Modelling the Dilution and Amplification Effects on Sin Nombre Virus (SNV) in Deer Mouse in GAMA 1.8

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Abstract Sin Nombre Virus (SNV) is a species of hantavirus that can cause hantavirus pulmonary syndrome in humans. To investigate the biodiversity effect on the SNV transmission in deer mouse, we formulated a stochastic agent-based model (ABM) to compare the impact between the presence of a dilution agent and an amplification agent in the deer mouse population. The ABM simulations were done in GAMA 1.8 and the results were then compared with the deterministic counterpart of the model. The deterministic results showed the dilution agent has better effectiveness in reducing the infected density compared to the amplification agent. However, this was not observed for the stochastic results with small populations. Instead, the infected densities were at a similar level for both dilution and amplification agent in the ABM results. This suggests that the investigation on the role of the community assemblage may not be relevant in reducing SNV transmission when the population density is small, and further research is needed to better understand the discrepancy between the stochastic and deterministic result and its implications. Our study highlights the importance of ABM in eco-epidemiological studies, and has established a methodological discussion regarding the usability of different simulation approaches e.g., deterministic and stochastic ABM in order to produce robust observations of eco-epidemiological phenomenon under consideration.

Keywords Sin Nombre Virus (SNV) · Biodiversity · GAMA

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M. H. Mohd et al. (eds.), *Modelling, Simulation and Applications of Complex Systems*,
Springer Proceedings in Mathematics & Statistics 359,
https://doi.org/10.1007/978-981-16-2629-6_3

1 Introduction

In 1993, the Four Corners region in the United States had a mysterious disease outbreak which killed many humans. The disease was later identified as the hantavirus pulmonary syndrome (HPS), which was caused by a hantavirus species called the Sin Nombre Virus (SNV) [1]. The SNV is primarily hosted by the deer mouse, *Peromyscus maniculatus*, and humans can be infected through contacts with the saliva, urine and excreta of the infected rodents [2]. To better understand the dynamics of SNV among the deer mouse population, Abramson and Kenkre [3] were one of the pioneers to mathematically model this eco-epidemiological problem. They proposed a susceptible-infected (SI) spatio-temporal model to investigate the dynamics of the SNV. Peixoto and Abramson [4] later extended the model to include the biodiversity effect on the SNV transmission in deer mouse. Based on their theoretical model, they observed that the presence of a non-host alien species was able to reduce the SNV prevalence. Empirical studies such as [5, 6] have further supported such hypothesis.

However, Randolph and Dobson [7] warned that the biodiversity effect may not necessarily reduce a disease prevalence as amplification effect may occur instead. Studies such as [8, 9] investigated the amplification effect (increase in disease prevalence with increase species diversity) and dilution effect (decrease in disease prevalence with increase species diversity) for the case of Lyme disease. Authors from [8] concluded that the occurrence of amplification or dilution effect is dependent on the mechanism of competition, the host contact rates with ticks and acquired host resistant to ticks. Furthermore, different work such as [10] has investigated the mechanisms which cause the dilution or amplification effect for the endemic case of a disease. They highlighted that factors such as the type of disease transmission, relationship between the host competence and community assembly, and identity of hosts contributing to disease transmission, should be investigated to uncover whether a dilution or amplification effect can occur. For the case of SNV in deer mouse, Luis et al. [11] observed the occurrence of both amplification and dilution effects from their empirical data. Both effects occurred concurrently, and a net dilution was observed due to the dilution effect being greater between the two effects. Hence, we were interested in formulating a mathematical model to better understand the dynamics of the amplification and dilution effects of a non-host species and the impacts of stochasticity on SNV transmission in deer mouse.

In recent years, stochastic agent-based model (ABM) has received much utilization in eco-epidemiology studies. Unlike deterministic model, an ABM manages to incorporate the noise feature, which allows it to better mimic the reality. Allen [12] showed the importance of stochastic modelling of epidemics especially when the number of infectious individuals is small, or when there occurs a variability in transmission, recovery, births, deaths, or the environment which impacts the epidemic outcome. Authors from [13] were able to observe the extinction of disease in a general multi-host epidemic model given that the level of prevalence in the spillover species is relatively low and the reproduction number in the reservoir host is less than one. However, such occurrence was not observed in their deterministic model;

thus, showcasing the capability of stochastic modelling in understanding mechanisms underlying natural phenomenon. Eco-epidemiology studies such as [14, 15] have utilized the stochastic approach in their studies for modelling dengue disease and disease transmission among Tilapia with Pelican respectively. Guzzeta et al. [16] highlighted the incorporation of stochastic effect into modelling the dynamics of zoonotic pathogen has allowed them to gauge the probability and severity of potential future outbreaks. Besides that, several studies on the dynamics of hantavirus [17–19] also utilized the stochastic approach. Therefore, we would like to opt for a stochastic ABM for this study as well. It is the goal of this paper to show the importance of ABM in eco-epidemiological studies, and to establish a methodological discussion regarding the usability of different simulation approaches e.g., deterministic and stochastic ABM in order to produce robust observations of eco-epidemiological phenomenon under consideration.

