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Research article

Limiting behaviors of a bi-virus model with two interaction factors

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Abstract: Traditional compartmental models describe the evolution of a virus over a population of nodes; one common model separates the population into compartments of susceptible, infected, and removed nodes. More complex bi-virus models describe this evolution for two competing viruses in the same population, having an additional parameter known as the virus interaction factor, which defines the effect one virus has on the rate of infection of another. Although these models are generally used in the context of infectious diseases, they could also describe the spread of ideas, or competing products in a market of consumers. In this paper, a new model was proposed that added separate interaction factors for each virus, differentiating the effects of viruses on one another. Adding this additional parameter will allow for more accurate interactions to be modeled and analyzed. A focus was placed on the limiting behavior of this model, an eventual end-state equilibrium where the number of nodes in each compartment remains constant. Relationships between the virus interaction factors and the strengths of the viruses on the limiting behavior were identified. Finally, a complete numerical solution to the model and a condition for real-valued limiting behaviors was calculated and tested against the simulation data.

Keywords: epidemiology; competition; co-existence; equilibrium

1. Introduction

The spread of viruses over a population network is an extremely important topic of study in today's society, especially after the events of the COVID-19 pandemic. In the field of epidemiology, compartmental models are commonly used to simulate the progression of a virus over a population space. There are many commonly used single-virus models, such as the SIR model: Consisting of susceptible, infected, and removed compartments. While modeling, nodes in populations travel between these compartments from one to another: Susceptible nodes are infected at a certain attack rate β, which are then removed at a certain healing rate δ. These three compartments can be modeled together using a system of ordinary differential equations (ODEs), which represent the change in the number of nodes in each compartment with respect to time. When analyzing the behavior of these compartmental models, after initial fluctuations, there is a limiting behavior of constant end-state values (a steady state) for each compartment [1]. This limiting behavior is demonstrated in the SIR model in Figure 1.

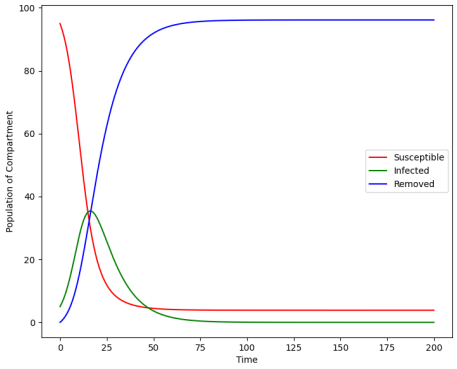

Figure 1. A classic susceptible-infected-removed (SIR) model run with parameters $\beta = 1/3$ and $\delta = 1/10$. This model demonstrates how all compartment populations eventually reach a constant end state.

However, SIR and other similar models fail to consider competition within the population. A type of model that adds another layer of complexity are bi-virus models, which model the progression of two viruses over the same population. One previously proposed bi-virus model is based off of the susceptible-infected-susceptible (SIS) single-virus model [1, 2], where infected nodes have no immunity and are susceptible to the virus afterwards (there is no recovered compartment). A new compartment I_{12} is created to represent nodes infected with both viruses [3]. In order to model competition between the viruses, the virus interaction factor ε is incorporated, which alters the attack rate of one virus on a node that has already been infected with the other virus. This model is named the susceptible-infected 1 or 2-susceptible ($SI_{1|2}S$) model. An important discovery with this model is a critical interaction factor value $\varepsilon_{critical}$, that represents an equilibrium point of coexistence between the two viruses [3, Theorem 1]:

$$\varepsilon_{critical} = \begin{cases} \frac{\sigma_1-\sigma_2}{\sigma_2(\sigma_1-1)}, & \text{if } \sigma_1+\sigma_2 \geq 2, \\ \frac{2(1+\sqrt{1-\sigma_1\sigma_2})}{\sigma_1\sigma_2}, & \text{if } \sigma_1+\sigma_2 < 2, \end{cases}$$

where $\sigma_1 \geq \sigma_2$, and σ represents the strength of a virus: the size of the population multiplied by its rate of infection (attack rate), divided by its healing rate (see Table 1).

