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RESEARCH ARTICLE

The biodiversity effect in regulating the prevalence of Sin Nombre virus (SNV)

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Abstract

Sin Nombre virus (SNV), a virus that can cause fatal disease among humans, is primarily hosted by the deer mouse, *Peromyscus maniculatus*. To better understand the biodiversity effect in curbing the prevalence of the SNV infection in the deer mouse population, we analyze the Peixoto & Abramson (2006) "one host, one non-host alien" deterministic model. In this study, we focus on the relationship of carrying capacity and interspecific competition strength of both host and non-host species in relation to the prevalence of the infection. Bifurcation analysis is carried out to examine the dynamics of this eco-epidemiological system. Our results show that the non-host species have a certain degree of influence in suppressing the SNV prevalence, given that the environmental conditions are similar and the interspecific competition strengths are relatively weak between the host and non-host species.

Keywords: Sin Nombre virus (SNV), interspecific interactions, biodiversity

INTRODUCTION

In the spring of 1993, the local authorities were alerted by the presence of a strange respiratory disease in the Southwestern United States after receiving death reports with the disease as its cause. Investigations were probed, and the disease was identified as hantavirus cardiopulmonary syndrome (HCPS) or hantavirus pulmonary syndrome (HPS). HPS primarily targets the lungs and may cause complications such as renal insufficiency, thrombocytopenia, myalgia and lethal outcomes (Jiang *et al.*, 2017). The mortality rate was reported to be around 40% in HPS cases (MacNeil *et al.*, 2011). The virus which caused HPS in that region was later termed as Sin Nombre virus (SNV) and the deer mouse, *Peromyscus maniculatus*, was later identified as the primary host of SNV (Jonsson *et al.*, 2010). SNV can be transmitted to humans through contacts with the saliva, excreta and urine of the infected animals (Jiang *et al.,* 2014). The outbreak of SNV was hypothesized to be linked to the large density increase in deer mouse population. This was confirmed through the Sevilleta LTER rodent studies where researchers were managed to observe 3-20 folds of density increment between 1992 and 1993 (Yater *et al.*, 2002). The occurrence of El Niño Southern Oscillation (ENSO) event, which brought favourable environmental conditions for the deer mouse population, in that period was attributed as the cause of the density increase (Dearing & Dizney, 2010). To our knowledge, Abramson & Kenkre (2002) were the first researchers that proposed a deterministic susceptible-infected mathematical model (AK model) to explain the dynamics of the SNV prevalence in the deer mouse population. Based on the AK model, they observed a critical carrying capacity threshold that needed to be attained for the infected population to prevail in the system, which coincided with the empirical observations after the ENSO event. The authors also extended the AK model in spatial term and managed to show the presence of "refugia" for the infected population when the environmental condition was less optimal. Abramson (2004) later tested the AK model with the empirical data from the Zuni capture sites in New Mexico. The AK model proved to be credible as the empirical data was well fitted. Thus, the AK model has become one of the pioneer models to many other variants; for example, the consideration of internal fluctuations in deer mouse population (Escudero *et al.*, 2004), the incorporation of age structural design (Reinoso & de la Rubia, 2015; Kenkre *et al.*, 2007), the extinction of SNV "refugia" in spatially heterogeneous environment (Kumar *et al.*, 2010). Furthermore, there are other researchers who studied the influence of external factors in suppressing the SNV prevalence; for example, Yusof *et al.* (2010) investigated several population harvesting strategies. They concluded that population harvesting could only reduce the intensity of the SNV prevalence when the environmental condition was favourable, and SNV prevalence could only be fully suppressed in the case of sub-optimal environmental condition. Following the case of biodiversity protects against Lyme disease (Ostfeld & Keesing, 2000), researchers such as Yusof *et al.* (2014) and Peixoto & Abramson (2006) modified the AK model by incorporating the biodiversity effects. Yusof *et al.* (2014) studied the effect of predator on the SNV prevalence and their simulation results showed the system stabilised at the extinction of both deer mouse and predator species. In our opinion, such situation is less ideal as we only want the SNV infection to subside while preserving the deer mouse species. On the other hand, Peixoto & Abramson (2006) studied the dynamics of SNV prevalence in the presence of a non-host alien species (which we will term it as PA model). Despite the interspecific competition strength between the deer mouse and non-host species was rather weak, they managed to observe the presence of the non-host species that was able to suppress the SNV prevalence while preserving the deer mouse population. Empirical studies such as (Dizney & Ruedas, 2009; Suzan *et al.*, 2009; Clay *et al.*, 2009; Luis *et al.*, 2018) managed to observe a negative relationship between rodent species diversity and SNV prevalence which supported the theoretical results of Peixoto & Abramson (2006). Hence, our goal in this paper is to provide a comprehensive analysis on the PA model, especially in terms

