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EFFECTS OF INVASION TIMING IN A ONE-DIMENSIONAL MODEL OF COMPETING SPECIES WITH AN INFECTIOUS DISEASE

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CHAPTER I

INTRODUCTION

In this thesis we study a model that combines two classical models from mathematical biology, the SIR model of disease and the competing species model. We start by presenting background on these two models.

1.1 Background on SIR Models

A general class of mathematical models that describe the spread of a disease in a population is the collection of SI models, or susceptible-infectious models. There are various special cases of this model, depending on how the disease affects each individual. The basic idea of this class of models is to divide the population between susceptible and infectious subpopulations, and the model shows how the individuals move between each subpopulation. For the case of permanent resistance, or immunity, in an individual who has recovered from the disease, an SIR model is used. Instead of having only susceptible and infected subpopulations, an SIR model adds a third subpopulation for individuals who recover from being infected [1]. Models of this type assume that those who recover from the disease do not leave the subpopulation of recovered individuals. If death is incorporated into the model, then those in the recovered class could drop out [2].

One standard form of the SIR model has the form

$$\frac{\partial S}{\partial t} = b - \beta SI, \tag{1.1}$$

$$\frac{\partial I}{\partial t} = \beta SI - \nu I, \tag{1.2}$$

$$\frac{\partial R}{\partial t} = \nu I, \tag{1.3}$$

where S, I, and R represent the susceptible, infectious, and resistant subpopulations, b is the birth rate, β represents the transmission coefficient, and ν is the recovery rate coefficient [3, 2]. This system allows for reproduction of healthy individuals, so the total population is not fixed. If we assume that the number of deaths approximately equals the number of births, then the Hong Kong Flu is an appropriate example of a disease that is modeled by the SIR model [4]. In the first equation, the birth rate is added to show that the susceptible subpopulation is growing but the βSI term is subtracted because as transmission happens, those who are infected with the disease move to the infectious subpopulation. This is how the pool of infectious individuals grows. As infectious individuals recover, they move to the recovered subpopulation at the rate ν and are subtracted from the infectious subpopulation and added to the pool of recovered individuals. The process is ongoing, as individuals continue to become diseased, and recover. At any given point in time, the total number of individuals in a population is $N = S + I + R$.

The SIR model is a sub-case of the SIRS model [1]. In the SIRS model, an individual has resistance to the pathogen for a period of time after having the disease, but eventually returns to the susceptible subpopulation [1]. Hence these

models assume that an individual can move from susceptible to infectious to recovered and then back to the susceptible subpopulation [5]. An example of a disease that can be modeled by the SIRS model is salmonella diarrhea [6]. One standard form of the SIRS model is

$$\frac{\partial S}{\partial t} = \gamma R - \beta SI, \tag{1.4}$$

$$\frac{\partial I}{\partial t} = \beta SI - \nu I, \tag{1.5}$$

$$\frac{\partial R}{\partial t} = \nu I - \gamma R, \tag{1.6}$$

where S, I, and R represent the susceptible, infectious, and removed, or resistant, subpopulations, β represents the transmission coefficient, ν is the recovery rate coefficient, and γ is the loss of immunity rate [3]. As individuals in the susceptible subpopulation become infectious, they move from the pool of susceptibles to the pool of infectious in the second equation, as shown in the term βSI that is subtracted from the first equation and added to the second. As infectious individuals recover, they move from the infectious subpopulation to the recovered subpopulation in the term νI that is subtracted from second equation and added to the third. After individuals are resistant for a time, they move back to the pool of susceptible individuals, which is described by the term γR. Resistance is not permanent in this model.

Like the SIR model, an SIRS model may incorporate death, in which case the total population decreases. If an individual goes back into the pool of susceptibles immediately after recovering from the disease, the model is called an SIS model [1]. Here, there is no equation that models the recovered, and instead the recovered

individuals go directly to the group of susceptible ones from the group of infectious [7]. In any of these cases, the model could assume that individuals die from disease or natural causes.

There are different methods to model pathogen transmission. Two commonly mentioned methods are mass-action (density-dependent) and standard (frequency-dependent) incidence (See Table 1.1) [8]. While both methods deal with transmission occurring from interaction with other members of the entire population, there is a clear line dividing the two. Saenz et al. gives a summary of the debate [1]. It is explained that a standard or frequency-dependent incidence is used when the model incorporates the number of new infections per unit time, or the frequency of new infections, in the susceptible population. In the general SIR and SIRS models summarized by equations (1.1)—(1.6), the βSI terms indicate that mass-action incidence is used.

Table 1.1: Two Forms of Disease Incidence

Form	Incidence
βSI	Mass Action
$\frac{\beta SI}{S+I}$	Standard

When density-dependent incidence is used, the transmission is a function of the density of infected hosts. Typically, density-dependent incidence is incorporated when the transmission is a result of random contact between species [9]. If the

transmission is not random, then it falls under the order of frequency-dependence. This dependence is typically used when transmission is systematic and there is a fixed number of contacts, which is why it is frequently used to model the spread of Sexually Transmitted Diseases [9].

