Two strain competition: *Trypanosoma cruzi*

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Abstract

Chagas disease is a tropical parasitic disease that affects Latin America. The parasite causing Chagas, *Trypanosoma cruzi*, is transmitted by an insect vector of the subfamily Triatominae. Chagas is uncommon in the United States, but is becoming more common in the southern part of the country. This may be due to a more virulent strain type of the parasite moving northward from Mexico and invading the less virulent, native strain type. A region in southern Texas where there is a mixing of two *Triatoma* species is modeled with two different modeling frameworks. A mathematical deterministic model is created to describe the interactions between one host and one vector and determine the outcome of an invasion of a non-native strain type into the region. A stochastic, agent based model is created to determine the effect space and randomness may have on the interactions. Within the models, three modes of parasite transmission are considered to account for the different characteristics of each strain type.

We vary the horizontal transmission ability of the invasive strain and run simulations for an equivalent time period of 30000 days (82 years). We find that the horizontal transmission potential of the invasive strain must be about 1.5 times as great as the other in order for over 50% of the runs to end with the invasive strain dominating. However, in the ODE model, the horizontal transmission of the invasive need only be 1.056 times that of the other strain. We also determine from the ODE model that considering three modes of transmission and no migration of vectors into the region being modeled, it is impossible for the two strains to coexist.

1 Introduction

Chagas disease is a parasitic disease affecting an enormous number of people throughout Latin America. The WHO estimated that before successful control programs were established in the Americas in the 1980’s, there were 16 to 18 million infections of the parasite causing Chagas, *Trypanosoma cruzi*. As much as 20% of Bolivia was infected in the eighties and 4.5% of rural Brazil was infected in 1970’s. An estimated 23,000 deaths occur annually from Chagas. Only acute respiratory infections, diarrhoeal disease, and AIDS produce a greater socioeconomic burden than Chagas disease in Latin America [12].
Chagas is a vector borne disease transmitted by blood feeding assassin bugs of the subfamily Triatominae. The typical prey of the insects include opossums, raccoons, woodrats, dogs, armadillos, birds, bats and many others. The parasite is typically transmitted by an infected *Triatoma* biting its prey and then defecating near the wound. If the feces enter the wound, the host may become infected by the parasite. The *Triatoma* are infected by ingesting blood containing the parasite. The parasite resides in the insect’s digestive tract where it remains for the remainder of the insect’s life. This is why the insect must defecate near the wound in order to infect the host; just biting is not enough. Two other ways a host may become infected are by eating an infected insect or by inheriting the infection congenitally. Amongst humans, blood transfusions play a significant role in parasite transmission in urban areas, responsible for approximately 10% of all cases [12].

*Trypanosoma cruzi* is not limited to Latin America. It is seen in alarmingly high rates among sylvatic hosts in the southern United States. For example, 104 out of 221 (47%) raccoons caught in S. Carolina and Georgia were infected with *Trypanosoma cruzi* [16]. There are six distinct classes of strains of the parasite circulating throughout Latin America and the United States; *T. cruzi* type I and type IIa through e. Only type I and IIa are prevalent in the United States. It is believed that the strains in the United States are less virulent than those existing in Latin America [10] and probably even non-chagasic. By virulence here, we are referring to the severity of chagasic symptoms which it induces in humans. In the southern part of Texas, there exists predominantly two species of *Triatoma* vector: *Triatoma sanguisuga* and *Triatoma gerstaeckeri*. *Triatoma gerstaeckeri* is generally associated with the northern part of Mexico while *Triatoma sanguisuga* is associated with the southern United States. It is possible that *Triatoma gerstaeckeri* is bringing with it a Chagasic strain of *T. cruzi* from Mexico. As evidence of this, three dogs died of Chagas disease at a residence in San Benito, Texas, 2003. Upon investigation, a severe infestation of *Triatoma gerstaeckeri* was found at the residence and many of the insects were determined to contain the parasite *T. cruzi* [2].

There has been limited research on modeling *T. cruzi* in sylvatic hosts. Kribs-Zaleta [8] investigated the significance of *T. cruzi* transmission through vector consumption. This is a single strain study which explores contact saturation in vector consumption. In this model a predator-prey structure is superimposed on a vector-host infection cycle in a system of ordinary differential equations. Cherif et. al. [4] created a model considering two
strains of *T. cruzi* in a population of one host and one vector. A two patch model is used to describe the migration of the infection in two regions. It was found that without migration between the two patches, the strains could not coexist, however when migration was included, coexistence was possible. A model considering non-sylvatic hosts was created by Devillers et. al. [6]. This model is an agent-based model which considers transmission of two strains of the parasite among humans. It was found that a reservoir host was needed as an additional agent in the model in order to obtain realistic simulation results of the prevalence of the two strains. This is in agreement with research in that it is generally sylvatic hosts which maintain the infection. In our model, we use an agent-based model for only a sylvatic population.

This paper is concerned with determining the capability of a virulent strain of *T. cruzi* to dominate in a region endemic with a less virulent strain of the infection. The region we will be modeling is in southern Texas where there is coexistence of *T. sanguisuga* and *T. gerstaeckeri*. The assumption is that *T. gerstaeckeri* is bringing a virulent strain of *T. cruzi* into a region dominated by a less virulent, non-chagasic strain [2]. A system of non-linear differential equations is used to model the transmission of *T. cruzi* between hosts and vectors. Three modes of disease transmission are considered along with natural death and births. A stochastic agent based model is also created to consider the effect randomness and space may have on the system. The agent based model focuses on matching precisely the rates in the differential equation model so that the two can be compared. It does not, however, consider particular host or vector behavior in the spatial domain.

## 2 Model

### 2.1 Model Considerations and Assumptions

As discussed previously, the models will consider a specific region in south Texas where *T. sanguisuga* and *T. gerstaeckeri* coexist. It will be assumed that a virulent, chagasic strain, hereafter referred to as strain I, is invading this region endemic with a less virulent non-chagasic strain, henceforth referred to as strain II. Two important hosts in this area are raccoons and woodrats. We consider only woodrats in the models because of their strong association with *Triatoma gerstaeckeri* [10], the vector that may be bringing strain I from Mexico. We model three different modes of transmission
in the models based on the evolutionary advantages of each strain. Vertical transmission is the process of giving birth to infected young, horizontal transmission is passage of the parasite through vector feeding, and oral transmission occurs when a host becomes infected by ingesting a vector. Hall et al. states, “Because of the increased potential for coevolution with placental mammals, the T. cruzi strains cycling in the southeastern United States may have adapted mechanisms to facilitate vertical transmission. Our own preliminary experiments in mice have confirmed that a Type IIa strain of T. cruzi isolated from one of the ring-tailed lemurs is twice as likely to be vertically transferred as a Type I isolate [7].” We consider it to be the case that strain II is better adapted to vertical transmission and treat strain II in the models with a much higher vertical transmission rate. Because of a less aggressive nature of establishing itself within a host, we consider that strain II is less adept in transmission through insect blood-feeding. We also include transmission to host through vector consumption. In this form of infection, very little data has been found on transmission probability and no studies to our knowledge have been done on strain difference in oral transmission [10]. We allow for the transmission rates to be different in the development and analysis of the model, however, we treat these two rates as equal in the simulation and numerical analysis. Because Chagas is an endemic disease and is known to have been around for very long periods of time, we consider both birth and death in the models. The death considered is only due to natural mortality and not to disease induced death.

