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Exploring the Impact of Reaction-Diffusion on an Ecological Diversity Mathematical Paradigm for Understanding Hantavirus Infection Dynamics

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ABSTRACT

Spatial dynamics are important in ecology because they provide insights into a myriad of spatial phenomena. Factor of these phenomena include the significant impact of habitat architecture and environmental variability on animal species survival, persistence, and harmony. To describe these complex spatial patterns, researchers are increasingly turning to reaction-diffusion systems as a valuable tool. One especially intriguing aspect of ecological research is understanding how the spatial implications of diffusion influence the dynamics of Hantavirus infection, with a particular emphasis on its interaction with alien predators and rodent species within a confined environment. When the diffusion constant, D and the spatial distance, x are taken into account, the relationship between diffusion and Hantavirus infection dynamics becomes very fascinating. When both D and x are relatively small, a remarkable phenomenon emerges: Hantavirus infection tends to reduce or even disappear within the ecosystem. This complex interaction of geographical dissemination and infection dynamics highlights the fragile balance that governs species coexistence within shared habitats. This research helps develop effective conservation strategies and provides critical knowledge for disease management and mitigation efforts, resulting in a more peaceful coexistence of humans, wildlife, and their ecosystems.

Keywords: Hantavirus infection, Competition, Reaction-Diffusion, Mathematics Model, Finite Difference

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INTRODUCTION

Hemorrhagic Fever Syndromes (HFRS and HPS) are illnesses triggered by Hantaviruses which are a borne zoonotic agents carried by rodents Hantaviruses are harbored by various of rodent species and worldwide without causing harm to harm hosts.

According to data from 2000, HFRS caused by the Hantaan virus had a mortality rate ranging from approximately 5% to 15%, while HPS resulting from the Sin Nombre virus was estimated to have a fatality rate exceeding 45% [1]. From 2005 to 2010, the most substantial occurrences human hantavirus infections were documented in Finland, Sweden, and Germany, reaching respectively peaks of 3259, 2195, and 1688 cases [2]. Abramson and Kenkre [3] introduced a basic mathematical model that considers rodent population decay due to mortality, population growth through births, and the impact of environmental factors to maintain population stability. The simulation outcomes of Abramson and Kenkre [3] mathematical framework can replicate two experienced characteristic of hantavirus affiliation in the field. The infection can vanish entirely from the rodent population in cases of unfriendly environmental situations, re-emerging only when these situations boost. Additionally, there is a spatial aspect, manifesting as localized infection "refugia" within the rodent population. These refugia have the capacity to either expand or contract, ultimately have an effect on the transmission of the infection to different regions [3]. The modeling for hantavirus has been explored by various researchers including [4]–[13]

Barbera et al. [14], Kumar et al. [15], and Kumar et al. [16] analyzed the Abramson and Kenkre model with spatial components. Kumar et al. [15] investigated the Allee phenomenon, with a focus on subcritical pitchfork bifurcation rather than the critical transition in infectious disease propagation. As the system under study is not symmetric under reflection, they state that the bifurcation is imperfect. The results revealed environmental spatial patterns (modulations) in homogeneities. The bifurcation they discovered was more evident when computing the average population densities, providing a linkage between the landscape structure of species' resource habitats and the surrounding matrix [15]. They demonstrated the presence of a critical spatial modulation wave number, at which point the system's behavior undergoes a profound shift, exhibiting bistable behavior contingent upon the initial conditions. In Kumar et al.'s [16] explores of infection, they concentrating on issues related to spatially dependent environmental component. Their research determined to establish a mathematical concept for understanding the reactions and behaviors of rodents with hantavirus interaction through time.

Barbera et al. [14] studied a hyperbolic reaction-diffusion paradigm to analyze the dynamics of hantavirus infection within the rodent populations of. They proposed this hyperbolic reaction-diffusion model to describe the hantavirus contagion in the rodent population and analyzed the steady value solutions for both structured and unstructured perturbations. The results of numerical simulation showed that the transmission of hantavirus pathogen is closely linked to an environmental parameter that characterizes the medium's capacity to sustain the rodent population.

In a natural ecosystem, rodents will usually need to share with others species that shall be called predator species. The populations of rodent and predator move from one spatial location to another to find the necessary resources (food, water, temperature, light, shelter). Consequently, the range of the rodents and predators are broad, comprising a various landscape by a diversity of habitats.

Reaction-diffusion equations were first introduced into the field of Ecology by [17]. According to Mohd [18], a reaction-diffusion system is defined as a spatial model that treats space as a continuum and depicts the population densities of interacting species over time. It arises naturally in systems consisting of many interacting components and is extensively used to illustrate pattern-formation phenomena in an assortment of chemical, physical, and biological systems. Mohd and Abu Hasan [19] stated that the existence of a diffusion mechanism in predator-prey interplay can change the attributes of the complete model.

Mohd and Abu Hasan [19] studied the spatial effect of diffusion and boundary on the extinction and persistence phenomena of species, using energy estimates. By using the classical energy method, it was shown that the occurrence of energy decays exponentially in the long run through the construction of the invariant region of the model. The results showed that the extinction of prey and predator populations occurs. When, $D > \frac{1}{\pi^2}$..Meanwhile $D << \frac{1}{\pi^2}$ both populations persist. For additional studies of the diffusion prey-predator models considered here, see [20-22].

Reaction-diffusion modeling has widespread applications across divers fields, extending beyond biology. The equations are in the form of semi-linear parabolic differential equations, which emphasize their versatility in solving problems in chemistry, physics, and social sciences. McCarthy et al. [23] investigated the stability of long-term gene regulation in an early Drosophila embryo using a simple reaction-diffusion model. They incorporated transcription rate, mRNA diffusion and decay rate, translation rate, and protein diffusion and decay rate within the model. Gnerucci et al. [24] published results on the scratch assay

microscopy used in the ‘in vitro’ technique applying the reaction-diffusion equation for common data and instruments. They are taking this approach to bridge the gap between simpler modeling and complex approaches. Galochkina et al. [25] studied the reaction-diffusion waves in blood coagulation employing a simplified one-equation method. Li and Sun [26] explored the dynamical behavior of a river network employing a reaction-diffusion model for one dimension, with two branches having the water flow at a constant speed. Zhao [27] investigated the application of reaction-advection-diffusion modeling on a one-dimensional urban crime system. Reaction-diffusion modeling is extensively studied in various fields, including biology [28-29], imaging [30], and computer viruses [31].

