

Modeling Basal Sprout Production in Vector-Borne Tree Disease

A Dissertation Presented for the

Doctor of Philosophy

Degree

The University of Tennessee, Knoxville

Kelly Ruth Buch

May 2022

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*To Raeleigh & Bryley, Robert & Riley, and Adrianna, the nieces and nephews I may never know
again but will always love.*

Acknowledgements

On a personal note, I would like to thank my advisor, Nina H. Fefferman, for her never-ending support of my professional and personal development. Thank you for always approaching me first as a person and second as an academic. I would also like to thank the members of my lab group and my fellow graduate students at University of Tennessee Knoxville for their support, advice, and kindness throughout my graduate career. While writing a dissertation is an individual effort, making it through graduate school is a group effort, and I could not have done it without the support of my peers. On a similar note, I would like to thank the Sylvia Bozeman and Rhonda Hughes EDGE Foundation and the entire EDGE network. Participating in the EDGE program prepared me for success graduate school and gave me a network of women in mathematics that will continue to support me for years to come. I probably could have done this without EDGE, but I am so thankful I did not have to. Lastly, I would like to thank my parents and sisters for their love and support all of my life and especially throughout my graduate career. This journey that I have taken was once unfamiliar to me and remains partially unfamiliar to my family. Thank you for supporting me as I embarked into the unknown and for always being on the other end of the line when I couldn't make it home.

Professionally, I would like to thank the members of my committee, Dr. Louis Gross, Dr. Olivia Prosper, and Dr. William Klingeman III for their advice and contributions which greatly improved this dissertation. I would like to thank the biologists and ecologists whose feedback shaped the scope of this dissertation. Specifically, I'd like to thank Dr. Albert (Bud) Mayfield and Dr. KaDonna Randolph for their early guidance in understanding the biological system of Laurel Wilt and determining relevant questions for wildlife managers. I would also like to thank the organizers and participants of the Laurel Wilt Workshop: Disease Biology, Diagnosis, and Management. In particular, we'd like to thank Dr. Denita Hadziabdic, Dr. Romina Gazis, Dr. Pedro Pablo Parra, and Meher Ony for sharing their insights on Laurel Wilt and other tree diseases which provided

relevant context for confirming the structure of our mathematical model. Finally, I would like to thank an anonymous reviewer for their comments which improved Chapter 2. I acknowledge no funding for sources which supported this research.

Abstract

The introduction of non-native pests that vector disease and kill trees poses a serious threat to native tree populations. Mathematical models of vector-borne tree disease can investigate the impact of certain pathogen and host characteristics on disease dynamics and provide a framework for evaluating management strategies that mitigate the effects of introduced vector-borne disease. This is especially useful for characteristics with impacts that are difficult to measure empirically. In this dissertation, I investigate the impact of basal sprout production in trees infected by an introduced vector-borne disease that is vectored by a non-native bark or ambrosia beetles (e.g. laurel wilt, dutch elm disease, among others). Basal sprouts are shoots or sprouts that grow from the root system of an existing tree, and the production of basal sprouts is commonly induced in response to vascular wilt disease. I develop a series of mathematical, epidemiological models that incorporate basal sprout production and secondary infection via vertical transmission. We interpret these models to study the effect of basal sprout production on the ecological and epidemiological outcomes, both locally and spatially, of the introduction of vector-borne disease into a novel environment. Results suggest that when secondary infection is low, the production of basal sprouts decreases the likelihood of local extinction of the host population, but simultaneously increases the likelihood of endemic disease or future waves of epidemic disease. I also provide a framework for evaluating the utility of basal sprout-centered management of vector-borne disease in a variety of disease and management contexts. This theoretical work provides a set of initial models and a basis upon which future field studies and more tailored models can be developed, together helping to protect trees as novel pathogens and introductions into new areas continue to threaten tree populations.

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Chapter 1

Introduction

Introductions of non-native forest pests and pathogens have had devastating effects on forest ecosystems across the globe and have resulted in an estimated loss of \$4.2 billion in forest products per year in the United States alone (Pimentel et al., 2005). This damage includes that caused by pathogens that are transmitted by abiotic factors (e.g. wind or soil), pests that kill trees independently without the presence of a pathogen, and vector-borne diseases that require an insect pest or other organism to transmit the pathogen (bacteria, virus, fungus, nematode, etc.) from plant to plant. Of the 16 non-native forest pathogens that have killed or damaged trees in the United States since the 1800s, five pathogens have been vectored by insects (Aukema et al., 2010), and in many introduced vector-borne diseases, pathogen and vector are introduced together. Several of these introduced vector-borne diseases (e.g. Dutch elm disease and laurel wilt) are caused by fungi associated with bark and ambrosia beetles, and other examples of disease vectored by bark and ambrosia beetles exist across the globe (e.g. Japanese oak wilt and mango sudden death disease) (Hulcr and Dunn, 2011). The associated fungi of bark and ambrosia beetles are not generally pathogenic in their native range, but upon introduction to a new range, these beetles and their associated fungi can cause high levels of mortality. With introductions of disease causing bark and ambrosia beetles becoming more frequent, the risk of local loss of forest trees is high. As world-wide trade increases, introductions of disease-carrying bark and ambrosia beetles are expected to continue (Roy et al., 2014), and the resulting risk to forest trees is also expected to increase. Accordingly, research that investigates potential outcomes and management strategies for current and expected diseases vectored by bark and ambrosia beetles is merited.

While diseases vectored by bark and ambrosia beetles each have their own unique characteristics, they share common traits. Bark and ambrosia beetles are wood-boring beetles that bore into and reproduce within the woody material of dead or weakened trees (Lieutier et al., 2004). Most bark and ambrosia beetles are associated with a fungus that is directly cultivated and consumed by the beetle or otherwise contributes to reproductive success. This associated fungus is the pathogenic agent in diseases vectored by bark and ambrosia beetles. Transmission can occur in several ways. In Dutch elm disease, fungal pathogen (*Ophiostoma novo-ulmi* or *Ophiostoma ulmi*) is introduced to susceptible elms when the elm bark beetle (*Hylurgopinus rufipes*) feeds on woody bark material, and the transmission of fungal pathogen induces vascular wilt (Tatar, 1989). In laurel wilt, primary transmission occurs through failed boring attempts by the redbay ambrosia beetle (*Xyleborus glabratus*). Healthy trees are resistant to colonization by the redbay ambrosia beetle, and boring attempts into healthy trees are abandoned without brood production. However, this process transfers fungal material (*Raffaelea lauricola*) into the vascular system of the tree, and for trees within the laurel family, the presence of foreign fungus can trigger an exaggerated immune response that ultimately causes vascular wilt, killing the main stem of the tree within weeks or months (Hanula et al., 2008). In both disease systems, the dying trees provide suitable habitat for successful brood production for the wood-boring beetles, including the bark or ambrosia beetles that originally infected the tree.

1.1 Basal Sprouting in Tree Disease Systems

Basal sprouts are shoots that grow from the base or root system of a tree. The production of basal sprouts is a common phenomenon and is induced in response to injury or sudden change in environmental conditions (Del Tredici, 2001). The production of basal sprouts can occur after traumatic damage caused by abiotic factors such as fire, strong winds, or logging. Damage caused by biotic factors like pests and pathogens can also trigger basal sprout production. For example, tanoak (*Notholithocarpus densiflorus*) trees infected with sudden oak disease produce basal sprouts, and green ash (*Fraxinus pennsylvanica*) trees infested with the Emerald Ash Borer (*Agilus planipennis*) produce basal sprouts following crown dieback (Cobb et al., 2012; Kashian, 2016).

Several vector-borne diseases are known to induce basal sprouting in response to disease, and the degree to which trees produce basal sprouts varies by species within a disease system. Many species of elm (*Ulmus* spp.) are susceptible to Dutch elm disease, and while basal sprouting is very rarely reported for most species of elm, infected wych elm (*Ulmus glabra*) commonly produce

basal sprouts (Peterken and Mountford, 1998). Several species within the laurel (Lauraceae) family are susceptible to laurel wilt, including redbay (*Persea borbonia*) and sassafras (*Sassafras albidum*). Redbay trees respond to laurel wilt infection by producing basal sprouts intensely, but basal sprouting is less common in laurel wilt infected sassafras (Cameron et al., 2015).

The production of basal sprouts in response to damage by the pest or pathogen in a vector-borne disease may affect the expected disease dynamics indirectly through altered host population demography. The production of basal sprouts by an infected tree essentially replaces a mature stem with several smaller stems. This both increases the total population size and shifts the stage structure towards a smaller average basal area. Population size can affect disease dynamics if transmission is density dependent, and population structure can affect disease dynamics if transmission is heterogeneous with respect to tree size (Hoppensteadt, 1974). Host reproduction is also known to affect disease dynamics (Anderson and May, 1979). If seed producing trees are highly susceptible to disease and the longevity of seed banking is limited, vegetative growth via basal sprouting can become essentially the only viable form of population growth remaining for infected populations.

The production of basal sprouts also adds an additional route of transmission. Although basal sprouts are produced by an infected tree, they are produced by healthy portions of the root system of the infected tree. Thus, upon production, basal sprouts do not harbor the pathogen. Many fungal diseases move systematically throughout the woody material of infected trees and can spread through interconnected root systems (Epstein, 1978). In this case, the basal sprouts are at risk of disease through vertical transmission. In the case that vector attraction to stems is differentially dependent on stem size, this may be the only method of transmission infecting immature basal sprouts.

The role of basal sprouts in vector-borne disease is relatively unknown. An untested hypothesis suggested that basal sprout production by wych elm could account for some of difference between disease dynamics compared to other species of elm (Peterken and Mountford, 1998; Gibbs, 1978). In this dissertation, I investigate the effect of basal sprouts in vector-borne tree diseases at both local and regional scales to consider this hypothesis mathematically and to contribute to the wealth of knowledge on forest pathogens. Many interesting questions are relevant to this investigation. In this dissertation, I identify and attempt to answer three specific questions of interest. In Chapter 2 I ask, how does the intensity of basal sprout production and the likelihood of vertical transmission of pathogen to basal sprouts change epidemiological and ecological outcomes in both short and long

time scales in a single forest patch? I answer that question specifically within the context laurel wilt in redbay and sassafras. With that answer in mind, I ask in Chapter 3, can altering disease dynamics through active management of basal sprouts eradicate disease and maintain the host population within the context of a generic disease vectored by bark or ambrosia beetles? Lastly, I ask in Chapter 4, again within the context of a generic disease vectored by a bark or ambrosia beetle, how does the production of basal sprouts affect the ability of a generic vector-borne disease to establish locally and spread spatially? These, of course, are not the only relevant (and perhaps not the most relevant), questions in this investigation. However, answers to these questions (and the models developed to answer them) are a start and provide a solid foundation upon which further research can expand.

1.2 Mathematical Modeling in Phytopathology

Mathematical modeling of plant diseases is a rich and well-established subdiscipline of mathematical ecology (Madden et al., 2007). Because of the economic impact of disease on plants that provide a direct resource to humans, a large majority of models within phytopathology are focused on diseases that affect either food crop species or species of trees used for lumber. However, numerous models have been developed for pathogens of forests trees (Cobb et al., 2012; Swinton and Gilligan, 1996; Yoshimura et al., 1999). Many plant diseases are modeled using compartmental models that separate the population into distinct categories and allow for movement between categories and flow into/out of the plant population. A common framework for compartmental models divides the population into subpopulations by their infection status (e.g. susceptible, infected, or recovered in an SIR model (Anderson and May, 1992) or healthy, latent, infectious, and recovered in a HLIR model (Madden et al., 2007)). There are many variations to the core SIR/HILR framework because assumptions in mathematical models are driven by both the disease and population dynamics of the system and the utility of those assumptions to provide answers to relevant questions.

Additional classes or subdivisions of classes may be necessary for the disease system or question at hand (see Gilligan (2008); Madden et al. (2007) for discussion of each of these following examples). The susceptible, infected, and recovered classes may be divided to represent heterogeneity in levels of susceptibility by age (e.g. size structure), immune strength (e.g. immunocompromised and non-immunocompromised), intensity of infection (e.g. mild and severe infection), and degree of immunity (e.g. recovery with full immunity or waning immunity). Additional classes beyond susceptible and infected may be included (e.g. recovered, treated, exposed, etc.) if plants can be

in additional, distinct epidemiological categories, and the recovered class may be not be included if recovery is not feasible (i.e. there is no immunity after recovery from infection or infection is always fatal). Additional non-plant classes may need to be modeled directly or indirectly if they affect disease processes. For example, if the source of infection comes from a reservoir within the system (e.g. soil or vector-borne pathogens), the total amount of inoculum in the environment may be modeled directly. In the case of vector-borne diseases, the vector population may be divided into carrier and non-carrier subpopulations.

In a compartmental, epidemiological model, the size of each subpopulation changes with time either through movement between classes (e.g. transmission, recovery, treatment), movement into the population (e.g. sprouting, planting), or removal from the population (e.g. natural death, disease-induced death, harvest). The most simple of epidemiological models include disease transmission, and additional flow within the model can be added as necessary. If the time-scale of disease is similar to or larger than that of plant life cycle events (e.g. seed production, vegetative growth, maturation, natural mortality), components of the plant life cycle may be incorporated into the model. In the case of crop or timber diseases, additional assumptions regarding planting, harvesting, and maintenance may be included in the model to account for the actions of humans in the system. When the total inoculum or the vector population is modeled, growth or decline of those populations may be included, and in the case of vector-borne diseases, population dynamics of the vector population and transmission of pathogen from carrier vector to non-carrier vector or from infected plant to non-carrier vector may be included.

1.3 Survey of Relevant Existing Mathematical Models

Because mathematical models are dependent on both the system and scope of the research question, many variations of mathematical models of vector-borne diseases exist. When modeling a new system or investigating a new question in an old system, if existing models are sufficient, they should be used. However, if existing models are insufficient to capture unique components of a new disease system or question, then different models should be developed, while still following the established best-practices. There are a wealth of mathematical models for plant and tree diseases. Here, I consider some existing mathematical models for vector-borne tree diseases and diseases in which basal sprouting is common and evaluate them according to their utility to answer these proposed questions. For examples of mathematical models for management of plant diseases in

other contexts (crop diseases, virus pathogens, etc), for example see Jeger et al. (2014, 2018); Cunniffe et al. (2016).

1.3.1 Pine Wilt Disease

Pine Wilt is a vector-borne, vascular disease that kills pine trees (*Pinus* spp.). It is caused by a nematode (*Bursaphelenchus xylophilus*) and vectored by pine sawyer beetles (*Monochamus* spp.). The larval stage of the nematode is transmitted to healthy pine trees by carrier bark beetles when the beetles feed on the leaves of healthy trees. The nematode then breeds in the xylem of the tree, causing wilting. Pine sawyer beetles are attracted to wilting trees for oviposition, and thus beetle pupae become carriers of the parasite when developing in an infected tree, completing the cycle (Shi and Song, 2013). In many regards, disease cycle of pine wilt is similar to the disease cycle of many fungal pathogens vectored by ambrosia and bark beetles.

Most models of pine wilt are variations of SI-SI models (i.e. host trees can be susceptible (S) or infected (I), and vectors can be carriers (S) or non-carriers(I)) (Shi and Song, 2013; Lee and Kim, 2013; Ozair, 2014; Lee, 2014). Extensions of the SI-SI model that include an exposed (E) class for trees only (SEI-SI) (Lee and Lashari, 2014) or for both trees and vector populations (SEI-SEI) (Awan et al., 2016; Khan et al., 2017, 2018a,b) have been proposed. The inclusion of an exposed tree class represented trees that are infected with the parasite but still maintain resistance to oviposition and accounted for the differing time scales of the two modes of transmission.

Transmission of disease from vector to tree has been modeled several ways. One preliminary model considered standard incidence infection rates that assumed that any beetle is equally likely to feed and oviposit on both susceptible and infected trees (Shi and Song, 2013). Later models considered non-linear incidence rates to account for the fact that adult pinewood beetles tend to avoid trees with high oviposition rates (Lee and Kim, 2013; Ozair, 2014; Awan et al., 2016; Khan et al., 2018a,b). In particular, they used a Holling type II functional response (Holling, 1965). Other models considered bilinear incidence for all transmission (Lee and Lashari, 2014; Lee, 2014; Khan et al., 2017). This is most likely a simplifying assumption to make the models more tractable for the application of optimal control. Many models considered two types of transmission of parasite from carrier beetles to susceptible trees (Lee and Kim, 2013; Ozair, 2014; Awan et al., 2016; Lee and Lashari, 2014; Lee, 2014; Khan et al., 2017, 2018a,b). The primary transmission mode occurs when carrier beetles can pass the parasite to healthy, susceptible trees when feeding. A secondary transmission occurs when a carrier beetle oviposits on a naturally dying susceptible tree, which has

lost its resistance to oviposition, resulting in infection when that act yields transfer of the nematode. Additionally, several models allowed for bilinear horizontal transmission of parasite between carrier and non-carrier beetles during mating (Ozair, 2014; Lee and Lashari, 2014).

Most models for Pine Wilt considered a constant birth rate for both host and vector, with one model matching parameter values such that the host population stays constant (Lee and Kim, 2013). The most recent model considered density dependent births for both host and vector (Khan et al., 2018a). To date, however, there are no models for pine wilt that define vector birth rate as dependent on the available host material.

1.3.2 Citrus Greening Disease

Citrus greening disease is a disease of citrus trees (*Citrus* spp.) caused by a bacterial pathogen (*Candidatus liberibacter asiaticus*) transmitted by a psyllid vector (*Diaphorina citri*). The disease is often seen in agricultural fields and threatens the citrus industry. This disease system has been modeled with two SI-SI variants (Chiyaka et al., 2012; Jacobsen et al., 2013). The disease transmission dynamics are very dependent on the stages of psyllid development, so one model included six state variables representing different developmental stages and infection levels of psyllid (Chiyaka et al., 2012). Moreover, the host population dynamics seen in both models were very specific to populations grown in managed agricultural fields.

1.3.3 Dutch Elm Disease

The disease dynamics of Dutch elm disease are described above. Two similar models for dutch elm have been introduced by the same authors (Swinton and Gilligan, 1996, 2000). Both differentiate tree by both size and infection status and assume that juvenile trees are not susceptible to disease. They include transmission of disease from vectors to susceptible, mature trees through beetle bark feeding as well as transmission from mature tree to mature tree through root grafting (i.e. transmission of fungal pathogen through interconnected root systems). They assume that the beetle population is directly proportional to the total number of infected trees and that infected trees become unsuitable for beetle breeding in time dependent manor (independent of vector population size). Reproductive output through seed production or basal sprouting by healthy trees is assumed to occur logistically with respect to the mature tree population, such that when the mature tree population is below carrying capacity, production of juvenile stems can occur to replace the lost mature trees. They specifically consider two competing strands of pathogenic fungus and include separate disease compartments for trees infected with each strand.

1.3.4 Sudden Oak Death

Sudden oak death, an air-borne disease caused by the fungal-like pathogen *Phytophthora ramorum*, infects (among others) tanoak (*Notholithocarpus densiflorus* and California bay laurel (*Umbellularia californica*) and has been modeled with a spatially explicit, density-dependent three-species model with population dynamics of the host populations (Cobb et al., 2012). The model includes both tanoak and California bay laurel populations, and a third class of trees that are not susceptible to sudden oak death is included to account for competition for unoccupied space for reproduction. They assume population growth is dependent on the amount of unoccupied space available for recruitment within a spatial cell. Transmission of disease can occur between species, but transmission is limited to trees within the same or adjacent spatial cells. Of the two susceptible species, one species is subdivided into 4 stage classes based on size, and susceptibility varies with size. In this species, the production of basal sprouts by infected trees is included, and they assume that saplings and basal sprouts are equivalent and thus are equally susceptible to disease. Accordingly, they do not consider the infection of basal sprouts through vertical transmission.