1.2 Background on Competing Species Models

Another general class of models in mathematical biology describes when two or more species compete for the same resources. These models are sometimes called Lotka-Volterra models. Competition leads to many effects on a population. The long-term dynamical changes in the size of the population directly stem from the inter-species and intra-species interactions, i.e., between individuals in difference species and between individuals in the same species [10]. We consider a version of the Lotka-Volterra model that features logistic growth for each population in the absence of competition. Each species grows until it hits the carrying capacity, or the maximum number of individuals that the environment can carry [11]. Logistic growth can be modeled as

$$\frac{\partial N}{\partial t} = rN\left[1 - \frac{N}{K}\right], \tag{1.7}$$

where N is the size of the population at time t, r is the intrinsic growth rate, which is the difference between the birth and death rates, and K is the carrying capacity. Expanding this to more than one population and incorporating the effect of competition

between species gives the Lotka-Volterra model

$$\frac{\partial N_1}{\partial t} = r_1 N_1 \left[1 - \frac{N_1}{K_1} - \frac{\alpha_{12} N_2}{K_1}\right], \tag{1.8}$$

$$\frac{\partial N_2}{\partial t} = r_2 N_2 \left[1 - \frac{N_2}{K_2} - \frac{\alpha_{21} N_1}{K_2}\right], \tag{1.9}$$

where N_i is the size of the population of species i at time t, r_i is the intrinsic growth rate, K_i is the carrying capacity, and α_{ij} is the competition coefficient, which describes the effect on species i of competition with species j [12]. Density-dependent incidence is used to express transmission in this simple Lotka-Volterra model. In both equations, the growth of each species is hindered by the intra-species and inter-species competition. Intra-specific competition, or the crippling done by species i on itself, is rendered by the $1/K_i$ factors. Not only does the species affect its own kind, but the other species competes so that the growth of species i is hindered by species j, as seen in the third terms in both equations. This widely used model shows the dynamics between species that compete for the same resource(s) in a region [1].

The long-term result of competition as modeled above has been proven to be the extinction of one species by the domination of the other, the extinction of both species, or a coexistence between the two species. In [3], the authors discuss the biological significance in and difference between each outcome. These outcomes are represented by different fixed points in the phase plane for (1.8) and (1.9). Using K_1 and K_2 as carrying capacity for the first and second species, respectively, two outcomes, or the fixed points at which the species are in steady state, include $(K_1, 0)$ and $(0, K_2)$. In the first, species 2 is driven to be absent in the long-run, while

the second shows the second species taking over and driving species 1 to be absent. These two fixed points are always present but their stability depends on the values of the parameters. In the case where neither species is able to keep their population alive, the long-run outcome is the point $(0, 0)$, but this fixed point is unstable for all parameter values. The last outcome is coexistence between the two species. This fixed point is described as

$$\Big(\frac{\alpha_{21}K_1 - K_2}{\alpha_{21}\alpha_{12} - 1}, \frac{\alpha_{12}K_2 - K_1}{\alpha_{21}\alpha_{12} - 1}\Big). \tag{1.10}$$

We only consider this fixed point when it is in the first quadrant, so the numerators and common denominator in (1.10) must have the same sign. This occurs under the conditions that (1) $\alpha_{21} > \frac{K_2}{K_1}$ and $\alpha_{12} > \frac{K_1}{K_2}$ OR (2) $\alpha_{21} < \frac{K_2}{K_1}$ and $\alpha_{12} < \frac{K_1}{K_2}$. From (1), it follows that $\alpha_{21}\alpha_{12} > 1$, thus, both x- and y-coordinates are positive and the fixed point is in the first quadrant. Likewise, from (2), it follows that $\alpha_{21}\alpha_{12} < 1$, so the coordinates are both positive and the fixed point is in the first quadrant. There are no other conditions under which the interior fixed point, (1.10) is in the first quadrant. We do not consider this fixed point in any other quadrant, because in any other quadrant it would represent a negative population.

Different relations among parameters in the model lead to sub-cases of each outcome. These relations are expressed in Table 1.2, which is reproduced from [3]. The stability of each fixed point may change from case to case. In case 1, there is only one point that is stable. That point is $(0, K_2)$. Case 2 yields stability at $(K_1, 0)$. Both $(K_1, 0)$ and $(0, K_2)$ are stable under the conditions in case 3, and in case 4, the

fourth fixed point, which is interior and given by (1.10), is stable. As noted above, the fixed point at $(0, 0)$ is never stable, and in fact is always called an unstable node [3]. The three fixed points that yield stability are shown in Figure 1.1.