In the next section, the deterministic model would first be introduced; then, the stochastic ABM counterpart would be introduced along with its implementation in GAMA version 1.8, a software which supports agent-based modelling. While some studies, e.g. Mohd [20] and Mohd [21], have simulated agent-based models (ABM) using Matlab package, we opt to study the dynamics of ABM by employing GAMA 1.8 to give an alternative approach on simulating the biological system using the techniques of stochastic process and differential equations. To the best of our knowledge, this approach has not been employed before (upon checking the literature review on GAMA), which is one of the main novelties of this study. This has contributed to the methodological discussions on the use of different modelling techniques and computer packages to examine the biological phenomena of interest. Similar to other platforms, GAMA provides similar flexibility to code the agent's characteristics and behavior according to the researcher's choice of techniques and assumptions. As mentioned above, what we would like to show is that GAMA can serve as an alternative towards the other platforms and it is up to the researcher's discretion to choose whichever platform they are comfortable with.

2 Model Formulation

2.1 Deterministic Model

The deterministic model we would like to introduce is based on the proposed model in our previous research [22]. It is a "single host, single non-host" endemic model that accounts for density-dependent restricted logistic growth, with the non-host having a certain amount of influence on the SNV transmission rate depending on its amplification or dilution role in a closed system. The biodiversity effect is accounted for through the inclusion of the non-host into the deer mouse community. The deterministic model is given as below:

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= N\left[b_1 - ar_1\left(\frac{N+q_1Z}{K_1}\right)\right] - S\left[d_1 + r_1(1-a)\left(\frac{N+q_1Z}{K_1}\right)\right] - \gamma(1+\delta Z)SI \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \gamma(1+\delta Z)SI - I\left[\mu + d_1 + r_1(1-a)\left(\frac{N+q_1Z}{K_1}\right)\right] \\ \frac{\mathrm{d}Z}{\mathrm{d}t} &= r_2\left[1 - \frac{Z+q_2N}{K_2}\right]Z\end{aligned} \tag{1}$$

where $N = S+I$ is the total average population density of the deer mouse per hectare, S is the average population density of the susceptible deer mouse per hectare, I is the average population density of the infected deer mouse per hectare, and Z is the average population density of the non-host individuals per hectare. The descriptions for the rest of the parameters can be found in Table 1. Most of the parameter values were based on [11] while the rest were based on the work of [22]. The parameter values obtained from [11] were based on their observational study at several sites, while the parameter values in [22] were based on a modelling study, which was motivated by the ecological studies of Luis et al. [11]. Interested readers can refer to [11, 22] for the assumptions and derivation.

Similar to our previous study, we shall present 2 case studies to account for the dilution and amplification role of the non-host in a small population community. Our interest in modelling for a small population was to observe for potential differences between the deterministic and stochastic results at this level. For case study 1, the non-host (dilution agent) has a relatively weaker interspecific competition strength compared to the deer mouse ($q_1 = 0.2$) but it does not contribute any positive influence towards the SNV transmission rate ($\delta = 0$). For case study 2, the non-host (amplification agent) has a relatively stronger interspecific competition strength compared to the deer mouse ($q_1 = 0.4$) and has a positive influence on the SNV transmission rate ($\delta = 0.0543$). These parameter values were chosen to best reflect the reality whereby a dilution agent, e.g. desert pocket mouse, is timid towards the deer mouse; while the amplification agent, e.g. Merriam's kangaroo rat, is aggressive towards the deer mouse and thus, the deer mouse would avoid encountering them [23]. In the presence of the Merriam's kangaroo rat (amplification agent), the activity area of the deer mouse becomes smaller and this may increase the stress in deer mouse, which indirectly influence its susceptibility towards the SNV infection [24].