1.4 Overview of Model Development

No model of the diseases described above is sufficiently reflective of the dynamics of fungal pathogens vectored by bark and ambrosia beetles or to investigate the function of basal sprouts in these disease systems. Models for pine wilt propose a useful framework for host population growth in a forest setting but include methods of transmission that do not accurately reflect situations in which all beetles carry the fungal pathogen. Models of citrus greening disease highlight the importance of modeling the vector population but include levels of specificity concerning vector growth and maturation that are unlikely to be known for forest pathogens. Because Dutch elm disease is a forest pathogen that is vectored by a bark beetle, existing models provide good starting point for investigating the role of basal sprouts in general diseases vectored by bark and ambrosia beetles (e.g. similar routes of transmission, underlying assumption that vectors are carriers of disease). Furthermore, existing models do include the production of basal sprouts. However, all young stems (basal sprouts and saplings) are tracked together, so the specific function of basal sprouts can not be examined using existing models. Moreover, existing models include additional assumptions (e.g. multiple pathogen strains) not relevant to this investigation. Models for sudden oak death provide a template for how to incorporate basal sprout production separate from germination, but the inclusion of spacial components is beyond the scope of this investigation, and because the disease

is air-borne, no vector population is modeled. Moreover, vertical transmission from infected parent tree to basal sprout (which is of vital interest in this investigation) is missing in the existing models for that system.

Because no existing model is sufficient for the purposes of investigating the role of basal sprouts in diseases vectored by bark and ambrosia beetles, I propose a new base model (and variations of it) that aligns with relevant components of the aforementioned models. In Chapter 2, I propose an ODE model following the SI-I framework (i.e. trees can be susceptible or infected, and all vectors carry disease) and incorporate host stage structure and density dependent reproduction via a short-lived seed pool. I assume host susceptibility varies with host size. I also assume that mature, infected trees produce basal sprouts in response to disease (e.g., induction of epicormic shoot production) and that those basal sprouts can become infected through vertical transmission from parent tree. I track the infected classes not by number of trees (as in susceptible classes) but instead by the volume of infected tree material that provides suitable habitat for beetle reproduction, and I assume that material degrades as beetles use the material for reproduction. The vector population growth is modeled explicitly using logistic growth, with the assumption that the carrying capacity depends on the total amount of infected tree material.

Variations to this base model are proposed in later chapters to better answer the specific question addressed. In Chapter 3, I present a simplification of this model that reduces the complexity of host stage structure and eliminates highly uncommon modes of transmission. The reduction in model size (both in dimension and number of terms) leads to analytical solutions that can more easily be evaluated for effectiveness of management strategies. In Chapter 4, I expand upon the simplified model presented in Chapter 3 by incorporating age structure in the vector population to better account for the growth of the vector population upon introduction, and I extend that model across N distinct patches of forest (rather than one) and allow for movement of the vector population between patches.

Together, these models developed for fungal pathogens vectored by bark and ambrosia beetles and the questions answered with them will provide a better understanding of disease dynamics to expect in, and how to manage, both existing and future vector-borne diseases. This work will enhance the disciplines of phytopathology and mathematical ecology by contributing a modeling framework for a specific but highly relevant subset of plant disease ecology that has gone unstudied thus far. In doing so, it lays the ground work for future models that will answer even more questions

that to help with real-world management of systems that have not yet otherwise been adequately captured in existing models.

Chapter 2

Epidemiological and Ecological Function of Basal Sprouts in Vector-Borne Tree Disease

This work has not yet been published.

Kelly Ruth Buch developed and analyzed the mathematical model presented in this chapter under the mentorship of Nina H. Fefferman. Kelly Ruth Buch drafted the manuscript, which was revised by both Kelly Ruth Buch and Nina H. Fefferman.

Abstract:

Some tree species respond to disease by producing shoots, called basal sprouts, from the base and root system of a dying tree. These basal sprouts have the potential to alter the epidemiology of tree infections by altering demography. In the case of many airborne lethal tree diseases, the production of basal sprouts has been shown to be a key contributor to post-epidemic host population resurgence, but their effect in lethal vector-borne tree diseases has not yet been studied. To determine the role of basal sprout production and secondary infection via the root system of infected parent trees for lethal vector-borne tree diseases, we develop a stage structured SI-X mathematical model and use laurel wilt, a vector-borne tree disease in which infected trees provide suitable material for vector reproduction, as our model system. The mathematical models show that basal sprouts do not affect the short-term dynamics of laurel wilt epidemics but profoundly alter the long-term dynamics. In particular, in the absence of basal sprout infection, basal sprout production yields a larger host population after disease establishment, but as secondary infection increases, the utility of basal sprouts to maintain the host population decreases. Results suggest management strategies for lethal vector-borne diseases should depend on the ratio of rates of basal sprout production to infection.

2.1 Introduction

Since 1850, sixteen non-native forest pathogens that kill or damage trees have been introduced to the United States, and at least five these pathogens are vectored by insects (Aukema et al., 2010). Introduced forest pathogens have devastating effects on forest ecosystems, affecting both the trees themselves and other plant and animal species that depend on the affected tree species. This results in an estimated loss of \$2.1 billion in forest products per year in the United States (Pimentel et al., 2005). It is important to develop management strategies quickly after the introduction of a new forest pathogen into an ecosystem to either eradicate it or mitigate its effects. An

understanding of the role of specific factors that influence disease dynamics can assist in developing better management strategies for current diseases and future disease introductions.

Below-ground basal sprouting or root suckering are commonly observed forms of vegetative growth in angiosperms in which new stems sprout from the root system of an existing tree and are often induced when damage (e.g. fire, logging, disease) kills the main stem of a tree but the root system remains intact (Del Tredici, 2001). Disease-induced production of basal sprouts after widespread infection of large stems is both ecologically and epidemiologically important. Of ecological importance, the production of basal sprouts by dying, infected trees can maintain (or increase) host population size during outbreaks. Because widespread mortality of trees can inhibit seed production, basal sprouting by infected trees represents the main path for host reproduction especially if the reliance on, or longevity of, seed banking is limited. Of epidemiological importance, replacement of mature stems with basal sprouts will cause a demographic shift towards smaller stemmed trees. Changes in the demography of the tree host can in turn affect disease dynamics. For example, demographic shifts towards older stems due to previous wildfire decreased disease incidence in Sudden Oak Death compared to areas without wildfire-induced demographic shifts (Simler-Williamson et al., 2021). Similarly, basal sprouts produced in response to pest attack and/or pathogen infection in a vector-borne tree disease system may then affect the epidemiological dynamics of the disease through changes in the overall demography of the host population (Del Tredici, 2001).

In vector-borne disease systems, the specific influence of basal sprout production on disease dynamics is relatively unknown, and existing mathematical models for vector-borne diseases are insufficient to determine it. Pine wilt is a vector-borne disease caused by a parasitic nematode (*Bursaphelenchus xylophilus*) and transmitted by pine sawyer beetles (*Monochamus* spp.) (Mamiya, 1983). This vector-borne disease kills pine trees (*Pinus* spp.) and has been modeled extensively (Shi et al., 2014; Lee and Kim, 2013). However, the infected gymnosperms do not respond to infection by producing basal sprouts. As such, production of basal sprouts is not included in models of pine wilt. Dutch elm disease, which is caused by *Ophiostoma* spp. and vectored by the elm bark beetle (*Scolytus multistriatus*), has had terrible impacts on elm (*Ulmus* spp.) trees across the globe, and some (but not all) species of elm respond to infection by producing basal sprouts (Collin et al., 2000). However, the vector that transmits Dutch elm disease is only attracted to mature trees, leading to disease cycles as young trees mature. An untested hypothesis suggested that basal sprout production could account for some of difference between disease dynamics in some species (Peterken

and Mountford, 1998; Gibbs, 1978). Dutch elm disease models that included the growth of root suckers predicted either an endemic disease state or a disease free state, depending on parameter values, but the model did not differentiate saplings from basal sprouts, so the specific role of basal sprouting could not be investigated independently (Swinton and Gilligan, 1996, 2000). Thus the specific role of basal sprouts in lethal, vector-borne disease systems is relatively unknown, despite the potential importance of this trait on disease dynamics and host population outcomes.

In producing the first model to investigate the role of basal sprouts in a vector-borne disease system, we focus on laurel wilt as our case study. Laurel wilt is a lethal vascular tree disease, caused by the fungus *Raffaelea lauricola* and vectored by the redbay ambrosia beetle (*Xyleborus glabratus*) (Fraedrich et al., 2008). The insect-pathogen complex is not considered a pest in its native range of Asia, but the fungus is pathogenic to trees within the laurel family (Lauraceae) in its introduced range in North America (Ploetz et al., 2017). Upon infection, the fungus moves systematically through the xylem of the host and causes a host response that leads to crown dieback, leaf wilting, and sapwood discoloration (Fraedrich et al., 2008). Disease kills the main stem of trees within weeks (at most two months), inhibiting seed production and inducing the production of basal sprouts from the still-living root system (Evans et al., 2014).

The redbay ambrosia beetle is a wood-boring beetle that has a symbiotic relationship with the pathogenic fungus and requires dead and dying laurel trees for successful brood production. Female ambrosia beetles bore into the woody material of dying host trees (male ambrosia beetles are flightless and do not attack trees), creating small tunnels called galleries in which they cultivate their symbiotic fungus to feed their larvae. In contrast to other species of ambrosia beetle, redbay ambrosia beetles will also bore into healthy host trees with no signs of distress, with a preference for large-stemmed trees. Boring attempts into healthy trees do not yield successful brood production and are abandoned; however, enough fungal pathogen is transferred to infect the tree with laurel wilt. This contact is the primary transmission route for laurel wilt (Fraedrich et al., 2008). As they start to die, infected trees provide suitable host material for redbay ambrosia brood production, but three material is eventually degraded by excessive gallery production.

Redbay (*Persea borbonia* (L.) Sprengel) and sassafras (*Sassafras albidum*) are two ecologically important members of the laurel family and are susceptible to laurel wilt. Redbay is an evergreen tree with natural range covering the southeastern coastal plain and Florida and sassafras is a deciduous tree that spans the majority of Eastern United States (Hanula et al., 2008; Randolph, 2017). Laurel wilt is known to cause mortality greater than 90% of redbay large stems in 2-3 years

(Cameron et al., 2015). About half the range of sassafras occurs in regions with winter temperatures consistent with redbay ambrosia beetle cold tolerance, but as of now, laurel wilt has is present in only a small portion of the range of sassafras (Formby et al., 2018). Accordingly, studies of laurel wilt infection in sassafras over long time scales have not been done yet and little is known about the disease dynamics in this new host range.

Newly infected redbay and sassafras trees can respond to laurel wilt infection by producing healthy basal sprouts (Cameron et al., 2015). These basal sprouts are not infected but can become infected via direct contact with the root system of an infected tree, representing the secondary transmission route for laurel wilt. Because redbay ambrosia beetles rarely attack small diameter stems, infection of basal sprouts via secondary transmission can occur more quickly than infection via primary transmission; a sprout must mature sufficiently to be attractive enough for primary transmission to occur (Ploetz et al., 2017). The degree to which trees produce basal sprouts in response to infection and the rate of secondary transmission via connected root systems are both species dependent, so these differences could lead to qualitatively different disease dynamics in different species of susceptible host populations.

Using mathematical modeling, we investigate the ecological and epidemiological dynamics of laurel wilt as a case study by which to provide broader insight into the processes of all lethal, vector-borne tree diseases. We use this model to determine the potential impact of basal sprout production and infection on stand-level disease progression and survivorship. Understanding the role of basal sprouts in epidemic progression will provide insight into how to best manage for lethal, vector-borne diseases and illuminate differences to expect in host species that respond with basal sprout production at varying intensities. Mathematical models can isolate the specific effects of specific components of tree disease dynamics in ways that cannot be manipulated for empirical study directly (i.e. limiting physical or temporal constraints); therefore, as introductions of new tree diseases threaten arboreal biodiversity across the globe, increasing the set of appropriate mathematical models available as tools to support management and mitigate long-term consequences will be critical.

2.2 Methods

We develop a mathematical model for laurel wilt using using ordinary differential equations following an SI-X framework (Gilligan, 2008) (with X representing vector population as a proxy for total inoculum) and incorporate discrete host stage structure. Host trees are considered infected if

they harbor the fungal pathogen and thus provide suitable host material for vector brood production and susceptible otherwise. We separate susceptible and infected trees into several discrete classes representing host size, consistent with size classes often reported in data collection [Cameron et al. \(2015\)](#). In particular, we separate susceptible trees into $P_S(t)$, $M_S(t)$, $L_S(t)$, and $B_S(t)$ representing the number of susceptible saplings, medium trees, large trees, and basal sprouts, respectively, in each size class and then separate infected trees into $P_I(t)$, $M_I(t)$, $L_I(t)$, and $B_I(t)$ representing the volume of suitable host material (rather than the number of trees) for vector brood production provided by saplings, medium trees, large trees, and basal sprouts respectively. The host seed bank is denoted by $D(t)$ and is considered neither susceptible nor infected. The final class, $X(t)$, represents the number of female adult vectors. Due to the obligate nature of the vector-pathogen system ([Hanula et al., 2008](#)), all vectors are assumed to carry the pathogen. The differential equations of the model are given by

$$\begin{aligned}
\frac{dD}{dt} &= r_M M_S + r_L L_S - \gamma D - \mu_D D \\
\frac{dP_S}{dt} &= \gamma D - g_P P_S - \mu_P P_S - \beta_P X P_S \\
\frac{dM_S}{dt} &= g_P P_S + g_B B_S - g_M M_S - \mu_M M_S - \beta_M X M_S \\
\frac{dL_S}{dt} &= g_M M_S - \mu_L L_S - \beta_L X L_S \\
\frac{dB_S}{dt} &= \rho_M \frac{M_I}{v_M} + \rho_L \frac{L_I}{v_L} - (\sigma + g_B + \mu_B) B_S \\
\frac{dP_I}{dt} &= v_P \beta_P X P_S - \delta_P P_I X \\
\frac{dM_I}{dt} &= v_M \beta_M X M_S - \delta_M M_I X \\
\frac{dL_I}{dt} &= v_L \beta_L X L_S - \delta_L L_I X \\
\frac{dB_I}{dt} &= v_B \sigma B_S - \delta_B B_I X \\
\frac{dX}{dt} &= \omega X \left(1 - \frac{v_0 X}{P_I + M_I + L_I + B_I} \right),
\end{aligned} \tag{2.1}$$

and a diagram representing the movement of the model is shown in [Figure 2.1](#).

2.2.1 Host Dynamics

Susceptible host dynamics include seed germination, tree growth, natural mortality, and seed production. Saplings and basal sprouts mature to medium susceptible trees at rates g_P and g_B , respectively. Susceptible medium trees mature to susceptible large trees at at rate g_M . Saplings,

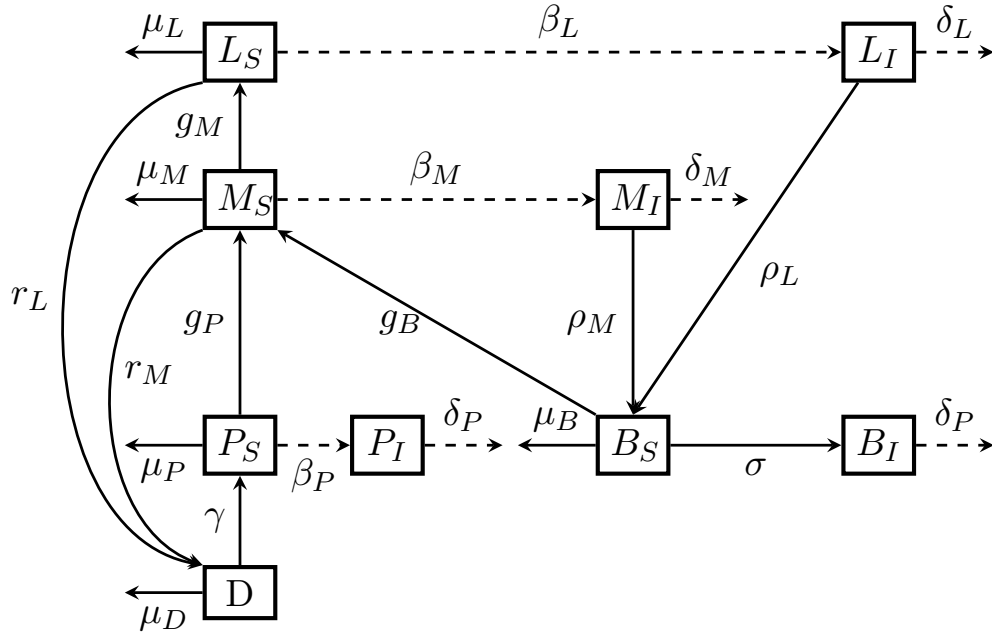


Figure 2.1: Flow diagram for Model 2.1. Solid lines represent movement independent of vector interaction, and dashed lines represent movement facilitated by vector interaction. Lines with upward movement represent maturation between host stages, curves with downward movement represent host seed production, and lines with downward movement represent the production of basal sprouts by infected trees.

medium trees, large trees, and basal sprouts suffer natural mortality at rates μ_P , μ_M , μ_L , and μ_B respectively. Both susceptible medium and large trees contribute to the seed pool at rates r_M and r_L respectively. Seeds either germinate into susceptible saplings at rate γ or deteriorate at rate μ_D . We consider large values for μ_D because laurels do not maintain long-term seed pools. Sassafras seeds can remain viable up to 5-6 years, with low germinative capacity after 5 years (Meadows et al., 2006), and redbay seed are not viable after two years (Evans et al., 2014).

Infected host classes do not grow nor contribute to the seed pool, but infected medium and large trees respond to laurel wilt infection by producing susceptible basal sprouts at rates ρ_M and ρ_L , respectively. This production is scaled by v_M and v_L , the volume of medium and large trees, respectively. Infected host material of all classes is degraded through the use of infected trees by the vector for brood production, rendering it unsuitable for future beetle brood production. This degradation is represented by the outgoing terms $\delta_Z Z_I X$ where Z is a place holder for the volumes of infected saplings, medium trees, large trees, and basal sprouts and δ_Z is the size-specific degradation rate.

2.2.2 Vector Dynamics

In laurel wilt, the vector requires infected host material for successful reproduction, and vector populations decline as disease prevalence declines (Potter et al., 2011; Maner et al., 2014). Accordingly, we assume the vector population growth depends on both the current beetle population size and the volume of infected trees. We capture these assumptions through logistic growth with a non-constant carrying capacity dependent on the total amount of infected host volume. We define the non-constant carrying capacity, K , as the ratio of the total available volume of infected tree and the volume required for each beetle gallery, i.e.

$$K = \frac{P_I + M_I + L_I + B_I}{v_0}.$$

For simplicity, we assume that beetles reproduce equally efficiently in any equivalent volume of infected material, regardless of the size of tree from which that material originated (despite some empirical evidence suggesting otherwise (Maner et al., 2014); extensions of this work to relax this assumption are planned for the future; preliminary sensitivity explorations suggest that the qualitative outcomes here described may be robust across sizes).

2.2.3 Infection Transmission

Transmission of the fungal pathogen occurs via two routes. The primary transmission occurs with contact between vector and susceptible tree and occur at rates β_Z , with incidence terms $\beta_Z Z_S X$, where Z is a place holder for the volumes of infected saplings, medium trees, large trees. This transmission route represents the vector creating and abandoning galleries. We take $\beta_P < \beta_M < \beta_L$ to account for the preference of the vector for large stems. Incidence terms are scaled by the volume of the tree, v_Z , producing a positive term $v_Z \beta_Z Z_S X$ into each of the differential equations for the volume of infected saplings, medium trees, and large trees. Secondary transmission is the direct transmission of pathogen from infected medium and large trees to susceptible basal sprouts through their shared root system and occurs at rate σ . Susceptible basal sprouts are scaled by v_B , the volume of a basal sprout, and transferred into the infected basal sprout class. This secondary transmission occurs without vector involvement because basal sprouts are physically attached to an infected tree via the root system.