2.2 ODE Model

Our deterministic model is a system of non-linear ordinary differential equations that describes the interactions and vital dynamics of a population of hosts and vectors. The susceptible vectors and hosts will be denoted by \( S_v \) and \( S_h \) respectively, the infected vectors and hosts will be denoted by \( I_{vj} \) and \( I_{hj} \) respectively where \( j \) represents the strain with which the individual is infected.

A flow chart for the compartmental model is given in Figure 1. In this model, individuals can enter the class of susceptible vectors by birth and they can leave by death or getting infected. Death is caused by predation of hosts or natural. By infection they will enter class of infected with strain I or II. Infected vectors leave their respective classes only by death due to predation by hosts or by natural death. Hosts enter the class of susceptible
hosts only by birth. They leave when they get infected with strain I or II due to oral or horizontal transmissions. They also leave the class due to natural death. Hosts can also enter an infected class with strain I or II by vertical transmission upon birth by an infected mother. They leave the class only by natural death.

The non-linear ordinary differential equations system is:

\[
\frac{dS_v}{dt} = r_v N_v \left(1 - \frac{N_v}{K_v}\right) - \beta_{v1} \frac{I_{h1}}{N_h} S_v - \beta_{v2} \frac{I_{h2}}{N_h} S_v - \mu_v S_v - \left(\frac{HN_h}{N_v}\right) S_v (1)
\]

\[
\frac{dS_h}{dt} = r_h (S_h + (1 - \gamma_1) I_{h1} + (1 - \gamma_2) I_{h2}) \left(1 - \frac{N_h}{K_h}\right) - \beta_{h1} \frac{I_{v1}}{N_h} S_h - \rho_1 \left(\frac{HN_h}{N_v}\right) \frac{I_{v1}}{N_h} S_h - \beta_{h2} \frac{I_{v2}}{N_h} S_h - \rho_2 \left(\frac{HN_h}{N_v}\right) \frac{I_{v2}}{N_h} S_h - \mu_h S_h (2)
\]

\[
\frac{dI_{v1}}{dt} = \beta_{v1} \frac{I_{h1}}{N_h} S_v - \mu_v I_{v1} - \left(\frac{HN_h}{N_v}\right) I_{v1} (3)
\]

\[
\frac{dI_{v2}}{dt} = \beta_{v2} \frac{I_{h2}}{N_h} S_v - \mu_v I_{v2} - \left(\frac{HN_h}{N_v}\right) I_{v2} (4)
\]

\[
\frac{dI_{h1}}{dt} = \beta_{h1} \frac{I_{v1}}{N_h} S_h + \rho_1 \left(\frac{HN_h}{N_v}\right) \frac{I_{v1}}{N_h} S_h + \gamma_1 r_h I_{h1} \left(1 - \frac{N_h}{K_h}\right) - \mu_h I_{h1} (5)
\]

\[
\frac{dI_{h2}}{dt} = \beta_{h2} \frac{I_{v2}}{N_h} S_h + \rho_2 \left(\frac{HN_h}{N_v}\right) \frac{I_{v2}}{N_h} S_h + \gamma_2 r_h I_{h2} \left(1 - \frac{N_h}{K_h}\right) - \mu_h I_{h2} (6)
\]

where

\[
N_v = S_v + I_{v1} + I_{v2}
\]

\[
N_h = S_h + I_{h1} + I_{h2}
\]

In the model above, we assume logistic growth rate for both vectors and hosts. We also assume host saturation, that is, there are many hosts compared to the number of vectors meaning that the ratio of vector to host is low. As a consequence, vectors have plenty to eat. We will show why this implies that $\beta_{hj}$, for $j = 1, 2$, has units $\frac{\text{hosts}}{(\text{vector})(\text{time})}$. The assumption of host saturation allows us to consider $\beta_{hj}$, for $j = 1, 2$, constant because the number of bites per vector per time can be considered as constant since there is no competition for food between vectors. Let $\lambda_v$ be the number of bites per vector per day. $\lambda_v N_v$ is the total number of bites per day for the vector
population. As a result of these bites, we will have new infected vectors and new infected hosts. With this, we will derive the terms $\beta_{hj} \frac{I_{vj}}{N_h} S_h$ and $\beta_{vj} \frac{I_{hv}}{N_v} S_v$, for $j = 1, 2$, in the equations above. Since we are concerned with the number of new infected hosts, we have to consider only the bites on susceptible hosts. We have $\lambda_v N_v \frac{S_h}{N_h}$, but these bites have to come from infected vectors with strain $j$, so we have $\lambda_v N_v \frac{S_h}{N_h} \frac{I_{vj}}{N_v} \pi_h$ where $\pi_h$ is the number of infected hosts per bite given that an infected vector bit a susceptible host. We see that the expression simplifies to $\pi_h \lambda_v \frac{I_{vj}}{N_v} S_h$, and $\pi_h \lambda_v$ has units of $\frac{\text{infected hosts}}{\text{vector}} \frac{\text{bites}}{\text{day}}$, so we conclude that $\beta_{hj} = \pi_h \lambda_v$; this completes the derivation of the term $\beta_{hj} \frac{I_{vj}}{N_h} S_h$.

In a similar way, the number of infected vectors per day will be $\lambda_v N_v \frac{S_h}{N_h} \frac{I_{hv}}{N_v} \pi_v$ where $\lambda_v N_v$ is the total number of bites per day for the vector population. We multiply by $\frac{S_v}{N_v} \frac{I_{hv}}{N_v}$ to consider only the cases in which a susceptible vector bites an infected host, then we multiply by $\pi_v$, the number of infected vectors per bite, given that a susceptible vector bit an infected host. $\lambda_v N_v \frac{S_h}{N_h} \frac{I_{hv}}{N_v} = \pi_v \lambda_v \frac{I_{hv}}{N_v} S_v$ and now $\pi_v \lambda_v$ has units of $\frac{\text{infected vectors}}{\text{vector}} \frac{\text{bites}}{\text{day}}$, which is equivalent to the units of $\frac{1}{\text{time}}$. We conclude that $\beta_{vj} = \pi_v \lambda_v$; this completes the derivation of the term $\beta_{vj} \frac{I_{hv}}{N_v} S_v$.