of the combined effects of carrying capacity and interspecific strength of both deer mouse and non-host species using bifurcation analysis techniques.

METHODOLOGY

To provide a better understanding for the readers, we shall first briefly present the AK model before going into the PA model. The AK model is built upon on the characteristics of the SNV transmission; such as, SNV can only be transmitted horizontally (physical contact between the susceptible and infected, for example fighting), the SNV infection is life-long (once the deer mouse is infected, it cannot recover), the SNV infection does not induce death in the infected deer mouse, and infected deer mouse can give births to healthy individuals (no horizontal transmission). The AK model is given as below:

$$\frac{dS}{dt} = b(S+I) - dS - \frac{S(S+I)}{K} - aSI \qquad (1)$$
$$\frac{dI}{dt} = -dI - \frac{I(S+I)}{K} + aSI$$

where S and I represent the susceptible and infected deer mouse population, b is the birth rate, d is the death rate, K is the carrying capacity, and a is the SNV transmission rate, which is the result of the product between the contact rate and the probability of being infected.

By setting $\frac{dS}{dt}$ and $\frac{dI}{dt}$ equal to 0 in system (1), we obtain the equilibrium points through some algebraic manipulations. There are 3 nonnegative equilibrium points; namely, the trivial solution, existence of susceptible population only, and existence of both susceptible and infection. Furthermore, it can be easily shown that the critical carrying capacity, $K_c = \frac{1}{a}\left(\frac{b}{b-c}\right)$ for the infection to exist in the system. Interested readers can refer to Abramson & Kenkre (2002) for more details.

Now, we shall introduce the PA model. The PA model is constructed based on the idea of AK model but with the inclusion of a non-host alien species to account for the biodiversity effect. The non-host alien species is defined as a rodent species other than the deer mouse which cannot be infected by the SNV. The deer mouse and non-host species can influence each other by pressuring the other party through their corresponding interspecific competition strength. Thus, the PA model is formulated as below:

$$\frac{dS}{dt} = b_1(S+I) - d_1S - \frac{S(S+I+q_1Z)}{K_1} - aSI$$
$$\frac{dI}{dt} = -d_1I - \frac{I(S+I+q_1Z)}{K_1} + aSI \qquad (2)$$
$$\frac{dZ}{dt} = b_2Z - d_2Z - \frac{Z[Z+q_2(S+I)]}{K_2}$$

where S, I and Z represent the susceptible deer mouse, infected deer mouse and non-host alien population. The description of the other parameters is presented in Table 1 along with the values which will be used in the subsequent analysis and results section.

Another interesting point to note is that by adding up the susceptible and infected equation in system (2), we will then get the 2-species Lotka-Volterra competition model as shown below:

$$\frac{dN}{dt} = b_1N - d_1N - \frac{N(N+q_1Z)}{K_1} \qquad (3)$$
$$\frac{dZ}{dt} = b_2Z - d_2Z - \frac{Z(Z+q_2N)}{K_2}$$

where $N = S + I$. This suggests that a bistability steady state will occur when the interspecific competition strength exerted by the deer mouse and non-host species are intense (meaning $q_1, q_2 > 1$ in cases where the environmental conditions (carrying capacity) of both deer mouse and non-host species are similar). Hence, the initial population density of both deer mouse and non-host species will dictate the presence-absence of the said species in the ecosystem.

Table 1 Descriptions and values of the parameters in system (2).