2.2.4 Model Parameterization and Analysis

We perform an equilibrium and stability analysis on Model 2.1. We do this by evaluating the sign of the real parts of the eigenvalues of the Jacobian of Model 2.1 evaluated at the equilibrium (see (Strogatz, 1996) or Brauer et al. (2012)). We do this both algebraically and numerically. We consider numerical approximations of solutions to the differential equations in, and equilibria of, Model 2.1 using a biologically feasible range of parameter values. The default value for each parameter is listed in Table 2.1. When possible, values are estimated from data (see Appendix A). In absence of data, values were assumed and then varied to test the sensitivity of model to the assumption. The biological system of course experiences seasonality, with life and infection transmission only occurring during portions of the year. We take seasonal growth and activity rates and distribute them over a 365 day year rather than including the non-growing season. We do this in order to report yearly measurements. The model was validated but its ability to reproduce trends similar to that in redbay plots as presented in Cameron et al. (2015).

To reduce the degrees of freedom among the parameters, we assume the following relationships among stage specific host parameters: the rate of seed production for medium trees is 90% of that of large trees; the medium tree and basal sprout maturation rates are equivalent and are 40% of that for large trees; and the natural mortality rates of saplings and medium trees are twice and thrice that of large trees, respectively. We further assume that size specific primary transmission

Table 2.1: Summary of stage specific parameters and their default values used in Model 2.1. See Appendix A for calculations and references. Values are presented with one significant digit.

Description	Units	Symbol	Default Value
Seed production rate	year^{-1}	r_L	0.3
		r_M	$0.9r_L$
Seed germination rate	year^{-1}	γ	0.1
Maturation rate	year^{-1}	g_P	0.08
		g_B	g_P
		g_M	$0.4g_P$
Natural mortality rate	year^{-1}	μ_D	0.3
		μ_L	0.01
		μ_M	$2\mu_L$
		μ_P	$3\mu_L$
		μ_B	0.001
Primary transmission rate	$\text{year}^{-1}\text{beetle}^{-1}$	β_L	0.02
		β_M	$0.3\beta_L$
		β_P	$0.01\beta_L$
Secondary transmission rate	year^{-1}	σ	1
Basal sprout production rate	year^{-1}	ρ_L	2
		ρ_M	ρ_L
Volume of host	units^3	v_L	4
		v_M	2
		v_P	1
		v_B	1
Host degradation rate	$\text{year}^{-1}\text{beetle}^{-1}$	δ_L	0.02
		δ_M	δ_L
		δ_P	δ_L
		δ_B	δ_L
Volume of beetle gallery	units^3	v_0	0.3
Vector per capita reproductive rate	year^{-1}	ω	8

rates increases with tree size such that the rates for sapling and medium trees are 1% and 25% that of large trees, respectively. As a gross estimate for volume of trees, we assume that infected saplings and basal sprouts contribute equivalent volume, and medium and large trees contribute twice and four times as much, respectively. Due to a lack of differentiating empirical information, we assume that basal sprout production is equivalent for large and medium trees ($\rho := \rho_M = \rho_L$) and host degradation rate is equivalent for all stems ($\delta := \delta_B = \delta_P = \delta_M = \delta_L$).

When parameters are changed from the default, we define low and high values as 50% and 200% of the default value, respectively. When considering a range of values of parameters, we use Latin Hypercube Sampling (Marino et al., 2008) and sample values between the low and high value for each parameter. Size-specific parameters (i.e. parameters for the same action for hosts of different sizes) are varied proportionally, and when a parameter is not varied, its default value is used. (Note: the set of parameters we assumed to be related were chosen such that independent variations would not affect the qualitative outcomes of the modeled dynamics, only the rapidity and magnitude of the outcomes.)

2.3 Results

We investigate both the short-term (transient) and long-term (asymptotic) dynamics of the mathematical model, focusing on the effects of the rates of basal sprout production (ρ) and secondary infection (σ). We first consider the dynamics of the system without basal sprouting. In the absence of basal sprouting (and otherwise at default parameter values), all host populations decline after disease introduction, with the large tree population hitting 80% mortality first after 1.46, and the vector population reaches a peak at 1.56 years (Figure 2.2, solid line). Within a parameter space of 250 samples varying r_Z, γ, g_Z, μ_D , and β_Z , trajectories follow similar dynamics, but the timing of crashes and peaks varies. In particular, with varied host growth and transmission parameters, the large tree population reaches 80% mortality between 1.0 and 2.6 years after infection, and the vector population peaks between 1.2 and 2.2 years. Upon varying ρ and σ to the 13 aforementioned possibilities for each of the 250 samples, the time to 80% mortality changes by at most 0.23 years (with the largest changes occurring when β is smallest). The time of the peak of the vector population is largely unaffected by basal sprouting, with the peak changing by at most 0.03 years for varied values of ρ and σ . However, the size of the beetle population at the peak does increase as either ρ or σ increase, by at most around 25 beetles. Trajectories of the large tree and

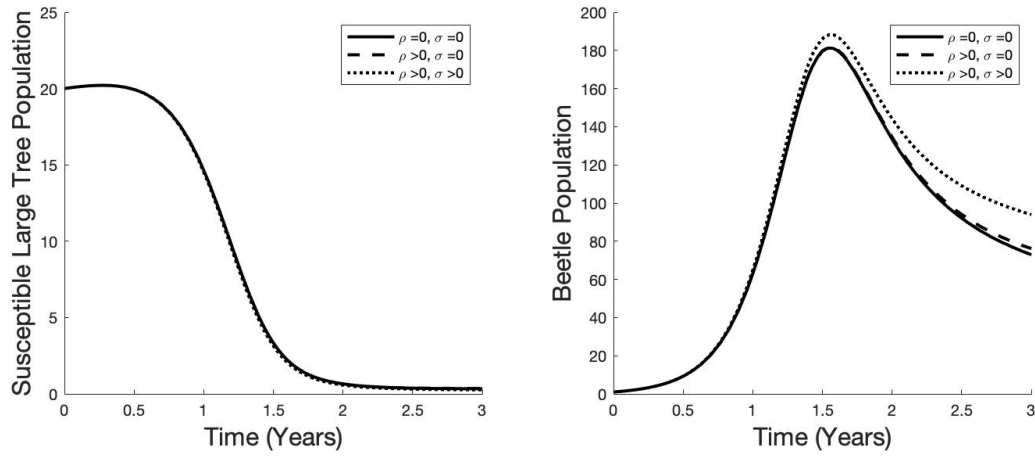


Figure 2.2: Susceptible large tree population (left) and vector population (right) over three years with no basal sprout production (solid line), basal sprout production with no secondary infection (dashed line), and basal sprout production with secondary infection (dotted line): (left) initial decline of susceptible population is invariant under basal sprout production and infection; (right) the vector population peaks at 1.56 years. The maximum size of the vector population is dependent on basal sprout production and infection, but when the maximum occurs is invariant with respect to basal sprout production and infection. Unless zero, all parameters take the default values.

vector populations for varied basal sprout production and infection at the default parameter values are given in Figure 2.2.

2.3.1 Equilibrium and Stability Analysis

We now consider long-term effects of basal sprouting with secondary infection and proceed by determining equilibria. From the last differential equation equated to zero, we have that either $X = 0$ or $X = K$, representing either extinction or maintenance of the vector population at equilibrium. We proceed with the former.

Extinction and Disease-Free Equilibria

Equating all differential equations to zero and assuming $X^* = 0$ immediately implies $B_S^* = 0$ and that M_I^* is a negative multiple of L_I^* (assuming all positive parameters). Thus, the only biologically feasible equilibrium without vectors has $B_S^* = M_I^* = L_I^* = 0$. Continuing, we obtain expressions for D^* , P_S^* , and L_S^* as positive multiples of M_S^* . First, supposing $M_S^* = 0$, we yield an extinction equilibrium in which all susceptible host classes are zero. We have insufficient information to obtain representations for P_I^* or B_I^* . Any non-negative value of these would yield a biologically feasible extinction equilibrium. (That is, extinction of vectors despite unused infected material of small diameter remaining is possible.) The extinction equilibrium is given by $(D^*, P_S^*, M_S^*, L_S^*, B_S^*, P_I^*, M_I^*, L_I^*, B_I^*, X^*)$, they are given by Eq₀ = $(0, 0, 0, 0, 0, P_I^*, 0, 0, B_I^*, 0)$

If M_S^* is non-zero at equilibrium (and thus other susceptible host classes are non-zero), we yield a non-extinction disease-free equilibrium when $\gamma = \gamma_0 > 0$ where

$$\gamma_0 = \frac{\mu_D \mu_L (g_P + \mu_P)(g_M + \mu_M)}{g_P(g_M r_L + \mu_L r_M) - \mu_L(\mu_P + g_P)(\mu_M + g_M)}. \quad (2.2)$$

This threshold value γ_0 is consistent with the minimum germination rate required for exponential growth (instead of exponential decay) of the host population in absence of disease.

Due to the lack of restriction of the representations of P_I^* or B_I^* , we obtain a plain of possible equilibria (rather than a unique point). Thus, unused infected tree material could remain in the disease-free non-extinction equilibrium. The disease-free non-extinction equilibrium expressed in the form $(D^*, P_S^*, M_S^*, L_S^*, B_S^*, P_I^*, M_I^*, L_I^*, B_I^*, X^*)$, is given by

$$\text{Eq}_1 = \left(\left(\frac{g_M r_L + \mu_L r_M}{(\gamma + \mu_D) \mu_L} \right) M_S^*, \left(\frac{g_M + \mu_m}{g_P} \right) M_S^*, M_S^*, \left(\frac{g_M}{\mu_L} \right) M_S^*, 0, P_I^*, 0, 0^*, B_I^*, 0 \right),$$

where M_S^* is positive and P_I^* and B_I^* are non-negative.

We claim that under reasonable assumptions, the non-extinction disease-free equilibrium exists.

Lemma 2.1. *Assume $r_L > \mu_L$ and $r_M > 3\mu_M$. Further, assume $g_M < g_P$, $\mu_M < \mu_P < g_P$, and all parameters are positive. Then $\gamma_0 > 0$.*

Proof. The numerator of Equation 2.2 is always positive. Thus Equation 2.2 is positive iff the denominator is positive. The denominator of Equation 2.2 is positive iff

$$\frac{r_L}{\mu_L} > 1 + \frac{\mu_M g_P + \mu_M \mu_P + g_M \mu_P - r_M g_P}{g_M g_P}.$$

By assumption, $\frac{\mu_P}{g_P} < 1$, $\frac{g_M}{g_P} < 1$, and $\frac{\mu_P}{\mu_M} < 1$. Thus,

$$1 + \frac{\mu_P}{g_P} + \frac{g_M}{g_P} \frac{\mu_P}{\mu_M} < 3.$$

Multiplying by μ_M and applying the lower bound of r_M yields

$$\mu_M \left(1 + \frac{\mu_P}{g_P} + \frac{g_M}{g_P} \frac{\mu_P}{\mu_M} \right) < 3\mu_M < r_M.$$

This inequality is equivalent to

$$\mu_M g_P + \mu_M \mu_P + g_M \mu_P < r_M g_P.$$

Consequently,

$$\frac{\mu_M g_P + \mu_M \mu_P + g_M \mu_P - r_M g_P}{g_M g_P} < 0$$

and

$$1 + \frac{\mu_M g_P + \mu_M \mu_P + g_M \mu_P - r_M g_P}{g_M g_P} < 1 < \frac{r_M}{\mu_L}.$$

This is sufficient to show γ in Equation 2.2 is positive. $\square$

The assumptions in Lemma 1 are reasonable under normal biological conditions. Adult trees produce a large quantity of seeds and suffer mortality infrequently. Thus, the lower bounds on the reproductive output of medium and large trees hold throughout the biologically feasible parameter

space. Regarding the growth rates of medium trees and saplings, we note that the medium class represents a larger spread of diameters than saplings (stems less than 2.5 cm DBH). As such, a tree that grows in DBH linearly will spend more time in the medium size class than in the sapling class. Thus, $g_M < g_P$. However, medium trees are more resilient than saplings and, as such, have a lower mortality rate, i.e., $\mu_M < \mu_P$. Finally, the assumption that $g_P > \mu_P$ requires that more saplings mature than die in a given time step. We maintain these inequalities throughout the swept parameter space.

Because the assumptions of Lemma 1 hold over the biologically feasible parameter space, we conclude the boundary representation for γ is positive throughout the biological feasible parameter space. Moreover, numerical experiments show that the value of γ when evaluated at parameters within the biologically feasible parameter space is of an order of magnitude that is also biologically feasible. Thus, we have, mathematically, a biologically feasible disease-free equilibrium with viable host population. However, this boundary case is unlikely to persist through natural perturbations of parameter values.

To determine the stability of these equilibria, we compute the Jacobian of the ODE system and substitute $A = 0$. The characteristic polynomial of the Jacobian contains no other state variables, so we proceed with stability of both equilibria that exclude vectors simultaneously. The characteristic polynomial in x of the Jacobian of the system at an equilibrium with no vectors is a tenth degree polynomial, which can be factored as

$$(\omega - x)(g_B + \mu_b + \sigma + x)p(x)x^4,$$

where $p(x)$ is a fourth degree polynomial in x . Thus, ω , $-(g_B + \mu_b + \sigma)$, and 0 are both eigenvalues. Because ω is assumed positive, both equilibria are unstable, despite the zero eigenvalues.

Endemic Disease Equilibrium

If $X^* \neq 0$, then $X^* = K$, where K is a function of the infected classes. That is, $X^* = \frac{B_I + L_I + M_I + P_I}{v_0}$.

We immediately obtain the expressions

$$\begin{aligned} P_I^* &= \frac{P_S \beta_P v_P}{\delta_P}, \\ M_I^* &= \frac{M_S \beta_M v_M}{\delta_M}, \text{ and} \\ L_I^* &= \frac{L_S \beta_L v_L}{\delta_L}. \end{aligned}$$

We proceed with the remaining differential equations, setting one equal to zero, solving for a variable, and substituting that expression into the other equations, yielding

$$B_S^* = \frac{B_I \delta_B X}{\sigma v_B},$$

$$P_S^* = \frac{\delta_P \left(M_S g_M v_0 - L_S v_0 \mu_l - L_S \beta_L \left(A - \frac{P_I}{v_0} \right) \right)}{L_S \beta_L \beta_P v_P},$$

$$B_I = \frac{\sigma v_0 v_B \left(\frac{L_S \beta_L \rho_L}{\delta_L} + \frac{M_S \beta_M \rho_M}{\delta_M} \right)}{\delta_B \left(L_I + M_I - \frac{v_0 \left(L_S \mu_L - M_S g_M + \frac{L_S \beta_L (L_I + M_I)}{v_0} \right)}{L_S \beta_L} \right) (g_B + \mu_B + \sigma)},$$

and

$$M_S^* = \frac{D (\gamma + \mu_D) - L_S r_L}{r_M}.$$

Doing so increases the degree of the remaining equations to be solved and the process ultimately fails when two variables remain, L_s and D , and two differential equations, $\frac{dP_s}{dt}$ and $\frac{dM_s}{dt}$. Each differential equation can be expressed in factored form as

$$\frac{dP_s}{dt} = \frac{a(D, L_s)}{L_s^2 c(D, L_s)} \text{ and} \quad (2.3)$$

$$\frac{dM_s}{dt} = \frac{b(D, L_s)}{L_s c(D, L_s)} \quad (2.4)$$

where $a(D, L_s)$ is a third degree polynomial in D and a fourth degree polynomial in L_s and $b(D, L_s)$ is a third degree polynomial in both variables. Neither differential equation can be solved for either remaining variable using symbolic algebra in MATLAB.

Despite not having expressions for D^* and L_s^* , we proceed in search of support that an endemic equilibrium exists. We see that we have an endemic equilibrium iff there exists a pair (D^*, L_s^*) such that $a(D^*, L_s^*) = b(D^*, L_s^*) = 0$. These equations cannot be solved explicitly for either remaining state variable. However, it can be shown (using an intermediate value theorem argument) that $a(D, L_s)$ and $b(D, L_s)$ each must have a non-empty set of positive roots. Moreover, in a sweep of the biologically feasible parameter space, we demonstrate that the curves of solutions to each equation always intersect.

Numerical investigations suggest that exists a pair (D^*, L_S^*) such that $a(D^*, L_S^*) = b(D^*, L_S^*) = 0$ throughout the biologically feasible parameter space. We use Latin Hypercube Sampling with $N = 1000$ samples varying $r_L, \gamma, g_P, \mu_D, \mu_L, \mu_B, \beta_L, \rho, \sigma, \delta_P$, and ω between $10^{-2}\alpha$ and $10^2\alpha$ where α is the default value listed in Table 2.1. Parameters that depend on the varied parameters were co-varied. To ensure that the sampled parameter space is biologically feasible, we remove samples in which $\gamma > \gamma_0 > 0$ does not hold. This removes, on average, approximately 25% of samples. In case this removal left regions of parameter space insufficiently sampled, we perform a second sweep that restricts parameters that positively influence host growth (r_l, γ , and g_p) to $10^{-2}\alpha$ and $10^1\alpha$ and parameters that negatively influence host growth (μ_D, μ_L , and μ_B) to $10^{-1}\alpha$ and $10^1\alpha$, where α is the default value listed in Table 2.1. We sample with $N = 10,000$ and again remove samples in which $\gamma > \gamma_0 > 0$ does not hold (approximately 95% of samples).

To find the pair (D^*, L_S^*) for each parameter combination, we discretize the set of positive roots $\{(L_S, D^a)\}$ to $a(D, L_S)$ and $\{(L_S, D^b)\}$ to $b(D, L_S)$ by evaluating each expression at values of L_S and approximating the values of D_a and D_b using the function `solve`. We inspect the discretized set of roots to each equation and search for an intersection of the curves in the form of a sign change in the expression $D^a - D^b$ at values of D^a and D^b that are positive and real. A positive, real pair (D^*, L_S^*) was located at every combination in each sweep. Thus, there appears to always be a pair of positive values for the seed population and the large susceptible tree population that are roots of both $a(D, L_S)$ and $b(D, L_S)$. Using these approximations of D^* and L_S^* , we can approximate the values of all states at the endemic equilibrium using the algebraic expressions above. For example, the endemic equilibrium at the default parameter values is given by $(D^*, P_S^*, M_S^*, L_S^*, B_S^*, P_I^*, M_I^*, L_I^*, B_I^*, X^*) = (0.5438, 0.4805, 0.7056, 0.0719, 0.6889, 0.0072, 0.5292, 0.4315, 3.2595, 12.6864)$. We conclude that numerical experiments support the existence of an endemic equilibrium throughout the biologically feasible parameter space.

Numerical Support of Stability of Endemic Equilibrium

To determine the stability of the equilibrium, we investigate the eigenvalues of the Jacobian of the system evaluated at the endemic equilibrium. The eigenvalues of this 10×10 system cannot be computed algebraically. Thus we resort to numerical experiments. For each parameter combination in the sweeps, we evaluate the Jacobian of the system at both the parameter values and at the approximations of the state variables at the endemic equilibrium computed previously. We compute

the eigenvalues of the resulting matrix. Throughout the biologically feasible parameter space, the eigenvalues of the Jacobian have negative real part. We conclude that numerical experiments support the asymptotic stability of the endemic equilibrium throughout the biologically feasible parameter space.

Numerical Support of Global Stability

To determine the global stability of the endemic equilibrium, we confirm that numerical approximations of the solutions to the ODE system (computed via the MATLAB function `ode45`) converge to the theoretical endemic equilibrium. For each parameter combination, we approximate the value of D^∞ and L_S^∞ by running the model for 10,000 years or until equilibrium is reached and note the number of seeds and large trees. We compare the approximations of (D^*, L_S^*) against the values of (D^∞, L_S^∞) . The Euclidean difference between these two points can be made less than 10^{-2} trees throughout the vast majority of the biologically feasible parameter space by using an increasingly fine mesh to compute D^* and L_S^* . (This is not the case when host life-cycle parameters are restricted and when β is less than 1% of the default value. In this case, solutions the pairs (D^*, L_S^*) and (D^∞, D^∞) can be distant, but both pairs are sufficiently far from the origin. We blame the inconsistency on numerical error within `ode45`, `solve`, or both for exceptionally small values of β .)