The term $\frac{HN_h}{N_v} S_v$ in (1) represents the number of susceptible vectors that die per unit of time by host predation. Let $H$ be the number of vectors eaten per host per day, so $HN_h$ represents the total number of vectors that get eaten per unit of time by the host population, however, we are only interested in susceptible vectors, so we multiply by $\frac{S_v}{N_v}$ to obtain $HN_h \frac{S_v}{N_v} = \frac{HN_h}{N_v} S_v$.

We can also derive the term $\rho_1 \left( \frac{HN_h}{N_v} \right) \frac{I_{h1}}{N_h} S_h$ in (5). This term represents the number of susceptible hosts per unit of time that become infected with strain I. As we said, $HN_h$ is the number of vectors eaten by the population of hosts per unit of time. We are interested in the case where a susceptible host becomes infected with strain I, so we must consider only the case in which a susceptible host eats an infected vector with strain I. We must multiply $HN_h$ by $\frac{S_h}{N_h} \left( \frac{I_{h1}}{N_v} \right)$ and consider that not always when a susceptible host eats an infected vector will the host become infected. To account for this, we
multiply \( HN_h \) by \( \rho_1 \), the number of hosts per vector that become infected given that a susceptible host ate an infected vector with strain I, to obtain
\[
HN_h \left( \frac{S_h}{N_h} \right) \left( \frac{I_v}{N_v} \right) \rho_1 = \rho_1 \left( \frac{HN_h}{N_v} \right) l_{vl} \frac{S_h}{N_v}.
\]

The terms in (5) and (6) with the form \( \gamma_j r_h I_{hj} \left( 1 - \frac{N_h}{K_h} \right) \) represent the number of hosts per unit of time that obtain strain \( j \), for \( j = 1, 2 \), by vertical transmission. \( \gamma_j \) represents the probability that an infected host with strain \( j \) produces an infected child upon giving birth.

The parameters \( \mu_v \) and \( \mu_h \) represent the natural death rates of vectors and hosts respectively. In our model we will assume that the hosts do not die from any of the two strains. There is no evidence that either host dies from the disease in nature.

### 2.3 Agent Based Model

The agent based model is created to be directly comparable to the ODE model, meaning the rates at which events take place should be derived from the ODE model. The ABM model serves as a discrete, stochastic version of the ODE’s which also considers movement and space. However, the model disregards host and vector behavior; all agents move randomly and in the same way in the program. Space is considered only to observe a crowding out effect; if an infected vector is surrounded by only infected hosts, the vector has no potential to infect the hosts. Movement among the agents tends to disperse these events, but also tends to create more of them in time. Three events may take place in the model: infection, birth, or death. The probability of an event happening in a given time step are taken from the rates in the ODE model.

Generally in ABM modeling, interactions between agents only occur when agents are within a certain distance of each other; however, we want to avoid the difficulty in model comparisons that can occur when modeling this way. Instead we poll a set of the agents who may have an interaction, and then find the closest agent for the that first agent to interact with. In this way, the problem of interaction based on a interacting distance is eliminated. We have two types of agents in this model: vectors and hosts. In the model we assume an area of one hectare and calculate the population size accordingly; see Table 2. We take the time step in which events can occur as one day. Each vector and host have a property indicating the infection state of the agent. The vectors can only become infected by biting infected hosts. The
hosts become infected by being bitten by an infected vector, giving birth to infected young, and by eating infected vectors. Each of these events may happen at every time step, here days, but only if a certain probability is satisfied. The probabilities are taken from the rates in the ODE model. To show how these rates are changed to probabilities in discrete time steps consider the following very simple ODE system for recovering infectives:

\[
\begin{align*}
\dot{I} &= -\gamma I \\
\dot{R} &= \gamma I
\end{align*}
\]

The solution for \( I(t) \) is given by

\[ I(t) = I(0)e^{-\gamma t} \]

So, after a time step \( T \) we have

\[ I(0)e^{-\gamma T} \]

individuals remaining in the infective class and

\[ I(0)(1 - e^{-\gamma T}) \]

individuals in the recovered class. Thus the proportion of recovered individuals in time step \( T \) is given by \( 1 - e^{-\gamma T} \). This can also be interpreted as the probability that an individual recovers in time step \( T \). The particular probabilities for our system will be given in the explanation of each process in the following paragraphs.

**Horizontal infection to vectors:** At each time step, each vector is asked to find the closest host; if the host is infected, then the vector will become infected with a certain probability which is dependent on strain type. If the host is uninfected, nothing will happen. Let’s assume the vector has found a strain I infected host. The ODE for susceptible vectors leaving due to horizontal infection of strain I is shown below.

\[
\dot{S}_v = -\beta_{v1} \frac{I_{h1}}{N_h} S_v
\]

In the ABM, the proportion of infected hosts, \( \frac{I_{h1}}{N_h} \), is dealt with directly in the layout of the model. When the vector finds the closest host, the probability that this host is infected is \( \frac{I_{h1}}{N_h} \). \( S_v \) is handled by polling only the susceptible
vectors. Thus the proportion \( \frac{I_{v1}}{N_h} \) in the rate can be ignored, yielding the probability
\[
1 - e^{-\beta_{v1} T},
\]
for a vector to be infected with strain I by horizontal transmission in a time step \( T \). The infection probability for strain II is found in exactly the same manner and is
\[
1 - e^{-\beta_{v2} T}.
\]

**Horizontal infection to hosts:** To infect the hosts, each susceptible host is asked to find the closest vector. If that vector is infected there is a probability that the host obtains the same infection. The ODE describing host infection by horizontal transmission of strain I only, is
\[
\dot{S}_h = -\beta_{h1} \frac{I_{v1}}{N_h} N_v S_h,
\]
which can be rewritten
\[
\dot{S}_h = -\left( \beta_{h1} \frac{N_v}{N_h} \right) \frac{I_{v1}}{N_v} S_h.
\]
Again, we have the proportion \( \frac{I_{v1}}{N_v} \) handled implicitly in the model, however, \( \frac{N_v}{N_h} \) must be included in the probability to account for host saturation just as is done in the ODE’s. Note that the units in \( \beta_{h1} \frac{N_v}{N_h} \) are 1/time, as is desired. The probability for a host to become infected after finding an infected vector with strain I and II respectively are:
\[
1 - e^{-\left( \beta_{h1} \frac{N_v}{N_h} \right) T},
\]
\[
1 - e^{-\left( \beta_{h2} \frac{N_v}{N_h} \right) T}.
\]

**Vector consumption:** To decide if a vector is consumed, each vector is polled, and if a probability is satisfied, the vector dies. The probability is derived from the ODE describing the rate of decrease of the vector population due to consumption. The ODE is
\[
\dot{N}_v = -\left( H \frac{N_h}{N_v} \right) N_v.
\]
Note that we write the rate as \( \left( H \frac{N_h}{N_v} \right) \) in order to make the units \( \frac{1}{time} \), see Table 3. Even though the number of vectors consumed is only dependent on
the number of hosts, we must poll the vectors to get the appropriate units in the rate. The probability that a vector dies in a time step, derived from this equation, is

\[ 1 - e^{-H \frac{N_v}{N_v} T}. \]

If this vector that is consumed happens to be infected, the vector will find the closest host and infect that host with a probability \( \rho \), but only if that host is susceptible. \( \rho \) is an experimentally measured fraction of hosts that become infected after consuming an infected vector. See Table 1.