In this paper, reaction-diffusion systems are employed to investigate spatial effects, including the movement of rodent and alien (as predator) populations between different spatial locations within a finite habitat. The work of Peixoto and Abramsons [5] on the biodiversity model is extended to study the impact of aliens as predators and spatial incidences of diffusion where the movement of the rodent and predator can be taken into account. According to Mackean [32], a host is an organism in which pathogens exists and propagate. Spatial effects mean that it includes the consequences of habitat outlay and heterogeneity of the atmosphere on the extinction, survival, and coexistence of species.

Predators are highly regarded for their capacity to control prey and, as an outcome, to maintain maximum positions of biodiversity [33]. The focus of this research has been to investigate the effects of the diffusion mechanism, especially the diffusion stiffness *D*, on the preservation and persistence aspects of the improved model through exact and approximate experiments.

MODEL DERIVATION

In the scenario where a commonly rodent species does not move from one spatial domain to another, the fundamental Abramson and Kenkre model can be expressed as

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

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

Distinguished by their respective symbols, the susceptible rodent (r_s), the infected rodents (r_i), and the total community of rodents ($r(t) = r_s(t) + r_i(t)$) are denoted. As for the variables, we represent the birth rate as 'b,' the natural mortality rate as 'c,' the transmission rate responsible

for infection (also referred to as the aggression parameter) as 'a,' and the environmental parameter as k. According to the research conducted by Abramson and Kenkre [3], the infection diminishes for [$k < k_c$] and flourishes under [$k > k_c$]. For further insights into this topic, refer to Yusof et al. [8].

The model can be spatially extended to incorporate the movement of rodents in a single dimension by adding a diffusive term. The equation describing the movement of rodents in the model is as follows

$$\frac{\partial r_s}{\partial t} = br - cr_s - \frac{r_s r}{k(x,t)} - ar_s r_i + D\nabla^2 r_s,$$

$$\frac{\partial r_i}{\partial t} = -cr_i - \frac{r_i r}{k(x,t)} + ar_s r_i + D\nabla^2 r_i.$$

The symbols and correspond to the second partial derivatives of the populations of susceptible $\nabla^2 r_s$ and infected rodents $\nabla^2 r_i$, respectively. These derivatives, r_s and r_i , are now functions of both *x* and *t*. According to Kumar et al. [16], ∇^2 corresponds to the one-dimensional Laplacian, i.e., $\frac{\partial^2}{\partial x^2}$. Yates et al. [34] have asserted that the Abramson and Kenkre model is characterized by key features shared with any population that acts as a reservoir for infectious diseases. The diffusion constant, denoted as *D*, is measured in m^2 per day. *D* represents the rate at which susceptible and infected rodents transfer across the terrain. Abramson et al. [35] define diffusion as a process that typically involves a transition from more coherent motion to intermittent scattering events. This description is particularly applicable when scattering events occur with high frequency.

Abramson and Kenkre [3] utilized numerical methods to investigate a one-dimensional spatial landscape. This landscape featured a central region with a high environmental parameter $(k > k_c)$ surrounded by a larger region characterized by a lower environmental parameter $(k < k_c)$. In their simulations, a steady state was reached where the infected population concentrated at the high-k central spot, starting from arbitrary initial population conditions.

When the diffusion coefficient had small to moderate values, the infected population only persisted in regions with a high environmental parameter, *k*, while going extinct in other areas. These pockets of infection served as reservoirs for the virus, presenting the highest risk for human exposure and transmission. This observation aligns with the findings reported by [3].

Expanding upon the *AK* model, Peixoto and Abramson [5] incorporated an alien population into the equation. The variables *r* and *z* represent the host and alien populations,

respectively. Consequently, the competition dynamics model proposed by Peixoto and Abramson can be elucidated through the following set of ordinary 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, all parameters are constants with positive values. The Peixoto and Abramson model includes the following parameter definitions: The model assigns specific labels and definitions to various parameters for both the host and alien populations: For the host population: The natural mortality rate is represented by *c*, the birth rate is denoted as *b* and the transmission rate, known as the aggression parameter, is labelled *a*. In the absence of the alien population, the environmental parameter is identified as *k*, while the influence of the alien community is quantified by *q*. Regarding the alien population: The environmental parameter is referred to as κ, β, γ and ε are utilized to characterize resource acquisition from other species.

Peixoto and Abramson [5] have proposed that when the competitor population exceeds a certain threshold, it becomes greater than the minimum required to drive the infected subpopulation to extinction. At this point, the system exhibits a positive prevalence of infection when certain conditions are met, and a specific point in the parameter space constitutes a critical point of the system. This critical point demarcates two distinct behaviors in terms of the stability of the equilibrium of the infected population. In cases where the intensity of interacting competition is not excessively high (when certain conditions are met), coexistence remains stable. However, when competition becomes strong enough (under specific conditions), biostability can occur, and the final outcome may depend on the initial conditions. Finally, in scenarios where particular conditions are met, only the strong competitor survives. For more in-depth discussions on this topic, please refer to Yusof et al. [9]. The fundamental model, which is based on the Lotka-Volterra predator-prey model introduced by Lotka and Volterra, is presented below

$$\frac{dr}{dt} = r(a-bz),$$
$$\frac{dz}{dt} = z(cr-d).$$

In these above equations, the population of prey is represented as *r* and the population of predators is represented as *z* at time *t*. Within this model, '*a*' signifies the intrinsic growth rate of the prey, '*b*' denotes the per-capita rate of predation by the predator, '*c*' represents the product of the per-capita rate of predation and the rate of conversion from prey to predator, and '*d*' stands

for the death rate of the predator. It is important to note that all these parameters are considered positive constants, as indicated by Guo and Chen [36].

In the absence of predation ar, the prey's population grows indefinitely in a Malthusian manner, leading to exponential growth over time. This is due to the absence of predators that would otherwise reduce the prey's per-capita $-brz$ growth rate by a factor related to the populations of both prey and predators. Additionally, the parameter crz signifies the prey's contribution to the predator's growth rate, which is proportional to the available prey and the size of the predator population. In the absence of prey for sustenance $-dr$, the predator's death rate results in exponential decline.