Table 1. Definitions of variables.

Variable	Definition
N	Size of population = $S + I_1 + I_2 + I_{12}$
β_1, β_2	Attack rates of viruses 1 and 2
δ_1, δ_2	Healing rates of viruses 1 and 2
σ_1, σ_2	Strength of viruses 1 and 2 $= \frac{N\beta}{\delta}$
$\varepsilon_1, \varepsilon_2$	Interaction factors of viruses 1 and 2
I_1, I_2, I_{12}	# of nodes infected with either or both viruses
S	Number of susceptible nodes

I am motivated by the reality where competing viruses have interactions that are individualized in their impact on the opposing virus's attack rate; as opposed to the $SI_{1|2}S$ model previously cited where the viruses exhibit the same effect on each other. Therefore, a new model is proposed that incorporates two virus interaction factor values, one for each virus. For example, this will differentiate the effect of having COVID-19 on catching the flu and the effect of having flu on catching COVID-19. This would allow for the ability to model more realistic interactions, expanding outside the scope of epidemiology in applications such as products in a market or political opinions over a population [4]. This model is then analyzed to determine different limiting behaviors based on initial parameters.

2. Methods

2.1. The model

The proposed model with two interaction factors is represented by the following system of ODEs:

$$\frac{dI_1}{dt} = \beta_1 S(I_1 + I_{12}) + \delta_2 I_{12} - \delta_1 I_1 - \varepsilon_2\beta_2 I_1(I_2 + I_{12}), \quad (2.1)$$

$$\frac{dI_2}{dt} = \beta_2 S(I_2 + I_{12}) + \delta_1 I_{12} - \delta_2 I_2 - \varepsilon_1\beta_1 I_2(I_1 + I_{12}), \quad (2.2)$$

$$\frac{dI_{12}}{dt} = \varepsilon_1\beta_1 I_2(I_1 + I_{12}) + \varepsilon_2\beta_2 I_1(I_2 + I_{12}) - (\delta_1 + \delta_2)I_{12}. \quad (2.3)$$

In this model, the size of the population N stays constant, which implies

$$\frac{dS}{dt} = -\frac{dI_1}{dt} - \frac{dI_2}{dt} - \frac{dI_{12}}{dt}. \quad (2.4)$$

As illustrated in Figure 2, susceptible nodes move into infected compartments at the attack rate β, and move back to the susceptible compartment at the healing rate δ.

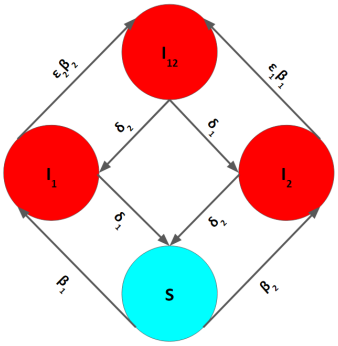

Figure 2. Visual depiction of the compartments in the model, with arrows representing the direction of movement between compartments and the variables that define the rate of movement.

Nodes infected with one virus also move into the I_{12} compartment at a rate defined by the interaction factor ε multiplied by the original attack rate of the other virus. Nodes infected with both viruses also move back into single-virus compartments with their respective healing rate.

2.2. Restrictions on domain

It is important to consider the positivity and boundedness [5] of the proposed model in order for it to have a physically relevant meaning, i.e., ensuring all values will stay positive and bounded within the population. The work is presented under the following assumptions, where $i_1 = \frac{I_1}{N}$, $i_2 = \frac{I_2}{N}$, $i_{12} = \frac{I_{12}}{N}$:

(1) $i_1, i_2, i_{12}, (1 - i_1 - i_2 - i_{12}) \in [0, 1]$
The initial fraction of infected and susceptible nodes are between 0 and 1.

(2) $\beta_1, \beta_2, \delta_1, \delta_2 \geq 0$
All attack rates and healing rates are nonnegative.