2.2 *Stochastic ABM*

To model the stochastic ABM counterpart of model (1), we utilized the discrete-time Markov chain approach. We assumed that each individual has a probability in executing one of the three events, namely "reproduce", "die" or "do nothing", in every small time step, Δt. When an individual executes the "reproduce" event in the $[t, t + \Delta t)$ interval, a new individual of the same category will be created in the system. For the "die" event, the individual would be permanently deleted from the system. Finally, nothing will happen to the individual if it executes the "do

Table 1 Descriptions and parameter values for model (1)

Parameters	Descriptions	Parameter Value	
		Case Study 1	**Case Study 2**
K_1	The carrying capacity of the deer mouse (per hectare)	20[b]	
K_2	The carrying capacity of the non-host. (per hectare)	15[b]	
r_1	The net density dependent growth rate for the deer mouse, $b_1 - d_1$. (per month)	3.1496×10^{-1} [a]	
b_1	The density dependent birth rate for the deer mouse. (per month)	0.315[a]	
μ	The disease induced mortality rate (per month)	0.085[a]	
d_1	The death rate of the deer mouse. (per month)	3.66×10^{-5} [a]	
q_1	The interspecific pressure exerted by the non-host onto the deer mouse	0.2[b]	0.4[b]
a	The proportion of density dependence due to density dependence of the deer mouse in birth rates	0.614[a]	
r_2	The net density dependent growth rate for the non-host, $b_2 - d_2$. (per month)	3.9996×10^{-1} [b]	
b_2	The density dependent birth rate for the non-host. (per month)	0.4[b]	
d_2	The death rate of the non-host. (per month)	4.0×10^{-5} [b]	
q_2	The interspecific pressure exerted by the deer mouse onto the non-host	0.3[b]	
γ	Initial disease transmission rate of the deer mouse without the influence of additional species. (hectare per month)	0.0130[b]	
δ	Proportional constant of the disease transmission rate with the non-host density. (hectare)	0[b]	0.0543[b]
Δt	Small time step (per month)	0.001	

[a]The values were based on [11]
[b]The values were based on [22].

nothing" event in the $[t, t + \Delta t)$ interval. Figure 1 shows the schematic diagram of the possible events of an individual.

Following the approach depicted in Fig. 1, we could then do the same for the susceptible and infected deer mouse, and the non-host. By expressing the equations in model (1) in per capita form, we could then equate the probabilities of the "die", "reproduce" and "do nothing" events for each susceptible, infected and non-host individuals. The per capita form of model (1) is as follow:

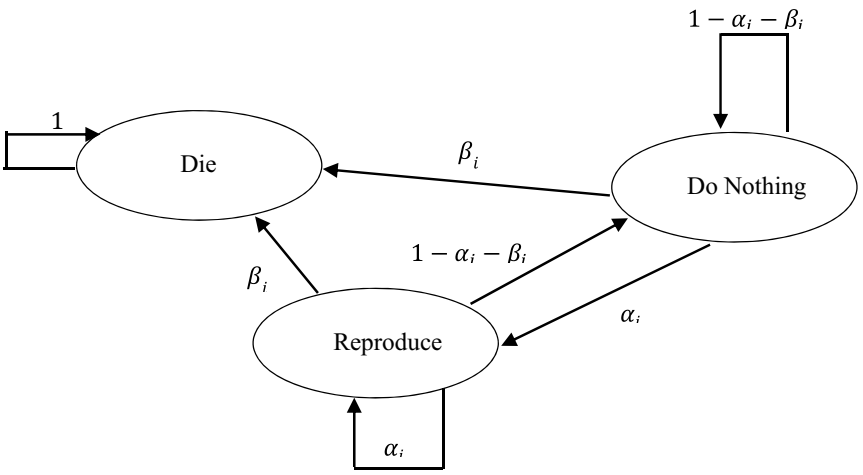

Fig. 1 Schematic diagram of the possible event path for an individual with α_i = probability of executing "reproduce" event and β_i = probability of executing "die" event in the $[t, t+\Delta t)$ interval, where $i = 1, 2, 3, \ldots$

$$\begin{aligned}
\tfrac{1}{S}\tfrac{\mathrm{d}S}{\mathrm{d}t} &= r_1 + \tfrac{I}{S}(b_1) - r_1\Big(\tfrac{N+q_1Z}{K_1}\Big)\Big[1-a\big(\tfrac{I}{S}\big)\Big] - \gamma(1+\delta Z)I \\
\tfrac{1}{I}\tfrac{\mathrm{d}I}{\mathrm{d}t} &= \gamma(1+\delta Z)S - \Big[\mu + d_1 + r_1(1-a)\Big(\tfrac{N+q_1Z}{K_1}\Big)\Big] \\
\tfrac{1}{Z}\tfrac{\mathrm{d}Z}{\mathrm{d}t} &= r_2\Big[1 - \tfrac{Z+q_2N}{K_2}\Big]
\end{aligned} \tag{2}$$