We also evaluate $|a(D^\infty, L_S^\infty)|$ and $|b(D^\infty, L_S^\infty)|$ and confirm the values is near zero to confirm the system is at equilibrium, which would suggest the equilibrium reached is the endemic equilibrium. The results are always less than 10^{-19} and does not vary with the size of the sampled parameter space or number of parameter combinations considered. This is numerical support of the global stability of the endemic equilibrium throughout the biologically feasible parameter space.

2.3.2 Long-Term Function of Basal Sprouts in Disease

The production and infection of basal sprouts does not affect the stability of the endemic equilibrium, but each are seen to have profound effects on the size of both host and vector populations at the endemic equilibrium. In the absence of secondary infection ($\sigma = 0$), the production of basal sprouts yields a larger the host population at endemic equilibrium. While the host population can persist without basal sprouting ($\rho = 0$) when seed production and germination are high (γ and r_Z 200% of default), the number of susceptible medium trees at endemic the equilibrium is nearly zero thus not biologically feasible when $\rho = 0$, In either case, the number of

susceptible medium trees at the endemic equilibrium increases approximately linearly as ρ increases (Figure 2.3). Other classes of susceptible host follow similar trends.

In the case of basal sprout production with secondary infection ($\rho > 0, \sigma > 0$), the production of basal sprouts increases the host population compared to that with no production for small rates of basal sprout infection. In particular, when the ratio of basal sprout infection to basal sprout production is low ($\frac{\rho}{\sigma} < 10^{-3}$), the effect of secondary infection is negligible. Host populations at equilibrium are only slightly smaller than they are with no secondary infection. However, as basal sprout infection increases relative to production, the utility of basal sprouts decreases and host populations at equilibrium decline significantly (Figure 2.4). When seed production and germination are low, high ratios of basal sprout infection to production yield medium tree population sizes at equilibrium near zero (Figure 2.4A), comparable to the population at equilibrium without basal sprout production ($\rho = 0$). When seed production and germination are low, high ratios of basal sprout infection to production yield medium tree population sizes at equilibrium much larger than zero. However, the population is smaller than that with no basal sprout production (Figure 2.4B).

2.4 Discussion

Our results show that the production and infection of basal sprouts by infected trees in response to disease are unlikely to affect the short-term dynamics of the disease but can profoundly affect the size of host population over larger time scales. Basal sprouts do not affect the initial spread of disease and tree mortality because the time scale of initial disease dynamics is much faster than the time scale of basal sprout production. If disease dynamics could be artificially slowed through active management (such as sanitation efforts to remove infected trees as has been done in beech bark disease (Heyd, 2005) and Dutch elm disease (Scheffer et al., 2008)), then early production of basal sprouts could affect the initial dynamics of disease spread.

Over larger time scales, basal sprout production yields a larger host population and is necessary to maintain the host population when reproduction via seeds is unlikely. The efficacy of basal sprout production in either case decreases as secondary infection increases. When compared to no sprouting at all, basal sprouting with very high rates of secondary infection yields a similarly exhausted host population when seed production and germination are low but an even smaller host population when the seed production and germination are high. This suggests that basal sprouts should be considered even when reproduction via seeds is not fully limited by disease. Moreover, this suggests

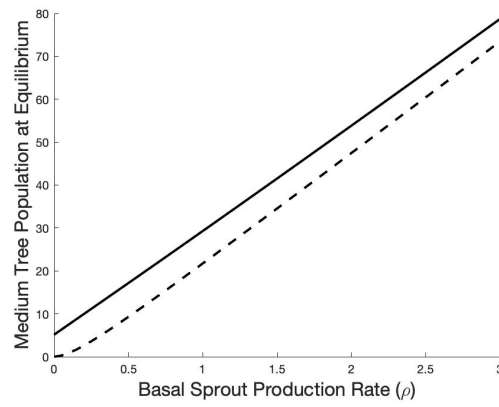


Figure 2.3: Medium tree population size at equilibrium with no secondary infection for low (dashed, 50% of default) and high (solid, 200% of default) seed production and germination: The size of the medium tree population at the endemic equilibrium increases as basal sprout production rate increases when there is no secondary infection in both cases.

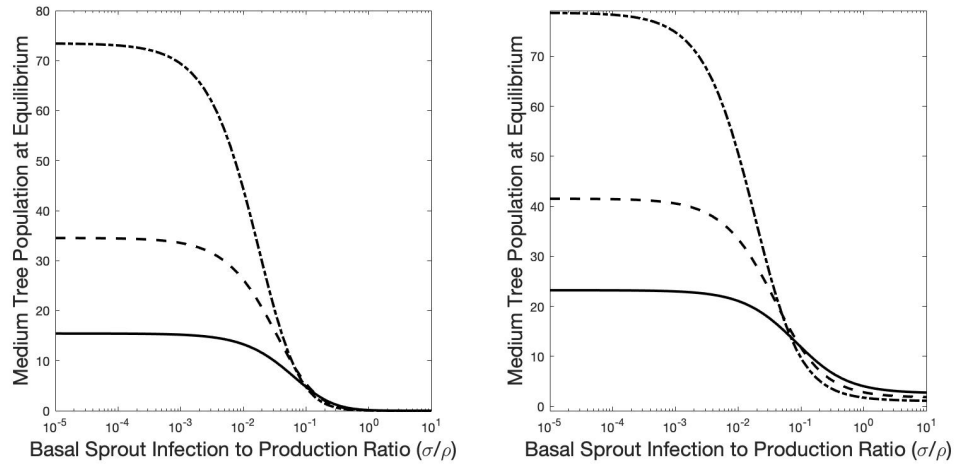


Figure 2.4: Medium tree population at equilibrium size with secondary infection for low (left, 50% of default) and high (right, 200% of default) values of seed production and germination: As basal sprout secondary infection increases, the medium tree population at equilibrium declines for low (solid), default (dashed), and high (dot dashed) basal sprout production.

that management strategies that protect susceptible basal sprouts from secondary infection could sufficiently increase the host population to save the host from local extinction.

The dynamics of this mathematical model are consistent with time-series data of disease progression in redbay. Field work in redbay shows that vector populations exhibit great increase immediately after introduction and that both host and vector populations exhibit substantial decline over 2-5 years. In many locations, vector populations are maintained at very low levels for many years after the initial infestation, and in others no beetles were trapped in post-epidemic plots (Cameron et al., 2015; Maner et al., 2014; Hanula et al., 2008). It was hypothesized that vector populations were being maintained through the use of small stem for brood production, despite their poor quality as host material. Our model supports this hypothesis because it yields an endemic equilibrium with positive host and vector populations, and those populations are very small under poor host growth and high basal sprout secondary infection. While our model does not predict extinction, it does allow for population levels so small that extinction is likely through stochastic events (e.g. increased vulnerability due to the small population paradigm (Caughley, 1994)).

Time series data on disease progression in sassafras (outside of the range of redbay) is not available, but our results give a framework of what to expect in laurel wilt in sassafras stands. Sassafras does not respond to laurel wilt infection by producing basal sprouts, but sassafras has been known to produce epicormic shoots off the trunk or lower crown of the tree. These epicormic shoots can grow into branches and persist without infection symptoms for several years, despite infection in the main stem (Cameron et al., 2015). It is unclear how the production of epicormic shoots will affect disease dynamics. If matured epicormic shoots function as independent trees themselves, then our mathematical model suggests that the high rate of epicormic shoot production and low rate of secondary infection will maintain the population size. However, if the placement of epicormic shoots on decaying stems inhibits them from maturing into independent, tree-like stems, then our mathematical model suggests that epicormic shoot production is unlikely to preserve host populations from extinction. Further study of the function of epicormic shoots in sassafras trees infected with laurel wilt is merited.

The formulation and results of our model aligns to some degree with other ordinary differential equation models that do not specifically consider basal sprout production in vector-borne plant disease systems. Models of Dutch elm disease include host stage structure and dependence of the vector population on infected host material as does our model, but they do not isolate basal sprout

production (Swinton and Gilligan, 1996). Models of pine wilt disease have similar transmission terms as in our model, but many assume a constant influx of vector population independent of available material, which is inconsistent with redbay ambrosia beetle reproduction (Shi et al., 2014; Lee and Kim, 2013). In the models for each of these diseases, vectors can be non-carriers of the pathogen. This assumption is also poor for laurel wilt due to the obligate nature of the vector-pathogen duplex. Despite these differences, the dynamics of our model generally align with the dynamics of these aforementioned vector-borne disease models that predict either a stable disease free equilibrium or a stable endemic equilibrium, depending on the size of the basic reproductive number. Our model also yields a stable endemic equilibrium, but does not yield a disease free equilibrium for a majority of the biologically feasible parameter space because a limit on host growth is not incorporated. A basic reproductive number thus was not calculated; however, we find that the endemic equilibrium is always stable, with trajectories never experiencing unbounded growth with or without disease.

Of course, there are disease systems to which this direct model does not apply. As a model for vector-borne tree disease, our model captures both the epidemiological impact of vector population size on disease progression and the ecological impact of disease progression on vector population size. Tree diseases transmitted without a vector are likely to have different dynamics and should be modeled differently. However, basal sprouts are also commonly produced in response to diseases with other modes of transmission, so similar questions are relevant and have been studied both empirically and with mathematical models.

In the case of airborne tree disease systems, the production of basal sprouts maintains the population despite high disease-induced mortality. For example, the fungal pathogen (*Cryphonectria parasitica*) that causes Chestnut Blight kills the main stem of American chestnut (*Castanea dentata*) and prevents seed production, but infected American Chestnut can regenerate through the still-living root system (Paillet, 2002). Most sprouts of mature American Chestnut are ripped out when the main stem falls, but the sprouts of infected saplings survive; thus, nearly all mature American Chestnut trees alive today in New England are the matured sprouts of blight infected saplings (Paillet, 1984, 2002). The tree disease Sudden Oak Death is caused by an airborne, fungus-like pathogen (*Phytophthora ramorum*) and causes high mortality in mature coast live oak (*Quercus agrifolia*) and tanoak (*Notholithocarpus densiflorus*). Infected trees respond to infection by producing basal sprouts while root systems are still intact, and mathematical models suggest that this production will maintain the tree populations post-epidemic (Cobb et al., 2012). In the

sense that basal sprouting in an airborne tree disease maintain the host population away from extinction, our results are consistent with models of airborne diseases. However, we find the case of vector-borne diseases that basal sprouting can only maintain the population for small rates of basal sprout infection via secondary transmission.

All mathematical models include assumptions about population interactions that could affect model outputs. Most assumptions in our model are supported by empirical evidence, but at times sufficient empirical evidence is lacking to support one assumption over another. In particular, little is known specifically about how the beetle and infected host trees interact, so assumptions regarding this interaction had to be made. We have assumed that vectors are more strongly attracted to large stems than small stems (i.e. $\beta_P < \beta_M < \beta_L$). If this assumption were relaxed, we expect host mortality to be spread more evenly over stage classes rather than being highest for large trees. This could slow the speed of disease transmission because medium and small trees offer less volume of infected material for vector reproduction. We have also assumed that all equivalent infected volumes support equivalent vector reproduction. While this is mathematically more tractable, in reality, galleries in small diameter stems produce smaller broods and have longer maturation times than galleries in large diameter stems (Maner et al., 2014). As a result, our model could overestimate the utility of infected basal sprouts for vector reproduction and thus overestimate vector population at equilibrium. This could mean that the vector population could go locally extinct (as per the small population paradigm) if infected basal sprouts are of insufficient quality for substantial vector reproduction. Lastly, it is unclear how vector utilization of host material for reproduction contributes to the degradation of material. It is possible that this degradation is density independent - occurring to the same degree regardless of vector population size - or density dependent, with more degradation occurring when the vector population is large. With an absence of empirical evidence, we have assumed density dependent degradation. An exploration of the consequences of this assumption is planned in future work.

We have created a mathematical model for the epidemiology of a vector-borne lethal tree disease with host stage structure and basal sprout production by infected trees using laurel wilt as a model system. We assumed all vectors carry the pathogen and that transmission could occur via contact with vector or through direct contact between infected trees and their attached basal sprouts. We found that in vector-borne tree disease systems, the production of basal sprouts by an individual infected tree affects the disease dynamics on the population level. The disease remains endemic regardless of basal sprout production or infection, but the host population is maintained at high

levels only when basal sprout infection is sufficiently infrequent when compared to basal sprout production. When basal sprout infection is high, the host population levels many years after initial introduction are the same or lower than in the absence of basal sprout production.

Basal sprout production and infection can have profound effects on disease dynamics in trees. When a new vector-pathogen duplex is introduced into an ecosystem, rapid information gathering about a number of factors will be critical to designing rapid, responsive best practices for management and mitigation. These, at least, include the rate of production of basal sprouts by infected trees, the rate of secondary infection of basal sprouts via root systems, the nature of disease transmission, host association, and vector biology. When these factors together reveal the potential reliance on basal sprout production for tree population persistence, management should potentially explore such interventions as active clearance of infected biomass, while otherwise strategies may be more effective when focusing on interruption of the spread of the vector. Models, such as the one here presented, provide a critical tool for proactive management as global spread of vectors and pathogens expose old forests to new threats.

Chapter 3

Basal Sprout Centered Management of Vector-Borne Diseases

This work has not yet been published.

Kelly Ruth Buch developed and analyzed the mathematical model presented in this chapter under the mentorship of Nina H. Fefferman. Kelly Ruth Buch drafted the manuscript, which was revised by both Kelly Ruth Buch and Nina H. Fefferman.

Abstract:

Non-native, lethal forest pathogens introduced via international trading routes pose a serious threat to forest ecosystems. After introduction, novel pathogens spread quickly and have devastating effects on susceptible tree populations. Pathogens that are introduced along with an insect vector have especially profound effects because the mobility of the insect substantially increases the spread of the disease. To eradicate the forest pathogen and/or control the spread of the pathogen-vector complex, rapid and effective management is necessary. Any management of a forest system is expensive and labor-intensive, so only methods with high chances of success should be used. Using laurel wilt as a model system, we consider various management strategies that prevent basal sprout infection or remove infected or susceptible basal sprouts. We test these strategies for effectiveness in reducing disease, lowering vector population, and maintaining host population in a vector-borne disease system by integrating management strategies into our mathematical model for lethal, vector-borne tree diseases. We analyze the existence and stability of equilibrium the ODE system and use this analysis to make recommendations to forest managers on how to effectively eradicate an introduced vector-borne tree disease.

3.1 Introduction

An increasingly common class of emerging diseases are vector-borne tree diseases (e.g. Dutch elm disease, laurel wilt, mango sudden death syndrome) caused by novel introductions of bark and ambrosia beetles (Coleoptera: Scolytidae) into non-native ranges (Hulcr and Dunn, 2011). Bark and ambrosia beetles live and reproduce within in tree material and are usually associated with a mutualistic fungus (Ceratocystis, Ceratocystiopsis and Ophiostoma among others) that is either directly consumed by beetle larva (e.g. ambrosia beetles) or positively influences beetle reproduction or utilization of host tree material (e.g. bark beetles) (Hofstetter et al., 2015). Within the native ranges of bark and ambrosia beetles, the beetle and its associated fungus are not considered vectors of disease. Healthy trees in the native range are resistant to both low-density beetle attack and

the associated fungus. Therefore, bark and ambrosia beetles typically only colonize dead or dying trees with immune systems too weakened to fight initial beetle attack (Lieutier et al., 2004).

Outside the native ranges of bark and ambrosia beetles, healthy trees have resistance to low-density beetle attack; however, some healthy trees are not resistant to the non-native fungus. In these cases, initial attack of healthy trees by non-native bark and ambrosia beetles is unsuccessful, but the transfer of fungal material kills or weakens the tree sufficiently such that the tree is vulnerable to future beetle colonization. In these cases, the beetle and its associated fungus is considered a vector-borne disease and can cause near population level die-offs in a short time span. For example, redbay populations experience 90% mortality in 3 years due to laurel wilt (Cameron et al., 2015).

Emerging diseases can threaten local populations of trees and have been known to drive them extinct, so when preventive measures fail stop the introduction of non-native bark and ambrosia beetles, active management is necessary to eradicate, contain, or control the beetle population (Victoria, 2010). In a vector-borne tree disease system, management could directly target either the host or vector population. (Management for non-disease induced die-offs might be different.) In a majority of forest systems, management strategies that directly attack the vector population (such as insecticides, repellents, etc) are not feasible due to both cost and environmental concerns (Hughes et al., 2015). Instead, management for the disease often surrounds the host population, and commonly suggested strategies include sanitation (removal of healthy or infected trees) or lowering transmission (i.e. application of fungicides, severing of root systems to prevent underground transmission) (Hughes et al., 2015). To maximize the efficacy, a proposed management plan may target a specific subset of trees rather than all trees. A plan might choose to only target trees of a certain size (e.g., only mature trees, only saplings or small stemmed trees) and only trees of a certain infection status (e.g., only susceptible stems or only infected stems).

Shoots that proceed from the base or root system of tree, called basal sprouts, represent a potential candidates for host-centered management strategies. The production of basal sprouts is a common form of vegetative growth in angiosperms and can be induced in response to death or damage to the main trunk of a tree (Del Tredici, 2001). Basal sprouts are often produced by trees in response to infection with pathogens (e.g. redbay (*Persea borbonia*) trees infected with laurel wilt and wych elm (*Ulmus glabra*) trees infected with Dutch elm disease) or infestation with pests (e.g. green ash (*Fraxinus pennsylvanica*) infested with the emerald ash borer (*Agrilus planipennis*)) (Cameron et al., 2015; Peterken and Mountford, 1998; Kashian, 2016). When disease-induced

mortality is widespread and seed banking capacity is limited, basal sprouting represent the only naturally occurring regeneration of the host species. In both airborne and vector-borne diseases, production of basal sprouts can lead to host persistence, but the utility of basal sprout production diminishes in vector-borne diseases when vertical transmission from infected parent tree to basal sprout is high (See Chapter 2).

Basal sprouts may provide an effective target size class for management strategies for several reasons. Due to the small size of basal sprouts, it may be easier to implement management targeted at basal sprouts. For example, sanitation of basal sprouts is logistically easier than removing full size-tree. Moreover, smaller quantities of expensive fungicides may be required to protect small stems. Severing roots to disconnect adult tree root systems in species with high degree of root grafting may be infeasible, but severing shallow roots to disconnect basal sprouts from parent trees may be more feasible. For newly emerging diseases in particular, basal sprouts may be an especially useful size class to target. The development, testing, and implementation of diagnostic testing tools or fungicides for newly emerging diseases can take years, and by the time techniques are ready to use in a management plan, a majority of large stems might already have succumbed to disease, yielding a population in which basal sprouts are the dominant size class. In such situations, management plans that target larger stems are unlikely to be effective, but management strategies targeted at basal sprouts could be highly effective.

Any management strategy for vector-borne disease should aim to decrease the vector population indirectly to stop or slow disease transmission (Gilligan and van den Bosch, 2008). Additionally, because host mortality is often quite high for the beetle-tree systems mentioned, management plans should also aim to support host population size to minimize the risk of local extinction. A management strategy should be evaluated against these two aims before widespread implementation. Field studies that validate the efficacy of management strategies are ideal but can be costly and slow. As a cost-effective and timely alternative, mathematical modeling can be used to test the efficacy of management plans prior to implementation.