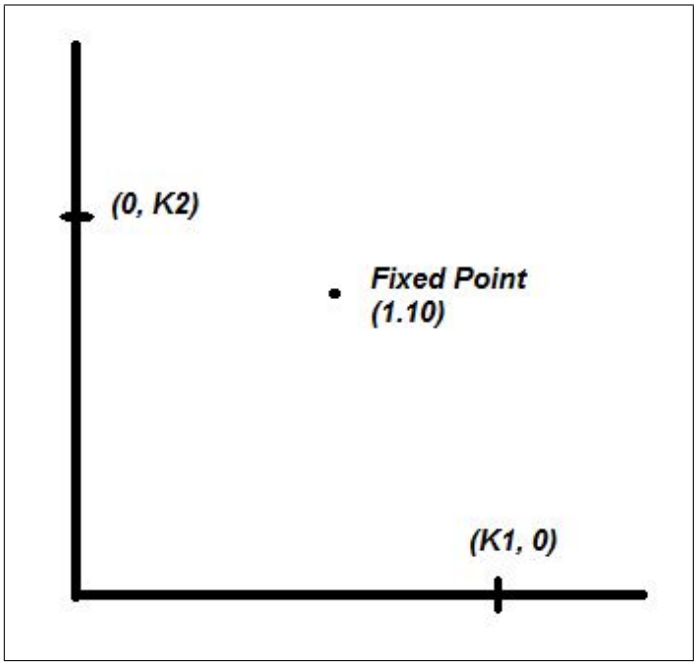

Figure 1.1: Fixed Points for Stability

Biologically, the stability of these fixed points shows that in Cases 1 and 2, any combination of initial population sizes will drive the outcome to the stable node, allowing only one species to thrive. In case 3, the initial population sizes will determine which species dies out and which one ends up at a density close to its carrying capacity. In this case, (1.10) is saddle point that has a stable manifold that divides the first quadrant into two. This distinguishes which initial conditions push the species to $(K_1, 0)$ or $(0, K_2)$. If the pair of initial conditions lies above

this manifold, the outcome will be $(0, K_2)$. If the initial conditions fall below this manifold, the outcome will be $(K_1, 0)$. Case 4 gives a coexistence between the two species no matter what their initial densities are. This coexistence shows that the size of each population is less than its carrying capacities.

Table 1.2: Cases to determine Stability for Fixed Points

Case	**Inequality 1**	**and**	**Inequality 2**	**Stability of Fixed Point**
Case 1	$\frac{K_2}{\alpha_{21}} > K_1$	and	$K_2 > \frac{K_1}{\alpha_{12}}$	$(0, K_2)$ only stable fixed point
Case 2	$K_1 > \frac{K_2}{\alpha_{21}}$	and	$\frac{K_1}{\alpha_{12}} > K_2$	$(K_1, 0)$ only stable fixed point
Case 3	$K_1 > \frac{K_2}{\alpha_{21}}$	and	$K_2 > \frac{K_1}{\alpha_{12}}$	Both $(K_1, 0)$ and $(0, K_2)$ stable
Case 4	$\frac{K_2}{\alpha_{21}} > K_1$	and	$\frac{K_1}{\alpha_{12}} > K_2$	(1.10) only stable fixed point

1.3 Modeling Spread of Disease Between Competing Species

Recently, a new line of research has been pursued, in which a Lotka-Volterra type model is combined with an SIR type model to show the effect on populations of the interaction between the spread of disease and competition [13, 14, 15, 16, 17, 18, 19]. Other recent research endeavors explore models in which an SIR type model is combined with a predator-prey model [20, 21, 22]. Here, we will discuss only the combination of competition and disease. An important recent paper in this area is [10], in which Bowers and Turner reveal how a native species can have an edge when

they are strong competitors or when their population is healthy. The edge that they have on another species may diminish when they do not compete very well against the other species or when their population is infected with a disease. In [10], there are two species analyzed in a habitat: the native species and the invasive species. The invasive species has a disease, and upon entering the natives' habitat, they introduce both the disease and competition.

Before an invasion, each species will be at an equilibrium at its carrying capacity. By the time they need to compete with another population, they will have already established how competitively strong they are against others in their species. Bowers and Turner introduce the idea of "intra-specific" and "inter-specific forces of competition." These forces are interpreted as certain combinations of parameters describing the carrying capacities and competition. For the natives, the force of competition within their own species will continue after the invasive species enters their habitat. The other type of competition force occurs when the two species are competing for resources against each other, instead of against members of the same species. Aside from competition, the other main "force" introduced into the situation is that of infection, or how the infection spreads and affects individuals of both species. Once an invasion begins, the population sizes may change, depending on the forces of competition between each species and the forces of infection [10].

Bowers and Turner state their results in terms of the balance among these forces. In the case of competition only, if the effect of the natives' force of competition on the invaders is weaker than the invaders' already-established competition on

themselves, the invasion will succeed and the natives will be diminished by the impact of the invaders. This is because the invaders were previously able to withstand a certain amount of competition, and the competition from the natives was not greater than the force they felt before. Similarly, if the effect of the invaders' competition on the natives is weaker than the effect of the natives' competition with themselves, the invasion will fail, and the natives will still dominate their habitat [10]. In the previous section, the main outcomes included the two metioned here: one where the invasion succeeded and the fixed point that was approached as time went on was $(0, K_2)$, and one where the invasion failed and the fixed point that was approached as time went on was $(K_1, 0)$.