For abbreviation purpose, let us express the following transition probabilities:

1. $\alpha_i(S) = Pr\{\text{a } S \text{ reproduces a new } S \text{ in } [t, t+\Delta t)\}$
2. $\beta_i(S) = Pr\{\text{a single } S \text{ dies in } [t, t+\Delta t)\}$
3. $1-\alpha_i(S)-\beta_i(S) = Pr\{\text{a single } S \text{ does nothing in } [t, t+\Delta t)\}$
4. $\alpha_i(I) = Pr\{\text{a } I \text{ reproduces a new } I \text{ in } [t, t+\Delta t)\}$
5. $\beta_i(I) = Pr\{\text{a single } I \text{ dies in } [t, t+\Delta t)\}$
6. $1-\alpha_i(I)-\beta_i(I) = Pr\{\text{a single } I \text{ does nothing in } [t, t+\Delta t)\}$
7. $\alpha_i(Z) = Pr\{\text{a } Z \text{ reproduces a new } Z \text{ in } [t, t+\Delta t)\}$
8. $\beta_i(Z) = Pr\{\text{a single } Z \text{ dies in } [t, t+\Delta t)\}$
9. $1-\alpha_i(Z)-\beta_i(Z) = Pr\{\text{a single } Z \text{ does nothing in } [t, t+\Delta t)\}$

Thus, we can now equate the transition probabilities as below:

$$\alpha_i(S) = \left[r_1 + \frac{I}{S}(b_1)\right]\Delta t$$

$$\beta_i(S) = \left\{r_1\left(\frac{N+q_1Z}{K_1}\right)\left[1-a\left(\frac{I}{S}\right)\right] + \gamma(1+\delta Z)I\right\}\Delta t$$

$$1-\alpha_i(S)-\beta_i(S) = 1 - \left[r_1 + \frac{I}{S}(b_1)\right]\Delta t - \left\{r_1\left(\frac{N+q_1Z}{K_1}\right)\left[1-a\left(\frac{I}{S}\right)\right] + \gamma(1+\delta Z)I\right\}\Delta t$$

$$\alpha_i(I) = \left[\gamma(1+\delta Z)S\right]\Delta t$$

$$\beta_i(I) = \left[\mu + d_1 + r_1(1-a)\left(\frac{N+q_1 Z}{K_1}\right)\right]\Delta t$$

$$1-\alpha_i(I)-\beta_i(I) = 1-\left[\gamma(1+\delta Z)S\right]\Delta t - \left[\mu + d_1 + r_1(1-a)\left(\frac{N+q_1 Z}{K_1}\right)\right]\Delta t$$

$$\alpha_i(Z) = r_2\Delta t$$

$$\beta_i(Z) = r_2\left(\frac{Z+q_2 N}{K_2}\right)\Delta t$$

$$1-\alpha_i(Z)-\beta_i(Z) = 1-r_2\Delta t - r_2\left(\frac{Z+q_2 N}{K_2}\right)\Delta t$$

With these probabilities, we implemented the stochastic simulations for both case studies in GAMA version 1.8. It should be reminded that any software which supports individual-based modelling such as MATLAB can also be used for the simulations.

2.3 Implementation in GAMA 1.8

GAMA 1.8 is a free modelling and simulation development software [25]. It specializes in spatially explicit agent-based simulations. It is developed by several teams from France and Vietnam under the IRD/SU international research unit UMMISCO. It has been widely used by researchers to study problems related to epidemiology, urban planning, transportation, etc. The stochastic ABM was implemented through the steps depicted by the flowchart in Fig. 2.

The simulations were first done with the parameter values from Table 1. Then, we reran the simulations with different values for K_2 and q_1 to observe for any potential differences between case study 1 and 2. Due to our computer limitation, we only ran 100 simulations for each scenario.