In this paper, we introduce a mathematical model for fungal forest pathogens transmitted by a symbiotic vector, such as bark or ambrosia beetles. We include in the model the production of basal sprouts as well as secondary transmission from parent trees. Using this model, we consider three management strategies that target basal sprouts: (1) methods that prevent basal sprout infection (i.e. application of fungicides, severing roots connected to infected parent tree), (2) methods that limit susceptible material (i.e. the removal of susceptible basal sprouts/prevention

of basal sprouting), and (3) methods that limit infected material (i.e. the removal of infected basal sprouts). We test the efficacy of these three management strategies in their ability to increase the host populations and decrease the vector populations at the long-term equilibrium, relative to their sizes without management. We consider the diversity of potential outcomes, parameterizing the model for both already existing diseases and hypothetical emerging diseases with characteristics consistent with known threats. We use the results of this analysis to make recommendations for active and efficient management to mitigate the effects of current or future vector-borne tree diseases.

3.2 Methods

We consider a simplification of the mathematical model presented in Chapter 2. Using an SI framework with host stage structure (e.g. as in [Castillo-Chavez et al. \(1989\)](#)), we model both the host and vector populations in a system of ordinary differential equations. We separate the host population into large stems (trees) and two classes of small stems (saplings if produced by healthy trees and basal sprouts if produced by infected trees). We further separate these classes according to infection status. The number of susceptible trees is denoted T_S , and the number of saplings is denoted P . Saplings are not considered susceptible to disease under the assumption that vectors would not attack small stems that lack the pathogen. However, due to the possibility of vertical transmission of fungal pathogen from an infected parent tree to a basal sprout, basal sprouts are considered susceptible to disease, with susceptible basal sprouts denoted B_S . Because the degree to which infected trees and basal sprouts contribute to vector reproduction depends on the total amount of tree material (rather than the number of trees), we measure the infected tree and basal sprout classes, T_I and B_I respectively, by total amount of infected material that is suitable material for vector brood production (using an arbitrary unit of measurement for volume). The vector population is noted by X and is assumed to always carry the fungal pathogen. We model obligate mutualism as it is the most severe potential outcome for tree hosts.

Susceptible trees produce saplings at rate r and infected trees produce susceptible saplings at rate ρ . Saplings and basal sprout (regardless of the infection status of their parent tree) mature into susceptible trees at rate g , and all non-infected host classes (P , T_S , B_S) suffer natural mortality at rates μ_P , μ_T , and μ_B , respectively. Infection transmission occurs in two ways. Susceptible trees can become infected via density-dependent contact with vectors, and susceptible basal sprouts become infected at rate σ (independent of vector population) through to vertical transmission from the

parent tree through the root system. All infected material degrades through vector utilization and leaves the infected classes in a density-dependent manner. The equations that capture these dynamics can be written as

$$\begin{aligned}
\frac{dP}{dt} &= rT_S - (g + \mu_P)P \\
\frac{dT_S}{dt} &= g(P + B_S) - \mu_T T_S - \beta X T_S \\
\frac{dB_S}{dt} &= \rho \frac{T_I}{v_T} - (\sigma + g + \mu_B)B_S \\
\frac{dT_I}{dt} &= v_T \beta X T_S - \delta T_I X \\
\frac{dB_I}{dt} &= v_B \sigma B_S - \delta B_I X \\
\frac{dX}{dt} &= \omega X \left(1 - \frac{v_0 X}{T_I + B_I} \right),
\end{aligned} \tag{3.1}$$

and diagram representing the flow of the host population is given in Figure 3.1. A description of parameter values are given in Table 3.1 (see Appendix A for parameter calculations).

3.2.1 Inclusion and Analysis of Management Strategies

We represent the implementation of basal sprout centered management as equivalent to decreasing specific parameters within the model. The prevention of basal sprout infection via fungicides or root severing is represented mathematically by decreasing the basal sprout infection rate, σ . The prevention of basal sprouting/removal of susceptible basal sprouts attached to infected trees can be represented by decreasing the basal sprout production rate, ρ . The removal of infected trees is represented by decreasing number of galleries supported by an infected basal sprout, n , where $n = v_B/v_0$. Partial efforts of any management strategies are represented by a percent decrease in each parameter, and immediate, fully-effective management is represented by a decrease of the associated parameter to zero. Unless otherwise stated, the parameter values used in numerical simulations presented are given in Table 3.1. The parameters are not fitted to data but instead chosen to represent a biologically reasonable values for the rates in a hypothetical system. When available, reasonable parameter values or magnitudes are calculated or estimated from data on various hardwoods (see Appendix A).

For each management strategy, we evaluate the effectiveness in the management strategy at increasing the host population and decreasing the vector population. Because the endemic equilibrium is stable throughout a majority of the biologically-feasible parameter space and neither

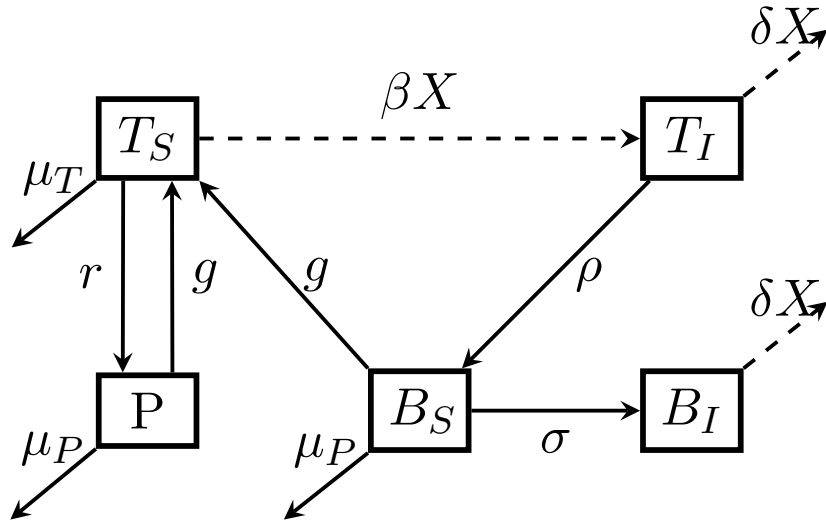


Figure 3.1: A flow diagram encapsulating growth and reproduction of the non-infected host population, infection transmission, and degradation of infected material.

Table 3.1: Description, notation, and values of parameters used in simplified model. Values are presented with two significant digits in decimal form.

Description	Symbol	Units	Value in Simulations
Host reproductive rate	r	1/day	$1.9 \times 10^{-4\dagger}$
Host maturation rate	g	1/day	$2.2 \times 10^{-4\dagger}$
Sapling natural mortality rate	μ_P	1/day	$1.0 \times 10^{-4\dagger}$
Tree natural mortality rate	μ_T	1/day	$5.2 \times 10^{-5\dagger}$
Basal sprout natural mortality rate	μ_B	1/day	$3.4 \times 10^{-6\dagger}$
Primary transmission rate	β	1/(day*beetle)	4.9×10^{-5}
Secondary transmission rate	σ	1/day	$2.2 \times 10^{-3\dagger}$
Basal sprout production rate	ρ	1/day	$2.2 \times 10^{-3\dagger}$
Vector per capita growth rate	ω	1/day	$2.3 \times 10^{-2\dagger}$
Infected material degradation rate	δ	1/(day*beetle)	$4.5 \times 10^{-5\dagger}$
Volume of infected tree	v_T	units ³	$4.0^\ddagger$
Volume of infected basal sprout	v_B	units ³	$1.0^\ddagger$
Volume of a beetle gallery	v_0	units ³	$1.0^\ddagger$

[†] See Appendix A for value citation and calculation. [‡] Value was assumed.

of the management-related parameters affect the stability (see Results), we consider the expressions for the non-infected trees (of all sizes) and the vector population as functions of each of the three management-related parameters. We specifically consider the derivative and y-intercepts of the functions, and we interpret these to conclude the compared outcomes increasingly effective management efforts and the outcomes of fully effective management efforts, respectively. We also consider the specific outcomes for several state variables as functions of each of the management-related parameters within various regions of the infection parameter space (i.e. with varied values of β , ρ , and σ) to show the diversity of potential outcomes.

3.3 Results

The system has two equilibria that exclude the vector population. A host-vector extinction equilibrium with all states zero (except possible B_I) always exists, and a vector-free equilibrium with P, T_S , and $B_I > 0$ and all other states zero exists when $r = r_0 := \frac{(g+\mu_P)\mu_T}{g}$. The Jacobian matrices evaluated at each equilibrium are identical, and its eigenvalues include $\omega > 0$. Thus, both are unstable for initial conditions with $X > 0$. For biologically-feasible initial conditions that exclude vectors, the non-infected host population either experience exponential growth ($r > r_0$) or decay ($r < r_0$).

The system has a unique endemic equilibrium, for which the algebraic expressions for certain state variables are given in Table 3.2. All states are positive for the entirety of the parameter space such that $r > r_0$, and the endemic equilibrium is stable throughout this space. In particular, existence and stability of the endemic equilibrium is independent values of σ, ρ , and n . Because the region corresponding to exponential growth in absence of disease is equivalent to the region of stability of the endemic equilibrium, we restrict further analysis to this region.

We simplify analysis by considering X^* , T_S^* and B_S^* as functions of each of the management-related parameters (and do not consider P^* directly). Indeed, the sapling population can both be expressed as multiples of the mature tree population at equilibrium, and the multiplicative factor does not depend on σ , ρ , or v_B . Thus, T_S^* and P^* will exhibit similar dynamics in all three situations. The expression for the sprout population at endemic equilibrium can also be expressed as a multiple of the mature tree population, but the multiple depends on both ρ and σ . Accordingly, we consider B_S^* independently from T_S^* , except when considering the effects of removing infected basal sprouts. A summary of the effects of each management strategy is given in Table 3.3, and a detailed explanation follows.

Table 3.2: Algebraic expressions for live host trees and vector populations at endemic equilibrium.

State Variable	Expression for State at Endemic Equilibrium
Saplings	$P^* = \frac{r}{g+\mu_p} T_S^*$
Susceptible Mature Trees	$T_S^* = \frac{[\beta g \rho (g+\mu_p) + \delta (g r - \mu_t (g+\mu_p)) (g+\mu_b + \sigma)]^2}{\beta^2 (g+\mu_p) (g+\mu_b + \sigma) \left[\beta \rho (g+\mu_p) \left(\frac{g v_t + \sigma v_b}{v_0} \right) + \frac{v_t}{v_0} \delta (g r - \mu_t (g+\mu_p)) (g+\mu_b + \sigma) \right]}$
Susceptible Basal Sprouts	$B_S^* = \frac{\beta \rho}{\delta (g+\mu_b + \sigma)} T_S^*$
Vector	$X^* = \frac{g \rho}{\delta (g+\mu_b + \sigma)} + \frac{(g r - \mu_t (g+\mu_p))}{\beta (g+\mu_p)}$

Table 3.3: Summary of management strategies and their effect on each on each state variable at endemic equilibrium.

Description	Management	Parameter	Effect on Population		
			Sapling & Tree	Basal Sprout	Vector
Application of Fungicides/Root Severing		σ	increase	increase	increase.
Prevention of Basal Sprout Production		ρ	decrease*	decrease	decrease
Removal of Infected Basal Sprouts		$n = v_B/v_0$	increase	increase	no effect

* Sapling and tree populations can increase slightly with decreasing ρ when σ is high.

3.3.1 Efficacy of Preventing Basal Sprout Infection

We first consider the effects of methods that prevent basal sprout infection (i.e. application of fungicides onto basal sprouts or root severing) by considering the X^* , T_S^* , and B_S^* as function of the rate of basal sprout infection σ . We see that all expressions are rational with respect to σ and that $\frac{dX}{d\sigma} < 0$, $\frac{dT_S^*}{d\sigma} < 0$, and $\frac{dB_S^*}{d\sigma} < 0$ for $\sigma \geq 0$. Thus, the application of fungicides on basal sprouts or severing of roots will increase the vector population and all non-infected host populations at equilibrium. Examples of the graph of T_S^* , B_S^* and X^* as functions of σ in various regions of the parameter space are shown in Figure 3.2. The asymptotic behavior of the graphs show that low efficacy of methods that prevent basal sprout infection yields a small change in all population sizes, but application with 100% efficacy ($\sigma = 0$) significantly increases all populations. When the primary transmission rate, β , is high, fully effective management yields a smaller population of mature trees than when β is low. However, β has little effect on the efficacy of the management strategy on the basal sprout and vector populations.

3.3.2 Efficacy of Removing Susceptible Basal Sprouts

We next consider the effects of removing susceptible basal sprout/preventing basal sprout production by considering the X^* , T_S^* , and B_S^* as functions of the rate of basal sprout production ρ . The expression for X^* is linear with respect to ρ and $\frac{dX}{d\rho} > 0$. The slope of the line decreases with increasing σ such that, when secondary infection is high, equivalent efforts of basal sprout removal has a lessened effect than when secondary infection rate is low. The y-intercept of X^* as a function of ρ decreases with increasing β . Thus, when the primary transmission rate is high, fully effective efforts of basal sprout removal will yield a smaller vector population than when primary transmission rate is low. This is demonstrated in Figures 3.3A and 3.3B.

Now, P^* is also rational with respect to ρ and is concave up for $\rho > 0$. It has a local minimum that occurs when $\rho = \rho_0$ for some ρ_0 . The sign of ρ_0 (and its biological feasibility) varies with σ and v_B . If $\sigma v_b < gv_T$, the location of the local minimum, ρ_0 , is negative and thus $\frac{dT_S^*}{d\rho} > 0$ for all $\rho \geq 0$. In this case, any attempt to remove or suppress susceptible basal sprouts will yield a reduced population of saplings and trees at equilibrium compared to no management, and larger efforts produce a further reduced population. Conversely, if $\sigma v_b > gv_T$, then P^* has a local minimum at $\rho_0 > 0$. In this case, larger efforts may not produce a further reduced population. This is demonstrated in Figures 3.3C and 3.3D, which show T_S^* as a function of ρ for several rates of secondary infection. When σ is large, the size of T_S^* is approximately the same for both 100%

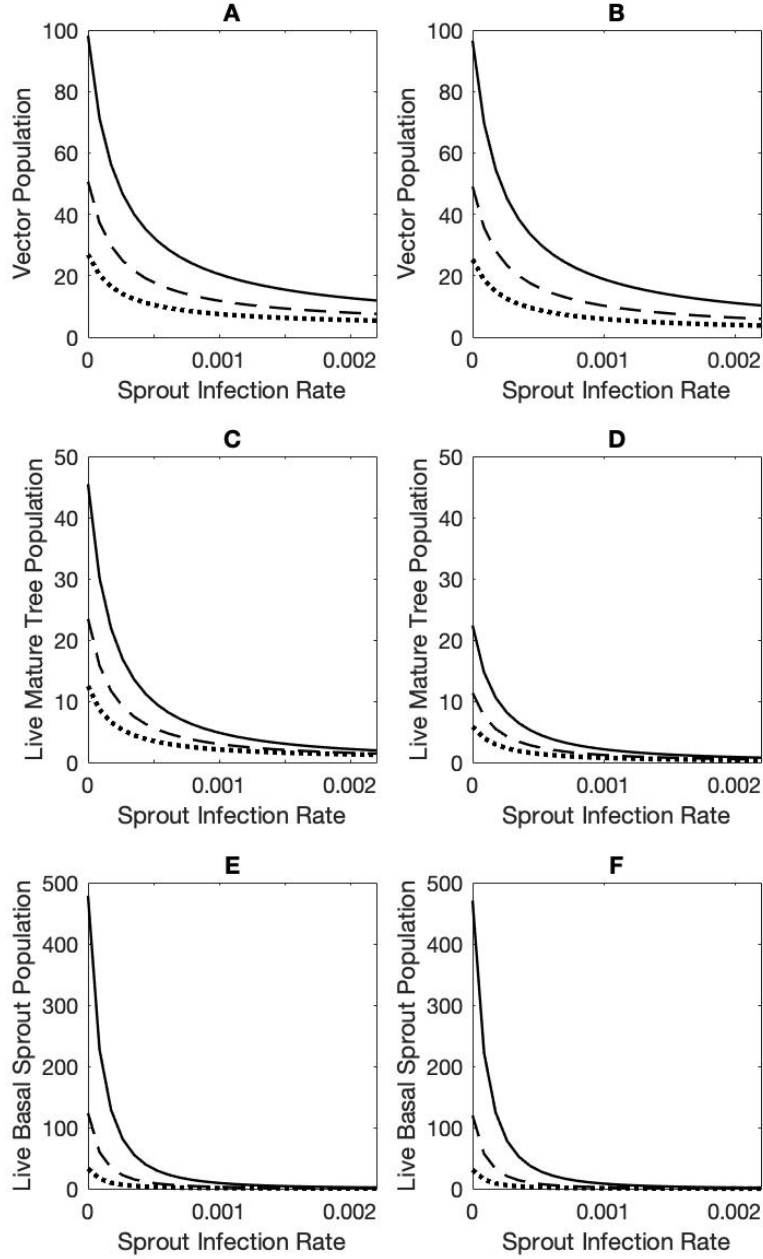


Figure 3.2: Abundance of vectors (top), mature trees (middle), and sprouts (bottom) for low ($\beta = 2.429 \times 10^{-3}$, left) and high ($\beta = 4.8857 \times 10^{-3}$, right) primary transmission for low ($\rho = 1.1 \times 10^{-3}$, dotted), moderate ($\rho = 2.2 \times 10^{-3}$, dashed), and high ($\rho = 4.3 \times 10^{-3}$, solid) rates of basal sprout production as functions of the basal sprout infection. All populations increase as basal sprout infection is suppressed.

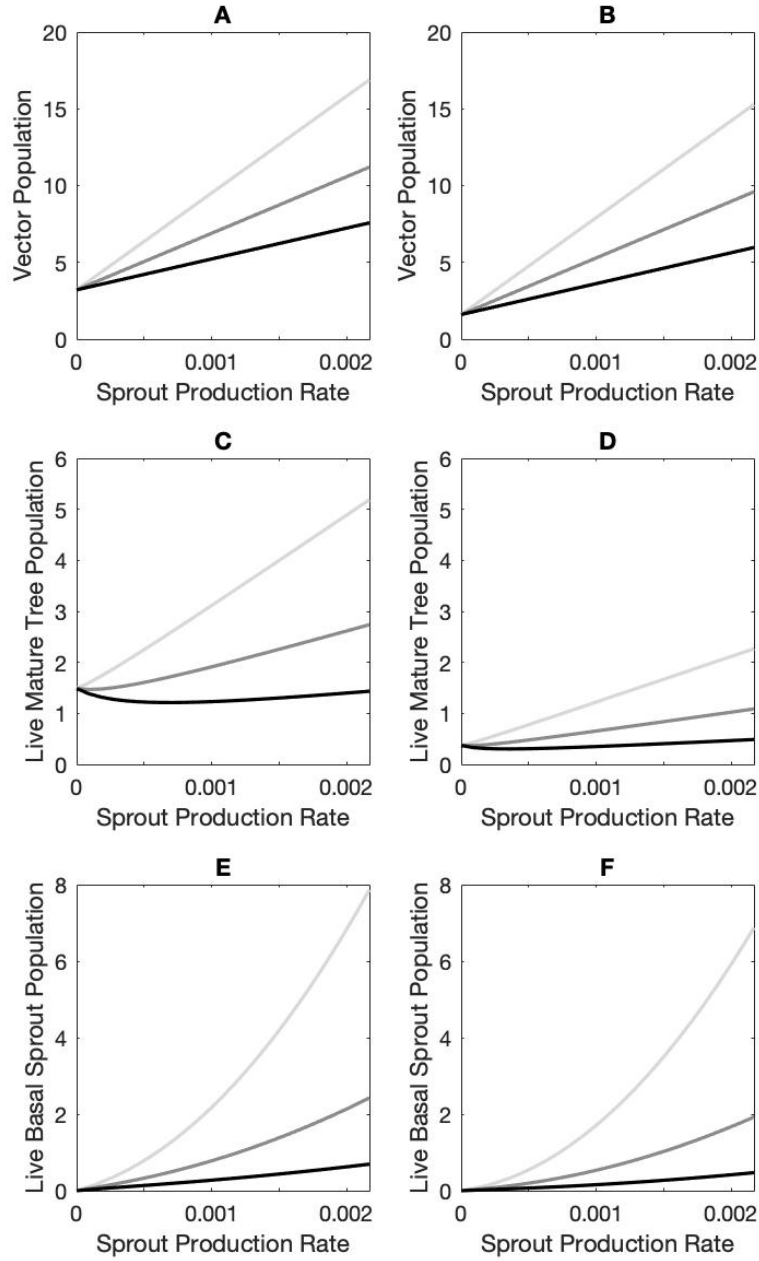


Figure 3.3: Abundance of vectors (top), mature trees (middle), and sprouts (bottom) for low ($\beta = 2.429 \times 10^{-3}$, left) and high ($\beta = 4.8857 \times 10^{-3}$, right) primary transmission for low ($\sigma = 0.6 \times 10^{-3}$, light grey), moderate ($\sigma = 1.1 \times 10^{-3}$, dark grey), and high ($\sigma = 2.2 \times 10^{-3}$, black) rates of basal sprout infection as functions of the basal sprout production. All populations decline as basal sprout production decreases.

effective efforts of basal sprout removal ($\rho = 0$) and for no efforts of basal sprout removal (ρ large). Both are larger than for semi-effective efforts of basal sprout removal (ρ moderate). When σ is moderate, the size of T_S^* for 100% effective efforts of basal sprout removal ($\rho = 0$) is slightly larger than for moderate efforts of basal sprout removal, but both are significantly larger than with no efforts of basal sprout removal. When σ is small, the size of T_S^* for 100% effective efforts of basal sprout removal ($\rho = 0$) smaller than for moderate efforts of basal sprout removal, which is smaller than with no efforts of basal sprout removal.