Parameters	Descriptions	Values
b_1	The birth rate of deer mouse population	1.0
d_1	The death rate of deer mouse population	0.6
K_1	The carrying capacity of the deer mouse population	50
a	The SNV transmission rate among deer mouse population	0.1
b_2	The birth rate of the non-host population	1.0
d_2	The death rate of the non-host population	0.5
K_2	The carrying capacity of the non-host population	30
q_1	The interspecific competition strength exerted by the non-host population onto the deer mouse population	0.4
q_2	The interspecific competition strength exerted by the deer mouse population onto the non-host population	0.3

RESULTS

The equilibrium points of system (2) can be found by setting the left-hand side equals to 0. There are a total of 6 nonnegative equilibrium points which includes $(S^*, I^*, Z^*) = (0,0,0)$, $\left(0,0,K_2(b_2-c_2)\right)$, $(K_1(b_1-c_1),0,0)$, $\left(\frac{b_1}{a}, K_1(b_1-c_1)-\frac{b_1}{a}, 0\right)$, $\left(\frac{K_1(b_1-c_1)-q_1K_2(b_2-c_2)}{1-q_1q_2}, 0, \frac{K_2(b_2-c_2)-q_2K_1(b_1-c_1)}{1-q_1q_2}\right)$ and $\left(\frac{b_1}{a}, \frac{K_1(b_1-c_1)-q_1K_2(b_2-c_2)}{1-q_1q_2} - \frac{b_1}{a}, \frac{K_2(b_2-c_2)-q_2K_1(b_1-c_1)}{1-q_1q_2}\right)$. To determine steady state of the system, we would need to solve for the corresponding eigenvalues of the equilibrium points. The Jacobian matrix for system (2) is given by

$$\begin{pmatrix} b_1 - c_1 - \frac{S+I+q_1Z}{K_1} - \frac{S}{K_1} - aI & b_1 - \frac{S}{K_1} - aS & -\frac{q_1S}{K_1} \\ -\frac{I}{K_1} + aS & -c_1 - \frac{S+I+q_1Z}{K_1} - \frac{I}{K_1} + aS & -\frac{q_1I}{K_1} \\ -\frac{q_2Z}{K_2} & -\frac{q_2Z}{K_2} & b_2 - c_2 - \frac{Z+q_2(S+I)}{K_2} - \frac{Z}{K_2} \end{pmatrix}$$

Due to the complexity of the equations, we utilise Maple 2016 to solve for the eigenvalues in which the stable steady state is identified when all 3 eigenvalues are less than 0 for the choice of parameter values used.

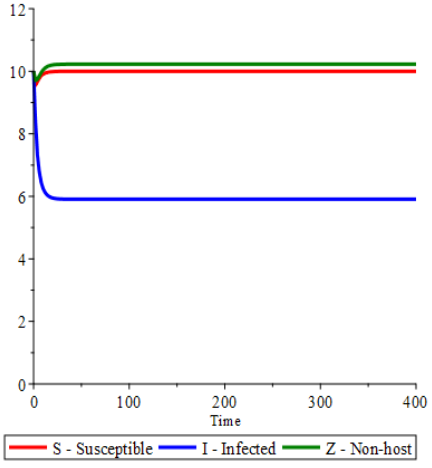

Fig. 1 Time series graph of S, I and Z with parameter values in Table 1 and initial $(S, I, Z) = (10, 10, 10)$.

By substituting the parameter values as in Table 1 into system (2) with the initial $(S, I, Z) = (10, 10, 10)$, a time series graph is plotted (refer to Fig. 1). From Fig. 1, we can see that the system achieves a stable steady state of the coexistence of the susceptible, infected and

non-host species. The corresponding equilibrium values are $(S^*, I^*, Z^*) = (10, 5.91, 10.23)$ and all 3 eigenvalues are indeed negative as shown in Table 2.

Table 2 The stability of system (2)'s equilibrium points based on parameter values as in Table 1.