Mohd and Abu Hasan [19] utilized the Lotka-Volterra predator-prey model as a foundation. They assumed specific parameter values and introduced a one-dimensional spatial dimension into the model, extending it to encompass the community dynamics of two species with diffusion. The resulting model can be described as follows:

$$\frac{\partial r}{\partial t} = r(1-z) + D\nabla^2 r,$$

$$\frac{\partial z}{\partial t} = z(r-1) + D\nabla^2 z,$$

where, the prey population is represented by *r* and the predator population is denoted by *z* at the time, *t*. $\nabla^2 = \frac{\partial^2}{\partial x^2}$ are the diffusion terms for both prey and predator populations. In an ecosystem, the presence of a diffusion process tends to result in a homogenized distribution of population density across space. It functions to enhance stability within a system comprising diverse populations and resources, as stated by Okubo and Levin [37].

According to Mohd and Abu Hasan [19], there is a diffusion constant $\left(D = \frac{1}{\pi^2}\right)$ that division between two distinct regimes., whereby if the diffusion constant *D* is greater than $\frac{1}{\pi^2}$, the population densities of the prey and predator go to extinction in one ecosystem. If the diffusion constant *D* is extremely small i.e., $D << \frac{1}{\pi^2}$, meaning that it approaches zero, the populations of prey and predators will flourish.

DIFFUSION ONE RODENT ONE ALIEN (PREDATOR)

Our model is derived from the ordinary differential equations of the Lotka-Volterra predator-prey model initially presented by Mohd and Abu Hasan [19]. It is represented as follows:

$$\frac{\partial r}{\partial t} = r(1-z) + D\nabla^2 r,$$

$$\frac{\partial z}{\partial t} = z(r-1) + D\nabla^2 z,$$

where, r and z are the populations of the prey and predator at the time t, respectively. $\nabla^2 r$ and $\nabla^2 z$ are the second partial derivatives of the populations of prey and predator populations, respectively, where r and z are now function of both x and t additionally the parameter D is diffusion constant.

In here, r and z represent the community of the prey and predator at time "t," consistently. The symbols $\nabla^2 r$ and $\nabla^2 z$ denote the second partial derivatives of the prey and predator populations, respectively. Notably, these derivatives are now functions of both r and z and the parameter D represents the diffusion constant.

Peixoto and Abramson [5] introduced an alternative mathematical model using ordinary nonlinear differential equations to analyze the outbreak of hantavirus infection. This model provides a structured framework for examining the competitive dynamics between two species:

$$\frac{dr}{dt} = (b-c)r - \frac{r}{k}(r+qz),$$

$$\frac{dz}{dt} = (\beta-\gamma)z - \frac{z}{\kappa}(z+\varepsilon r).$$

In this specific model, the host population is represented as r and the alien population is denoted as z. For the host population, b represents the birth rate, c is the natural mortality rate, k is the environmental parameter when there is no presence of the alien population, and q signifies the impact of the alien population. On the other hand, for the alien population, the parameters β, γ and ε are associated with obtaining resources from the other species, while κ represents the environmental parameter.

Now, we can proceed to create a combined model for the diffusion of one rodent and one alien (acting as a predator), incorporating elements from both the Mohd and Abu Hasan [19] model and the Peixoto and Abramson [5] model. In the Peixoto and Abramson model, we adjust the host population by introducing the diffusion term, D. Additionally, we modify the alien population by including the diffusion term, D, and replacing it with the predator

population represented by the variable *z*. As a result, the diffusion one rodent, one alien (as a predator) model is presented below:

$$\left.\begin{aligned}\frac{\partial r}{\partial t} &= (b-c)r - \frac{r}{k}(r+qz) + D\nabla^2 r,\\ \frac{\partial z}{\partial t} &= -(\beta-\gamma)z + \varepsilon rz + D\nabla^2 z\,.\end{aligned}\right\} \quad \ldots (1)$$

In this above equation *r* represents the host population, *z* stands for the predator population, and *D* signifies the diffusion constant. The symbols $\nabla^2 r$ and $\nabla^2 z$ represent the second partial derivatives of the host and predator populations, respectively. Importantly, both *r* and *z* are now functions of both space (*x*) and time (*t*). Regarding the predator population, β and γ are parameters associated with acquiring resources from other species, while ε represents the product of the per-capita rate of predation and the rate of conversion of rodents into predator offspring.

Assume an internal categorization of the rodent model is implemented, wherein r_s denotes the susceptible rodents, r_i represents the infected rodents, and "*r*" stands for the overall rodent population,

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

Hence, the model for the diffusion of one rodent and one alien (acting as a predator) is as follows:

$$\left.\begin{aligned}\frac{\partial r_s}{\partial t} &= br - cr_s - \frac{r_s}{k}(r+qz) - ar_s r_i + D\nabla^2 r_s\\ \frac{\partial r_i}{\partial t} &= -cr_i - \frac{r_i}{k}(r+qz) + ar_s r_i + D\nabla^2 r_i\\ \frac{\partial z}{\partial t} &= -(\beta-\gamma)z + \varepsilon rz + D\nabla^2 z\end{aligned}\right\} \quad \ldots (2)$$

where, r_s represents the population of susceptible rodents, r_i stands for infected rodents, and *z* signifies the predator population $r(t) = r_s(t) + r_i(t)$ denotes the total rodent population. The parameter a denotes the transmission rate of the infection, *b* represents the birth rate, and *c* corresponds to the natural death rate. The parameter *k* is a time-dependent environmental parameter, while *q* measures the impact of the predator population. *D* represents the diffusion constant. The symbols $\nabla^2 r_s$ $\nabla^2 r_i$, and $\nabla^2 z$ represent the second partial derivatives of the populations of susceptible rodents, infected rodents, and predators, respectively. It's important to note that r_s , r_i and *z* are now functions of both space (*x*) and time (*t*). For the predator populations, β and γ are the corresponding parameters for acquiring resources from other

species, while ε represents the product of the per-capita rate of predation and the rate of conversion of consumed rodents into predator offspring.

LOCAL STABILITY ANALYSIS

In this paper, the stability of the model of diffusion organisms (i.e., single rodent and single alien as a predator) around the equilibrium is determined by studying the corresponding characteristic equations.