Special cases of the model in [10] can describe when there is no competition between the species, but the species can infect each other. Strict infection here follows a similar pattern to the competition criterion mentioned above. Before an invasion, each species is stable and able to handle a certain amount of infection present in its own population. Once the invasion begins, if the intra-species force of infection that the invaders tolerated before is greater than the force that the natives exert on the invaders, the invaders will be able to invade the natives [10]. This scenario could be reversed, and if the invaders pose a greater force of infection on the natives than the force they already equilibrated with within their population, then the natives may be in danger of being invaded.

Though the natives are unable to repel the invaders by strictly outcompeting the invaders or by transmitting the disease more quickly than the invaders do, the

two forces may be able to combine to help the natives overcome the invasion [10]. Table 1.3 relates the invadability criteria found in [10] and relates each outcome to its respective fixed point mentioned in the previous section.

Table 1.3: Cases to determine Stability for Fixed Points

Case	**Species 1**	**Species 2**	**Outcome**	**Stability of Fixed Point**
Case 1	Not invadable	Invadable	Species 1 survives and Species 2 is eliminated	Stable at $(K_1, 0)$
Case 2	Invadable	Not invadable	Species 1 is eliminated and Species 2 survives	Stable at $(0, K_2)$
Case 3	Not invadable	Not invadable	Species 1 survives and Species 2 is eliminated OR Species 1 is eliminated and Species 2 survives	Stable at either $(K_1, 0)$ or $(0, K_2)$
Case 4	Invadable	Invadable	Species 1 and Species 2 coexist	Stable at the fixed point (1.13)

In Table (1.3), conditions under which species are invadable, or not invadable, follow from either competition effects in the absence of infection, or infection effects in the absence of competition. Bowers and Turner further describe outcomes when forces of competition and infection are combined. If both species realize an outcome of infection, there are more outcomes for each case. Though both species are infected and competing with one another, only one survives, despite having the disease. These outcomes follow from Cases 1–3 in Table (1.3), though now, each species that survives

will carry the disease. Case 4 differs in that there are two types of coexistence outcomes: one in an infectious state and one in a healthy state. Since each species here ends in an infected state, a normal conclusion would be that Case 4 would yield an infected coexistence. However, if the equilibrium is feasible and stable under strict competition, and if the linear approximation guarantees stability against pathogen invasion, Case 4 could yield an uninfected coexistence [10]. If both species do not realize an outcome of infection, these cases may change, depending on whether they both end up in a healthy state or if only one species ends up infected.

Garcia-Ramos et al. also present a model in which both the Lotka-Volterra and SIRS models are combined [23]. Though the forces of infection and competition are combined to seek outcomes as is done in [10], they include one more feature in the dynamics between species: disease resistance. The model describes the relationship between a native species and an invading species that carries a disease. As the disease is introduced to the native population, the natives start to evolve resistance to the disease as a response to the threat posed on their existence. Garcia-Ramos et al. present the dynamics over a one-dimensional spatial domain, so the governing equations are PDEs instead of simply ODEs.

The level of resistance plays a part in the natives' birth rate and in the transmission rate of the disease to the natives from their own kind or from the invaders. The transmission rate encompasses disease transmission, establishment, and development. As resistance goes up, the transmission rate goes down. The rate goes down, but many still could be affected as fewer and fewer individuals become infected over

time. However, there is a cost associated with a heightened resistance. This cost is the damaging effect on fecundity, or the ability to produce healthy offspring, of the native species. As the natives develop a higher resistance, their ability to have healthy offspring goes down [23].

A significant effect in the model presented in [23] is that becoming infected and evolving resistance does not happen instantaneously. Being able to evolve resistance to the pathogen helps the natives not only stay alive as a whole, but it also allows them to recover so they can compete against the invaders. Two concepts that are introduced include the "disease front" and the "competition front." Because the invaders enter the territory of the natives at one edge of the domain, they do not immediately come into contact with all of the native individuals. Though the invaders may not directly come into contact and transmit the disease to each native individual, they could infect a native, and that native could move to another place in its domain and thus infect another native. This could repeat until there are many natives infected who were not in direct contact with infected invaders. This phenomenon is called the "disease front." The "competition front" refers to the arrival of invading species to points in the domain where there are natives who compete for the same resource. So, if the disease front arrives much faster than the competition front, there is a better chance that the natives are able to deal with the disease and evolve a resistance before they have to compete for their lives [23].