Consider a time t where $i_1 = 0$. Under Assumption 2, Eq (2.1) now implies that $\frac{dI_1}{dt} \geq 0$. The same applies to i_2 and i_{12}. If $i_1 = 1$ at time t, then under Assumption 2 and Eq (2.1), $\frac{dI_1}{dt} \leq 0$. The same applies to i_2 and i_{12}.

Under Assumption 1 and the above arguments, we can conclude $i_1, i_2, i_{12}, i_1 + i_2 + i_{12} \in [0, 1]$ for all $t \geq 0$ and that the set

$$D = \{(i_1, i_2, i_{12}) \mid i_1 \geq 0, i_2 \geq 0, i_{12} \geq 0, i_1 + i_2 + i_{12} \leq 1\} \quad (2.5)$$

is positively invariant in the context of the system of ODEs. The analysis of the model will be carried out with these assumptions and therefore exclusively within domain D.

2.3. Stability analysis

The stability of the model during a steady state must be confirmed before analyzing its limiting behaviors. In a steady state

$$\frac{dI_1}{dt}, \frac{dI_2}{dt}, \frac{dI_{12}}{dt} = 0, \quad (2.6)$$

which implies

$$\frac{dS}{dt} = 0. \quad (2.7)$$

2.3.1. Stability with respect to time

Consider the derivative of Eq (2.1) with respect to time:

$$\begin{aligned}\frac{d^2I_1}{dt^2} =& \beta_1 S\left(\frac{dI_1}{dt} + \frac{dI_{12}}{dt}\right) + \beta_1 \frac{dS}{dt}(I_1 + I_{12}) + \delta_2 \frac{dI_{12}}{dt} - \delta_1 \frac{dI_1}{dt} \\ & - \varepsilon_2 \beta_2 \frac{dI_1}{dt}(I_1 + I_{12}) - \varepsilon_2 \beta_2 I_1 \left(\frac{dI_2}{dt} + \frac{dI_{12}}{dt}\right). \quad (2.8)\end{aligned}$$

Given Eqs (2.6) and (2.7), $\frac{d^2I_1}{dt^2} = 0$. As time elapses, the derivatives of the compartments are not effected, demonstrating stability across the dimension of time. The same applies to the other compartments in the model.

2.3.2. Stability with respect to compartments

Let

$$I_1'(t) \equiv \frac{dI_1}{dt}. \quad (2.9)$$

Consider the partial derivative of (2.9) with respect to I_1

$$\begin{aligned}\frac{\partial I_1'(t)}{\partial I_1} =& \beta_1 S + \beta_1 \frac{\partial S}{\partial I_1}(I_1 + I_{12}) \\ & - \delta_1 - \varepsilon_2 \beta_2 (I_2 + I_{12}). \quad (2.10)\end{aligned}$$

Substituting from Eq (2.6) and $\frac{\partial S}{\partial I_1} = -1$

$$\begin{aligned}\frac{\partial I_1'(t)}{\partial I_1} =& \beta_1 S - \beta_1 (I_1 + I_{12}) - \delta_1 - \frac{\beta_1 S (I_1 + I_{12}) + \delta_2 I_{12} - \delta_1 I_1}{I_1} \\ =& -\beta_1 (I_1 + I_{12}) - \frac{\beta_1 S I_{12} + \delta_2 I_{12}}{I_1}. \quad (2.11)\end{aligned}$$

Under Assumptions 1 and 2, $\frac{\partial I_1'(t)}{\partial I_1} \leq 0$. Any fluctuations in I_1 will result in

$$I_1'(t) = \frac{dI_1}{dt}$$

being a force that pulls I_1 back to the stable state, demonstrating stability. The same applies to the other compartments in the model.

2.4. Goals

This model is simulated *in silico* by integrating the system of ODEs along a unitless timescale (code can be found at: `https://github.com/benz5460/bi-virus-model`). The procedure is split into two parts with different goals:

(1) Observations of the effect of varying virus interaction factors ε_1 and ε_2 on the number of nodes infected with each virus at its end behavior.