This analysis is carried out based on the approach of Murray [38]. Firstly, the local stability analysis of model (1) around each fixed point is investigated. The steady value solutions of model (1) do not depend on time but satisfy,

$$\begin{aligned}(b-c)r-\frac{r}{k}(r+qz)+D\nabla^2 r=0,\\ -(\beta-\gamma)z+\varepsilon rz+D\nabla^2 z=0.\end{aligned}$$

And rewriting as

$$\begin{aligned}&\frac{dr}{dx}=U,\ \frac{d^2r}{dx^2}=\frac{dU}{dx}=-\frac{1}{D}\left((b-c)r-\frac{r}{k}(r+qz)\right),\\ &\frac{dz}{dx}=V,\ \frac{d^2z}{dx^2}=\frac{dV}{dx}=\frac{1}{D}((\beta-\gamma)z-\varepsilon rz).\end{aligned}$$

In consequence, the phase plane representing the solutions is given by

$$\begin{aligned}&\frac{dU}{dr}=-\frac{1}{D}\left(\frac{(b-c)r-\frac{r}{k}(r+qz)}{U}\right),\\ &\frac{dV}{dz}=\frac{1}{D}\left(\frac{(\beta-\gamma)z-\varepsilon rz}{\mathrm{V}}\right).\end{aligned}$$

To compute the equilibrium points, we solve $\frac{dU}{dr}=0$ and $\frac{dV}{dz}=0$. So, the model (1) has two equilibriums: namely $(r^*,z^*)=(0,0)$ and $(r^*,z^*)=(k(b-c),0)$.

The linearization of model (1) about (r^*,z^*) is

$$\begin{bmatrix}\frac{dU}{dx}\\ \frac{dV}{dx}\end{bmatrix}=\begin{bmatrix}-\frac{1}{D}\left(b-c-\frac{1}{k}(2r^*+qz^*)\right) & \frac{q}{Dk}r^*\\ -\frac{1}{D}z^* & \frac{1}{D}(\beta-\gamma-\varepsilon r^*)\end{bmatrix}\begin{bmatrix}U\\ V\end{bmatrix}.$$

The general Jacobian matrix of model (1) with respect to any state variable as follows

$$J(r^*,z^*)=\begin{bmatrix} -\frac{1}{D}\left(b-c-\frac{1}{k}(2r^*+qz^*)\right) & \frac{q}{Dk}r^* \\ -\frac{1}{D}z^* & \frac{1}{D}(\beta-\gamma-\varepsilon r^*) \end{bmatrix}$$

when, $(r^*,z^*)=(0,0)$ then the Jacobian matrix as follows

$$J(0,0)=\begin{bmatrix} -\frac{1}{D}(b-c) & 0 \\ 0 & \frac{1}{D}(\beta-\gamma) \end{bmatrix}.$$

Hence, the characteristic equation for $(r^*,z^*)=(0,0)$ is

$$\left(\lambda+\frac{(b-c)}{D}\right)\left(\lambda-\frac{1}{D}(\beta-\gamma)\right)=0.$$

At the equilibrium $(r^*,z^*)=(0,0)$, we find the eigenvalues $-\frac{b-c}{D}$ and $\frac{1}{D}(\beta-\gamma)$.

Thus, at this equilibrium $(r^*,z^*)=(0,0)$ it becomes saddle point.

At the equilibrium, the Jacobian matrix, $(r^*,z^*)=(k(b-c),0)$ is given by

$$J(k(b-c),0)=\begin{bmatrix} \frac{1}{D}((b-c)) & \frac{q}{D}(b-c) \\ 0 & \frac{1}{D}(\beta-\gamma-\varepsilon k(b-c)) \end{bmatrix}$$

The characteristic equation for the equilibrium of $[(k(b-c),0)]$ is:

$$\left(\frac{1}{D}(b-c)-\lambda\right)\left(\frac{1}{D}(\beta-\gamma-\varepsilon k(b-c))-\lambda\right)=0$$

where around the equilibrium $(r^*,z^*)=(k(b-c),0)$.

The eigenvalues are $\frac{1}{D}(b-c)$ and $\frac{1}{D}(\beta-\gamma-\varepsilon k(b-c))$.

In this case the equilibrium $(r^*,z^*)=(k(b-c),0)$ is called an unstable node.

The steady-state roots of approach (2) are time independent on time and satisfy

$$\begin{aligned} br-cr_s-\frac{r_s}{k}(r+qz)-ar_sr_i+D\nabla^2 r_s&=0,\\ -cr_i-\frac{r_i}{k}(r+qz)+ar_sr_i+D\nabla^2 r_i&=0,\\ -(\beta-\gamma)z+\varepsilon rz+D\nabla^2 z&=0. \end{aligned}$$

The above equations can be write as follows

$$\frac{dr_s}{dx} = U,\ \frac{d^2 r_s}{dx^2} = \frac{dU}{dx} = -\frac{1}{D}\left(br - cr_s - \frac{r_s}{k}(r + qz) - ar_s r_i \right),$$
$$\frac{dr_i}{dx} = V,\ \frac{d^2 r_i}{dx^2} = \frac{dV}{dx} = -\frac{1}{D}\left(-cr_i - \frac{r_i}{k}(r + qz) + ar_s r_i \right),$$
$$\frac{dz}{dx} = W, \frac{d^2 z}{dx^2} = \frac{dW}{dx} = \frac{1}{D}((\beta - \gamma)z - \varepsilon rz).$$

Now, all the equations in consequence, the phase plane as solutions is given by

$$\frac{dU}{dr_s} = -\frac{1}{D}\left(\frac{br - cr_s - \frac{r_s}{k}(r + qz) - ar_s r_i}{U} \right),$$
$$\frac{dV}{dr_i} = -\frac{1}{D}\left(\frac{-cr_i - \frac{r_i}{k}(r + qz) + ar_s r_i}{\mathrm{V}} \right),$$
$$\frac{dW}{dz} = \frac{1}{D}\left(\frac{(\beta - \gamma)z - \varepsilon rz}{W} \right).$$

To compute the equilibrium point, we solve as follows:

$$\frac{dU}{dr_s} = 0,\ \frac{dV}{dr_i} = 0 \text{ and } \frac{dW}{dz} = 0.$$

There are three equilibrium points in the dynamical model (2):

(i) The trivial equilibrium point at $E_1(0, 0, 0)$.