Results of [23] explain the positive outcomes that evolution of resistance has on the native population and show under which conditions the natives are able to halt

the invasion. In a step-by-step analysis, Garcia-Ramos et al. began by modeling the interaction between the native and invading species with respect to many different disease-induced mortality rates. The criteria used for the interaction between the two species was taken from [10]. Their model shows that when the native species is able to evolve a resistance to the pathogen, there is a better chance that they survive and do not die out completely. With resistance, there is also a bigger window that would allow for coexistence of the two species. This means that, even if the mortality rate of the natives is higher due to disease, their development of resistance will assist them in becoming stronger more quickly and keep their population in existence. This coexistence is typically seen when the repulsion force from the invaders onto the natives is stronger than the force exerted from the natives onto the invaders. Developing resistance could also lead to an invasion collapse. The conditions for a collapse include an intermediate disease-mortality rate in the native species and a repulsive force from the natives onto the invaders that is greater than the force from the invaders onto the natives. [23].

The simulations shown in [23] illustrate two situations, including a species replacement, where the competitive effect of the invading species is so great that it overtakes the natives, and invasion collapse, where the natives are able to evolve a resistance quickly enough to fight off the invading species and survive in their habitat. Though resistance helps a population stay alive as a whole, developing resistance does not ensure that a population will not be overtaken by another, specifically when a competing population is powerful enough to have a detrimental effect on the other

[23].

The evolution of resistance described in [23] is based on a model presented in [24]. In [24], the evolution of a phenotypic trait is described by

$$\frac{\partial z}{\partial t} = \frac{\sigma^2}{2}\frac{\partial^2 z}{\partial x^2} + \sigma^2 \frac{\partial \ln n}{\partial x}\frac{\partial z}{\partial x} + h^2\beta, \tag{1.11}$$

where z is the trait mean, σ^2 is the dispersal variance, h^2 is the heritability, x is the spatial dimension, t is time, n is the density of surviving individuals, and β is the selection intensity at point x [24]. The terms on the right-hand side of equation (1.11) describe several different effects. The first term describes regular diffusion, or how dispersal of individuals changes the trait mean of the local population to be more like the mean of the surrounding population. The second term describes how the trait mean is affected if there is a more dense population nearby [24]. We will discuss the effects of the mean trait of the dense population in our explanation of Equation (1.12). The last term on the right-hand side of (1.11) describes selection within the local population [24].

As noted above, equation (1.11) is the basis for the equation describing the evolution of resistance in [23]. This equation is

$$\frac{\partial R}{\partial t} = h^2 V_p \Big\{ \frac{\partial}{\partial R}\Big[\frac{1}{S_0}\frac{\partial S_0}{\partial t}\Big]\Big\} + \frac{2D_0}{H_0}\frac{\partial H_0}{\partial x}\frac{\partial R}{\partial x} + D_0\frac{\partial^2 R}{\partial x^2}, \tag{1.12}$$

where R denotes the mean of the trait resistance, h^2 is heritability of resistance, V_p is variance of resistance, S_0 respresents the density of the native susceptible subpopulation, H_0 represents the density of the entire native population, and D_0 is the diffusion coefficient. The last two terms on the right-hand side of equation (1.12) cor-

respond to the first two terms on the right-hand side of equation (1.11). Hence, these terms account for gene flow in the spatial domain and how dispersal of individuals affects the mean resistance at other places.

The second term on the right-hand side of (1.12) corresponds to the second term on the right-hand side of Equation (1.11). It describes how the mean value of resistance at a point x changes based on the mean level of resistance and the size of the population at nearby points. For example, if there is a higher density group with a higher resistance level nearby, the smaller group's mean trait may be pulled up if they mate with individuals from the bigger group. Likewise, if the more populous group has a lower resistance level, the resistance level of the smaller group may be pulled down as they mate. Consider a population that is centered around a specific point in the domain, where the total number of individuals in the population is increasing across the domain. This scenario is depicted in Figure 1.2.

The mean trait is increasing over the domain. There is an influx of individuals to the center of the domain, which means that both individuals with a higher mean trait and individuals with a lower mean trait are trickling in from the right and left, respectively. Though the individuals with a lower mean trait are causing the mean trait at the center to decrease, there are fewer individuals entering from the left than at the right. This means that the ones coming from the right are affecting the mean trait at the center of the domain more so than the ones coming from the left, as they are greater in population [24].

The third term accounts for diffusion. Diffusion shows the scattering of indi-

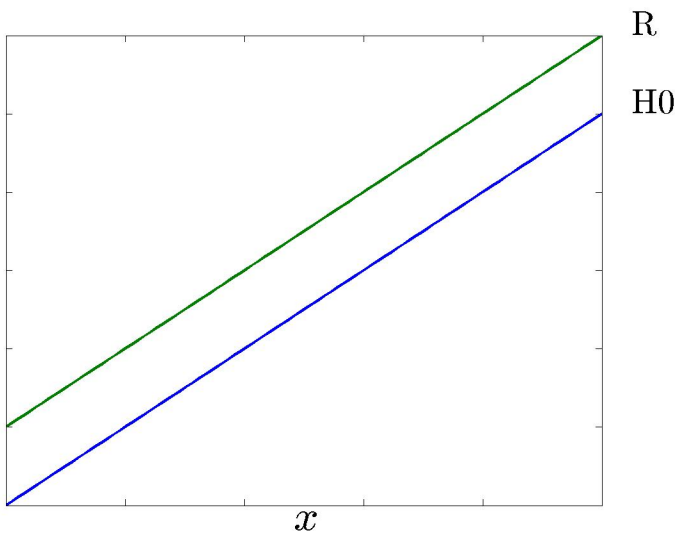

Figure 1.2: Mean trait and population size about center of spatial domain

viduals of both species and how resistance is spread throughout the spatial domain [23]. In this model, it was assumed that all dispersing individuals from one spot in the spatial domain shared the same resistance, since they were at the average resistance of individuals in that area before scattering [23].