Equilibrium Point	Eigenvalues	Stability
$(S, I, Z) = (0, 0, 0)$	$\lambda_1 = -0.60, \lambda_2 = 0.50, \lambda_3 = 0.40$	Unstable
$(S, I, Z) = (0, 0, 15)$	$\lambda_1 = -0.50, \lambda_2 = -0.72, \lambda_3 = 0.28$	Unstable
$(S, I, Z) = (20, 0, 0)$	$\lambda_1 = -0.40, \lambda_2 = 1.00, \lambda_3 = 0.30$	Unstable
$(S, I, Z) = (15.91, 0, 10.23)$	$\lambda_1 = -0.44, \lambda_2 = -0.21, \lambda_3 = 0.59$	Unstable
$(S, I, Z) = (10, 10, 0)$	$\lambda_1 = -0.40, \lambda_2 = -1.00, \lambda_3 = 0.30$	Unstable
$(S, I, Z) = (10, 5.91, 10.23)$	$\lambda_1 = -0.44, \lambda_2 = -0.21, \lambda_3 = -0.59$	Stable

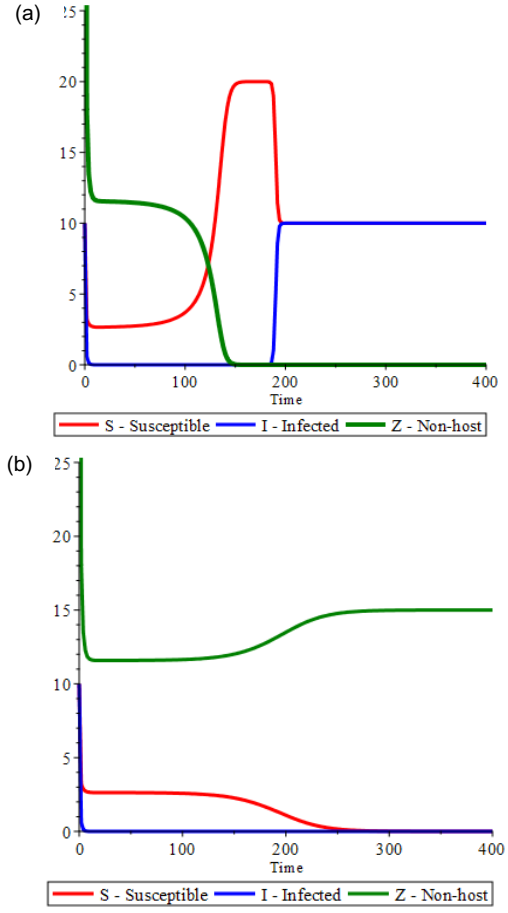

Fig. 2 Time series graphs of bistability steady state with $q_1 = 1.5$, $q_2 = 1.3$, initial $(S, I, Z) = (10, 10, 135)$ for case 1 (a) and initial $(S, I, Z) = (10, 10, 136)$ for case 2 (b).

To showcase the possibility of a bistability steady states of system (2), we plot two additional time series graphs with the same parameter values as in Table 1 but with $q_1 = 1.5$, $q_2 = 1.3$, initial $(S, I, Z) = (10, 10, 135)$ for case 1 and initial $(S, I, Z) = (10, 10, 136)$ for case 2 instead. By comparing Fig. 2(a) and 2(b), we clearly see that the system is either converged to $(S^*, I^*, Z^*) = (10, 10, 0)$ as seen in Fig. 2(a) or converged to $(S^*, I^*, Z^*) = (0, 0, 15)$ as seen in Fig. 2(b). The numerical values of the equilibrium points along with their respective stability properties are calculated using Maple 2016 as shown in Table 3. These results agree with our hypothesis of the bistability steady state existence, in which it exhibits sensitivity to the small change in initial population. Unfortunately, the bistability steady state is not an ideal situation in epidemiological ecology point of view. This is because the SNV infection either persists in the case where the deer mouse triumphs over the non-host or be eradicated but at the cost of the deer mouse extinction. This does not align to our goal of suppressing SNV infection while preserving the deer mouse and non-host population.

Table 3 The stability of system (2)'s equilibrium points based on parameter values as in Table 1 except for $q_1 = 1.5$, $q_2 = 1.3$.