(ii) The predator free equilibrium point at $E_2(r_s^*, r_i^*, 0)$

where, $r_s^* = \frac{b}{a}$, $r_i^* = k(b - c) - \frac{b}{a}$

(iii) The interior equilibrium point at $E_3(r_s^*, r_i^*, z^*)$

where, $r_s^* = \frac{b}{a}$, $r_i^* = \frac{(\beta - \gamma)}{\varepsilon} - \frac{b}{a}$ *and* $z^* = \frac{k\varepsilon(b - c) - (\beta - \gamma)}{q\varepsilon}$

The linearization of the system (2) about the inner equilibrium $E_3(r_s^*, r_i^*, z^*)$ is

$$\begin{bmatrix} \frac{dU}{dx} \\ \frac{dV}{dx} \\ \frac{dW}{dx} \end{bmatrix} = \begin{bmatrix} -\frac{1}{D}\left(b - c - \frac{1}{k}(2r_s^* + r_i^* + qz^*) - ar_i^* \right) & -\frac{1}{D}\left(b - \frac{r_s^*}{k} - ar_s^* \right) \\ -\frac{1}{D}\left(-\frac{r_i^*}{k} + ar_i^* \right) & -\frac{1}{D}\left(-c - \frac{1}{k}(r_s^* + 2r_i^* + qz^*) + ar_s^* \right) \\ \frac{1}{D}(\varepsilon z^*) & \frac{1}{D}(\varepsilon z^*) \end{bmatrix}$$

$$\begin{bmatrix} \frac{q}{kD} r_s^* \\ \frac{q}{kD} r_i^* \\ \frac{1}{D}\left(-(\beta-\gamma)+\varepsilon(r_s^*+r_i^*)\right) \end{bmatrix} \begin{bmatrix} U \\ V \\ W \end{bmatrix}.$$

The general Jacobian matrix of model (2) at any state variable as follows:

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

$$\begin{bmatrix} \frac{q}{kD} r_s^* \\ \frac{q}{kD} r_i^* \\ \frac{1}{D}\left(-(\beta-\gamma)+\varepsilon(r_s^*+r_i^*)\right) \end{bmatrix} \quad \ldots (3)$$

The corresponding characteristic equation is

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

where, $A = -(a_{11}+a_{22}+a_{33}), B = a_{11}a_{22}+a_{11}a_{33}+a_{22}a_{33}-a_{13}a_{31}-a_{23}a_{32}-a_{12}a_{21}$

$$C = a_{11}a_{23}a_{32}+a_{12}a_{21}a_{23}+a_{13}a_{22}a_{31}-a_{11}a_{22}a_{33}-a_{12}a_{23}a_{31}-a_{13}a_{21}a_{32},$$

$$a_{11} = -\frac{1}{D}\left(b-c-\frac{1}{k}(2r_s^*+r_i^*+qz^*)-ar_i\right)^*,\ a_{12} = -\frac{1}{D}\left(b-\frac{r_s^*}{k}-ar_s^*\right)$$

$$a_{13} = \frac{q}{kD} r_s^*,\quad a_{21} = -\frac{1}{D}\left(-\frac{r_i^*}{k}+ar_i\right)^*$$

$$a_{22} = -\frac{1}{D}\left(-c-\frac{1}{k}(r_s^*+2r_i^*+qz^*)+ar_s\right)^*,\ a_{23} = \frac{q}{kD} r_i^*$$

$$a_{31} = \frac{\varepsilon z}{D}^*,\ a_{32} = \frac{\varepsilon z^*}{D}\ \text{and}\ a_{33} = \frac{1}{D}\left(-(\beta-\gamma)-\varepsilon(r_s^*+r_i^*)\right)$$

$$r_s^* = \frac{b}{a},\ r_i^* = \frac{(\beta-\gamma)}{\varepsilon}-\frac{b}{a}\ \text{and}\ \ z^* = \frac{k\varepsilon(b-c)-(\beta-\gamma)}{q\varepsilon}$$

Following the work of Bairagi et al. [39], the predator free equilibrium E_2 is locally asymptotically stable if all roots of characteristic equation of model (2) have negative real parts. If we choose $A>0$, $C>0$ and $AB-C>0$, then the inner equilibrium point E_2 is locally asymptotically steady. Now, $A>0$ implies $a_{11}+a_{22}+a_{33}<0$ and $C>0$ implies $a_{11}a_{23}a_{32}+a_{12}a_{21}a_{23}+a_{13}a_{22}a_{31}-a_{11}a_{22}a_{33}-a_{12}a_{23}a_{31}-a_{13}a_{21}a_{32}<0$. So, for $a_{11}+a_{22}+a_{33}>0$ and $a_{11}a_{23}a_{32}+a_{12}a_{21}a_{23}+a_{13}a_{22}a_{31}-a_{11}a_{22}a_{33}-a_{12}a_{23}a_{31}-a_{13}a_{21}a_{32}<0$ instability E_2 may occur. Secondly, $A>0$ with $C<0$ may cause instability around E_2. The selection of the values, a, b, c, k, q, β, γ, ε and D influence the steps of rodent population at which this equilibrium is achieved.

When the trivial equilibrium $E_1(0,0,0)=0$, then the Jacobian matrix as follows

$$J(0,0,0)=\begin{bmatrix} -\frac{1}{D}(b-c) & -\frac{b}{D} & 0 \\ 0 & \frac{c}{D} & 0 \\ 0 & 0 & -\left(\frac{\beta-\gamma}{D}\right) \end{bmatrix}.$$

Hence, the properties equation of $E_1(0,0,0)$ is

$$\left(\lambda+\frac{(b-c)}{D}\right)\left(\lambda-\frac{c}{D}\right)\left(\lambda+\left(\frac{\beta-\lambda}{D}\right)\right)=0.$$

The eigenvalues of the corresponding properties equation to equilibrium $E_1(0,0,0)$ are $\lambda_1=-\left(\frac{b-c}{D}\right)$, $\lambda_2=\frac{c}{D}$ and $\lambda_3=-\left(\frac{\beta-\gamma}{D}\right)$. Clearly, λ_1 and λ_3 always have two negative eigenvalues and λ_2 will be negative if,$c>0$ hence, the trivial equilibrium $E_1(0,0,0)$ is locally asymptotically stable.

The Jacobian matrix (3) evaluated at $E_2(r_s{*},r_i{*},0)$ as follows

$$J(r_s{*},r_i{*},0)=\begin{bmatrix} -\frac{1}{D}\left(b-c-\frac{1}{k}(2r_s{*}+r_i{*})-ar_i{*}\right) & -\frac{1}{D}\left(b-\frac{r_s{*}}{k}-ar_s{*}\right) & \frac{q}{kD}r_s{*} \\ -\frac{1}{D}\left(-\frac{r_i{*}}{k}+ar_i{*}\right) & -\frac{1}{D}\left(-c-\frac{1}{k}(r_s{*}+2r_i{*})+ar_s{*}\right) & \frac{q}{kD}r_i{*} \\ 0 & 0 & \frac{1}{D}\left(-(\beta-\gamma)+\varepsilon(r_s{*}+r_i{*})\right) \end{bmatrix}$$

where $r_s{*}=\frac{b}{a}$, $r_i{*}=k(b-c)-\frac{b}{a}$.