To explain the first term on the right-hand side of (1.12), note that S_0 is the density of susceptible individuals and hence $(1/S_0)(\partial S_0/\partial t)$ is the per-capita growth rate of native susceptibles. This quantity measures the fitness of the healthy natives. This fitness is a function of R, denoted $F(R)$. Hence, ignoring migration, (1.12) looks like

$$\frac{\partial R}{\partial t} \propto \frac{\partial F(R)}{\partial R}. \tag{1.13}$$

Equation (1.13) explains how effectively the pathogen makes individuals sick [2]. A

similar proportion is used in [2] to express the fitness of a pathogen strain as its virulence changes. Virulence measures deadliness of a disease, and the level of virulence goes up when the mortality rate in the infected species is high or if the time between infection and disease-induced death is short [25]. In [2], they describe the fitness of the pathogen. This fitness is determined by the change in size of the infectious population. If the fitness increases as virulence increases, then natural selection should cause the level of virulence to increase. This means that the growth rate of the infectious population is increasing. Likewise, if fitness decreases as virulence decreases, the level of virulence will be driven to decrease. Equation (1.13) says that if $\frac{\partial F(R)}{\partial R}$ is positive, which means that fitness as a function of R is increasing, then natural selection will cause resistance to increase. In a similar manner, if $\frac{\partial F(R)}{\partial R}$ is negative, which means that fitness decreases as R increases, then natural selection will drive resistance down with time.

1.4 Summary of Main Results

Evolution of resistance in a native species that undergoes forces of infection and competition was introduced and discussed by Garcia-Ramos, et al [23]. Our model differs in that the disease is introduced into the relationship by the native species and the invading species evolves a resistance to the pathogen to stay alive. The conditions under which species replacement, invasion failure, and coexistence are discussed.

In this thesis, we adapt the model of Garcia-Ramos et al. In their model, the invaders entered into the habitat of the natives, bringing a pathogen with them. The

natives developed resistance to that pathogen. Here, we present a model in which the natives initially have the disease and the invaders must develop resistance once they enter the habitat. This model takes the form of five coupled reaction-diffusion equations. We use our model to perform numerical simulations using MATLAB's pdepe solver.

We not only use simulations to explore and illustrate basic outcomes that are predicted by the model, but also to analyze whether the timing of the invasion matters. Normally, the invaders enter a habitat in which the natives are equilibrated with the disease. To answer the question of invasion timing, we simulate results using initial conditions that place the natives in a transient state, battling the disease.

This thesis is organized as follows. In Chapter II, we present our model. In Chapter III, initial conditions for native and invasive species are derived and the outcomes of the model are presented in seven simulations. We conclude with explaining our results and presenting an adaption of our model for future work.

CHAPTER II

INVASION MODEL

2.1 Governing Equations

In this chapter we present our model. As noted in the previous chapter, our model is a variation of the model presented in [23]. Our model takes the form of a system of five partial differential equations (PDEs). Four of these describe the change over time of the densities of the sub-populations: native susceptibles (S_0), native infectious (I_0), invasive susceptibles (S_1), invasive infectious (I_1). The fifth PDE describes the evolution of resistance (R) of the invaders. Each dependent variable is a function of time ($t \geq 0$) over the spatial domain $x \in [0, L]$. Mass-action, or density-dependent, incidence is used. Variables $H_0 = S_0 + I_0$ and $H_1 = S_1 + I_1$ are used to express the combined sub-populations of susceptible and infectious for each the native and invasive species.

In this thesis, we study questions similar to those studied in [23]. We analyze the effect that evolution of resistance has on an invasive species when it competes for resources. As in [23], the SIS model is combined with Lotka-Volterra competition to show both the effect of disease and effect of competition. The invading species develops resistance to the disease that is indigenous to the natives. Instead of the

invaders bringing the disease with them to help with the invasion, they battle with the disease upon entering the habitat of the natives. The birth and transmission rates of the invading species are dependent on the resistance. Because the development of resistance is tracked and acts on transmission and birth, an SIR model is not used, i.e., the model does not track a sub-population of recovered or resistant individuals.