The expression B_S^* is rational with respect to ρ , and $\frac{dB_S^*}{d\rho} > 0$ for $\rho \geq 0$ regardless of the value of σ . Thus, any attempt to prevent basal sprout production will decrease the vector population at endemic equilibrium, with a more severe decrease when primary transmission is high. Fully effective efforts will reduce the population to zero in either case. This is demonstrated in Figures 3.3E and 3.3F.

3.3.3 Efficacy of Removing Infected Basal Sprouts

Finally, we consider the effects of removing susceptible basal sprout/preventing basal sprout production by considering the X^* , T_S^* , and B_S^* as functions of the number of galleries supported by an infected basal sprout, n . Neither v_B nor v_0 appear in the expression for X^* , so X^* is constant with respect to n . Thus, the removal of infected basal sprouts does not affect the size of the vector population at equilibrium. In T_S^* , n only occurs linearly in the denominator, and $\frac{dT_S^*}{dv_B} > 0$ for all v_B . Because the B_S^* is a multiple of B_S^* , and that multiple does not depend on v_B , we have that $\frac{dB_S^*}{dv_B} > 0$ for v_B as well. This implies the removal of infected basal sprouts will increase all live host populations. Efforts are more successful when primary transmission is low, but fully effective removal of infected basal sprouts can maintain the the live tree populations, especially when sprout infection is low and/or sprout production is high, as shown in Figure 3.4.

3.4 Discussion

An ideal management strategy for a novel, vector-borne tree disease should both maintain the host population and decrease the vector population. However, the results of this mathematical model suggest that basal sprout centered management cannot achieve both goals. Of the three management strategies considered, two (removing susceptible basal sprouts and preventing basal sprout infection) increase the host population. Of these two, neither yield a reduced vector population. Methods that prevent basal sprout infection increase the vector population substantially, while the removal of infected basal sprouts has no affect on the long-term size of

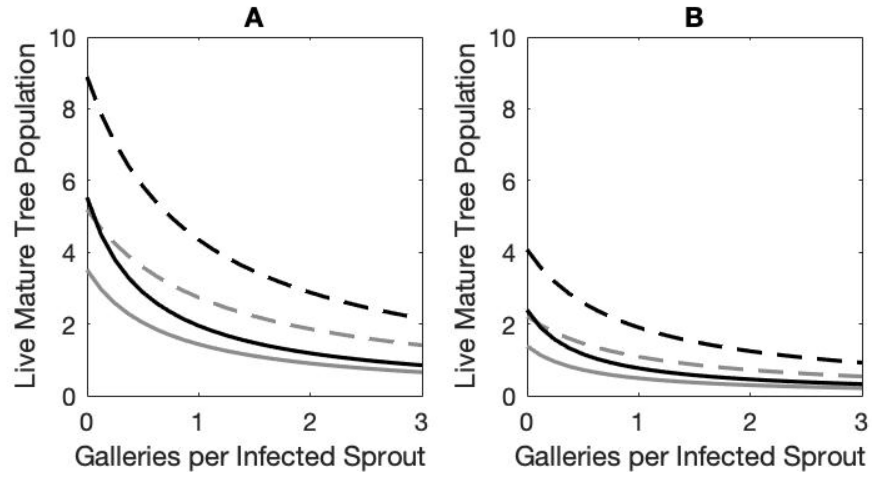


Figure 3.4: Abundance of mature trees for low ($\beta = 2.429 \times 10^{-3}$, left) and high ($\beta = 4.8857 \times 10^{-3}$, right) primary transmission for moderate ($\sigma = 1.1 \times 10^{-3}$, dark grey) and high ($\sigma = 2.2 \times 10^{-3}$, black) rates of basal sprout infection and moderate ($\rho = 2.2 \times 10^{-3}$, dashed) and high ($\rho = 4.3 \times 10^{-3}$, solid) rates of basal sprout production as functions of the number of galleries supported by an infected basal sprout. The mature population of trees increase as the number of galleries supported by an infected basal sprout is reduced.

the vector population. Preventing basal sprout production is the only method considered that will decrease the vector population. However, this strategy naturally yields a decreased basal sprout population. Low to moderate attempts at preventing the growth of or removing basal sprouts will also yield a lower tree population. Depending on the natural rates of basal sprout production and infection, extreme efforts to reduce basal sprout production can yield a larger tree population than low or moderate efforts. However, this change is likely still lower than the expected number of trees without management, so we conclude that prevention of basal sprouts production will not yield a larger tree population in most circumstances.

Because none of the strategies examined can attain both goals of an ideal management strategy, wildlife managers must prioritize one over the other. The decision between these management strategies is dependent on secondary priorities including the importance of the size of the vector population and the desired size of the host population and well as the burden of implementing the control in the specific system including both financial and logistical considerations of implementing the control.

In some cases, it might be more important to prevent the depletion of the host population in the forest system. This approach might be most important if the at-risk host species is of high economic-value, a dominant species in the forest, or depended upon by threatened fauna. In this case, managers might opt to remove infected basal sprouts or use management strategies that cure or prevent basal sprout infection.

In other situations, it might be more reasonable to prioritize indirectly limiting the vector population through host-centered management, especially if limiting the spread of the fungal pathogen is of high concern. Large vector populations can spread the fungal pathogen spatially through establishment in nearby forests without pathogen presence and locally through lateral transmission to native bark and ambrosia beetles (Carrillo et al., 2014). In this case, managers, might opt to remove susceptible basal sprouts and prevent the production to new ones. It is important to note, however, that this management strategy can decrease the vector population but cannot reduce the vector population to zero. (The numerator of the expression for X^* contains constant terms with respect to σ .) As such, managers should not expect eradication of the vector.

Of course, each management strategy considered involves biological and physical limitations in addition to those suggested by our mathematical evaluation. The protection granted by fungicides is short-lived, and fungicides must be reapplied within 1-1.5 years to remain effective at both preventative and curative efforts (Hughes et al., 2015). Prevention of basal sprout infection through

severing the root system requires heavy machinery, and some have questioned its feasibility in forest settings (Randolph, 2017). Furthermore, it is likely that only sprouts that have a sufficiently large, independent root system can survive being severed from a parent tree. The removal of basal sprouts also has biological limitations. The removal of basal sprouts, either susceptible or infected, will require routine surveillance of infected patches. If early infection of sprouts is asymptomatic, diagnostic methods must be used to determine infection status, and if diagnostic methods damage sprouts, susceptible sprouts may suffer higher rates of induced mortality. This could reduce the efficacy of removing infected basal sprouts.

Indeed, this mathematical model suggests that most host centered management is insufficient to eradicate the non-native vector population. Under the assumption that the size of the vector population depends on the total amount of infected material, the vector population will always be positive as long as any infected material is available. Mathematical analysis suggests that the only way to yield a long term state that excludes the vector population is to include a negative term in the differential equation for the vector population (for example, $\frac{dX}{dt} = \omega X \left(1 - \frac{v_0 X}{T_I + B_I}\right) - cX$ or $\frac{dX}{dt} = \omega X \left(1 - \frac{v_0 X}{T_I + B_I}\right) - c'$ for density dependent removal rate c or density independent removal rate c'). Biologically, this would represent active management against vectors such as the use of insecticides, traps, and repellents. The structure of the model suggests that these methods in combination with the aforementioned methods could drive the population to zero. However, these methods are generally quite expensive, difficult to implement in a forest settings, and damaging to native insects (Hughes et al., 2015), so this method has not been formally analyzed here. However, deeper analysis of this method is warranted if an introduced vector-borne disease infected trees of very high economic value or those that were critical to the stability of the forest ecosystem.

We have developed a mathematical model to capture the disease dynamics of a non-native fungal tree pathogen vectored by an introduced ambrosia or bark beetle. The model includes the production of basal sprouts by infected trees as well as possible transmission to basal sprouts via infected root systems and can be parameterized to represent past or present forest pests and pathogens (such as Dutch elm disease, laurel wilt) as well as future emerging diseases. Using the model, we considered three management methods that center around basal sprouts, including the prevention of basal sprout infection via application of fungicides or root severing, the removal/prevention of basal sprouts (pre-infection), and the removal of infected basal sprouts, and evaluated their ability to increase the host population and decrease the vector population (relative to no management). We determined that no basal sprout centered management strategy

can attain both goals. If the use of insecticides or repellents is not cost-effective for a disease system, managers should expect to have to prioritize maintaining the host population or reducing the vector population when determining management plans.

Chapter 4

Basal Sprouts and the Establishment and Spread of Vector-Borne Disease

This work has not yet been published.

Kelly Ruth Buch developed and analyzed the mathematical model presented in this chapter under the mentorship of Nina H. Fefferman. Kelly Ruth Buch drafted the manuscript, which was revised by both Kelly Ruth Buch and Nina H. Fefferman.

Abstract:

The establishment of a non-native pest that kills trees and its ability to spread spatially depend on the individual characteristics of the host and pest populations as well as characteristics of the interactions between them. Potentially important factors influencing these interactions include the susceptibility of the host population to pest attack, the production of basal sprouts by the host population in response to disease, and the degradation of host material following utilization by the pest. We develop a mathematical model for a vector-borne disease vectored by an introduced pest and extend the model over linearly interconnected patches with movement of the pest between patches. We analyze the mathematical model to determine characteristics that lead to either endemic or epidemic disease locally, and that lead to spread of disease spatially. When a wave of epidemic disease is predicted, we determine characteristics that affect the duration of the epidemic and the vulnerability of the local host population to a second wave of epidemic disease. Our results suggest that the production of basal sprouts by host species in response to disease increases the likelihood of endemic disease, and when epidemic disease spread occurs, the epidemic lasts longer and hosts are vulnerable to a second wave sooner, compared to species that do not respond to disease by producing basal sprouts. Because of this increased risk, management strategies for introduced pests that vector tree disease should specifically consider the production of basal sprouts when anticipating the impact of a novel introduction.

4.1 Introduction

When a non-native pest that kills trees as a necessary part of its reproduction is first introduced into a novel area, the fate of both the pest and the forest ecosystem are uncertain, falling into distinct cases (Gilligan and van den Bosch, 2008; Anderson and May, 1986). To establish, the introduced pest must be able to locate and use resources for consumption and reproduction in an unfamiliar environment. If there are not sufficient resources (or if the pest is inefficient at finding available resources), the introduced pest population could die out after little to no reproduction,

and the tree population could suffer little to no mortality. If instead the pest can locate sufficient resources for reproduction, the pest population will grow and the tree population could suffer high mortality. If the host population declines enough to become a limited resource, the pest population will decline as well. This is especially true of specialist pests, which can only consume or reproduce in specific species of trees (Godefroid et al., 2015). If reproduction of the affected host population can keep pace with pest-included mortality, then the pest may become endemic in the introduced area. If not, the pest population could go locally extinct as resources become scarce. If the pest can spread spatially and establish in new areas, the decline in locally available resources can favor spatial spread. This wave of either epidemic or endemic disease can devastate new areas, just as in the area of the introduction. Whether or not a newly introduced disease will trigger spatial spread (and if that spread leads to epidemic or endemic disease) is of importance to wildlife managers who must often enact management strategies before the long-term outcome of an introduction is known.

If an introduced pest causes an epidemic, the host population may recover (either naturally or through active management) after pest populations die out locally. If the pest epidemic also spreads spatially (and does not die out globally), the host may be vulnerable to a second wave of pest attack, if the pest is reintroduced after the host population has regenerated sufficiently. This has been the case with beech bark disease, in which a second wave of disease has caused mortality in beech (*Fagus grandifolia*) populations devastated by disease fifty years beforehand (Busby and Canham, 2011). Similarly, a stronger strain of pathogen caused a second wave of Dutch elm disease-induced mortality of elm (*Ulmus* spp.) just a few decades after a weaker strain ravaged Europe, Asia, and North America (Brasier, 1991). The susceptibility of the host population to a second wave of disease depends on a variety of factors including: inherited immunity or resistance in the regenerated host population, the ability for the pest to be reintroduced, and the size/composition of the host population at the time of introduction. The latter is especially important because, if the host population has not regenerated sufficiently, the pest population will not be able to reproduce and may again die out locally before causing substantial mortality to the host population. If a second wave of a pest epidemic is possible, special consideration must therefore be taken to avoid reintroductions.

Because these factors are of importance in making decisions and policies regarding new introductions of disease, theory that classifies pest and host characteristics that yield each of these outcomes is merited. In particular, classification of characteristics that affect the ability of the pest to spread spatially and establish or cause waves of disease would help wildlife managers

enact effective management strategies to prevent host decline. Important characteristics may include elements of the the host and pest lifespan, life cycle, and reproductive output. Additional characteristics of the pest population may include how the pest populations interacts with the host population, including attraction to hosts and the degree to which multiple generations of pests can reuse the same host material. Characteristics of the host population may include its reaction to pest attack, including both the time frame of pest-induced mortality and the production of basal sprouts following mortality of the main trunk of mature trees.

In particular, the way a pest interacts with a host population and the host response to pest attack affect the host composition and the ability of the host population to recover after the pest has died out locally. Some pests have preference for the size of stems they can utilize ([Kendra et al., 2014](#)). If pests have preference for large stems, regeneration via seedlings can be difficult due to the loss of large, seed-producing stems. Moreover, the host population may remain robust against reintroduction of disease for several generations, as the remaining saplings mature to a sufficiently large population of trees that are attractive to the pests. If trees respond to disease by producing basal sprouts, regeneration of the host population may be more rapidly attainable, even with the loss of large stems. While the production of basal sprouts can maintain the host population after disease (see [Chapter 2](#)), it could also increase the likelihood of low levels of endemic disease if pests can survive on smaller stems ([Cameron et al., 2015](#); [Maner et al., 2014](#)). If the pest cannot utilize smaller stems and the pest population dies out, the production of basal sprouts may increase the vulnerability of the host population to a second wave of disease through an increased population size and altered average basal area. Indeed, it has been suggested that the maturation of basal sprouts could lead to a second wave of laurel wilt in areas that experienced disease years before ([Cameron et al., 2010](#)).

In this paper, we investigate host and pest characteristics that may lead to each of the differential outcomes following pest introduction into novel areas and host populations. We specifically consider the introduction of a bark or ambrosia beetle that reproduces in dead/dying trees and that carries a fungus with is pathogenic to the host species (e.g. laurel wilt, Dutch elm disease). We develop a mathematical model for the introduction of a pest that causes vector-borne disease and extend the model over linearly connected patches of forest. Within the model, we allow for the movement of the pest population between interconnected patches, as well as the production of basal sprouts by infected trees. We analyze the model to consider the following questions: 1) Under what conditions does the introduction of a pest that vectors disease yield minimal disease with no spread,

endemic disease with spatial spread, or epidemic disease with spatial spread and 2) In the case of epidemic disease with spatial spread, what characteristics affect the vulnerability of the local host population to a second wave of epidemic disease? We interpret our results to suggest a framework for management of newly introduced pests that kill trees as the result of a vectored pathogen.

4.2 Methods

We introduce a mathematical model that is both a simplification and an extension of the model presented in Chapter 3, and we quote directly from its methods, as needed. We simplify the previous model by removing the secondary infection of basal sprouts. This is motivated by the results of Chapter 2 that indicate that high basal sprout infection yield similar equilibrium structures as when there is no basal sprout production at all. We extend the previous model to add complexity and specificity to the growth of the beetle population and includes stage structure, separating the pest population into adult and juvenile beetles (i.e. all pre-flight, larval stages of development). This is motivated by the need to separate the phenomenon of initial introduction of adult founding beetles, which can infect trees but do not require infected material for survival, from the phenomenon of beetle reproduction by adult beetles that requires sufficient infected material.

The resulting model is a six-dimensional epidemiological model with stage structure for both host and pest population. We separate the host population into large stems (trees) and two classes of small stems (saplings if produced by healthy trees and basal sprouts if produced by infected trees). We further separate these classes according to infection status. The number of susceptible trees is denoted T_S , and the number of saplings and basal sprouts are denoted P and B , respectively. Saplings and basal sprouts are not considered susceptible to disease under the assumption that pests would not attack small stems that lack the pathogen. Because the degree to which infected trees contribute to pest reproduction depends on the total amount of tree material (rather than the number of trees), we measure the infected tree class, T_I , by total amount of infected material that is suitable material for pest brood production (using an arbitrary unit of measurement for volume). The adult and juvenile beetle populations are denoted by X_A and X_J , respectively.

Susceptible trees produce saplings at rate r , and infected trees produce susceptible saplings at rate ρ . Saplings and basal sprouts mature into susceptible trees at rate g , and all non-infected host classes (P , T_S , B) suffer natural mortality at rates μ_P , μ_T , and μ_B , respectively. Susceptible trees can become infected via density-dependent contact with adult pests (juvenile beetles are flightless and do not infect new trees). All infected material degrades through pest utilization, leaving the

infected classes in a density-dependent manner with respect to juvenile beetles (brood development degrades tree material).

Birth of juvenile beetles is logistic with per a capita birth rate, b , dependent on the size of the adult beetle population and carrying capacity $\frac{T_I}{v_0}$, where v_0 is the volume of infected tree required for the formation of a beetle gallery. Juvenile beetles mature and suffer density dependent death at rates m and μ_J respectively. Adult beetles mature from the juvenile stage and suffer natural mortality at rate μ_A . We model obligate mutualism (the most severe potential outcome for tree hosts) and assume all beetles (adult and juvenile) carry the fungal pathogen. A diagram representing the flow of the host population is given in Figure 4.1 and a description of parameter values are given in Table 4.1. The full one-patch model with host and pest age structure is given by

$$\begin{aligned}
\frac{dP}{dt} &= rT_S - (g + \mu_P)P \\
\frac{dT_S}{dt} &= g(P + B) - \mu_T T_S - \beta X_A T_S \\
\frac{dB}{dt} &= \rho \frac{T_I}{v_T} - (g + \mu_B)B \\
\frac{dT_I}{dt} &= v_T \beta X_A T_S - \delta T_I X_J \\
\frac{dX_A}{dt} &= mX_J - \mu_A X_A \\
\frac{dX_J}{dt} &= bX_A \left(1 - \frac{v_0 X_J}{T_I}\right) - (m + \mu_J)X_J.
\end{aligned} \tag{4.1}$$

We extend this one-patch model over N patches (e.g. counties) containing forest connected linearly and include movement of adult beetles between connected patches. We assume adult beetles are equally likely to move in either direction, regardless of the size of the host population in adjacent patches. Accordingly, we set

$$\frac{dX_A^i}{dt} = mX_J^i - \mu_A X_A^i + c(X_A^{i-1} - X_A^i + X_A^{i+1})$$

for $1 \leq i \leq N$ where X_A^i and X_J^i denote the adult and juvenile populations in patch i , c is the rate of adult beetle movement, and $X_A^0 \equiv X_A^{N+1} \equiv 0$.