Equilibrium Point	Eigenvalues	Stability
$(S, I, Z) = (0, 0, 0)$	$\lambda_1 = -0.60, \lambda_2 = 0.50, \lambda_3 = 0.40$	Unstable
$(S, I, Z) = (0, 0, 15)$	$\lambda_1 = -0.50, \lambda_2 = -1.05, \lambda_3 = -0.05$	Stable
$(S, I, Z) = (20, 0, 0)$	$\lambda_1 = -0.40, \lambda_2 = 1.00, \lambda_3 = -0.37$	Unstable
$(S, I, Z) = (2.63, 0, 11.58)$	$\lambda_1 = 0.04, \lambda_2 = -0.48, \lambda_3 = -0.74$	Unstable
$(S, I, Z) = (10, 10, 0)$	$\lambda_1 = -0.40, \lambda_2 = -1.00, \lambda_3 = -0.37$	Stable
$(S, I, Z) = (10, -7.37, 11.58)$*	$\lambda_1 = 0.04, \lambda_2 = -0.48, \lambda_3 = 0.74$	Unstable

*Ecologically infeasible.

To further explore the dynamical system (2), bifurcation analysis up to co-dimension 2 is carried out on K_1, K_2, q_1 and q_2 with the values indicated in Table 1, unless stated otherwise, through the utilisation of XPPAUT. The co-dimension 1 results can be found in Fig. 3 while the co-dimension 2 results can be seen in Fig. 4.

Co-dimension 1 bifurcation analysis

In Fig. 3(a), we can see that a minimum K_2 (reference to the first transcritical bifurcation point, $\alpha = 12$) is required for the non-host population to exist. For any value of K_2 below $\alpha = 12$, the system will only reach a stable steady state at (10,10,0), indicating the absence of the non-host population. When K_2 is in between $\alpha = 12$ and $\beta = 56$, the system will reach the coexistence of susceptible, infected and non-host stable steady state and the population density of the infected deer mouse starts to decrease along with increasing K_2. When K_2 is beyond $\beta = 56$, the infected population will be eradicated, leaving behind the coexistence of the susceptible and non-host population only. This indicates that a critical carrying capacity threshold for the non-host species, $\beta = 56$ needs to be attained in order to fully eliminate the SNV while any K_2 between $\alpha = 12$ and $\beta = 56$ will only reduce the infection intensity in the system.

For the bifurcation analysis of q_1 (Fig. 3(b)), we can see that the inclusion of a non-host population interacting with the deer mouse population will reduce the prevalence of SNV even if the interspecific competition pressure exerted by the non-host species is relatively weak. Furthermore, the system reaches a coexistence of susceptible and non-host population stable steady state when q_1 is larger than $\gamma = 0.833$. This indicates that a relatively moderate interaction strength q_1 is required to eliminate the infected population when the carrying capacity of the non-host is less abundant than the deer mouse population ($K_2 < K_1$) given that the interspecific competition pressure exerted by the deer mouse is relatively weak ($q_2 = 0.3$).

In Fig. 3(c), a co-dimension 1 bifurcation diagram for Z is plotted against q_1 with the parameter values as in Table 1 but $q_2 = 1.3$ instead. There are two stable steady states in the figure, where one corresponds to the coexistence of the susceptible and infected deer mouse while the other corresponds to the survival of the non-host population only. As these two branches of stable steady state do not intersect with one another, this indicates the presence of the bistability steady states and the initial abundance of either the deer mouse or non-host population will dictate the presence-absence of one another. It is also interesting to note that the transcritical bifurcation point $\theta = 1.333$ represents the switch of stable steady states from a negative solution branch to $(0, 0, Z^*)$ branch. This indicates that it is ecologically infeasible for Z to exist when $q_1 < \theta$ and care should be given on the initial population density as bistability occurs when $q_1 > \theta$.