(2) A numerical method to calculate the end behavior of the system using the initial parameters.

Throughout the simulations, the end behavior is measured at time $t = 5000$. This was empirically determined to be a reasonable stopping point that was far into the end behavior of all tests. Initial compartment values of $I_1 = 50$, $I_2 = 50$, and $I_{12} = 0$ were used, although it is observed that initial values do not influence end state behavior.

3. Results and discussion

3.1. Observations

When running simulations on this model, there are three possible end states: Both viruses survive, one virus survives while the other dies out, or both viruses die out. It is found that the strengths σ_1 and σ_2 have a large effect on the end behaviors of this model when varying the virus interaction factors ε_1 and ε_2. Two conditions based on strength values are investigated by graphing the effect of changes in virus interaction factor, on end state values of nodes infected with virus 1 ($I_1 + I_{12}$) and virus 2 ($I_2 + I_{12}$).

3.1.1. Condition 1: $\sigma_1 + \sigma_2 \geq 2$

When the strengths of the two viruses have a sum greater than or equal to two, then

$$\varepsilon_{critical} = \frac{\sigma_1 - \sigma_2}{\sigma_2(\sigma_1 - 1)}$$

in the single interaction factor model. The stronger virus will be able to dominate the weaker until ε_2 is high enough to ensure survival through collaboration. In Figures 3 and 4, example simulations are shown where the weaker virus will die out unless its virus interaction factor ε_2 is greater than or equal to the aforementioned $\varepsilon_{critical}$ value. This indicates that within this condition, the model behaves in a similar way to if there were only one virus interaction factor.

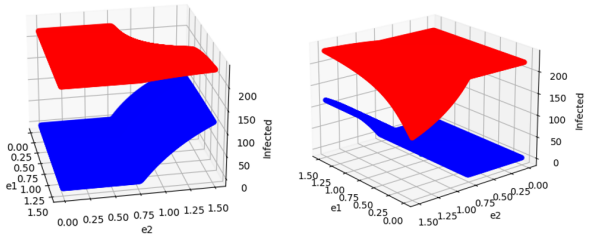

Figure 3. Simulations run with parameters: $N = 300$, $\beta_1 = 0.005$, $\delta_1 = 0.4$, $\beta_2 = 0.0002$, $\delta_2 = 0.05$.$\varepsilon_{critical} = 0.773$. Axes e1 and e2 represent varying virus interaction factors, and the end-state population of infected nodes for each virus is given by the Infected axis. The location of the fold up seen in the weaker (blue) virus at $\varepsilon_{critical}$.

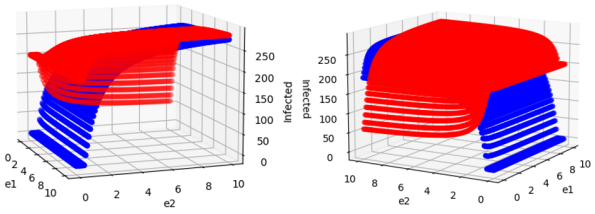

Figure 4. Simulations run with parameters: $N = 300$, $\beta_1 = 0.002$, $\delta_1 = 0.2$, $\beta_2 = 0.001$, $\delta_2 = 0.2$. The stronger virus will be overpowered by the weaker if its interaction factor is much lower, as seen with the red virus dropping at low values of ε_1.

3.1.2. Condition 2: $\sigma_1 + \sigma_2 < 2$

Within this condition, either both viruses have strengths less than 1, or one virus has a strength greater than 1. In the first case, both viruses do not have enough strength to survive on their own, and collaboration is required for survival. This results in a graph that shows a relationship between interaction factors that act as a threshold between the flat region of extinction and raised region of survival (see Figure 5).

