Animal and Plant Health Inspection Service
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Risk of *Phytophthora ramorum* to the United States

Agency Contact:
Plant Pest Risk Analysis
Science and Technology
Plant Protection and Quarantine
Animal and Plant Health Inspection Service
United States Department of Agriculture
920 Main Campus Drive, Suite 400
Raleigh, NC 27606

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Executive Summary

In 2008, Plant Protection and Quarantine (PPQ) Plant Epidemiology and Risk Analysis Laboratory (now Plant Pest Risk Analysis) conducted a risk assessment for *Phytophthora ramorum* Werres, De Cock & Man in't Veld and concluded *P. ramorum* was a high-risk pathogen for large areas of the United States. However, as of 2023 in the United States, *P. ramorum* is still only established and causing disease in the coastal areas of California and Oregon despite repeated nursery and stream detections throughout the country. In this document, we revised our 2008 assessment of the risk *P. ramorum* poses to areas of the United States where it is not present using the latest scientific information on its biology, ecology, and epidemiology.

Outside of California and Oregon forests, there is an asynchrony between the infective stage of *P. ramorum* and the susceptible stage of the host complex when environmental conditions are favorable for infection, disease development, and spread. Three interrelated factors are likely causing this asynchrony and making the occurrence of diseases caused by *P. ramorum* unlikely:

1. Environmental stress, such as heat stress, decreases inoculum survival of *P. ramorum.*
2. Inoculum does not build up in sufficient amount to produce significant disease.
3. The infectious stage of the *P. ramorum* lifecycle, (i.e., zoospore production) does not overlap with the susceptible stage of the host or host complex for sufficient time, reducing the likelihood of infection and disease development.

Thus, while *P. ramorum* may survive in the environment, the necessary conditions for the development of ramorum blight, ramorum shoot dieback, and sudden oak death in forests are not occurring, and consequently hosts are not symptomatic—at least not on a noticeable scale. While it is possible that repeated incursions of the pathogen, or changes in climate conditions, in an area could increase inoculum pressure enough to cause infection and disease under the right conditions, twenty years of observations with this pathogen and numerous movements of infected plant material outside of California and Oregon, lead us to conclude that this is unlikely. Therefore, unless conditions change, *P. ramorum* is unlikely to pose a high risk to the United States outside of forests in California and Oregon.

There are three important sources of uncertainty in this assessment. First, there is still a lot unknown about the competency and susceptibility of eastern U.S. hosts and it is still unclear if there is synchrony between inoculum production and times of host susceptibility. Second, modeling climatic suitability remains challenging. Some climatic factors important for disease development cannot be reliably modeled for forest conditions. Third, a host range expansion due to the introduction of new clonal lineages or the emergence of new lineages due to sexual recombination may increase the adaptability of *P. ramorum* in the United States and potentially alter the consequences of introduction. The jump of *P. ramorum* to larch (*Larix* spp.) in Europe in 2009, and the introduction of the EU1 clonal lineage to the United States, which occurred around 2016, suggests a host range expansion could affect U.S. conifers or other plants. If EU1 expanded in range, or if other lineages were to establish, the chances for sexual recombination may increase, which could result in changes in the biology and epidemiology of this pathogen.

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1. Introduction

Phytophthora ramorum Werres, De Cock & Man in't Veld is a pathogenic, fungus-like organism that attacks aboveground parts of many woody and herbaceous plant species. Symptoms of sudden oak death, one of the diseases it causes, were first detected in the United States in the mid-1990s when diseased oaks (*Quercus agrifolia*, *Q. kelloggii*, and other *Quercus* species) and tanoaks (*Notholithocarpus densiflorus*) were recognized almost simultaneously in several coastal locations of California (Rizzo et al., 2002). This disease was named sudden oak death because tree canopies appeared to rapidly turn brown and die (Svihra, 1999). In 2001, the pathogen was officially named "*Phytophthora ramorum"* by Werres, De Cock, & Man in't Veld (2001), and in 2002 it was published as the causal agent of sudden oak death by Rizzo et al. (2002). In a nursery in California in 2001, *P. ramorum* was recovered from rhododendron (COMTF, 2022b) and in 2003 it was detected on camellia nursery stock (*Camellia* spp.) (COMTF, 2022b). Around the same time, in Oregon nurseries, *P. ramorum* was also detected on camellias, *Pieris* spp., rhododendrons (*Rhododendron* spp.), and viburnums (*Viburnum* spp.) (Parke et al., 2004); the disease it caused on those hosts was named ramorum blight. There are currently over 130 plant species known to be susceptible including trees, shrubs, plants, and ferns (USDA-APHIS, 2023).