The eigenvalues of the Jacobian matrix around E_2 are the solutions of the following equation:

$$\left(\lambda-\frac{1}{D}\left(-\left(\beta-\gamma\right)+\varepsilon k\left(b-c\right)\right)\right)\left(\lambda^2+\frac{1}{D}\left(-2b+c-ak\left(b-c\right)\right)\lambda+\right.$$

$$\left.\left(\frac{1}{D^2}\left(b-c-\frac{1}{k}\left(\frac{b}{a}+k\left(b-c\right)\right)-ak\left(b-c\right)+b\right)\right)\left(\left(-c-\frac{2}{k}\left(b-c\right)+b\right)\right)\right)=0.$$

The corresponding properties equation to equilibrium E_2 has three eigenvalues. An eigenvalue $\frac{1}{D}\left(-\left(\beta-\gamma\right)+\varepsilon k\left(b-c\right)\right)$ has negative real part.

Solving the quadratic equation yields the other two eigenvalues

$$\lambda^2+A\lambda+B=0$$

where, $A=\frac{1}{D}\left(-2b+c-ak\left(b-c\right)\right)$

$$B=\left(\frac{1}{D^2}\left(b-c-\frac{1}{k}\left(\frac{b}{a}+k\left(b-c\right)\right)-ak\left(b-c\right)+b\right)\right)\left(\left(-c-\frac{2}{k}\left(b-c\right)+b\right)\right).$$

Both roots of this quadratic equation have negative real parts if and only if its coefficients are positive $\left(A>0 \text{ and } B>0\right)$. So, all of the eigenvalues of the characteristic equation are negative real parts. Therefore, the predator free equilibrium at E_2 of the model (2) is locally asymptotically stable.

NUMERICAL EXPERIMENTS AND DISCUSSION OF RESULTS

In this study, a numerical simulation of a reaction-diffusion model is refugium conducted using the standard finite-difference approach. Two scenarios will be considered; each characterized by different values of the parameter q, ε and diffusion constant, D for diffusion organisms model i.e. $\left(q<1 \text{ and } \varepsilon<1\right)$, $\left(q>1 \text{ and } \varepsilon<1\right)$, $D=0.001$ and $D=20$. The values of $a=0.1, b=1.0, c=0.6,$ $\beta=1.0$ and $\gamma=0.5$, are utilized as they were utilized by Peixoto and Abramson [5]. In our model, we've selected the same parameter q value for both the susceptible and infected rodent populations to ensure their equal competitiveness against the predator's population. We assume that the rodents and the alien species (acting as predators) diffuse at similar rates, with the goal of preventing the emergence of diffusion-driven instability. In the attendance of any minor fluctuation, diffusion-driven instability, as defined by Huang et al. [40], is the propensity of the uniform positions of a system to spontaneously develop patterns.

Hence, $k_c = 25$ represents the critical environmental condition, while $k = 150$ signifies a favorable environmental condition. As a results the infection is rapidly spreading in the basic *AK* model. The duration of approximate simulation is 20 years.