3 Results

3.1 Simulations Based on Parameter Values from Table 1

The deterministic and stochastic results for both case study 1 and 2 are depicted in Fig. 3 respectively. By comparing the deterministic results from both cases, we could see that the non-host population (Z) stabilized at similar levels; whereas, the

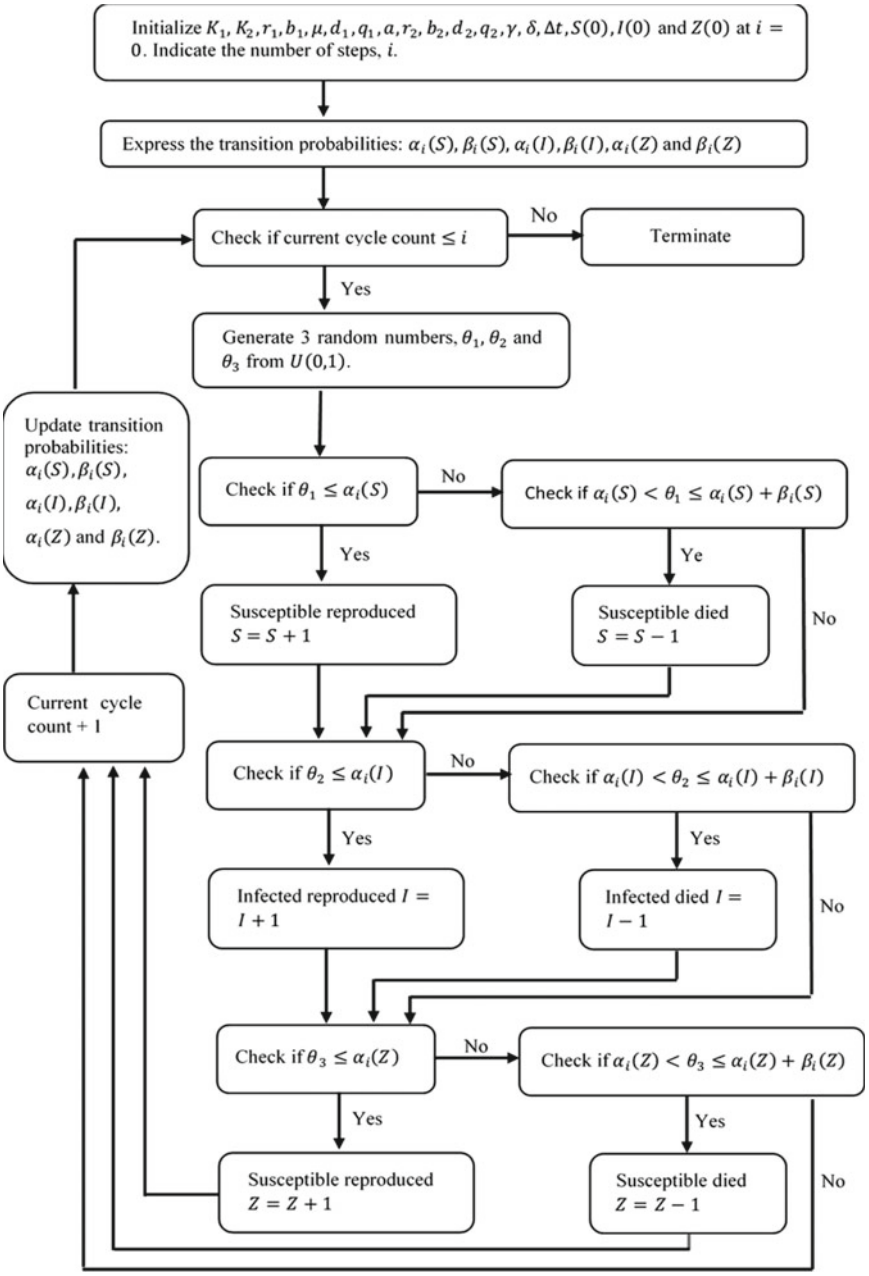

Fig. 2 Flowchart for simulating the stochastic ABM in GAMA

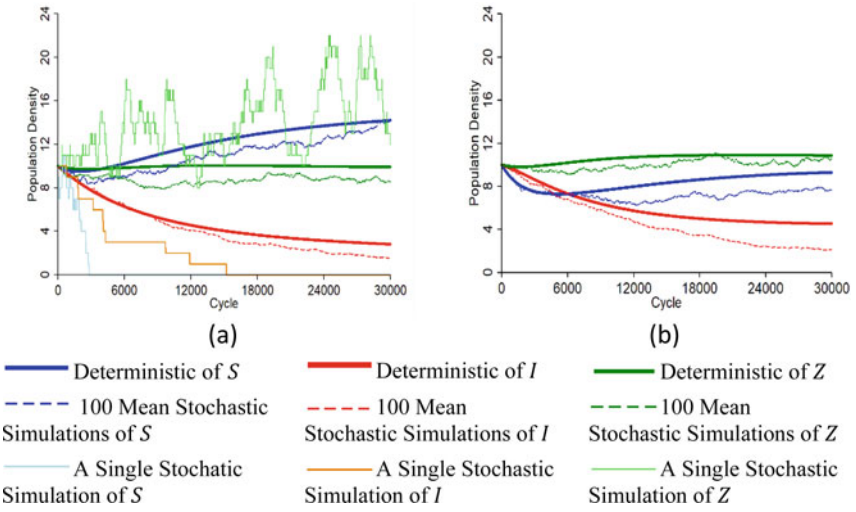

Fig. 3 Population density versus time for (a) case study 1 and (b) case study 2 with initial population $(S(0), I(0), Z(0)) = (10, 10, 10)$ and parameter values from Table 1. The (S^*, I^*, Z^*) densities at the end of the 30,000 cycles simulation were (a) Deterministic: (14.2, 2.8, 9.9); Stochastic: (14.4, 1.5, 8.6) (b) Deterministic: (9.3, 4.5, 10.9); Stochastic: (7.6, 2.1, 10.5)

susceptible deer mouse density was higher when the non-host acted as a dilution agent compared to it being an amplification agent. Despite the lower density in the susceptible, the population density for the infected deer mouse was higher for the case of amplification agent; and it is also interesting to note that the infected density was higher than the susceptible density before the simulation reached 6000 cycles. This showed that a dilution agent not only reduces the SNV transmission but also preserve the healthy deer mouse population; whereas, the inclusion of an amplification agent in the system was rather ineffective in reducing SNV transmission and the reduction of the SNV was at the cost of further reduction of the healthy deer mouse population.