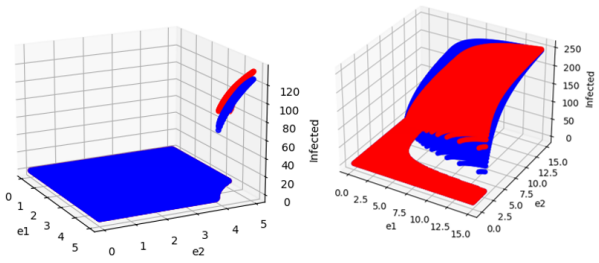

Figure 5. Simulations run with parameters: $N = 300$, $\beta_1 = 0.0006$, $\delta_1 = 0.2$, $\beta_2 = 0.0008$, $\delta_2 = 0.3$. The flat plane in both images describes both viruses dying out (0 infected), until the virus interaction factors are large enough. The image on the right extends the range of values for ε_1 and ε_2 shown.

In the case where one virus has a strength greater than 1, a drastically different behavior is observed. The stronger virus

will dominate until ε_2 reaches a threshold where both viruses will jump to increased numbers of infected nodes. This threshold is observed to be near the $\varepsilon_{critical}$ values but not exact, through the observation of several simulations (see Figure 6).

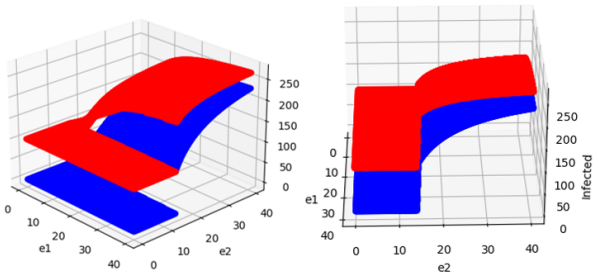

Figure 6. Simulations run with parameters: $N = 300$, $\beta_1 = 0.00025$, $\delta_1 = 0.05$, $\beta_2 = 0.0003$, $\delta_2 = 0.5$, where one virus has a strength greater than 1 and the sum of strengths is less than 2.

3.2. *Calculations for a numerical method*

An end state equilibrium is achieved when all three ODEs are equal to 0. With this, a concrete condition for real-valued, positive end state behavior is described, as well as a numerical method for calculating end state behavior given initial parameters

$$\beta_1 S(I_1 + I_{12}) + \delta_2 I_{12} - \delta_1 I_1 - \varepsilon_2\beta_2 I_1(I_2 + I_{12}) = 0, \quad (3.1)$$

$$\beta_2 S(I_2 + I_{12}) + \delta_1 I_{12} - \delta_2 I_1 - \varepsilon_1\beta_1 I_2(I_1 + I_{12}) = 0, \quad (3.2)$$

$$\varepsilon_1\beta_1 I_2(I_1 + I_{12}) + \varepsilon_2\beta_2 I_1(I_2 + I_{12}) - (\delta_1 + \delta_2)I_{12} = 0. \quad (3.3)$$

These equations are then manipulated into the following:

$$N\kappa_1\beta_1\left[1 - \kappa_1 - (1 - \varepsilon_1)\, i_2\right] = \delta_1\kappa_1, \quad (3.4)$$

$$N\kappa_2\beta_2\left[1 - \kappa_2 - (1 - \varepsilon_2)\, i_1\right] = \delta_2\kappa_2, \quad (3.5)$$

$$N\left(\varepsilon_1\beta_1\kappa_1 i_2 + \varepsilon_2\beta_2\kappa_2 i_1\right) = (\delta_1 + \delta_2)\, i_{12}, \quad (3.6)$$

where

$$\kappa_1 = \frac{I_1 + I_{12}}{N},\ \kappa_2 = \frac{I_2 + I_{12}}{N},\ i_1 = \frac{I_1}{N},\ i_2 = \frac{I_2}{N},\ \ i_{12} = \frac{I_{12}}{N}$$