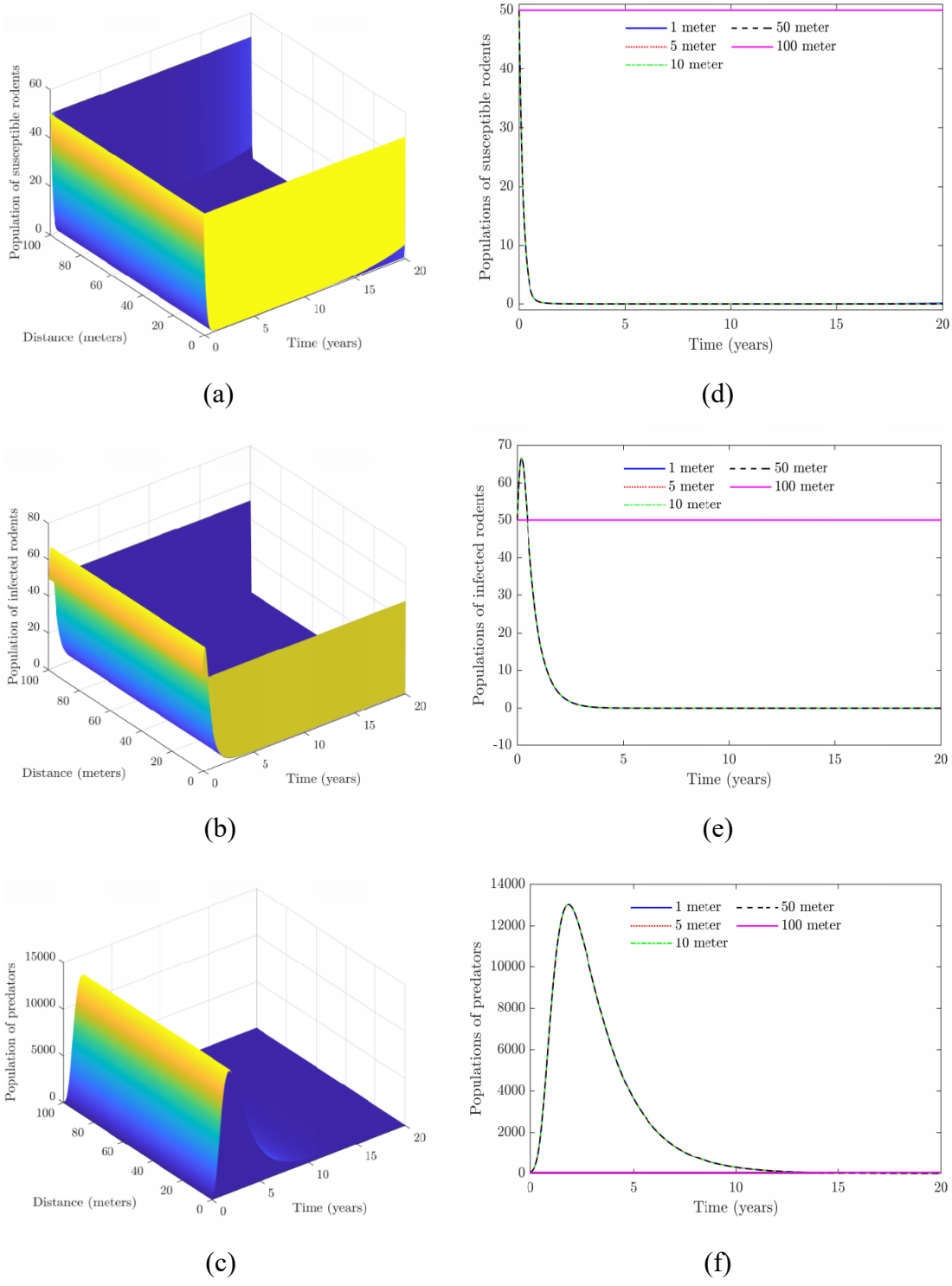


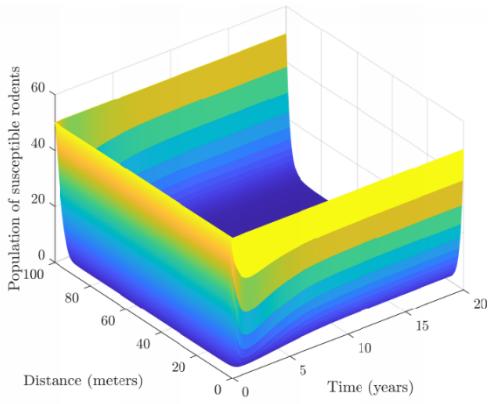
Figure 1: presents a three-dimensional plot illustrating the relationships between distance, time, and the populations of (a) susceptible rodents, (b) infected rodents, and (c) predators, with a focus on their respective effects on the community of (d) susceptible rodents, (e) infected rodents, and (f) predator in the refugium for $D = 0.001$.

Figure 1 shows the organisms (i.e. single rodent and alien a as predator) community for the case of $k(=150) > k_c$ and diffusion constant, $D = 0.001\left(D << \frac{1}{\pi^2}\right)$ when diffusion organisms (i.e. single rodent and alien a as predator) model is answered, using the various parameters of distances, x in meter $(x = 1, 5, 10, 50, 100)$, $q = 0.2$, $\varepsilon = 0.1$ $(q < 1 \text{ and } \varepsilon < 1)$ and the similar starting parameters for r_s, r_i and z $(r_s = r_i = z = 30)$.

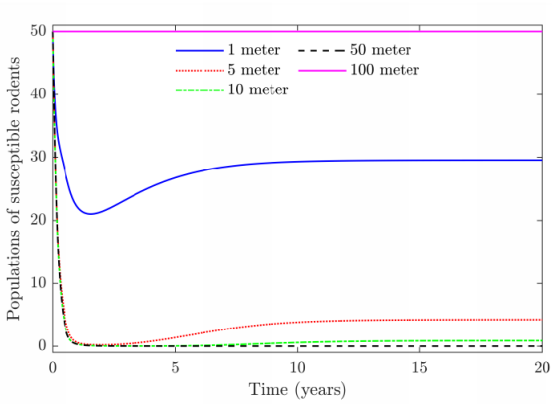
The positive solution shown in the graphs (a), (b), and (c) of Figure 1 is contained in the numerical design developed for the model (2). It shows the results of the numerical simulations taken at steady state when the diffusion mechanism is very small $\left(D(=0.001) << \frac{1}{\pi^2}\right)$. Meanwhile, the graph of organisms depends on the period, t with $k(=150) > k_c$ is as given in Figures 1(d), 1(e), and 1(f). These Figures demonstrate the susceptible rodent, infected rodent and alien (as predator) remain stable for various values of distance, x through solutions reach to zero for a smaller value of distance, x. In a broader sense, with the increase of the time, the peak value of alien (as predator) population z decreases exponentially for the model. Both populations of rodents are found to be zero after one year (Figures 1(d) and 1(e)). After one to 10 years, z shows a symmetric dome-shaped patch (Figure 1(f)). The position of the patch remains fixed and the shape of the patch changes with time. After one year, the extinction of rodent population occurs when the diffusion mechanism is very small $\left(D(=0.001) << \frac{1}{\pi^2}\right)$. The alien population (as predator) will increase in response to an abundance of resources such as food (rodents). As a result, the rodent's population decreases. This allows the alien (as predator) to eat and kill the rodent population due to the slow movement of the rodent population over the terrain and diversity of the landscape when the diffusion coefficient D is very small. Eventually, the rodent population going extinct in a short period of time. If x $(\text{i.e. } 100\,\text{m})$ is too large, the infection is thrived with the absence of the alien (as predator) community. This could

be attributed to the unrestricted movement of the rodent population, while the alien species (acting as predators) experience natural mortality and continue to reproduce. As a result, the diffusion coefficient, D and the distance x have stabilization effect and may aid to remove the intensity of the hantavirus contagion. When the diffusion mechanism is too small, the reaction-diffusion hantavirus infection model can be approximated over the long-term using a scheme of ordinary differential equations.

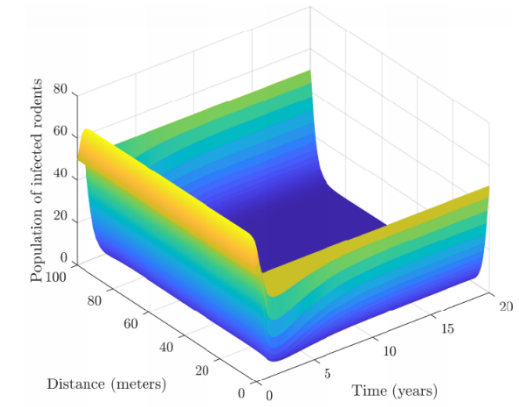
We have changed distance (x) throughout our numerical experiment. For the values $x = 1, 5, 10$ and 50, finally leads to trivial equilibrium $(0, 0, 0)$ in the solution of model (2). As demonstrated in Figures 1(d), 1(e), and 1(f), in the appearance of the infected rodents, the population dynamic is stable around the interior equilibrium point $(50, 50, 50)$ when $x = 100$. The trivial equilibrium point $(0, 0, 0)$and the interior equilibrium point $(50, 50, 50)$ of the model (2) are globally asymptotically stable as shown in Figures 1(d), 1(e), and 1(f). Therefore, we can finalize the whole scenarios of the domain. All populations become extinct if $x = 1, 5, 10$ and 50.