The PDEs for the population densities and resistance are

$$\begin{aligned}
\frac{\partial S_0}{\partial t} &= \left[a_0 - (\frac{a_0 - b_0}{K_0})(H_0 + c_{01}H_1)\right](S_0 + f_0 I_0) - b_0 S_0 \qquad (2.1)\\
&\quad -\beta_{00} S_0 I_0 - \beta_{01} S_0 I_1 + \gamma_0 I_0 + D_0 \frac{\partial^2 S_0}{\partial x^2}\\
\frac{\partial I_0}{\partial t} &= \beta_{00} S_0 I_0 + \beta_{01} S_0 I_1 - b_0 I_0 - \alpha_0 I_0 - \gamma_0 I_0 + D_0 \frac{\partial^2 I_0}{\partial x^2} \qquad (2.2)\\
\frac{\partial S_1}{\partial t} &= \left[a_1(R) - (\frac{a_1(R) - b_1}{K_1})(H_1 + c_{10}H_0)\right](S_1 + f_1 I_1) - b_1 S_1 \qquad (2.3)\\
&\quad -\beta_{11}(R) S_1 I_1 - \beta_{10}(R) S_1 I_0 + \gamma_1 I_1 + D_1 \frac{\partial^2 S_1}{\partial x^2}\\
\frac{\partial I_1}{\partial t} &= \beta_{11}(R) S_1 I_1 + \beta_{10}(R) S_1 I_0 - b_1 I_1 - \alpha_1 I_1 - \gamma_1 I_1 + D_1 \frac{\partial^2 I_1}{\partial x^2}, \qquad (2.4)\\
\frac{\partial R}{\partial t} &= \frac{h^2 V_p}{S_1}\Big\{ - k_a a_1(R)\left(1 - \frac{H_1 + c_{10}H_0}{K_0}\right) \times \{S_1 + f_1 I_1\} \qquad (2.5)\\
&\quad + k_b \left[\beta_{10}(R) I_1 + \beta_{11}(R) I_0\right] S_1 \Big\} + \frac{2D_1}{H_1}\frac{\partial H_1}{\partial x}\frac{\partial R}{\partial x} + D_1 \frac{\partial^2 R}{\partial x^2}.
\end{aligned}$$

The five equations above are based on the model presented in [23]. The first term on the right-hand side of the first equation is a product of two expressions, $\left[a_0 - (\frac{a_0 - b_0}{K_0})(H_0 + c_{01}H_1)\right]$ and $(S_0 + f_0 I_0)$. The quantity describes the logistic growth of and competition in the native susceptible population. These terms include the birth rate a_0, death rate b_0, and the effect of competition on the net growth, c_{01}. They also include the carrying capacity, or point of highest saturation, of native individuals in the habitat K_0, and the total densities of both the native and invasive subpopulations,

H_0 and H_1. The first quantity describes the net growth as new individuals in the subpopulation are born (a_0) and as they die (b_0). The difference between these two rates is divided by the carrying capacity K_0. That factor is multiplied by the factor that sums to the total population density with the competition affecting the invading population. This product is subtracted from a_0 to show that the growth is hindered by death and competition. The sum of infected and susceptible individuals ($S_0 + f_0 I_0$) is multiplied and shows that growth of the susceptible population is contingent upon the birth of new individuals, whether the births occur in susceptible, or healthy populations, with regular fecundity, or in infected populations with reduced fecundity, where f_0 is a parameter that describes this reduction.

The next term on the right-hand side of (2.1), $b_0 S_0$, accounts for disease-free death, or death from natural causes. Transmission rates β_{00} and β_{11} relate how the disease spreads within each species, while β_{01} and β_{10} describe transmission from one species to another. Native individuals become infected from being exposed to infectious natives or infectious invaders. The next two terms involving the rates β_{00} and β_{01} are subtracted to show that there are individuals becoming ill and leaving the subpopulation of the susceptible, or healthy, individuals. Equations (2.1)—(2.4) each represent a subpopulation. As native individuals become infected, they move to the subpopulation that is diseased. Here, this movement takes them from the susceptible subpopulation, represented by Equation (2.1) to the infected subpopulation, represented by Equation (2.2). After these terms, there is a term which represents recovered individuals that return to the pool of susceptibles, which recover at the

rate γ_0. The last term represents the diffusion of individuals in this subpopulation.

The second equation, (2.2) for the native infectious, contains terms which represent growth in the infectious subpopulation as the disease spreads. The first two terms, $\beta_{00}S_0I_0$ and $\beta_{01}S_0I_1$, were originally seen in the first equation, where they were subtracted to show that there were some individuals becoming ill and leaving the healthy subpopulation. Here, they are added to show that, as the individuals become ill, they are classified as infectious. The next two negative terms, b_0I_0 and α_0I_0, account for the rate at which disease-free and disease-induced death affect infectious individuals. These terms describe individuals who die and are completely removed from the native population. The second to last term, γ_0I_0, describes the recovered individuals leaving the infectious population, and who reenter the susceptible population as a positive number to show an increase in that population. The last term, again, accounts for diffusion.