However, the stochastic simulations showed a slightly different result in terms of the comparison between case study 1 (dilution agent) and 2 (amplification agent) for the infected deer mouse population. By comparing the stochastic results of the infected density in Fig. 3a and 3b, we could see the densities between both dilution agent and amplification agent cases were quite similar (stochastic infected deer mouse density at 30000th cycle for: study case 1 = 1.5; study case 2 = 2.1). This indicated that the role of the non-host might not be significant in reducing the SNV prevalence in small deer mouse population compared to the deterministic results. Furthermore, small gaps were observed between the deterministic and stochastic results. This phenomenon was due.

to the inherent characteristic of the extinction probability in the stochastic ABM model which matches the observation in [26].

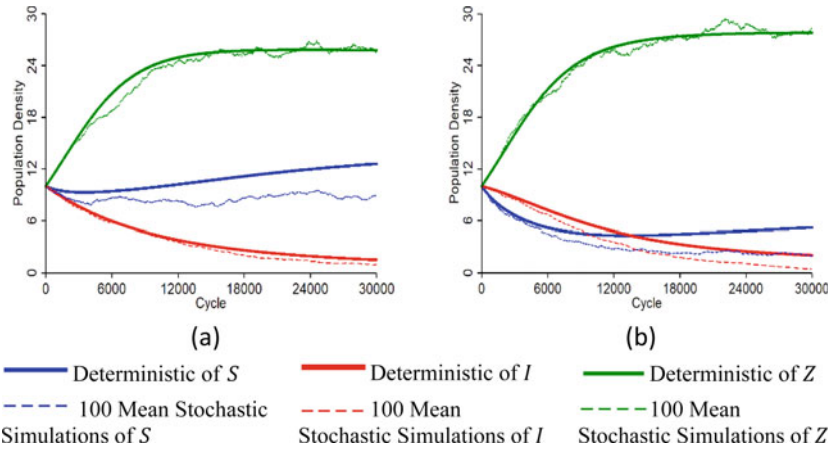

Fig. 4 Population density versus time for (a) case study 1 and (b) case study 2 with initial population $(S(0), I(0), z(0)) = (10, 10, 10)$ and parameter values from Table 1 except for $K_2 = 30$. The (S^*, I^*, Z^*) densities at the end of the 30,000 cycles simulation were (a) Deterministic: (12.6, 1.5, 25.8); Stochastic: (9.0, 1.0, 25.6) (b) Deterministic: (5.2, 2.1, 27.8); Stochastic: (2.0, 0.4, 28.2)

3.2 *Varying K_2 and q_1*

To investigate the impact of the carrying capacity and the interspecific strength of the non-host, we reran the simulations with an initial population of $(S(0), I(0), Z(0)) = (10, 10, 10)$ with $K_2 = 30$ (Fig. 4), and $(S(0), I(0), Z(0)) = (10, 10, 10)$ with $q_1 = 0.6$ (Fig. 5), while keeping the other parameter values as of Table 1. $K_2 = 30$ represents a much more favorable environment for the non-host while $q_1 = 0.6$ indicates the increase in aggressiveness of the non-host towards the deer mouse.