**Vector and host birth:** Vector and host birth is assumed to be logistic. Consider the logistic birth ODE for vectors:

\[
\dot{N}_v = r_v N_v \left(1 - \frac{N_v}{K_v}\right).
\]

Each vector or host respectively will give birth to one other vector or host in a time step with the probability

\[ 1 - e^{-r_v \left(1 - \frac{N_v}{K_v}\right)T}, \]

and

\[ 1 - e^{-r_h \left(1 - \frac{N_h}{K_h}\right)T}. \]

Note that \( N_v \) is never greater than \( K_v \) as long as the initial set up of the system creates population lower than the carrying capacity of the region. The hosts have the added complication of possibly giving birth to infected young. If the host that gives birth is infected, the young will be infected with the probability \( \gamma_1 \) or \( \gamma_2 \) depending on the strain. \( \gamma_1 \) and \( \gamma_2 \) are simply the proportion of young born infected from infected mothers; see Table 1.

**Vector and host death:** Vectors and hosts are assumed to die at a linear rate, shown in the following ODE:

\[
\dot{N} = -\mu N.
\]

The probability that a given agent dies in one time step is given by

\[ 1 - e^{-\mu T}, \]

where \( \mu \) will vary depending on whether agent being observed is a host or vector.
3 Analysis

In this section we analyze the ODE model presented previously. The analysis of this model will be carried out under the assumption that the birth rates are greater than the death rates: \( r_v > \mu_v, \ r_h > \mu_h \). It can be shown that the behavior of our model is asymptotic by showing that the two populations, \( N_v \) and \( N_h \), are asymptotically constant. The ODE’s describing the vital dynamics of the two populations are given by:

\[
\frac{dN_v}{dt} = r_v N_v \left( 1 - \frac{N_v}{K_v} \right) - \mu_v N_v - H N_h \quad (7)
\]

\[
\frac{dN_h}{dt} = r_h N_h \left( 1 - \frac{N_h}{K_h} \right) - \mu_h N_h \quad (8)
\]

It can be shown that under our assumptions, the population of hosts is asymptotically constant, by proving that all solutions of (8) converge to \( \frac{K_h (r_h - \mu_h)}{r_h} \), under the model’s assumptions. For details, see appendix. A theorem of Thieme [13] and [14] suggests that we can analyze (7) while treating \( N_h \) as its limiting value as \( t \) approaches infinity:

\[
\frac{dN_v}{dt} = r_v N_v \left( 1 - \frac{N_v}{K_v} \right) - \mu_v N_v - H \left( \frac{K_h (r_h - \mu_h)}{r_h} \right). \quad (9)
\]

This can be rewritten as an equation with logistic growth and constant yield harvesting:

\[
\frac{dN_v}{dt} = (r_v - \mu_v) N_v \left( 1 - \frac{N_v}{K_v (r_v - \mu_v)} \right) - H \left( \frac{K_h (r_h - \mu_h)}{r_h} \right). \quad (10)
\]

For details of this type of differential equation, please refer to [3]. The equilibria of (10) can be written as

\[
N_{e1} = \frac{A - \sqrt{A^2 - \frac{4BA}{D}}}{2}
\]

\[
N_{e2} = \frac{A + \sqrt{A^2 - \frac{4BA}{D}}}{2}
\]

where \( A := \frac{K_v (r_v - \mu_v)}{r_v} \), \( B := H \left( \frac{K_h (r_h - \mu_h)}{r_h} \right) \), \( D := r_v - \mu_v \) and \( B_c := \frac{DA}{4} \). We assume that the population \( N_v \) is sufficiently large and the harvesting
is small enough such that the population does not go extinct, meaning that \( N_v > N_{v1} \) and \( B < B_c \). Under the last assumption, \( N_{v1} \) is unstable and \( N_{v2} \) is asymptotically stable. Considering both of the assumptions, the population, under any initial conditions, will tend to \( N_{v2} \).

We have justified why we can consider both populations constant in the model. The respective values for \( N_v \) and \( N_h \) will be considered as

\[
N_v^* = \frac{A + \sqrt{A^2 - 4BA}}{2} \\
N_h^* = \frac{K_h (r_h - \mu_h)}{r_h}
\]

### 3.1 Disease Free Equilibrium

We will write the equilibrium points of the system as an ordered vector with the following form: \((S_v, S_h, I_{v1}, I_{v2}, I_{h1}, I_{h2})\) where the state variables will be fixed.

The first step to finding the disease free equilibrium is to let \( I_{v1} = I_{v2} = I_{h1} = I_{h2} = 0 \), and solve for \( S_v \) and \( S_h \). At the disease free equilibrium, \( S_v = N_v \) and \( S_h = N_h \) and equations (3-6) are equivalent to zero. We now solve for \( S_v \) and \( S_h \) considering \( S_v = N_v \) and \( S_h = N_h \). The 2-dimensional system consisting of (1) and (2) becomes

\[
\frac{dN_v}{dt} = r_v N_v \left( 1 - \frac{N_v}{K_v} \right) - \mu_v N_v - H N_h \\
\frac{dN_h}{dt} = r_h N_h \left( 1 - \frac{N_h}{K_h} \right) - \mu_h N_h
\]

which we have already analyzed, using (7) and (8). We have the following disease free equilibrium point:

\[
E_0 := \left( \frac{A + \sqrt{A^2 - 4BA}}{2}, \frac{K_h (r_h - \mu_h)}{r_h}, 0, 0, 0, 0 \right)
\]
3.2 Basic Reproductive Number

To calculate the basic reproductive number ($R_0$) we use the second generation operator approach [15]. Before we show $R_0$, we will define the next terms:

\[ \tilde{\mu}_v := \mu_v + H \frac{N_h}{N_v} \]  
\[ \tilde{\beta}_h := \beta_h \frac{N_v}{N_h} + \rho_1 H \]  
\[ R_{h1} := \sqrt{\frac{\beta_{h1} \beta_{v1}}{\mu_h \tilde{\mu}_v}} \]  
\[ \tilde{\beta}_{h2} := \beta_{h2} \frac{N_v}{N_h} + \rho_2 H \]  
\[ R_{h2} := \sqrt{\frac{\beta_{h2} \beta_{v2}}{\mu_h \tilde{\mu}_v}} \]  

The terms $\tilde{\mu}_v$, $\tilde{\beta}_h$, and $\tilde{\beta}_{h2}$ are effective rates with units $\frac{1}{\text{time}}$ that only consider horizontal transmission. Oral transmission is included in the terms since it is specific type of horizontal transmission. $R_{h1}$ and $R_{h2}$ are the basic reproductive numbers that come from the system only considering horizontal transmission.