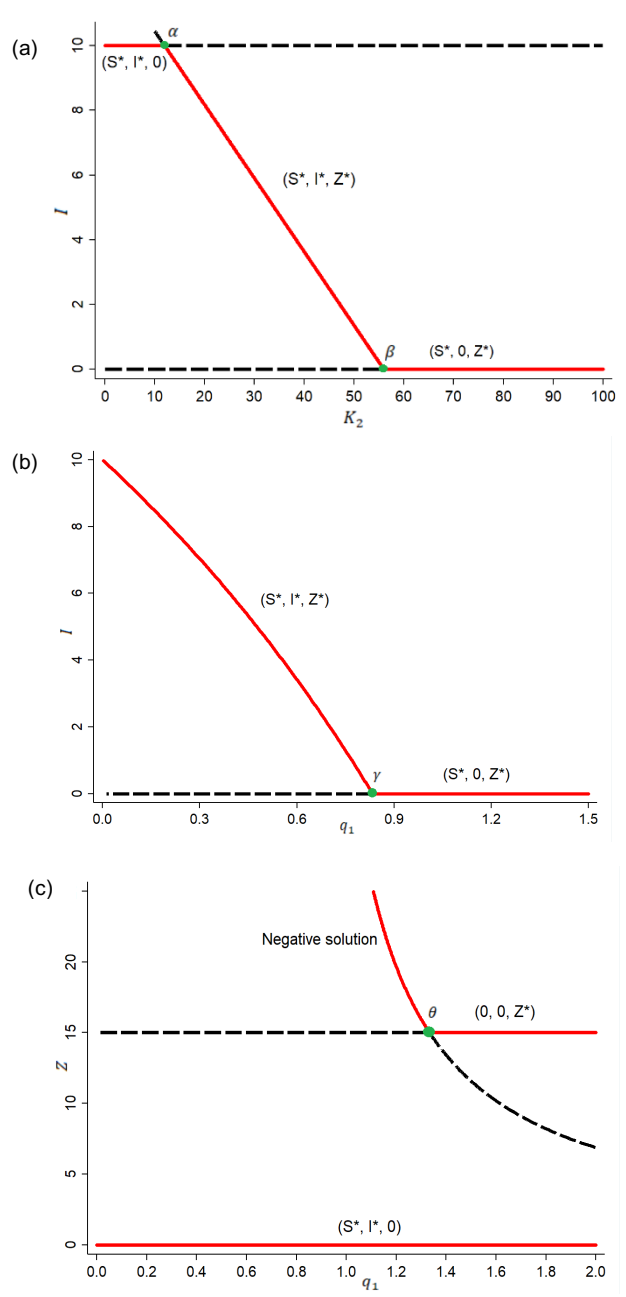

Fig. 3 Co-dimension 1 bifurcation diagrams where (a) and (b) are plotted by varying K_2 and q_1 against the infected population while (c) is plotted by varying q_1 against the non-host population with $q_2 = 1.3$. The red solid lines represent the stable steady state while the black dashed lines represent the unstable steady state.

Co-dimension 2 Bifurcation Analysis

By extending the analysis from the transcriticial bifurcation points in Fig. 3, we are able to obtain the co-dimension 2 bifurcation diagrams as shown in Fig. 4. In the diagrams, the regions between the lines are corresponded to distinct stable steady states of the system for some particular choices of parameters; for example, by choosing $K_1 = 10$ and $K_2 = 10$ in Fig. 4(a), this corresponds to the coexistence of susceptible and non-host population stable steady state; and if we increase K_1 by 20 ($K_1 = 30$), the system will cross over the transcritical bifurcation line and the stable steady state will switch over to the coexistence of the susceptible, infected and non-host population.

In Fig. 4(a), there are five stable steady state regions in total. When $K_1, K_2 < 20$, there is no SNV prevalence, and the coexistence of the susceptible deer mouse and non-host can be ensured when K_1 and K_2 are rather similar to each other. However, either the susceptible or non-host population can survive when carrying capacity of a species is relatively much larger than the other in this region; for example, only the non-host will survive when $K_1 = 5$ and $K_2 = 15$ in the system. When the value of K_1 is more favourable (e.g, $K_1 = 40$), the infected population starts to prevail. The abundance of K_2 can only increase the critical carrying capacity of the deer mouse (K_1) needed for the infected population to exist. This is indicated by the slanted slope separating the presence and absence of infected population regions. Hence, the more abundant K_2 is, the higher the K_1 required for the infected population to invade the system when the relatively interspecific competition strength between deer mouse and non-host species is relatively weak ($q_1 = 0.4, q_2 = 0.3$).

