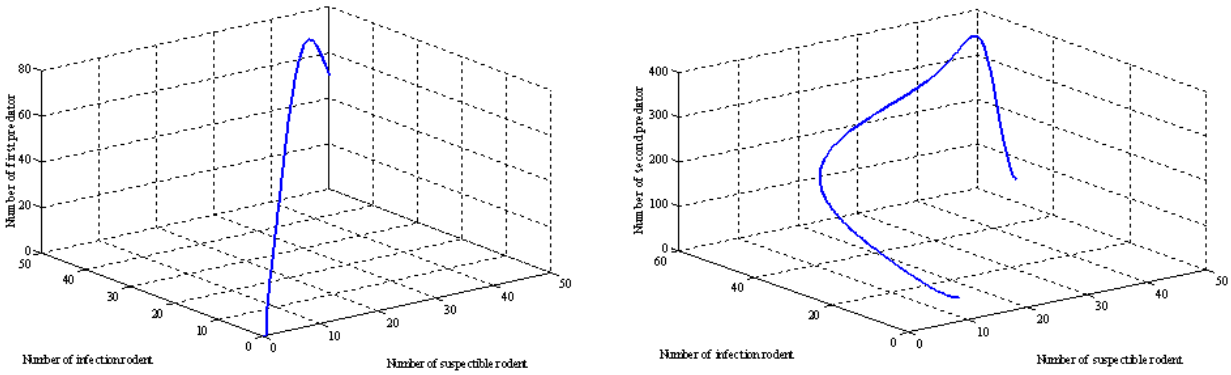

Figure 11: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 1.1$ and $\varepsilon = 1.5\,(q > 1\,\text{and}\,\varepsilon > 1)$

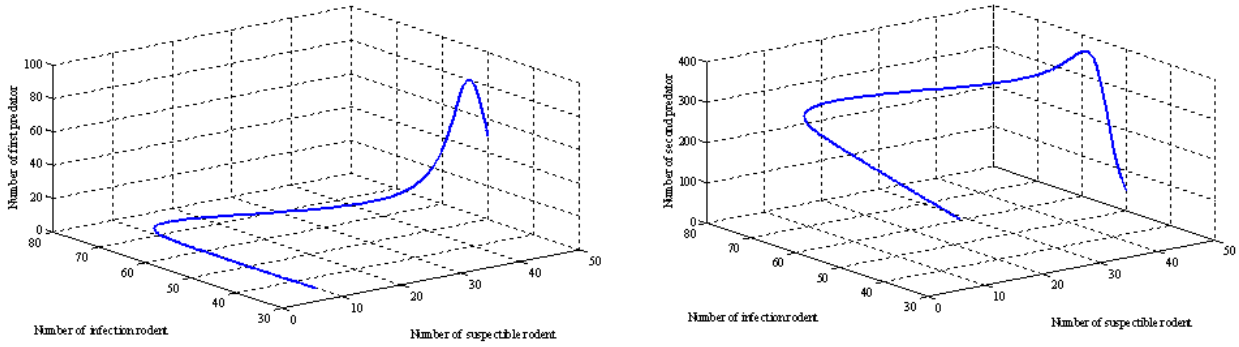

Figure 12: Values of r_s, r_i, z_1 and z_2 for One Rodent Two Predators Model with Initial Values $r_s = 50$, $r_i = 50$, $z_1 = 50$, $z_2 = 50$, $q = 0.1$ and $\varepsilon = 1.5$ ($q < 1$ and $\varepsilon > 1$)

The increase the environment parameter k and the values of q and ε is maintained, the numerical results in Figures 7, 8 and 9 displays similar graphical pattern to those of the Figures 1, 2 and 3 for susceptible rodent r_s, infected rodent r_i, first predator z_1 and second predator z_2. The reason is that an increase in water and food availability does not affect z_1 while the steady values for r_s, r_i and z_2 would change.