The basic reproductive number is $\max\{R_1, R_2\}$, where

\[ R_1 = \frac{1}{2} \left( \gamma_1 + \sqrt{(\gamma_1)^2 + 4(R_{h1})^2} \right) , \]  
\[ R_2 = \frac{1}{2} \left( \gamma_2 + \sqrt{(\gamma_2)^2 + 4(R_{h2})^2} \right) . \]  

$R_0 < 1$ implies that $E_0$ is asymptotically stable. If $R_0 > 1$, then $E_0$ will be unstable. Also note that if $\gamma_1 = 0$ and $\gamma_2 = 0$, then $R_1 = R_{h1}$ and $R_2 = R_{h2}$.

3.3 Endemic Equilibria

The endemic equilibrium for strain 1 ($E_1$), is:

\[ (N_v^* - I_{v1}^*, N_h^* - I_{h1}^*, I_{v1}^*, 0, I_{h1}^*, 0) \]
where

\[ I_{v1}^* = \frac{\tilde{\mu}_v \mu_h ((R_1)^2 - 1) N_v}{\beta_{h1} (\tilde{\mu}_v + \beta_{v1})}, \quad (18) \]

\[ I_{h1}^* = \frac{\tilde{\mu}_v \mu_h ((R_1)^2 - 1) N_h}{\beta_{v1} (\tilde{\beta}_{h1} + \mu_h (1 - \gamma_1))}. \quad (19) \]

By (18) and (19), it can be seen that \( E_1 \) will be biologically significant (will be positive) if and only if \( R_1 > 1 \).

The endemic equilibrium for strain II (\( E_2 \)) is:

\( (N_v^* - I_{v2}^*, N_h^* - I_{h2}^*, 0, I_{v2}^*, 0, I_{h2}^*) \)

where

\[ I_{v2}^* = \frac{\tilde{\mu}_v \mu_h ((R_2)^2 - 1) N_v}{\beta_{h2} (\tilde{\mu}_v + \beta_{v2})}, \quad (20) \]

\[ I_{h2}^* = \frac{\tilde{\mu}_v \mu_h ((R_2)^2 - 1) N_h}{\beta_{v2} (\tilde{\beta}_{h2} + \mu_h (1 - \gamma_2))}. \quad (21) \]

By (20) and (21), it can be seen that \( E_2 \) will be biologically significant (will be positive) if and only if \( R_2 > 1 \).

### 3.4 Invasive Reproductive Numbers

The invasive reproductive numbers describe the ability of a particular strain to invade a population that is endemic with other strain. If the reproductive number is greater than one, the invading strain has the ability to become endemic in a population that is already endemic with another strain. The invasive reproductive numbers for strain I and II are \( \tilde{R}_1 \) and \( \tilde{R}_2 \). These are obtained using the second generation operator approach [15] as well and are given by

\[ \tilde{R}_1 = \frac{1}{2} \left( \gamma_1 + \sqrt{(\gamma_1)^2 + 4(1 - \gamma_2) \frac{(R_{h1})^2}{(R_{h2})^2}} \right), \quad (22) \]

\[ \tilde{R}_2 = \frac{1}{2} \left( \gamma_2 + \sqrt{(\gamma_2)^2 + 4(1 - \gamma_1) \frac{(R_{h2})^2}{(R_{h1})^2}} \right). \quad (23) \]
By observing the basic and invasive reproductive numbers we can prove that $\tilde{R}_j < R_j$ iff $\frac{1 - \gamma_j}{(R_{hi})^2} < 1$ (for $j \neq i$). With some algebra, it is straightforward to show that $R_i > 1$ iff $\frac{1 - \gamma_i}{(R_{hi})^2} < 1$ and conclude that $R_j > 1$ iff $\tilde{R}_i < R_i$. These results biologically mean that the ability of one strain to invade a population where the other is already endemic, is less than the strain’s ability to invade a completely susceptible population. This result is expected since the strains considered are in competition with each other; only one parasite strain can persist in a single host or vector. In the case of mutualism, we would expect that $R_j > 1$ iff $\tilde{R}_i > R_i$.

If $\tilde{R}_1 > 1$ and $\tilde{R}_2 > 1$ then there is the possibility of coexistence within the population. However, if $\tilde{R}_1 > 1$ implies $\tilde{R}_2 < 1$ and $\tilde{R}_2 > 1$ implies $\tilde{R}_1 < 1$ then there is no possibility that coexistence can occur.

**Proposition 1**: $\tilde{R}_j > 1 \iff \tilde{R}_i < 1$, where $j \neq i$.

**Proof:**

($\implies$) Suppose $\tilde{R}_j > 1$. So we have that

$$\frac{1}{2} \left( \gamma_j + \sqrt{(\gamma_j)^2 + 4(1 - \gamma_i)\left(\frac{R_{hi}}{R_{hi}}\right)^2} \right) > 1$$

$$\left(2 - \gamma_j\right)^2 - (\gamma_j)^2 < 4(1 - \gamma_i)\left(\frac{R_{hi}}{R_{hi}}\right)^2$$

$$4 - 4\gamma_j < 4(1 - \gamma_i)\left(\frac{R_{hi}}{R_{hi}}\right)^2$$

$$4(1 - \gamma_j) < 4(1 - \gamma_i)\left(\frac{R_{hi}}{R_{hi}}\right)^2$$

$$(1 - \gamma_j)\left(\frac{R_{hi}}{R_{hi}}\right)^2 < 1 - \gamma_i$$
We know that,
\[ \tilde{R}_i = \frac{1}{2} \left( \gamma_i + \sqrt{\gamma_i^2 + 4(1 - \gamma_i)\left(\frac{R_{hi}}{R_{hj}}\right)^2} \right) \]

Since \( 0 \leq \gamma_i \leq 1 \), see Table 3, we are assured that \( 1 - \gamma_i \geq 0 \) and \( \gamma_i^2 + 4(1 - \gamma_i) \geq 0 \). This allows the following step:

\[ \tilde{R}_i < \frac{1}{2} \left( \gamma_i + \sqrt{\gamma_i^2 + 4(1 - \gamma_i)} \right) \]
\[ = \frac{1}{2} \left( \gamma_i + \sqrt{\gamma_i^2 - 4\gamma_i + 4} \right) \]
\[ = \frac{1}{2} \left( \gamma_i + \sqrt{\gamma_i - 2} \right)^2 \]
\[ = \frac{1}{2} (\gamma_i + |\gamma_i - 2|) \]
\[ = \frac{1}{2} (\gamma_i + (-(\gamma_i - 2))) \]
\[ = 1 \]

Therefore \( \tilde{R}_j > 1 \Rightarrow \tilde{R}_i < 1 \).