Assuming positive, non-zero values of κ_1 and κ_2, and substituting

$$\frac{1}{\sigma} = \frac{\delta}{N\beta},\ i_1 = \kappa_1 - i_{12},\ i_2 = \kappa_2 - i_{12}$$

into the three equations:

$$1 - \kappa_1 - (1 - \varepsilon_1)\, i_2 = 1/\sigma_1, \quad (3.7)$$

$$1 - \kappa_2 - (1 - \varepsilon_2)\, i_1 = 1/\sigma_2, \quad (3.8)$$

$$\kappa_1\kappa_2\left(\varepsilon_1\sigma_1\delta_1 + \varepsilon_2\sigma_2\delta_2\right) = i_{12}(\delta_1 + \delta_2 + \varepsilon_1\sigma_1\delta_1\kappa_1 + \varepsilon_2\sigma_2\delta_2\kappa_2). \quad (3.9)$$

Equation (3.10) is a rearrangement of Eq (3.9), while Eq (3.11) is the rearrangement of

$$Eq(3.7) \times (1 - \varepsilon_2) - Eq(3.8) \times (1 - \varepsilon_1)\,.$$

Equation (3.12) is an expanded form of Eq (3.7), substituting $i_2 = \kappa_2 - i_{12}$

$$i_{12} = \frac{\kappa_1\kappa_2\left(\varepsilon_1\sigma_1\delta_1 + \varepsilon_2\sigma_2\delta_2\right)}{\delta_1 + \delta_2 + \varepsilon_1\sigma_1\delta_1\kappa_1 + \varepsilon_2\sigma_2\delta_2\kappa_2}, \quad (3.10)$$

$$\kappa_1 = \frac{(1 - \varepsilon_1)\varepsilon_2}{(1 - \varepsilon_2)\varepsilon_1}\kappa_2 + \frac{\varepsilon_1 - \varepsilon_2 - \frac{1-\varepsilon_2}{\sigma_1} + \frac{1-\varepsilon_1}{\sigma_2}}{\varepsilon_1(1 - \varepsilon_2)}, \quad (3.11)$$

$$1 - \kappa_1 - (1 - \varepsilon_1)\,(\kappa_2 - i_{12}) = 1/\sigma_1. \quad (3.12)$$

Substituting Eqs (3.10) and (3.11) into Eq (3.12) results in a quadratic equation in the form of:

$$a(\kappa_2)^2 + b(\kappa_2) + c = 0, \quad (3.13)$$

where:

$$a = -\delta_1\,(\varepsilon_2)^2\,(\sigma_1)^2\,(\sigma_2)^2 + \delta_1\varepsilon_1\,(\varepsilon_2)^2\,(\sigma_1)^2\,(\sigma_2)^2\ + \delta_2\varepsilon_1\,(\varepsilon_2 - 1)\,\varepsilon_2\sigma_1\,(\sigma_2)^3\,,$$

$$b = -2\delta_1\varepsilon_2\,(\sigma_1)^2\,\sigma_2 + 2\delta_1\varepsilon_1\varepsilon_2\,(\sigma_1)^2\,\sigma_2 + \delta_2\varepsilon_1\,(\varepsilon_2 - 1)\,\sigma_1\,(\sigma_2)^2 - \delta_1\varepsilon_1\,(\varepsilon_2 - 1)^2\,\sigma_1\,(\sigma_2)^2 - \delta_1\,(\varepsilon_2 - 1)\,\varepsilon_2\sigma_1\,(\sigma_2)^2 + \delta_2\,(\varepsilon_2 - 1)\,\varepsilon_2\sigma_1\,(\sigma_2)^2 + \delta_1\varepsilon_1\,(\varepsilon_2 - 1)\,\varepsilon_2\sigma_1\,(\sigma_2)^2 - \delta_1\varepsilon_1\varepsilon_2\,(\sigma_1)^2\,(\sigma_2)^2 + 2\delta_1\,(\varepsilon_2)^2\,(\sigma_1)^2\,(\sigma_2)^2 - \delta_1\varepsilon_1\,(\varepsilon_2)^2\,(\sigma_1)^2\,(\sigma_2)^2 - \delta_2\varepsilon_1\,(\varepsilon_2 - 1)\,\varepsilon_2\sigma_1\,(\sigma_2)^3,$$