Based on Fig. 4 and 5, we could see that the intensity of the susceptible and infected densities for both cases in terms for both deterministic and stochastic results were generally lower compared to $K_2 = 15$, or $q_1 = 0.2$ (for case study 1) and $q_1 = 0.4$ (for case study 2). This showed that the carrying capacity and the interspecific strength of a non-host has a positive relationship in reducing the SNV prevalence irrespective of its role being an amplification or dilution agent. By comparing the total density of the deer mouse (N) between the varied K_2 and q_1 cases with the original parameter values in Table 1 at the end of the simulations, we could see there was a significant reduction in the deer mouse density; e.g. the N in Fig. 3(a) was at 17 (deterministic) and at 15.8 (stochastic) while the N in Fig. 4(a) was at 14.1 (deterministic) and at 10.0 (stochastic). Hence, we hypothesized that the reduction of the infected population was through the mechanism of decreasing the host density. Interestingly, the infected stochastic results for both dilution and amplification cases appeared to decrease to a similar level as opposed to the deterministic counterpart. The infected density at the end of the simulations for varied K_2 was 1.0 (Fig. 4(a)) and 0.4 (Fig. 4(b)); whereas it was 1.6 (Fig. 5(a)) and 2.2 (Fig. 5(b)). These observations were similar to the results

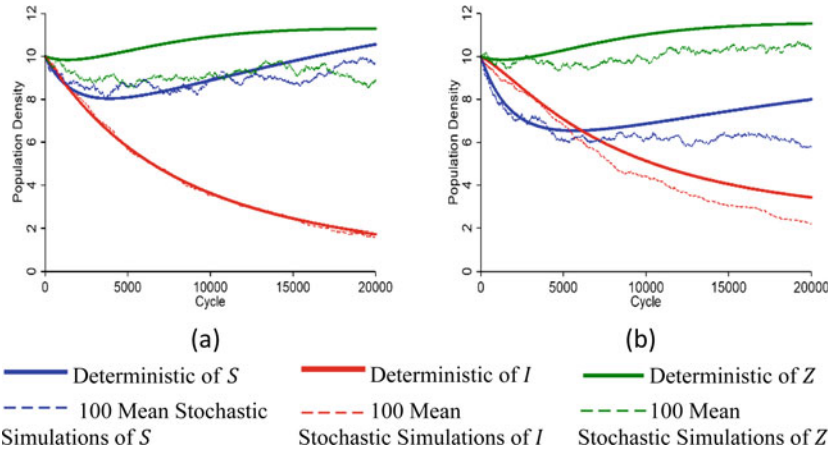

Fig. 5 Population density versus time for (a) case study 1 and (b) case study 2 with initial population $(S(0), I(0), z(0)) = (10, 10, 10)$ and parameter values from Table 1 except for $q_1 = 0.6$. The (S^*, I^*, Z^*) densities at the end of the 20,000 cycles simulation were (a) Deterministic: (10.6, 1.7, 11.3); Stochastic: (9.7, 1.6, 8.9) (b) Deterministic: (8.0, 3.4, 11.5); Stochastic: (5.8, 2.2, 10.5)

in Fig. 3, which led us to question the importance of investigating the community assemblage in terms of the biodiversity effect on SNV transmission in deer mouse especially when the population densities are small.

4 Discussion

From the above results, we managed to show and compare the impact of a non-host being a dilution agent and amplification agent towards the SNV transmission in deer mouse from deterministic and stochastic perspectives. Our deterministic model has shown that there were inherent differences in the infected density in the presence between a dilution agent and an amplification agent. The results showcased that a dilution agent performed much better in reducing the infected deer mouse density as well as preserving a much larger susceptible deer mouse population compared to an amplification agent. This finding aligns with the work of Milholland et al. [27] which highlights the identification of a species' role within the assemblage is as crucial as other factors (e.g. environmental conditions and species competition strength) to identify the biodiversity effect on disease transmission in an eco-epidemiological problem. However, such results were not observed in the case of stochastic ABM simulations. Our stochastic simulations revealed that the infected density levels were similar in the presence between a dilution agent and an amplification agent in small population. This suggests that the role of a non-host species may not have much of a difference between being a dilution or amplification agent