\((\Leftarrow)\) If one supposes that \( \tilde{R}_i < 1 \) then it can be proven that \( \tilde{R}_j > 1 \). We will not prove it since it does not contribute to showing the impossibility for coexistence in our model. ■

It can be proven in a similar manner that \( \tilde{R}_j = 1 \) if and only if \( \tilde{R}_i = 1 \). Proposition 1 suggests that in our deterministic model there is no possibility for coexistence, which is surprising since vertical transmission is included and coexistence between the two strains in the population is expected. In other words you do not expect competitive exclusion.

We see that in the bifurcation graph, Figure 2, there are three distinct regions. In region I, the disease free equilibrium is stable and no biologically relevant endemic equilibriums exists. In region III \( E_2 \) exists and is positive and in region II, \( E_1 \) exists and is positive. At the boundary of region I and II a transcritical bifurcation is exhibited where switching from region I into II, the endemic equilibrium \( E_1 \), becomes positive and stable, and the disease free equilibrium becomes unstable. At the boundary of region I and III,
there exists another transcritical bifurcation where switching from region I into III, \( E_2 \) becomes positive and stable, and \( E_0 \) and becomes unstable. At the boundary of region II and III, there is a degenerate transcritical bifurcation where \( E_1 \) and \( E_2 \) trade stability. We can see from the graph, and guess intuitively based on the aforementioned competitive exclusion, that if \( R_1 > R_2 \geq 1 \), then strain I becomes endemic in the population and strain II dies out and vice versa if \( R_2 > R_1 \geq 1 \).

4 Numerical Results

4.1 Parameter Estimation

All of our parameter estimations come from Kribs-Zaleta’s (2009) exhaustive study on contact process saturation in sylvatic hosts. From this paper, we have compiled a list of parameters and infection rates for woodrats and Triatoma gerstaeckeri in Texas shown in Table 1. Since no direct estimates for the horizontal transmission rates \( \beta_v \) and \( \beta_h \) exist, we estimate them by using \( T. \ cruzi \) prevalence data from Texas, see Table 2, and assume that this data reflects an equilibrium state. This is a reasonable assumption since \( T. \ cruzi \) is endemic in this region and has been for a long period of time. We disregard strain variation in the region and assume there is only one strain in the region. The estimation method is shown below.

\[
\begin{align*}
\dot{I}_h &= \beta_h \frac{I_v}{N_h} S_h + \rho H \frac{I_v}{N_v} S_h + \gamma I_h r_h \left(1 - \frac{N_h}{K_h}\right) - \mu_h I_h = 0 \\
\dot{I}_v &= \beta_v \frac{I_h}{N_v} S_v - \mu_v I_v - H \frac{I_v}{N_v} N_h = 0
\end{align*}
\]

We can then solve for \( \beta_v \) and \( \beta_h \):

\[
\begin{align*}
\beta_h &= \frac{\mu_h I_h - \rho H \frac{I_v}{N_v} S_h - \gamma I_h r_h \left(1 - \frac{N_h}{K_h}\right) \mu_h I_h}{\frac{I_v}{N_h} S_h} \\
\beta_v &= \frac{\mu_v I_v + H \frac{I_v}{N_v} N_h}{\frac{I_h}{N_h} S_v}
\end{align*}
\]

Parameter values, infection ratios, and population values from tables 2 and 1 are substituted into the equation. For example, from Table 2, we know
the infection rate for vector is 45%. We can make the calculation for one hectare, so \( N_v = 316 \). From these two pieces of information we see that \( I_v = .45 \cdot 316 = 142.2 \). The same can be done for the hosts. The vertical transmission \( \gamma \) is calculated as the average of \( \gamma_1 \) and \( \gamma_2 \). These values are assumed to be the transmission terms, \( \beta_{v2} \) and \( \beta_{h2} \), associated with strain II since this is the strain prevalent in the region the data was taken from. This is a reasonable estimate since strain II is the dominant strain in this region. The values calculated are shown in Table 1. Since the other values, \( \beta_{v1} \) and \( \beta_{h1} \), are to have higher horizontal transmission ability, they will be assumed to be equal to \( \beta_{v2} \) and \( \beta_{h2} \) multiplied by a value \( q \) that is greater than or equal to one:

\[
\begin{align*}
\beta_{v1} &= q \beta_{v2} \\
\beta_{h1} &= q \beta_{h2}
\end{align*}
\text{ where } q \geq 1.
\]

4.2 ODE Approximations

In the ODE model, for a particular value of \( q \), one behavior is always observed because this is a deterministic model. At a threshold value for \( q \), strain II dies and strain I dominates. We calculate this value by finding for what value of \( q \), \( \tilde{R}_1 = 1 \). This value is \( q = 1.056 \). We plot the ODE’s while varying \( q \) from 1 to 2 in order to determine how quickly one strain dominates; see Figure 4. We also evaluate \( R_1 \), \( R_2 \), and \( \tilde{R}_1 \) for every \( q \) value we use; see Table 4.

4.3 Simulations

In the simulations, we experiment with the value of \( q \) in order to determine when strain I will dominate. In the ABM model, since it is a stochastic model, strain I may dominate in one run and strain II in another; see Figure 3. This is very different from the ODE model, see section 4.2. We run 30 simulations for \( q \) ranging between 1 and 2 and average the results. We also record how many times a particular strain dominates and how long it takes for the other strain to die completely. We run the simulation a total of 30000 time steps, equivalent to 30000 days or 82 years. We want to observe an invasion of strain I, so we assume a 5% infection rate of strain I in the vector population. The initial set up of the system 5% infection of strain I in vectors, 40% infection of strain II in vectors, and 33% infection of strain II in hosts; see Table 2.
For all values of $q$, both strains on occasion are the dominant strain. For example, when $q = 1$, two times out of the 30 runs, strain one dominates despite it being a less effective strain; see tables 4 and 5. The reason we can say that strain I is the less effective strain when $q = 1$ is because the horizontal transmission probability for both strains are the same, and strain II has the advantage with a greater vertical transmission probability. The stochasticity of the system allows for either strain to dominate for a particular $q$ value; strain I may be lucky in infecting many hosts while strain II is unlucky in having hosts and vectors infected with strain II die. On occasion both strains would survive the entire 30000 time steps; this happened a total of 2 times out of 330 (0.6%). More frequently, though, both strains would go extinct; this happened a total of 14 times out 330 (4.24%). Of course the most usual outcome was for one or the other of the two strains to dominate.

When the graphs of all runs for a particular $q$ value are averaged, a plot more similar to the ODE plots is seen; see figures 3 and 4. There is, however, one major difference between the averaged plots and the deterministic plots. In the deterministic plots, when $q \geq 1$, strain II goes to zero while strain I approaches an endemic equilibrium. In the averaged ABM plots, it appears that strain II persists, as if there is coexistence; this is however not the correct interpretation. In almost all simulations, one or the other of the strains dominate. There are only 4 runs out of 330 where neither of the strains die. The appearance of strain II not going to zero comes from the occasional runs where strain I dies before it can take hold, and then strain II persists the remainder of simulation creating a persistent non-zero average.