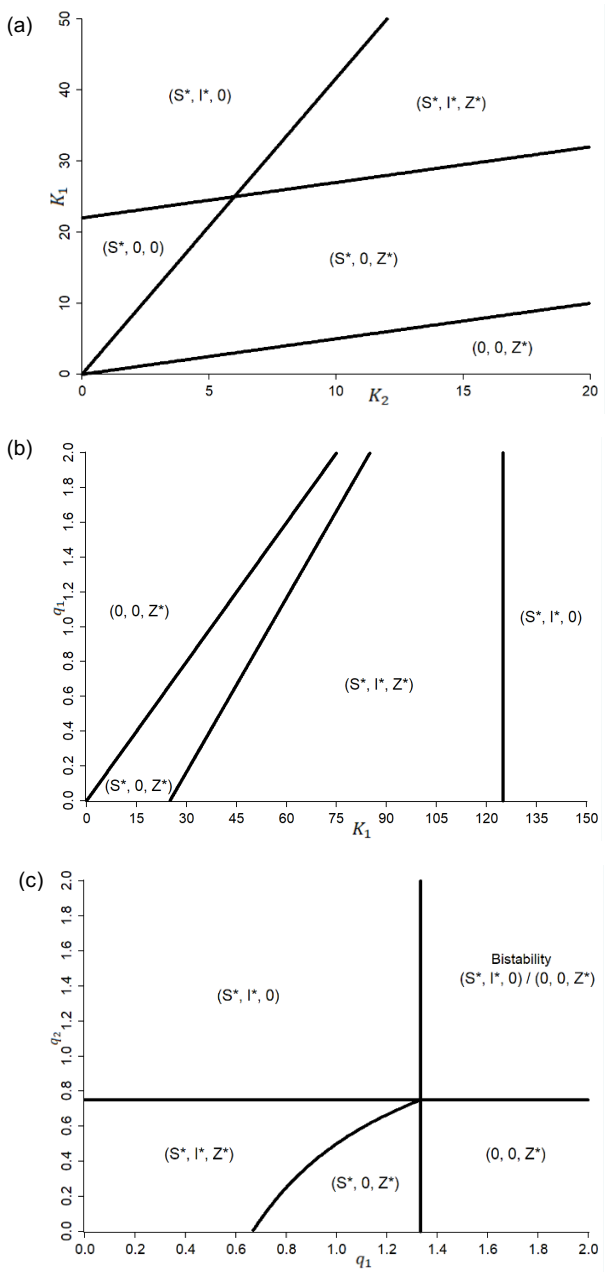

Fig. 4 Co-dimension 2 bifurcation diagrams where (a), (b) and (c) investigate the relationship between K_2 versus K_1, K_1 versus q_1, and q_1 versus q_2 correspondingly.

For the relationship between K_1 and q_1, we can refer to Fig. 4(b). Based on Fig. 4(b), there are four distinct stable steady state regions. When K_1 is very low, the non-host population with moderate or high q_1 can easily triumph over the deer mouse population which leaves us with the extinction of the deer mouse in the system. By increasing K_1 slightly to the right, the susceptible deer mouse and non-host can coexist. However, the region for the coexistence between the susceptible deer mouse and non-host gets narrower with the increase of K_1 and q_1. This suggests that the interspecific competition strength of the non-host population when K_1 is abundant, may not be as effective as when K_1 is

less than optimal even if the non-host population is very aggressive towards the deer mouse population. If we continue increasing K_1 to a certain extend (e.g. $K_1 = 130$), we can see the extinction of the non-host population irrespective of how strong q_1 is given that K_2 is relatively low compared to the now abundant K_1.