In the N-patch model, we include limitations on the life cycle of the beetles when the beetle population or the amount of infected material available for reproduction is too small. We assume that infection transmission cannot occur unless the adult beetle population in that patch is at least

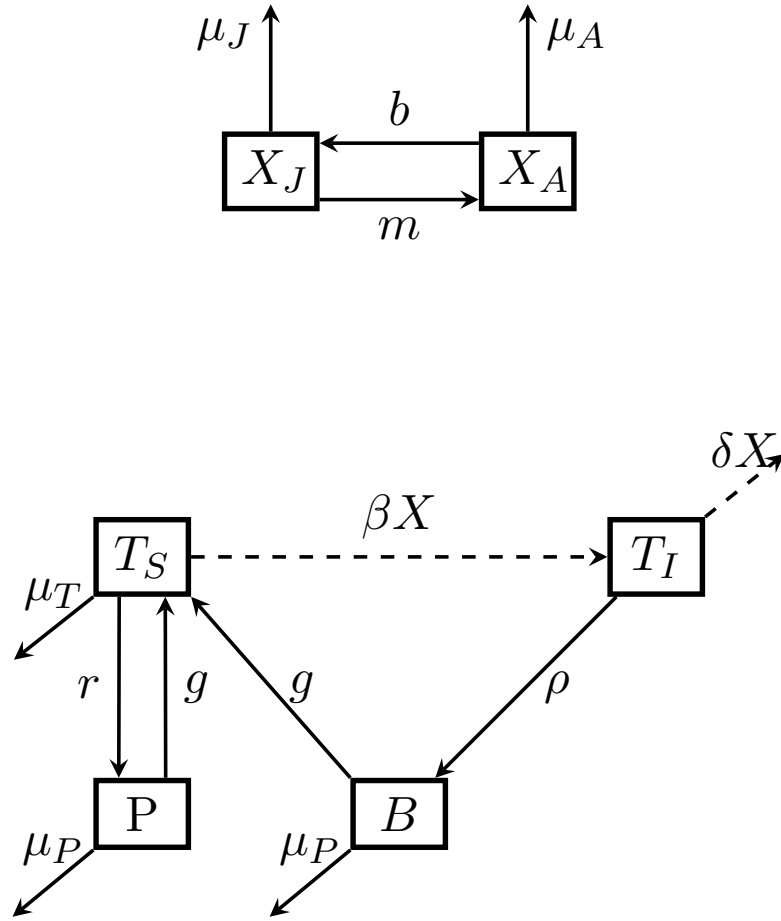


Figure 4.1: A flow diagram of the 1-Patch model, encapsulating growth and reproduction of the non-infected host population, infection transmission, and degradation of infected material, as well as birth, maturation, and death of the pest population.

Table 4.1: Description, notation, and values of parameters used in the 1-patch and N-patch models. Values are presented with two significant digits in decimal form.

Description	Symbol	Units	Value in Simulations
Host reproductive rate	r	1/day	$1.2 \times 10^{-4\dagger}$
Host maturation rate	g	1/day	$2.2 \times 10^{-4\dagger}$
Sapling natural mortality rate	μ_P	1/day	$8.2 \times 10^{-5\dagger}$
Tree natural mortality rate	μ_T	1/day	$4.1 \times 10^{-5\dagger}$
Basal sprout natural mortality rate	μ_B	1/day	$3.4 \times 10^{-6\dagger}$
Transmission rate	β	1/(day*beetle)	Varied [‡]
Basal sprout production rate	ρ	1/day	$4.1 \times 10^{-3\dagger}$
Infected material degradation rate	δ	1/(day*beetle)	Varied [‡]
Volume of infected tree	v_T	units ³	$4.0^\dagger$
Volume of a beetle gallery	v_0	units ³	$4.0 \times 10^{-1\dagger}$
Adult beetle per capita birth rate	b	1/(day*beetle)	$2.2^\dagger$
Juvenile Beetle Maturation Rate	m	1/day	$3.3 \times 10^{-2\dagger}$
Juvenile Beetle Natural Mortality Rate	μ_J	1/day	$6.7 \times 10^{-2\dagger}$
Adult Beetle Natural Mortality Rate	μ_A	1/day	$2.0 \times 10^{-2\dagger}$
Adult Beetle Migration Rate	c	1/day	$2.7 \times 10^{-3\dagger}$

[†] See Appendix A for value citation and calculation. [‡] Value was assumed.

one (i.e. if $X_A^i(t) < 1$, then $\beta = 0$ in patch i). We assume that the adult beetle population cannot reproduce unless there is sufficient infected material available (i.e. $T_I^i(t) > v_0$) and there is at least one adult beetle (i.e. $X_A^i(t) > 1$). If either condition is not satisfied, we set $b = 0$ in patch i . These assumptions mimic the biologically realistic limitations set on a founding beetle introduced into a new patch.

4.2.1 Analysis of Mathematical Model

We consider regions of the parameter space that could lead to endemic disease in the 1-patch model by considering the equilibrium of the system. Motivated by the inclusion of limitations on the actions small populations in the N-patch model, we analyze the endemic equilibrium of the 1-patch model to determine when the endemic equilibrium yields a true endemic (all states are sufficiently large) and when the endemic equilibrium yields an epidemic (at least one state is below its threshold value). We also consider regions of the parameter space that yield disease movement from introduction in patch 1 to patch N in the N-patch model. We combine those results to determine regions of parameter space that yield endemic or epidemic dynamics in patch 1, which then spreads to all N patches. Within the region that yields a spatially spreading epidemic, we consider the time until the epidemic wave ends and the minimum time until a reintroduction after the epidemic ends would yield a second epidemic wave throughout all N patches.

4.3 Results

We consider regions of parameter space in which the introduction of a small population of beetles into a fully susceptible tree population can lead either to an endemic disease state or to an epidemic with the possibility of a second wave after the first introduction. For either to occur, the pest and tree populations must experience growth (rather than decline) when decoupled from disease dynamics. Removing infection transmission and the pest population from the 1-patch model yields a two-dimensional system (saplings and trees). This sub-system experiences exponential growth of both tree sizes when $r > r_0 := \frac{\mu_T(g+\mu_P)}{g}$ and exponential decline when $r < r_0$. Removing the tree population in the full 1-patch model and replacing the pest carrying capacity with a constant quantity $K > 0$ yields a two-dimensional system (adult and juvenile beetles). In this sub-system, the juvenile population grows to size $\left(\frac{bm-\mu_A(\mu_J+m)}{bm}\right)K$ when $b > b_0 := \frac{\mu_A(\mu_J+m)}{m}$ and dies out otherwise.

The combined effects of tree and pest population growth rates on establishments outcomes are shown in Figure 4.2. Region A represents a potentially successful introduction of vector-borne

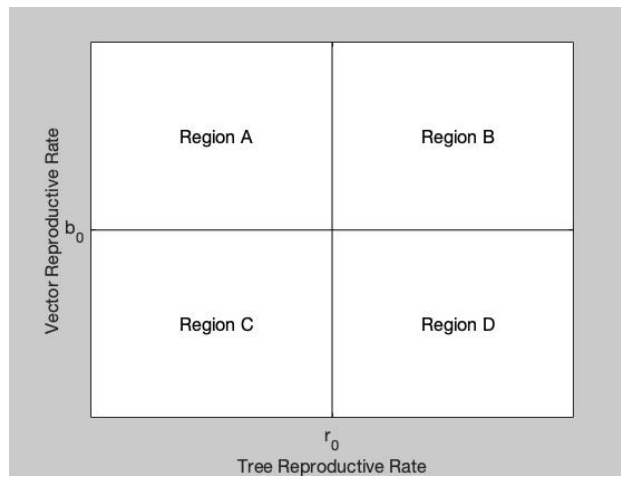


Figure 4.2: Regions of host growth and pest birth rates that lead to differential outcomes in model reductions. In regions A and B, pest birth rate is sufficient for positive population growth, and in regions C and D, the beetle population declines. In regions B and D, host tree reproduction is sufficient for positive host population growth, and in regions A and C, the host population decays exponentially.

disease into an already dying host population. While widespread disease can occur in an already dying host population, the introduction of disease will likely speed up the local extinction of an already dying population. Region D represents an unsuccessful introduction into a thriving tree population that does not result in disease establishment. Region C represents an unsuccessful introduction in a dying tree population. The tree population is likely to go extinct despite the absence of disease. The final region, Region B, represents a potentially successful introduction of vector-borne disease in patch 1. This region is the most likely candidate for the possibility of either endemic disease or epidemic disease with possible reintroduction. Accordingly, we choose to focus on the most likely scenario and limit our continued analysis to region B.

We next consider portions of region B that yield endemic or epidemic disease in the 1-patch model. The 1-patch model has a biologically feasible endemic equilibrium that exists through Region B. The adult beetle population at equilibrium is given by

$$X_A^* = \frac{\mu_A \delta (gr - \mu_t(g + \mu_p))(g + \mu_b) + \beta g m \rho(\mu_p + g)}{\beta \mu_A \delta (g + \mu_p) (g + \mu_b)}.$$

Motivated by the inclusion of limitations on the actions of adult beetles when $X_A < 1$ in the N-patch model, we consider regions of the parameter space that lead to an adult beetle population greater than 1. When $\rho = 0$, the expression simplifies nicely, and we have $X_A > 1$ when $\beta < \beta_X$ where

$$\beta_X := \frac{gr}{g + \mu_P} - \mu_t.$$

That is, the adult beetle population is larger than 1 when the rate at which trees are lost due to disease or natural deal is smaller than the rate at which trees produce saplings that will grow into mature trees. When $\rho > 0$, we have that $X_A^* = 1$ when $\delta = \delta_X(\beta)$ where

$$\delta_X(\beta) := \left(\frac{m}{\mu_A} \right) \left(\frac{g\rho}{g + \mu_B} \right) \left(\frac{\beta}{\beta - \beta_X} \right).$$

When $\beta < \beta_X$, the quantity $\frac{\beta}{\beta - \beta_X}$ is negative, and $X_A^* \leq 1$ as before. When $\beta > \beta_X$, we have that $X_A^* > 1$ only when $\delta < \delta_X(\beta)$. That is, an endemic equilibrium with an adult beetle population larger than 1 is only possible when the degradation rate of infected tree material is sufficiently less than the rate at which new infected material becomes available through the infection of matured basal sprouts produced by infected trees.

We also consider regions of the parameter space that yield a sufficiently large amount of infected material for pest utilization (i.e. $T_I^* > v_0$). The total amount of infected material at endemic equilibrium is given by

$$T_I^* = \frac{b v_0 (\beta g m \rho (g + \mu_p) + \mu_A \delta (g r - \mu_T (g + \mu_p)))}{\beta \delta (g + \mu_b) (g + \mu_p) (m b - \mu_A (\mu_J + m))}.$$

For $\rho = 0$, we have that $T_I^* < v_0$ only when $\beta < \beta_T$ where

$$\beta_T := \frac{\mu_A b (g r - \mu_T (g + \mu_p))}{(g + \mu_P) (m b - \mu_A (\mu_J + m))}.$$

When $\rho > 0$, we have that $T_I^* = 1$ when $\delta = \delta_T(\beta)$ where

$$\delta_T(\beta) := \frac{g \rho}{g + \mu_B} \frac{m b}{m b - \mu_A (\mu_J + m)} \frac{\beta}{\beta - \beta_T}.$$

When $\beta < \beta_T$, the quantity $\delta_T(\beta)$ is negative and thus $T_I^* > v_0$. When $\beta > \beta_T$, we have that $T_I^* > v_0$ only when $\delta < \delta_0(\beta)$ as well.

Letting $\beta_0 := \min(\beta_X, \beta_T)$ and $\delta_0(\beta) := \min(\delta_X(\beta), \delta_T(\beta))$, we can divide our parameter space into distinct regions that yield endemic or epidemic disease with and without basal sprout production in the 1-patch model. When $\rho = 0$, we have endemic disease when $\beta < \beta_0$ (regions I and III in Figure 4.3, top), and epidemic disease otherwise (regions II and IV in Figure 4.3, top). When $\rho > 0$, endemic disease is possible regardless of the degradation rate when $\beta < \beta_0$ and for degradation rate $\delta < \delta_0(\beta)$ when $\beta > \beta_0$ (regions I and III in Figure 4.3, bottom). Epidemic disease is possible when $\beta > \beta_0$ and $\delta > \delta_0(\beta)$ (regions II and IV in Figure 4.3, bottom).

In the N-patch model, the ability of epidemic or endemic disease in patch 1 to spread to patch N also depends on the transmission and degradation rates. There is a threshold curve, $\delta = g(\beta)$ above which epidemic or endemic disease cannot spread from patch 1 to patch N , regardless of basal sprouts. This threshold further divides our parameter space into distinct regions in terms of the outcome of the establishment and spread possibilities (Figure 4.3). We have that in regions I and II, endemic disease and epidemic disease, respectively, is possible in patch 1 alone, but the addition of movement between patches reduces ability of pests to establish, and disease does not spread beyond patch 1. In regions III and IV, disease can spread from patch one to patch N . In Region III, the disease spread can yield endemic disease, and in Region IV, disease spread can yield

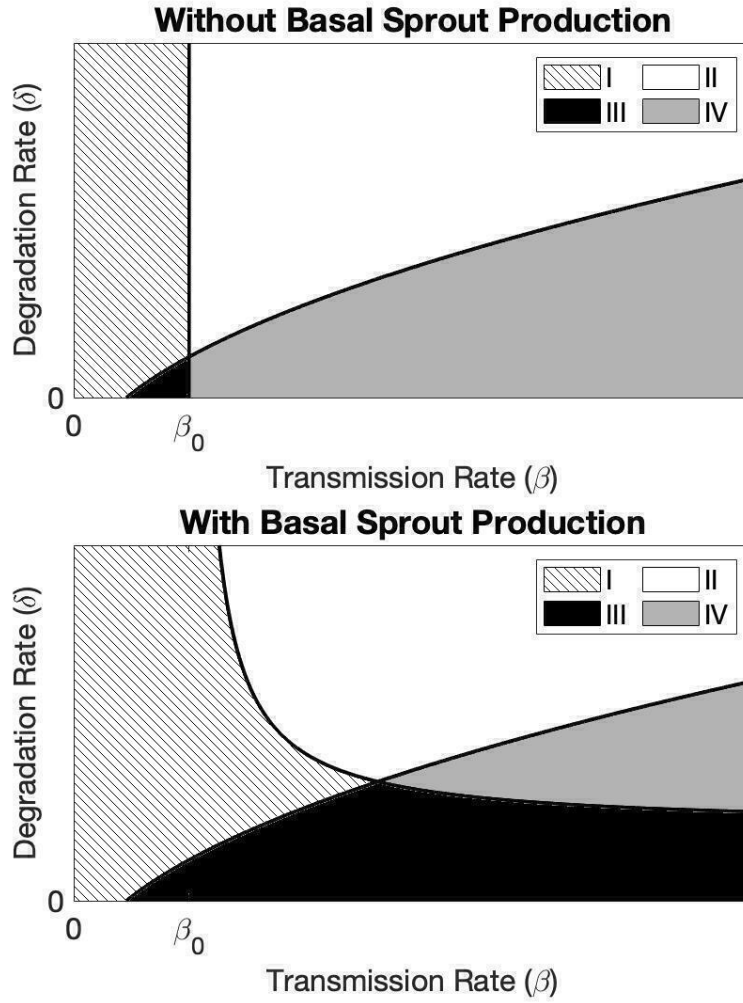


Figure 4.3: Potential outcomes of establishment and spread of disease without (top) and with (bottom) basal sprout production for ranges of transmission and degradation rates. In region I, endemic disease without spacial spread is predicted, and In region II, epidemic disease without spacial spread is predicted. In region III, endemic disease with spacial spread is predicted, and in region IV, epidemic disease with spacial spread is predicted.

epidemic disease (in which pest population dies out). Region III is larger and (region IV is smaller) when basal sprouts are produced in response to disease.

Within region IV, we consider the length of the epidemic, as measured as the time from the introduction of adult beetles in patch 1 until disease the pest population dies out in patch 5. (The dynamics within an individual patch are independent of the total number of patches. Patch 5 is chosen to balance computation needs with small but measurable distance from patch 1.) Regardless of basal sprouting, the length of the epidemic decreases as the degradation rate increases, but for small degradation rates, the production of basal sprouts leads to a longer epidemic (Figure 4.4). When the transmission rate is much larger than β_0 , the length of the epidemic does not depend on the transmission rate, but when the transmission rate is near β_0 , the length of the epidemic decreases as the transmission rate increases. (The epidemic continues to spread beyond patch 5, and the duration of the epidemic as measured as time until the pest population dies out in a patch greater than 5 will surely be longer. However, increasing the patch number yields the same trends.)

The production of basal sprouts in response to disease has a large impact on the time until the tree population is susceptible to a second wave of a vector-borne epidemic (measured by the minimum number of years after the pest has died out in patches 1-5 that an introduction of 10 beetles in patches 1 will trigger an epidemic in patches 1-5). Without the production of basal sprouts, the time until the population is vulnerable to a second wave is largely independent of the degradation rate, except when the degradation rate is low (Figure 4.5, top). However, with the production of basal sprouts, the time until the population is vulnerable to a second wave increases as the degradation increases for all values of δ (Figure 4.5, bottom). In either case, the time until the population is vulnerable to a second wave decreases with increasing transmission rate.

4.4 Discussion

The production of basal sprouts affects the expected dynamics of a newly introduced bark or ambrosia beetle with a pathogenic, symbiotic fungus. The production of basal sprouts increases the likelihood that introduced disease will be endemic rather than epidemic, and if the disease will die out, the production of basal sprouts increases the duration of the epidemic. When a new vector-borne disease is introduced, forest managers should note whether or not infected trees respond to disease by producing basal sprouts. When basal sprouts are produced, more intense management is merited because of the high risk of endemic disease or, at least, long-lasting epidemic disease.

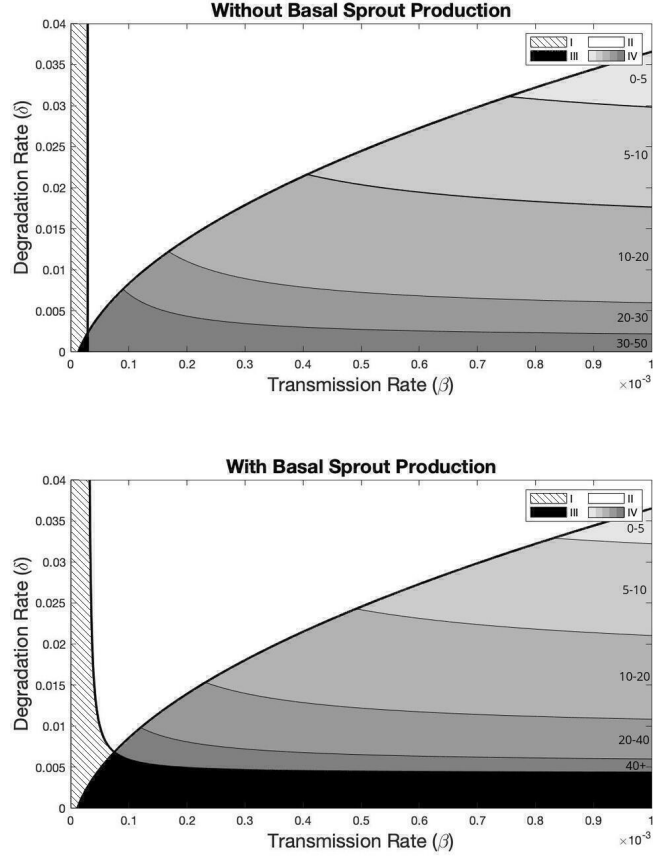


Figure 4.4: Duration, measured in years, of epidemic disease within region IV without (top) and with (bottom) basal sprout production, measured in years since introduction in patch 1 in numerical simulations of the N-patch model, using parameter values listed in Table 4.1 and ranges of transmission and degradation rates. Regions I-IV correspond to regions described in Figure 4.3.