As $q$ was increased from 1 to 2, the number of times strain I dominated went from 2 times to 22 times out of 30; see Table 5. With $q = 1$, strain I dominates 7% of the time. If $q$ is increased to only $q = 1.1$, strain I dominates 20% of the time. This dramatic increase can be explained by observing the reproductive numbers for strain I and strain II and in particular the invasive reproductive number for strain I in Table 4. We see that when $q = 1$, $R_2$ is only slightly larger than $R_1$. Vertical transmission only plays a small role in the strengths of the respective reproductive numbers. When $q = 1.1$, $R_2$ surpasses $R_1$, and more importantly, $\tilde{R}_1 \geq 1$, thus strain II has a harder time defeating the invasion of strain I. Compare this to the deterministic plots, and we see that as long as the invasive reproductive number for strain I is greater than one, then strain one will dominate. This is exactly as is to be expected.

Observing Table 5, we see that as $q$ increases, the time it takes both
strains to dominate decreases. This can be explained by noting that as $q$ increases the invasive reproductive number for strain I increases (see Table 4) and therefore the number of new infectives per infective individual with strain I increases. As a result, the speed of infection spread will increase as well. This means that the time it takes for strain I to dominate decreases. The time it takes for strain II to dominate, when it does dominate, decreases as well since usually the reason it dominates is because strain I dies out before it can take hold. This happens quickly when it happens.

5 Conclusions and Future Directions

In conclusion, the difference in the ODE versus the ABM model are quite significant. In the ABM model, strain I does not reach over 50% domination in the runs until $q$ is about 1.5. In the ODE's, $q$ need only be 1.056 and strain I will dominate. This can be explained by observing how one infected individual affects the different systems. In the ODE model, one infected individual will immediately begin infecting the susceptible population at a certain rate. In the stochastic model, one infected vector may die before it can spread its infection. The stochastic model better interprets reality in this respect; interactions happen discretely and probabilistically in nature. Another interesting finding is that it appears that the adaption to vertical transmission for strain II is not a significant advantage in preventing an invasion of strain I. This can be seen in the threshold value of $q = 1.056$ for strain I to dominate.

It is apparent from the results in Table 4 that the model does not consider enough biology to give strain II an ability to compete with strain I. Also the finding of competitive exclusion in the model suggests more must be considered. Strain II may have other strengths not considered in the model that may allow for coexistence in the region. For example, this model does not consider constant migration of strain I and strain II into the region. Including migration into the model may well create a possibility of coexistence as is shown in [4]. Perhaps if a larger area was considered, for example many square miles, the effect of space would be more apparent in our model and migration would imitated. One infectious region may die out while another region flourishes with the infection. One hectare is too small to observe this type of behavior. One may also want to include differences in oral transmission between the strains; strain II may have the advantage of being better
adapted to oral transmission as well as vertical transmission.

Another key factor that may affect the spread of the disease is host behavior. A consideration of multiple hosts and a careful modeling of their behavior would reveal information about how the parasite may move through a region. As an example, consider the two hosts woodrats and raccoons. The two hosts have distinctly different behavior. Male woodrats tend to stay in a relatively small region of about 0.19 hectares [5] and have a density of about 21 woodrats per hectare [10]. A woodrat in general has only a few nests at which it sleeps and only forages around in a limited region around these nests. When the hosts sleep is when they have the potential to be bitten by the *Triatoma*. Raccoons have a home-range of about 60.5 hectares [1] with a density of about 0.144 raccoons per hectare [10]. Within this home-range, they have many places at which they may sleep. Thus they cover a wide area where they have the possibility to become infected and a larger area over which they can carry the infection once they are infected. This model would most likely have to be made in a way such that it would be difficult to analyze side by side with a system of ODE’s; however, the results may well be interesting and useful.

6 Acknowledgments

The authors of this paper would like to thank Christopher Kribs-Zaleta, Britnee Crawford and Anuj Mubayi for all the help they gave us and for their supervision during this research project. We would also like to thank Kehinde Salau (A.K.A. Stack Mo’ Bricks) for the help with Netlogo and programming in general, Emmanuel Morales for the help with Matlab, and Carlos Castillo-Chávez for the opportunity to participate in the program. Without the help of all these people this paper could not be completed. This project has been partially supported by grants from the National Science Foundation (NSF - Grant DMPS-0838704), the National Security Agency (NSA - Grant H98230-09-1-0104), the Alfred P. Sloan Foundation and the Office of the Provost of Arizona State University.
References


Proposition 2: $N_h(t = \tau) \neq 0$ for $\tau$ arbitrary $\Rightarrow$ $N_h$ will be asymptotically constant to

$$N_h = \frac{k_h(r_h - \mu_h)}{r_h}$$

Proof:

Let's assume that $N_h(t = \tau) \neq 0$ and that $\tau$ is arbitrary. The general solution of (8) is

$$N_h = \frac{1}{\frac{r_h}{k_h(r_h - \mu_h)} + Ce^{-(r_h - \mu_h)t}}$$

(24)

where $C$ is an arbitrary constant. Under the assumption that $r_h > \mu_h$, we see that as $t \to \infty$, $N_h \to \frac{k_h(r_h - \mu_h)}{r_h}$. Referring to [11], since $\frac{dN_h}{dt}$ and $\frac{\partial N_h}{\partial N_h}$ are continuous on any rectangle in the $t - N_h$ plane, we are guaranteed that any two solutions of the family of solutions for (8) will never intersect. This implies the solution of (8) with initial condition $N_h(\tau) = N_{h\tau}$ where $\tau$ and $N_{h\tau}$ are arbitrary but $N_{h\tau} \neq 0$ will be unique and can be obtained from (24) and will always tends to $\frac{k_h(r_h - \mu_h)}{r_h}$. In the case that $N_{h\tau} = 0$ there is no real value for $C$ such that (24) is equal to zero but we know that $N_h = 0$ is a particular solution of (8), so the solution of (8) with initial condition $N_h(\tau) = 0$ for $\tau$ arbitrary must be $N_h = 0$. The fact that the general solution of (8) will never be zero and if there is another possible solution that can cross the $t$ axis implies that it will be $N_h = 0$, guarantees that all the solutions of (8) in our model will tend to $\frac{k_h(r_h - \mu_h)}{r_h}$ as $t \to \infty$, since we assumed that $N_h(t = \tau) \neq 0$. ■
Figure 1: Flow chart for the ODE model.
Figure 2: Bifurcation graph.
Figure 3: Averaged results for the vector population of ABM simulation: y axis is percentage and x axis is time where 1 unit represents 50 days. Green (S) is the percentage of the population that is susceptible, Red (I1) is the percentage of the population that is infected with strain I and Yellow (I2) is the percentage of the population that is infected with strain II.
Figure 4: Numerical approximation of the ODE’s: y axis is percentage and x axis is time in days. **Green**(S) is the percentage of the population that is susceptible, **Red**(I1) is the percentage of the population that is infected with strain I and **Yellow**(I2) is the percentage of the population that is infected with strain II.
### Tables