In Fig. 4(c), there are five stable steady state regions where the upper right region is bistability steady state. For instance, when both species have relatively low interspecific competition strength ($q_1, q_2 < 0.6$), the SNV prevalence will persist in the system. Only when q_1 is moderate or moderately high (e.g., $0.7 < q_1 < 1.3$) while q_2 is relatively low (e.g., $q_2 < 0.6$), the infected population can only be eradicated from the system. When the interspecific competition strength of one species is relatively much higher than the other, then only the species with the higher interspecific competition strength can survive while the other goes extinct (as seen in the upper left and lower right region in Fig. 4(c)). However, a bistability will occur when the interspecific competition strength of both deer mouse and non-host population are high. Thus, the species with a larger initial population density will survive in this case. If the initial deer mouse population density triumphs over the non-host, then the non-host population will go extinct while the susceptible and infected deer mouse population will flourish.

In summary, the bifurcation analysis shows that the presence of the non-host population can indeed reduce the intensity of the SNV prevalence in the deer mouse population; and to some extent, the SNV infection can be fully eradicated if the carrying capacity and interspecific competition strength of the deer mouse population is rather low and weak, respectively. However, this is not the case when the carrying capacity and interspecific competition strength of the deer mouse population are abundant and high. The infected population is likely to persist in the system despite the influence of the non-host population.

DISCUSSION

From the aforementioned results, it can be clearly seen that the inclusion of the non-host population has a certain effect in regulating the SNV prevalence in deer mouse population. When the interspecific competition strength of both deer mouse and non-host population are relatively weak and the carrying capacity of both species are similar, the non-host is able to reduce the intensity of the SNV prevalence and in some cases, eliminating the infected population. However, the influence of the non-host population on the SNV prevalence is rather limited when its carrying capacity is relatively much lower than the deer mouse population or the deer mouse population exhibits a rather high interspecific competition pressure onto the non-host population. In such extreme cases, the infected deer mouse population will still prevail. Thus, this shows that the effectiveness of the biodiversity effect in reducing SNV prevalence largely depends on the similar environmental conditions required by both deer mouse and non-host population, and the interspecific competition strength between the two species should be relatively weak against each other.

Our study has a few shortcomings. One of them is that we only provide a more comprehensive analysis on the temporal PA model. Perhaps the PA model can be further extended in the spatial term like what Abramson & Kenkre (2002) did on the AK model. To account for the spatial term, the authors included diffusion coefficients into the AK model and their simulation result showed the presence of "refugia" for the SNV infection when the environmental condition was less favourable. Hence, this may explain the reappearance of the SNV in some scenarios when the environmental condition for the deer mouse has returned to favourable. In another ecological study, Mohd *et al.* (2018) showed that the bistability phenomenon could interact with dispersal mechanisms of species to determine multi-species community assemblies. Thus, we may have missed some interesting features when we omit the spatial term and dispersal effects in the PA model. Besides that, our analysis is presented with a hypothetical non-host alien species instead of a real one due to the difficulties of obtaining empirical data. Hence, our results may differ from the observations of a field experimental study, but this can be remedied by varying the parameter values in Table 1 for comparison. Furthermore, the PA model only considers the interaction with a non-host species; though other researchers have pointed out that other *Peromyscus* species (Abbott *et al.*, 1999) can serve as secondary reservoirs to SNV. In some cases, the presence of a secondary host may reduce the SNV prevalence, depending on its quality in disease transmission. Interested readers may refer to O'Regan *et al.* (2015) for more details on the dynamics in disease transmission between two host species. Perhaps we can also explore the PA model through stochastic approach as the stochastic framework may better reflect the reality and at times, its result may impose a slight difference from the deterministic model; e.g. Mohd *et al.* (2016) managed to observe contrasting results on alternative stable states in multiple species between their stochastic model and deterministic model.

CONCLUSION

Our study was aimed to provide a comprehensive analysis on the PA model, especially on the carrying capacity and interspecific competition strength of the deer mouse and non-host species, in assessing the influence of biodiversity effect on the SNV prevalence. We were able to show that the non-host population has a certain degree of influence on suppressing the SNV prevalence in deer mouse but its effect may seem negligible when the environmental condition very much favours the deer mouse population and that interspecific competition strength of deer mouse is relatively strong. We hope future studies can further analyse the biodiversity effect on SNV prevalence; perhaps, by studying the PA model in spatial term or constructing the PA model in stochastic frameworks as these may provide interesting results compared to the current temporal PA model.

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