$$c = -\delta_1\,(\sigma_1)^2 + \delta_1\varepsilon_1\,(\sigma_1)^2 - \delta_1(\varepsilon_2 - 1)\sigma_1\sigma_2 + \delta_2(\varepsilon_2 - 1)\sigma_1\sigma_2 + \delta_1\varepsilon_1(\varepsilon_2 - 1)\sigma_1\sigma_2 - \delta_1\varepsilon_1\,(\sigma_1)^2\,\sigma_2 + 2\delta_1\varepsilon_1\,(\sigma_1)^2\,\sigma_2 - \delta_1\varepsilon_1\varepsilon_2\,(\sigma_1)^2\,\sigma_2 + \delta_2(\varepsilon_2 - 1)^2\,(\sigma_2)^2 - \delta_1\varepsilon_1(\varepsilon_2 - 1)\sigma_1\,(\sigma_2)^2 + \delta_1(\varepsilon_2 - 1)\varepsilon_2\sigma_1\,(\sigma_2)^2 - \delta_2(\varepsilon_2 - 1)\varepsilon_2\sigma_1\,(\sigma_2)^2 + \delta_1\varepsilon_1\varepsilon_2\,(\sigma_1)^2\,(\sigma_2)^2\ - \delta_1(\varepsilon_2)^2\,(\sigma_1)^2\,(\sigma_2)^2\,.$$

Equation (3.13) can be used in conjunction with Eq (3.11) to calculate the end state behavior of both viruses given initial parameters $\delta_1, \delta_2, \varepsilon_1, \varepsilon_2, \sigma_1, \sigma_2$. This agrees with the earlier observation that initial compartment values do not influence the end behavior.

For κ_2 to be real-valued, the discriminant of the quadratic must be positive: $b^2 - 4ac \geq 0$. In addition, only accepting roots where $\kappa_2 \in [0, 1]$ will further constrain the condition. This condition describes a numerical constraint for real-valued end state conditions based on initial parameters.

The original $SI_{1|2}S$ model exists as a unique case in the new model where $\varepsilon = \varepsilon_1 = \varepsilon_2$. When this is true, the constraint can be reduced to the form:

$$\sigma_1\sigma_2\varepsilon^2 - 4\varepsilon + 4 \geq 0. \tag{3.14}$$

This agrees with the result found for the original model [3, Lemma 4], demonstrating the new model's validity.

3.3. Testing the numerical method

Results from the numerical method are compared to those from the simulations. To achieve this, parameter values from selected conditions in Section 3.1 are plugged into Eq (3.13). Parameters from Figure 3 are first tested: $\beta_1 = 0.005, \delta_1 = 0.4, \beta_2 = 0.0002, \delta_2 = 0.05, \sigma_1 = 3.75, \sigma_2 = 1.2$. Plugging this into Eq (3.13),

$$\begin{aligned}
&[0.324\varepsilon_1(\varepsilon_2 - 1) - 8.1\varepsilon_2^2 + 8.1\varepsilon_1\varepsilon_2^2]\kappa_2^2 + \\
&[0.27\varepsilon_1(\varepsilon_2 - 1) - 2.16\varepsilon_1(\varepsilon_2 - 1)^2 - 13.5\varepsilon_2 + 5.4\varepsilon_1\varepsilon_2 \\
&- 1.89(\varepsilon_2 - 1)\varepsilon_2 + 1.836\varepsilon_1(\varepsilon_2 - 1)\varepsilon_2 + 16.2\varepsilon_2^2 - 8.1\varepsilon_1\varepsilon_2^2]\kappa_2 \\
&- 5.625 - 1.125\varepsilon_1 - 1.575(\varepsilon_2 - 1) - 0.36\varepsilon_1(\varepsilon_2 - 1) \\
&+ 0.072(\varepsilon_2 - 1)^2 + 13.5\varepsilon_2 + 1.35\varepsilon_1\varepsilon_2 + 1.89(-1 + \varepsilon_2)\varepsilon_2 \\
&- 8.1\varepsilon_2^2 = 0.
\end{aligned} \tag{3.15}$$

Given ε_1 and the condition $\kappa_2 \in [0, 1]$, the corresponding lower bound for ε_2 is found from Eq (3.15) (e.g., let $\varepsilon_1 = 1$, then $\varepsilon_2 \geq 0.772727$). This is repeated for a range of ε_1 values and results are compared to the original simulation.