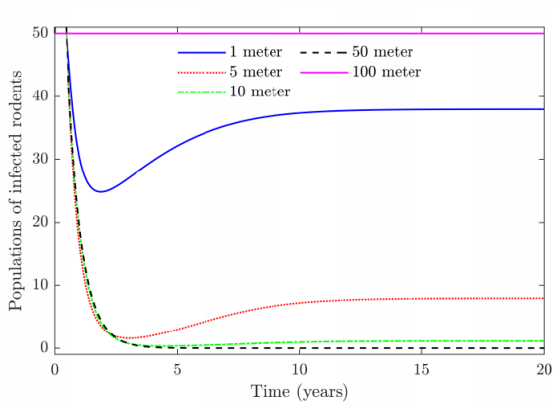
(a)



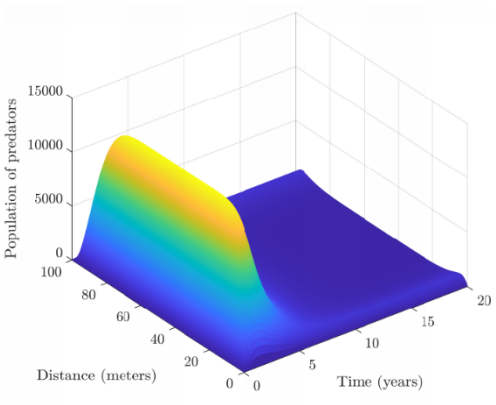
(d)



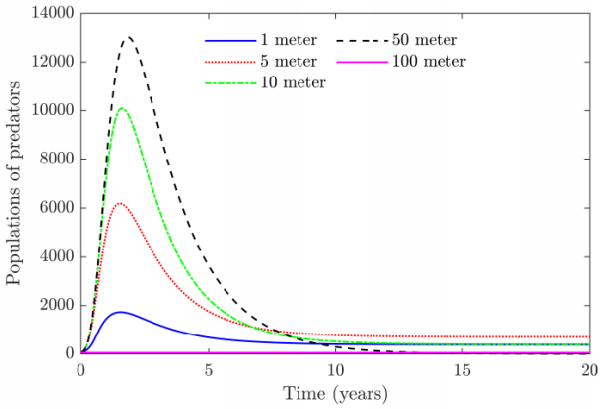
(b)



(e)



(c)



(f)

Figure 2: presents a three-dimensional plot illustrating the relationships between distance, time, and the populations of (a) susceptible rodents, (b) infected rodents, and (c) predators, with a focus on their respective effects on the community of (d) susceptible rodents, (e) infected rodents, and (f) predator in the refugium for $D = 20$.

Figure 2 shows the species (rodents and alien) community for the case of $k(=150) > k_c$ and increasing the diffusion constant D, i.e. $D = 20$ $\left(D > \frac{1}{\pi^2} \right)$, when diffusion single rodent single alien (as predator) method is answered, using the various parameters of distances, x in meter $(x = 1, 5, 10, 50, 100)$ and $q = 0.2, \varepsilon = 0.1$ $(q < 1 \text{ and } \varepsilon < 1)$. Results in a similar three-dimensional plot of the solutions are derived for the cases $D = 0.001$ and $D = 20$ based on Figures 1(a), 1(b), 1(c) and Figures 2(a), 2(b), 2(c). In all cases at an early stage of the dynamics model (2) (for $t = 1$), the population of alien (as predator) z shows the symmetric dome-shaped patch being formed. Meanwhile the populations of rodents are found to be drop after one year. The peak value of alien (as predator) population is higher than the peak value of rodent populations. For $x = 50$, the populations of rodents go extinct after one year while the peak

value of z decreases exponentially when increases the value of time. When the mobility of the rodent's community throughout the terrain and heterogeneity is moderate, an increase in the population of alien species (acting as predators) within the ecosystem will lead to a decline in the rodent population, eventually resulting in extinction. The aliens' (as predator) population tends to decrease because the population of rodents cannot survive, that is food for alien (as predator) is lacking. Finally, all the populations become extinct. The reason is that the alien (as predator) consumes all the rodents' community. As a result, the diffusion factor, D and the distance x have stabilization effect and may aid to thrive the intensity of the hantavirus infection.

Model (2) has four interior equilibrium: $(29.5429, 37.9350, 384.9105)$ $(4.2519, 7.9615, 706.0022)$, $(0.08720, 1.1262, 397.8449)$ and $(50, 50, 50)$with the distances, $x = 1, 5, 10, 100$, respectively. For $x = 50$, the solution converges to a trivial equilibrium $(0, 0, 0)$ eventually. The trivial equilibrium point $(0, 0, 0)$ and the interior equilibrium points $(29.5429, 37.9350, 384.9105)$, $(4.2519, 7.9615, 706.0022)$, $(0.08720, 1.1262, 397.8449)$, $(50, 50, 50)$ of the model (2) are globally asymptotically stable as shown in Figures 2(d), 2(e), and 2(f). Therefore, we can obtain the conclusion: all populations survive if $x = 1, 5, 10, 100$.

All the populations become extinct within a short time for the diffusion constant D is very small case compared with the diffusion constant D is moderate.

CONCLUSION

We demonstrated that the diffusion in a single rodent single alien (as predator) approach with the reaction-diffusion effect for the alien as predator resulted in a similar pattern of species spread as observed in the spread of hantavirus. We studied the propagation of competition among the susceptible and exposed rodents' populations, along with the alien (acting as a predator) in a reaction-diffusion model when higher available resources. The inclusion of diffusion constant D and distance x in this model played an important role in pattern formation. A similar pattern is produced when varying the diffusion constant D and distance x values. Investigations have been conducted to assess the local stability of the equilibrium points in the reaction-diffusion hantavirus infection approach.

Using numerical experiments, we demonstrated the occurrences of extinction and persistence phenomena. The peak value of alien (acting as a predator) population is continuously advanced than the peak rate of rodent populations when ($q < 1$ and $\varepsilon < 1$), the

distance, x is 50, the diffusion constant D is very small $\left(D(=0.001) << \frac{1}{\pi^2} \right)$ and it is moderate $\left(D(=20) > \frac{1}{\pi^2} \right)$. It is observed that the populations of rodents and alien (as predator) decline towards the end of the simulations and eventually become extinct when we choose a very small diffusion constant D $\left(D(=0.001) << \frac{1}{\pi^2} \right)$ for the distance, x are 1, 5, 10 and, 50 and ($q < 1$ and $\varepsilon < 1$). However, when the diffusion constant D is moderate $\left(D(=20) > \frac{1}{\pi^2} \right)$ and the distance, x are 1, 5, 10 and 100 and ($q < 1$ and $\varepsilon < 1$), The rodent and predator population have survived. What is important to note here is that the infection will die away when the rates of diffusion constant D is very minor or moderate and the distance, x less than 10 meter.

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