Equations three and four, (2.3) and (2.4) for the invading species, have right-hand sides with terms similar to the terms on the right-hand sides of the first two equations. However, the growth and transmission rates now depend on resistance R, so that these rates will change as the invaders evolve resistance to the disease. Specifically, the birth rate will decrease according to

$$a_1(R) = A_1e^{-k_aR}, \tag{2.6}$$

and the transmission rates will decrease according to

$$\beta_{11}(R) = B_{11}e^{-k_b R}, \tag{2.7}$$

$$\beta_{10}(R) = B_{10}e^{-k_b R}. \tag{2.8}$$

These are the same rates presented in [23].

Equation (2.5) describes how the development of resistance gives a mean value of resistance. The interpretation of (2.5) is similar to the interpretation of equation (1.11) presented in Chapter I. The first term on the right-hand side of (2.5) is h^2V_p times $\frac{\partial}{\partial R}\frac{(1/S_0)}{(\partial S_0/\partial t)}$. Here, $(1/S_0)(\partial S_0/\partial t)$ measures the fitness of the invasive susceptible population. The fitness of the healthy invasive susceptible population changes with respect to change in resistance. Hence, if $\frac{\partial F(R)}{\partial R}$ is positive, fitness as a function of R is increasing, and resistance will increase. Likewise, if $\frac{\partial F(R)}{\partial R}$ is negative, resistance will decrease. The product of the phenotypic variance V_p and heritability h^2 of resistance is multiplied by the rate at which resistance changes. These two parameters are multiplied in this term to put bounds on how much resistance can change within the species.

Clearly, if transmission goes down, there are fewer becoming infected, however, developing such a resistance does not come for free. The resistance is changing for the better as long as the low transmission is greater than the reduced reproduction. Heritability is multiplied by the phenotypic variance of the resistance, and this quantity if proportional to the rate of per-capita rate of change of native susceptibles changes with resistance at a fixed location [23]. This represents how effectively the

invaders evolve their resistance to the pathogen that was originally carried by the natives. If resistance goes up, fewer individuals become infectious and the pool of susceptibles increases in number.

The last two terms on the right-hand side of (2.5) account for gene flow in the spatial domain and diffusion of the invasive species. They correspond to the first two terms in (1.11) and the last two terms in (1.12), found in Chapter I.

The parameters in the model are listed in Table 2.1 [23].

Table 2.1: Parameter Values

Parameter	**Definition**	**Magnitude and Unit**
a_0	Birth rate (native susceptible)	0.5 $time^{-1}$
A_1	Birth rate (invasive susceptible) where $R = 0$	0.5 $time^{-1}$
b_0	Disease-free death rate (native)	0.1 $time^{-1}$
b_1	Disease-free death rate (invasive)	0.1 $time^{-1}$
α_0	Disease-induced death rate (native)	0.1 $time^{-1}$
α_1	Disease-induced death rate (invasive)	0.1 $time^{-1}$
c_{01}	Competitive effect of invaders on natives	1, dimensionless
c_{10}	Competitive effect of natives on invaders	1, dimensionless
c_{00}	Competitive effect of natives on natives	1, dimensionless
c_{11}	Competitive effect of invaders on invaders	1, dimensionless
f_0	Fecundity reduction from disease (native)	0.9, dimensionless
f_1	Fecundity reduction from disease (invasive)	0.9, dimensionless
K_0	Carrying capacity (native)	180, $individual * length^{-1}$
K_1	Carrying capacity (invasive)	175, $individual * length^{-1}$
β_{00}	Intraspecific transmission rate	0.06, $length * individual^{-1} time^{-1}$
β_{01}	Interspecific transmission rate	0.06, $length * individual^{-1} time^{-1}$
B_{11}	Intraspecific transmission rate (invasive) where $R = 0$	0.06, $length * individual^{-1} time^{-1}$
B_{10}	Interspecific transmission rate (native to invasive) where $R = 0$	0.06, $length * individual^{-1} time^{-1}$
k_a	Fecundity reduction parameter	0.015 $resistance^{-1}$
k_b	Disease transmission reduction parameter	0.25 $resistance^{-1}$
γ_0	Recovery rate (native)	0.3 $time^{-1}$
γ_1	Recovery rate (invasive)	0.3 $time^{-1}$
D_0	Diffusion coefficient (native)	1.2 $distance^2 time^{-1}$
D_1	Diffusion coefficient (invasive)	1.2 $distance^2 time^{-1}$
h^2	Heritability of resistance	0.08, dimensionless
V_p	Phenotypic variance of resistance	1 $resistance^2$

CHAPTER III

CONCLUSION

We have presented a model in which an invasive species enters the diseased habitat of a native species. Our model, an adaption of the model presented in [23], incorporates models that describe interaction of species through disease and competition. The invasive species evolves resistance to the disease. Because of this added edge, the invasive species has a chance to become healthy in order to compete well with the natives.

We will run simulations with different parameter values to see the simple outcomes of the invasion. We will also explore invasion timing by using different initial conditions for the native species.

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APPENDICES