The abundance of resources such as water and food at the initial stage will lead to the infected rodent and second predator populations to increase rapidly at first and reaches a certain maximum before reducing down and stabilizing at a steady value of 43.8 and 61.8, respectively, as shown in Figure 7. The second predator population increases, spurring a reduction in the populations of rodents and first predator due to more predation. The populations of susceptible rodent and predator behaving in the dissimilar way which is quite expected since more resources means more rodents are being infected while second predator killing and eating population of rodents and first predator, consequently lessen the populations size of susceptible and first predator. The susceptible population r_s will drop and stabilize at a steady value of 10 while the first predator population die off. The steady value for second predator population $z_2$is higher than the values of rodent and first predator populations. This could be due to the population of second predator z_2 maintaining their breeding and consuming both rodents and first predator. There results show that the infection was able to maintain in the ecosystem at a high size. When abundance of resources is high ($k > k_c$), the virus can persist between new generations of susceptible rodent. From simulations, the positive equilibrium (10, 43.8, 0, 61.8) of model (3) is globally asymptotically stable and then the populations of rodents and second predator can coexist in a positive equilibrium$(r_s^*, r_i^*, 0, z_2^*)$. The model can be stable around $(r_s^*, r_i^*, 0, z_2^*)$ when environmental condition is favourable ($k > k_c$) and $q < 1$ and $\varepsilon < 1$.

In Figure 8, the infected rodent decreased after 4 years and thus the spread of the virus through the susceptible rodent reduced. The population of infected rodent will drop, spurring the rapid expansion in the population of susceptible rodent. The population of susceptible and infected rodents will stabilize to steady values of 10 and 2.1, respectively. Thus, the results showed that infection will spread through the whole population in the ecosystem. The population of second predator increased rapidly within the first year due to the more abundant resources that second predator can use to thrive. After the first year, the populations of rodents and first predator become a small that size contributes to a drop in the population of second predator. The second predator population will stabilize to a steady value of 44 while the first

predator population becomes extinct. For $q > 1$ and $\varepsilon > 1$, the competition to survive is very strong between rodents and predators populations. The model (1) reaches the equilibrium point (10, 2.1, 0, 44) when we fix parameter as $a = 0.1$, $b = 1$, $c = 0.6$, $\beta = 1.0$, $\gamma = 0.5$, $q = 0.1$ and $\varepsilon = 0.5$. Thus, our numerical experiment results show that the positive equilibrium (10, 2.1, 0, 44) is global asymptotically stable.

The results displayed in Figure 9 is quite similar with the graphical pattern of the results displayed in Figures 4 and 5 which have the same value of z_1 but with varying value of r_s, r_i, z_2 and t. From Figure 6, when $k > k_c$, $q > 1$ and $\varepsilon > 1$, the first predator population z_1 goes extinct. Meanwhile the susceptible r_s, infected r_i and second predator z_2 populations will stabilize to steady values of 10, 35.5 and 42, respectively. The positive equilibrium (10, 35.5, 0, 42) of model (3) is globally asymptotically stable, all populations can coexist except the first predator population becomes extinct in a positive equilibrium when $k > k_c$, $q < 1$ and $\varepsilon > 1$.

For all of the above cases, the increase of the value of environment parameter k which means increasing for the availability of water, food and other resources for the rodents to thrive in the ecosystem. Our simulation results illustrate that the model cannot have a disease-free equilibrium state and have only the equilibrium point in which the infection persists in the population for a long time. Before one year, the numbers of infected rodent r_i are higher than the numbers of susceptible rodent r_s. This is due to the susceptible and infected rodents breeding infected rodent and the second predator consuming of susceptible rodent, infected rodent and first predator as the food. The numerical simulation shows that the steady value of infected rodents is always smaller for the model of one rodent two predators. It has potential to decrease and control the spread of hantavirus infection when the environment parameter k was favourable.

6 Conclusion

In this paper, we have introduced a one rodent two predators model and analyse model of the mathematical behaviours The important result obtained was the second predator play a role in the process of reducing and controlling of hantavirus infection. When the second predator are present, rodents population can coexist with the higher values of environment parameter, $k\,(k > k_c)$. To prevent the extinction of populations, one must look closely at some of the parameters, namely, the environmental parameter, k, the influence of the predators population, q and the rate of increase of the predators population, ε. For the situations where abundant resources are available with the presence of the second predator population, the spread of hantavirus infection have been reduced but not eliminated completely The stability of the equilibrium points by using Routh Hurwtiz stability criterion and global analysis by constructing Lypunov function has been investigated.

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