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value</th>
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</thead>
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<td>[10]</td>
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<td>$r_h$</td>
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<td>1/year</td>
<td>[10]</td>
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<td>host/vector</td>
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Table 1: Parameter Values

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<th>Species</th>
<th>Infection</th>
<th>per Hectare</th>
<th>Source</th>
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<tr>
<td>woodrat</td>
<td>33%</td>
<td>23</td>
<td>[10]</td>
</tr>
<tr>
<td><em>T. gerstaeckeri</em></td>
<td>45%</td>
<td>316</td>
<td>[10]</td>
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Table 2: Population densities and infection prevalence in Texas
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<td>$r_v$</td>
<td>Reproduction rate for vectors</td>
<td>1/time</td>
</tr>
<tr>
<td>$r_h$</td>
<td>Reproduction rate for hosts</td>
<td>1/time</td>
</tr>
<tr>
<td>$\mu_v$</td>
<td>Death rate for vectors</td>
<td>1/time</td>
</tr>
<tr>
<td>$\mu_h$</td>
<td>Death rate for hosts</td>
<td>1/time</td>
</tr>
<tr>
<td>$K_v$</td>
<td>Carrying capacity for vectors</td>
<td>vectors</td>
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<tr>
<td>$K_h$</td>
<td>Carrying capacity for hosts</td>
<td>hosts</td>
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<tr>
<td>$\beta_{v1}$</td>
<td>Infection rate of strain 1 f</td>
<td>hosts/(vectors year)</td>
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<td>Infection rate of strain 2 f</td>
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<td>$\beta_{h1}$</td>
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<td>$\beta_{h2}$</td>
<td>Infection rate of strain 2 f</td>
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<tr>
<td>$H$</td>
<td>Number of vector consumed per host per time</td>
<td>vectors/(hosts year)</td>
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<tr>
<td>$\gamma_1$</td>
<td>Proportion of young born infected from an infected mother with strain 1</td>
<td>unitless</td>
</tr>
<tr>
<td>$\gamma_2$</td>
<td>Proportion of young born infected from an infected mother with strain 2</td>
<td>unitless</td>
</tr>
<tr>
<td>$\rho_1$</td>
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<td>host/vector</td>
</tr>
<tr>
<td>$\rho_2$</td>
<td>Proportion of hosts infected with strain 2 after consuming on vector with strain 2</td>
<td>host/vector</td>
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Table 3: Parameter definitions
Table 4: Values of strain I reproductive number, strain II reproductive number, and strain I invasive reproductive number from parameter estimates in Table 1, and percent of the time strain I dominates in 30 simulations.

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<thead>
<tr>
<th>q value</th>
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<th>$R_2$</th>
<th>$R_1$</th>
<th>% Strain I dominates</th>
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<td>1.60</td>
<td>1.65</td>
<td>0.95</td>
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<tr>
<td>$q = 1.1$</td>
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<td>1.65</td>
<td>1.03</td>
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<tr>
<td>$q = 1.2$</td>
<td>1.88</td>
<td>1.65</td>
<td>1.12</td>
<td>30%</td>
</tr>
<tr>
<td>$q = 1.3$</td>
<td>2.02</td>
<td>1.65</td>
<td>1.20</td>
<td>33%</td>
</tr>
<tr>
<td>$q = 1.4$</td>
<td>2.16</td>
<td>1.65</td>
<td>1.28</td>
<td>43%</td>
</tr>
<tr>
<td>$q = 1.5$</td>
<td>2.29</td>
<td>1.65</td>
<td>1.36</td>
<td>57%</td>
</tr>
<tr>
<td>$q = 1.6$</td>
<td>2.43</td>
<td>1.65</td>
<td>1.44</td>
<td>70%</td>
</tr>
<tr>
<td>$q = 1.7$</td>
<td>2.57</td>
<td>1.65</td>
<td>1.52</td>
<td>60%</td>
</tr>
<tr>
<td>$q = 1.8$</td>
<td>2.70</td>
<td>1.65</td>
<td>1.61</td>
<td>57%</td>
</tr>
<tr>
<td>$q = 1.9$</td>
<td>2.84</td>
<td>1.65</td>
<td>1.69</td>
<td>77%</td>
</tr>
<tr>
<td>$q = 2.0$</td>
<td>2.98</td>
<td>1.65</td>
<td>1.77</td>
<td>73%</td>
</tr>
</tbody>
</table>

Table 5: Here column A is the number of time Strain I dominates, B is the number of time Strain II dominates, C is the average time in years for strain II to go to extinction with standard deviation, and D is the average time in years for strain I to go to extinction with standard deviation.

<table>
<thead>
<tr>
<th>q</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>$q = 1.0$</td>
<td>2</td>
<td>28</td>
<td>49 (20)</td>
<td>15 (13)</td>
</tr>
<tr>
<td>$q = 1.1$</td>
<td>6</td>
<td>22</td>
<td>57 (16)</td>
<td>13 (13)</td>
</tr>
<tr>
<td>$q = 1.2$</td>
<td>9</td>
<td>21</td>
<td>38 (12)</td>
<td>12 (10)</td>
</tr>
<tr>
<td>$q = 1.3$</td>
<td>10</td>
<td>20</td>
<td>31 (10)</td>
<td>9 (4)</td>
</tr>
<tr>
<td>$q = 1.4$</td>
<td>13</td>
<td>17</td>
<td>32 (8)</td>
<td>9 (7)</td>
</tr>
<tr>
<td>$q = 1.5$</td>
<td>17</td>
<td>13</td>
<td>30 (13)</td>
<td>6 (3)</td>
</tr>
<tr>
<td>$q = 1.6$</td>
<td>21</td>
<td>9</td>
<td>23 (9)</td>
<td>7 (3)</td>
</tr>
<tr>
<td>$q = 1.7$</td>
<td>18</td>
<td>12</td>
<td>23 (7)</td>
<td>9 (5)</td>
</tr>
<tr>
<td>$q = 1.8$</td>
<td>17</td>
<td>13</td>
<td>23 (6)</td>
<td>7 (3)</td>
</tr>
<tr>
<td>$q = 1.9$</td>
<td>23</td>
<td>7</td>
<td>19 (4)</td>
<td>6 (3)</td>
</tr>
<tr>
<td>$q = 2.0$</td>
<td>23</td>
<td>7</td>
<td>21 (7)</td>
<td>5 (2)</td>
</tr>
</tbody>
</table>