when the population density is small, as both manage to reduce the infected deer mouse to a similar density from a stochastic perspective. It may not be surprising to observe such discrepancies between our deterministic and stochastic model as Mohd et al. [28] managed to observe contrasting results on alternative stable states between their stochastic model and deterministic multiple species models. As such, our study managed to highlight the importance of ABM in eco-epidemiological studies as well as providing a methodological discussion regarding the usability of different simulation approaches, e.g. deterministic and stochastic ABM in order to produce robust observations of eco-epidemiological phenomenon under consideration.

Nonetheless, further investigation, especially comparison with experimental or field data, is required to confirm the discrepancies between the deterministic and stochastic results. If possible, we would suggest field researchers to conduct experimental studies at both small and large scales to investigate the transmission of SNV among deer mouse in the presence of a dilution agent, e.g. desert pocket mouse, in a close community. The results should then be compared to the experimental study conducted with an amplification agent, e.g. Merriam's kangaroo rat. The small and large scales studies are intended to represent the stochastic and deterministic simulations respectively. However, such experimental studies may be extremely difficult to perform. Alternatively, observational studies, which investigate the comparison of SNV transmission through role identification within a species assemblage at low and high densities population level, may be a more feasible approach. The challenges posed for such observational studies would then lie in the role identification played by the species on disease transmission, and the possible indirect effects caused by other species interactions within the assemblage. It should be noted the experimental and observational studies mentioned were just proposals based on our ideas. Opinions from experts and researchers on other viable study designs are much welcome to validate our findings.

Our study has successfully employed GAMA 1.8 as an alternative approach on simulating the biological system using the techniques of stochastic process and differential equations. This has contributed to the methodological discussions on the use of different modelling techniques and computer packages to examine the biological phenomena of interest. Similar to other platforms, GAMA provides similar flexibility to code the agent's characteristics and behavior according to the researcher's choice of techniques and assumptions. Some beginners may find GAMA a bit intimidating as it requires hard coding to simulate the ABM of their choice. They will need to browse through the tutorials to understand the available GAMA functions and their logic to construct their models of choice. Understanding these struggles, the GAMA developers and its community are actively providing skeleton codes along with examples in several fields to help with the learning process. All in all, GAMA is a good alternative platform for modelling biological systems and it is up to the researcher's discretion to choose whichever platform they are comfortable with.

There are a few limitations to this study. For starters, we only considered temporal modelling; but in reality, the rodents move around the environment. The lack of spatial considerations might cause us to miss some important observations, e.g. [3]

observed the presence of "refugia" for the SNV infected deer mouse in a spatio-temporal model. When the overall environmental condition is less favorable, the SNV infected deer mouse will find "refuge" in an area with better environment, which harbors the SNV and will then act as a source of transmission when the environmental condition has improved. We probably could follow the footstep of [17] in utilizing the agent-based modelling to incorporate not only the spatio-temporal feature but also to include detailed rodents' characteristics in a simulation. We also limited ourselves by only considering a non-host species. Other studies observed that other *Peromyscus* species [29] and desert woodrat [30] can serve as secondary reservoirs for the SNV. As pointed out by Ostfeld and Keesing [31], the quality in disease transmission by the secondary reservoir needs to be investigated as the presence of the secondary reservoir may not necessarily amplified the infection but may dilute it instead. Hence, it would be interesting to model for such scenario to better understand its dynamics and mechanisms.

5 Conclusion

This study managed to simulate and compare the effects of an amplification and dilution agent in regulating the SNV transmission in small deer mouse population from deterministic and stochastic perspectives. We implemented our stochastic ABM model in GAMA 1.8. Our deterministic results showed the effectiveness of a dilution agent comparatively to an amplification agent in reducing SNV. However, our stochastic results showed rather indifferent results between the dilution and amplification agent in small population density. Based on these contrasting results between the deterministic model and stochastic ABM, further investigations are required to better understand this discrepancy. As such, we would like to highlight the importance of utilizing ABM, especially in the eco-epidemiological field, as its usage might produce additional information which the deterministic models might fail to capture.

Acknowledgements The authors would like to thank the School of Mathematical Sciences and the Universiti Sains Malaysia (USM) for the support. Mohd Hafiz Mohd is supported by the USM Fundamental Research Grant Scheme (FRGS) No. 203/PMATHS/6711645.

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