The same procedure is repeated with the parameters from Figure 5, with the results shown in Figure 7. As shown in Figures 7 and 8, the calculated threshold values for mutual survival of both viruses of ε_2 match closely with simulation data for both observation conditions of virus strength values outlined in Section 3.1.

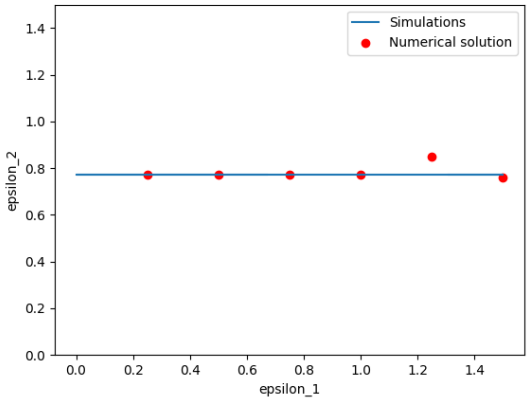

Figure 7. Using parameter values from Figure 3 and selected $\varepsilon_1 = 0.25, 0.5, 0.75, 1.0, 1.25, 1.5$; comparison of the threshold value of virus interaction factor and ε_2 (where both viruses will survive) between simulation data from the ODEs and the numerical method.

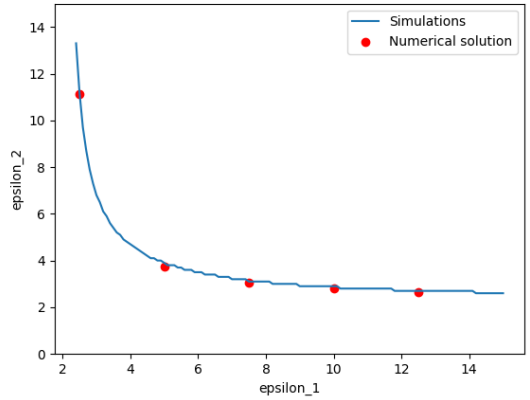

Figure 8. Similar to Figure 7, now using parameters from Figure 5 and selected $\varepsilon_1 = 2.5, 5.0, 7.5, 10.0, 12.5$.

4. Conclusions

A new bi-virus model is proposed and analyzed using a system of ODEs, which introduce unique virus interaction factors for each virus. This allows the impact of one virus on another to be fine-tuned and model more realistic

interactions. With the analysis, the following is concluded:

- End behaviors in this model are somewhat reliant on the original $\varepsilon_{critical}$ value. This is seen most clearly in the case where $\sigma_1 + \sigma_2 \geq 2$, while a less concrete dependence is seen when $\sigma_1 + \sigma_2 < 2$.
- When $\sigma_1+\sigma_2 < 2$ and both are less than 1, the threshold of survival for the interaction factors are related in the form of a curve.
- A numerical solution of the model's end behavior is found in the form of a quadratic equation using initial parameters. The solution is tested against simulation data and found to be accurate.
- Similarly, a numerical constraint for real-valued solutions is found, of which the original model's constraint is uncovered as a unique case of the new model.
- Future work can be done to identify the exact relation between the interaction factors when $\sigma_1 + \sigma_2 < 2$ and both are less than 1. Additionally, fitting the model to data of epidemics or sales of products can demonstrate the model's flexibility and applicability in the real world.
- Other interesting possibilities include models with greater than 2 viruses, with interactions determined through a virus interaction matrix, or one that incorporates the effect of interaction on healing rates.

Use of AI tools declaration

The author declares he has not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The author declares that he has no conflict of interest in this paper.

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