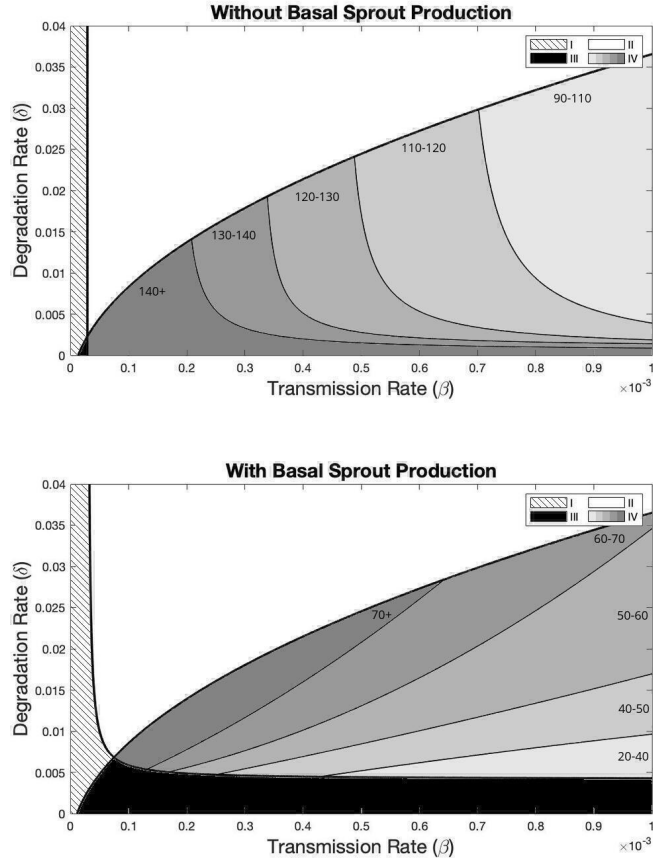


Figure 4.5: Minimum time, measured in years, after the first wave of epidemic disease ends before the a re-introduction of 10 adult beetles in patch-1 triggers a second wave of epidemic disease spread in region IV, from numerical simulations using parameter values from Table 4.1 and ranges of transmission and degradation rates. Regions I-IV correspond to regions described in Figure 4.3.

The production of basal sprouts also profoundly impacts the vulnerability of susceptible trees to a second wave of disease after the epidemic has ended. In particular, it takes at least 1.5 times longer for a tree population to be vulnerable to a second wave of disease when basal sprouts are not produced than when they are. Accordingly, managers should expect the possibility of a second wave of disease in a short time scale in systems in which host trees respond to disease by producing basal sprouts.

The introduction of vector-borne disease often occurs through the movement of firewood, which can harbor adult or juvenile forms of the vector. If firewood is moved from an area with an active vector-borne disease outbreak to an area with a previous outbreak, a second wave could be triggered if the host population is sufficiently large/mature to support beetle reproduction. The production of basal sprouts yields a population that is vulnerable to disease within a short time frame. If the geographic range of susceptible trees is quite large, the first wave of disease could still be active in areas of late arrival when the first-infected areas become vulnerable to disease again. Accordingly, bans on the movement of firewood should last at least until the first wave has fully ended and should prohibit movement both from and to areas with a history of infestation.

Commonly, basal sprouts produced by infected trees are at risk of secondary infection via vertical transmission from parent trees. When secondary infection is high, mathematical models predict similar long-term results as to cases in which there is no basal sprout production at all (see Chapter 2). While this mathematical model does not explicitly include the infection of basal sprouts, we suggest that high levels of basal sprouting combined with high rates of secondary infection should lead to outcomes similar to the results we have shown for no basal sprouting, and similar management decisions should be made.

This work is of specific relevance to laurel wilt, which is a vascular wilt disease caused by the fungus *Raffaelea lauricola* and vectored by the beetle *Xyleborus glabratus* (Hamula et al., 2008). Laurel wilt was introduced in Georgia in 2002, and is spreading throughout the eastern United States. The areas that experienced loss of redbay (*Persea borbonia*), in the early years of the laurel wilt epidemic are now recovering from disease, but it will be decades before laurel finishes its rampage across the eastern United States. Redbay produces basal sprouts but also suffers high rates of secondary infection, so counties in Georgia, Florida, and South Carolina should anticipate the possibility of reintroduction of laurel wilt in the coming decades and should make management decisions accordingly. As laurel wilt spreads into new regions, new species are susceptible to laurel wilt (e.g. sassafras (*Sassafras albidum*)). Forest managers should take special note of the production

(and infection) of basal sprouts by sassafras. Different disease dynamics (e.g. endemic or epidemic) and time scales might be expected depending on the frequency and rate at which sassafras produces basal sprouts.

The mathematical model includes limitations on the life cycle of beetles when the adult population size is too small or when the available material for reproduction is too limited. This represents the limitations imposed upon a founding female introduced into a new area, as well as the limitations on the beetle population as resources become limited due to over-utilization. The model does not, however, include similar limitations on the host population (e.g. restrictions on tree reproduction when $T_S < 1$). However, small host populations are possible. The ratio of the susceptible tree population to adult pest population at equilibrium is given by

$$\frac{T_S^*}{X_A^*} = \frac{\beta m v_t (mb - \mu_A (\mu_J + m))}{\mu_A^2 \delta b v_0}.$$

When this ratio is less than 1, the susceptible tree population may reach size 1 around or before the pest population declines. Despite the possibility of tree populations going below 1, we do not include limitations on their life cycle for two reasons. First, production of saplings is still possible after the host population passes below 1 through germination of seeds produced by trees just before dying. For small time scales, this is possible even for species that do not maintain long-term seed pools (such as is the case for laurels ([Evans et al., 2014](#); [Meadows et al., 2006](#))). Secondly, excluding limitations on small populations can represent the possibility of short-term, active management just after the epidemic has ended locally. Reseeding or planting saplings of the species devastated by vector-borne disease just after the epidemic ends locally captures similar dynamics as a lack of limitation on life cycle events of too-small tree populations. However, if disease causes the local-extinction of tree species and active management is not done to reestablish the species, the results of this model are not applicable.

We have developed a mathematical model for the spread of fungal, vector-borne disease vectored by a non-native bark or ambrosia beetle via movement of beetle through N linearly connected patches. Within this model, we have found thresholds that differentiate spatial spread of endemic disease, spatial spread of epidemic disease, and no spatial spread of disease. When spatial spread of epidemic disease occurs, we have quantified both the time until the epidemic ends locally and the minimum time required until the host population recovers from disease and is again vulnerable to disease, triggering a second wave of disease. Within this context, we have shown that, disease

is more likely to be endemic, that epidemic outbreaks last longer, and that tree populations are vulnerable to a second wave of disease much sooner when basal sprouts are produced than when basal sprouts are not produced. This is a case in which an evolved trait in response to disease-induced damage is detrimental to the tree population. This suggests that this type of disease was likely not a major threat over the evolutionary history of tree species that produce basal sprouts. However, the high likelihood of novel introduction of new beetles and pathogens in the modern anthropocene poses a major threat to tree species that produce basal sprouts. Because of this threat, the production of basal sprouts should not be overlooked when predicting outcomes and developing management strategies for introduced vector-borne disease.

Chapter 5

Conclusion

I have developed a series of mathematical models that incorporate the production and infection of basal sprouts in the context of introduced vector and vector-borne, tree-killing disease. I have interpreted these models to study the effect of basal sprout production on the ecological and epidemiological outcomes, both locally and spatially, of introduction of vector-borne disease in a novel environment. The mathematical models produced are distinct from, but inline with, other mathematical models that have informed management strategies in other disease systems (Swinton and Gilligan, 1996; Cobb et al., 2012). The epidemiological models presented here also follow the SI framework, separating the host population into susceptible and infected subcategories, and incorporate stage structure into one or both of the host and vector populations.

The models introduced here are designed to capture the dynamics of vector-borne disease caused by the fungal symbiont of a bark or ambrosia beetle (e.g. laurel wilt, dutch elm disease) and are sufficiently general to be applied to future, emerging diseases vectored by bark and ambrosia beetles that have similar dynamics. In particular, the models assume that the introduced beetle requires dead and dying (i.e. infected) host material for successful reproduction. If the vector of an introduced vector-borne disease instead required healthy, susceptible host material for successful reproduction, these mathematical models are not applicable, although it is possible that the addition of healthy tissue through basal sprouting would also affect the dynamics of such a disease similarly. Furthermore, the models assume that the vector requires the pathogen for successful reproduction and that vector and pathogen are introduced together. This is likely the case for bark and ambrosia beetles, which can carry their fungal symbiont in specialized mouth parts (Sauvard, 2004), in the case that the fungal symbiont is pathogenic to trees. If, instead, a novel pathogen is vectored by a

native beetle that does not explicitly require the introduced pathogen for successful reproduction, these mathematical models do not apply. The dynamics of lateral transfer of pathogenic material between native vectors and the ability of the vector to reproduce in absence of the pathogen could change outcomes dramatically. The limitations of a disease system whose dynamics are captured by these mathematical model may seem large; however, as international shipping continues to increase, beetles and pathogens with these characteristics are likely to continue to be introduced (Hulcr and Dunn, 2011; Roy et al., 2014). These limitations are imposed to yield specific recommendations for a smaller class of disease rather than general recommendations for a larger class of disease.

The mathematical models developed here are not the only models that could include basal sprout production in vector-borne disease systems. Due to the current limitations of available knowledge about the specific life cycle of beetles and their interaction with the host species in a forest setting, general, common-sense assumptions were made. The models assume that the degradation of infected host material is due to vector utilization and that the total degradation rate increases at the vector-population increases. It could also be that the degradation of infected host material is a time dependent process, independent of the size of the vector population. Different short-term or long-term dynamics could be expressed in models with time dependent degradation. Because the degradation rate affects the establishment and spread of vector-borne disease (Chapter 4), exploration of this assumption in future work is merited. The dynamics of the beetle population are modeled phenomenologically rather mechanistically. As empirical work expands the understanding of biology of bark and ambrosia beetles in novel environments, a more detailed model with mechanistic drivers of the vector population could be developed, but the utility of such modes should be weighed against the added analytical difficulty; more complicated models are not guaranteed to give different or better results.

The development and analysis of these models develop the theory of vector-borne tree disease and provide support for management strategies for such diseases. I have shown that the production of basal sprouts by infected trees does not affect the short-term dynamics of disease, but can affect the persistence of the host population in long time scales, lessening the likelihood of disease-driven local extinction of the host population. However, the positive effects of basal sprout production are minimized when the rate of secondary infection of basal sprouts via vertical transmission is high (Chapter 2). I have also shown that active management targeted at basal sprouts (e.g. reducing basal sprout infection, removal or infected or susceptible basal sprouts) can be effective in either reducing the vector population or maintaining the host population, but no basal-sprout

centered management strategy can accomplish both goals. Forest managers should consider active removal/limitation of beetle population in combination with basal-sprout centered management to achieve both goals (Chapter 3). Lastly, I have shown that the production of basal sprouts in response to disease increases the likelihood of endemic disease spread spatially, and when epidemic disease spread occurs, the epidemic lasts longer and hosts are vulnerable to a second wave sooner, compared to species that do not respond to disease by producing basal sprouts. Because of this increased risk, management strategies for introduced pests that vector tree disease should specifically consider the production of basal sprouts when anticipating the impact of introduction (Chapter 4).

Before this work, basal sprouts had generally been ignored in mathematical models of tree disease. Previous models that include the production of basal sprouts either fail to distinguish saplings from basal sprouts or allow for the production of basal sprouts from all trees (not just infected trees). Accordingly, secondary infection of basal sprouts via secondary infection had not yet been included. On a broader scope, no process analogous to basal sprout production by infected hosts exists outside the plant community. Because the production of basal sprouts in response to disease is unique to the epidemiology of plant diseases, no existing mathematical model for non-plant diseases can account for the dynamics like that of basal sprout production. Thus, the mathematical models developed here represent a unique and novel dynamical system.

These models suggest several meaningful recommendations for forest managers. Perhaps most importantly, this work highlights the degree to which the production of basal sprouts by infected trees can affect the long-term outcomes of vector-borne disease. Because basal sprouts have little impact on the short-term dynamics of disease systems, they are often overlooked. In contrast, this work suggests basal sprout production (and secondary infection) strongly affect the long-term outcome of introduced vector-borne disease. In particular, when secondary infection is low, the production of basal sprouts decreases the likelihood of local extinction of the host population but also increases the likelihood of endemic disease or future waves of epidemic disease. This is a significant trade-off that should be considered when formulating management strategies. Accordingly, early field work on an emerging disease should pay special attention to the production and infection of basal sprouts so that differential decisions can be made. Furthermore, this work highlights the utility of basal sprouts as a potential target class for active management to either increase the host population or decrease the vector population locally. While both goals cannot be attained simultaneously, forest managers can use these results to make rapid but informed decisions after the introduction of a novel vector-borne disease, given the context of the specific disease

system and the management goals. This theoretical work provides a basis upon which future field studies can be developed, which will help to protect trees as novel pathogens and introductions into new areas continue to threaten tree populations.

The mathematical models developed here contribute to the body of mathematical modeling within phytopathology and provide a starting point for the mathematical investigation of the role of basal sprout production in other classes of tree disease. Furthermore, the results of these models and future models that follow in their footsteps provide a framework for theory-based management of vector-borne tree diseases. While there are many steps remaining, this work represents one of many required to protect old forests from new threats in the era of the Anthropocene.

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Appendix

A Calculation of Parameter Values

A.1 Host Demography

Estimates on the diameter growth, survival, and volume of sassafras and other hardwoods is given in [Smith \(1984\)](#).

Host Tree Mortality

Average size-based yearly survival probability for other hardwoods is 0.967 for saplings, 0.972-0.987 for medium trees, and 0.987-0.997 for large trees. Thus we assume each year 1% of large trees die ($\mu_L = .01$ trees/year), 2% of medium trees die ($\mu_M = .02$ trees/year), and 3% of saplings die ($\mu_P = .03$ trees/year), in [Chapter 2](#). In models with reduced stage structure, the mature tree category includes both medium and large trees, so we chose values of mature tree mortality between 1-2% yearly (and divide by 365 to get daily rates). The seed bank is removed in simplified models, so the effect of seedling mortality are incorporated into sapling mortality. We assume a yearly sapling mortality rate between 30 and 40% (which corresponds to $\mu_P \in (8.2 \times 10^{-5}, 1.1 \times 10^{-4})$ trees/day) in [Chapters 3 and 4](#).

Seed Mortality

Redbay seeds remain viable for 1-2 years ([Evans et al., 2014](#)) and sassafras remain viable for 5-6 years ([Meadows et al., 2006](#)). We choose an intermediate viability of 3 years, and the yearly mortality rate is given by the reciprocal of the average lifespan ($\mu_D = \frac{1}{3}$ seeds/year).

Host Maturation

Size based maturation rates are calculated from data on the mean annual diameter growth by diameter class [Smith \(1984\)](#). Within the category other hardwoods (data for sassafras not given individually), yearly growth rates corresponding to medium trees (2.5cm-10cm) vary between 0.11-.98 cm/year. We choose a conservative annual diameter growth rate of 0.24 cm/year for medium trees. Accordingly, trees spend 31.25 years in this category ($\frac{10\text{cm}-2.5\text{cm}}{0.24\text{cm/year}}$) which yields maturation rate of $g_M = 1/(31.25\text{years}) = 0.032$ trees/year. Data is unavailable for yearly diameter growth rate for stems under 2.cm. We assume a slightly smaller growth rate of 0.2cm/year which implies trees spend 12.5 years in the sapling category ($\frac{2.5\text{cm}-0\text{cm}}{0.2\text{cm/year}} = 12.5$ years). We then take the maturation rate to be the reciprocal of the time spent as a sapling (i.e. $g_P = 0.08$ trees/year). We assume that basal sprouts and saplings mature at the same rate (i.e. $g_B = g_P = 0.08$ trees/year). In models with reduced stage structure, we take $g = g_P$.

Seed Production and Germination

The seed production rate was difficult to obtain from data. The mean yearly seed rain for sassafras is 0.0063 seeds/m²/year (Clark and Lavine, 2005). Assuming one tree spreads seeds over an area of 50 m² (arbitrarily selected), we have a per tree output of approximately $r_L = 1/3$ seed per year. We assume medium trees have a slight reduction in seed production and take $r_M = .9r_L$. To an average reader, these quantities seems quite small. No data is available for seed germination rates in a forest setting. Accordingly, we select a seed germination rate of 10% which yields a positive but population growth rate in absence of disease. This rate seems quite high, perhaps in part because it is compensating for a small seed production rate. In Chapters 3 and 4, the seed bank was excluded. In this case, the host reproductive rate r is selected to yield a positive but population growth rate.

Tree Volume

Regarding the volume of trees, we assume that large trees provide twice as much suitable material as medium trees. Moreover, we assume medium trees provide twice as much suitable material as saplings and basal sprouts.

Basal Sprout Parameters

In the absence of data to the contrary, we assume that basal sprouts and saplings mature at the same rate (i.e. $g_B = g_P = 0.08$ trees/year). Evans et al. (2014) reports a very high redbay sprout mortality rate (79%) years after introduction of disease, but this statistic is not decoupled from mortality due to secondary infection. It is likely that most of the basal sprout mortality reported is due to secondary infection rather than naturally occurring mortality. We assume that natural (not disease induced) mortality of basal sprouts is much lower than that of saplings because basal sprouts have the support of an existing root system. Thus we take $\mu_B = 0.0013$ trees/year.

In redbay plots, two-thirds of infected trees had at least one sprout 1.5 years after infection, and infected trees had on average 5 basal sprouts 7 years after infection (Cameron et al., 2015). Accordingly, rates for basal sprout production and infection were chosen to be on the magnitude of one basal sprout produced every 6 months to 2 years, and basal sprout infection was chosen on a similar magnitude.

A.2 Beetle Demography

In absence of data on a variety of bark and ambrosia beetles, we use demographic data for the redbay ambrosia beetle. The first adults emerge approximately thirty days after gallery initiation Brar et al.

(2013), so we take the beetle maturation rate as the reciprocal (i.e. $m = 1/30$ beetles/day). Data on the lifespan of redbay ambrosia beetles is unavailable. Assuming that juvenile are twice as likely to die than mature, we set the $\mu_J = 1/15$ beetles/day. Finally, we assume that an adult beetle lives for 50 days (i.e. $\mu_A = 1/50$ beetles/day).

Vita

Kelly Buch is from O'Fallon, Illinois, and received two undergraduate degrees from Southern Illinois University Edwardsville. She earned a Bachelor of Arts degree in Foreign Language and Literature with a Spanish major and a Bachelor of Science degree in Mathematical Studies with a major in Applied Mathematics and minor in Biological Sciences. She followed her love of mathematics and nature by pursuing a PhD in Mathematics with a concentration in Mathematical Ecology at the University of Tennessee Knoxville. Her research interests involve using mathematical modeling as a tool to answer questions in wildlife conservation and restoration. The subject matter of Kelly's dissertation was inspired by her love of tree identification. After graduation, she will stay in Tennessee to begin her new position as an Assistant Professor at Austin Peay State University in Clarksville, TN. She is excited to mentor undergraduate research in the next phase of her career and hopes to enjoy the beauty of sassafras trees for years to come.