In 2008, Plant Protection and Quarantine (PPQ), Plant Epidemiology and Risk Analysis Laboratory (now Plant Pest Risk Analysis, PPRA) conducted a comprehensive risk assessment on *P. ramorum* and concluded its overall risk—in terms of both the likelihood and the consequences of its introduction into the United States—was high, and that a large portion of the country, including areas containing eastern hardwoods (*Quercus* spp.), were vulnerable (PPQ, 2008).

Nearly three decades since its discovery and over a decade since PPQ conducted its initial risk assessment, *P. ramorum* continues to damage forests in California and Oregon where it is established[1]. It has been estimated to have killed over 40 million trees from 2012 to 2019 (Cobb et al., 2020) but no symptoms have been observed in hosts in natural settings elsewhere in the United States, even though it has over 130 reported hosts (USDA-APHIS, 2023). This is surprising given the numerous detections of *P. ramorum* in nurseries and streams throughout much of the country and has led to questions about why the pathogen is not causing impacts in natural areas originally believed to be highly suitable for disease development, such as the eastern United States.

Since the initial risk assessment in 2008, there have been numerous scientific advances in our understanding of *P. ramorum* (Figure 1), and the way PPRA conceptualizes and evaluates risk has evolved. Given these advances, we had two objectives:

1) to summarize the current state of knowledge of *P. ramorum,* and
2) to reevaluate the risk this pathogen poses to areas of the United States where it is not present.

To reevaluate the risk, we considered what has been learned recently about the biology and epidemiology of the pathogen. Part of reevaluating the risk also includes mapping where

[1] Establishment (of a pest): perpetuation, for the foreseeable future, of a pest within an area after entry (IPPC, 2022).

temperatures are favorable for *P. ramorum* survival and infection, or extreme enough to stress *P. ramorum* and hinder its survival. The maps from these analyses alone should not be considered predictive of where the pathogen might establish; we use them along with our current understanding of host susceptibility and competency[2] and where and how the pathogen has been detected to reevaluate the risk.

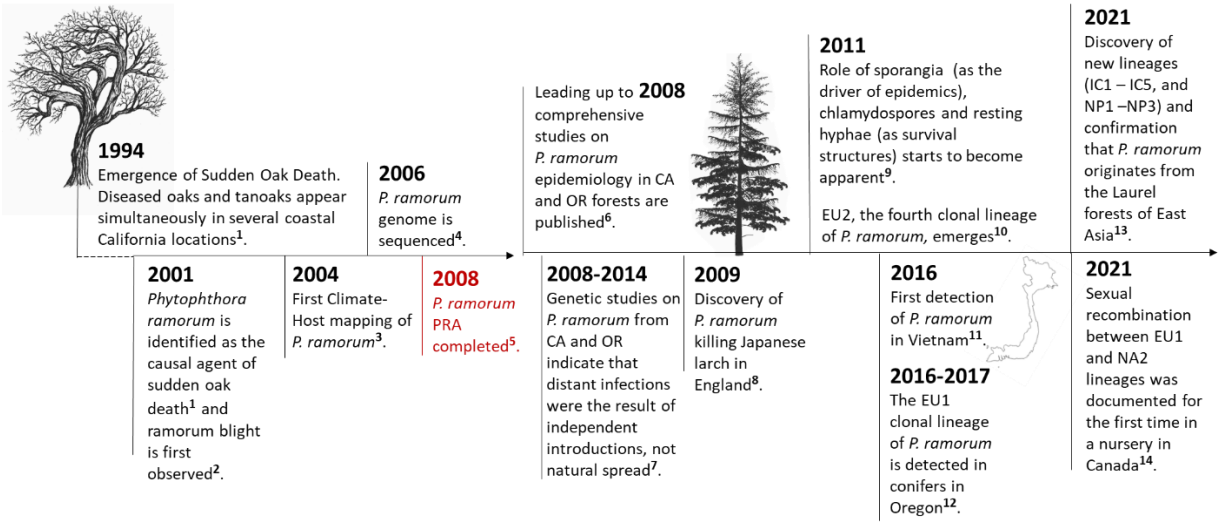


Figure 1. Timeline of major research milestones during the global emergence of the sudden oak death pathogen *P. ramorum* showing what was known in 2008 when the original risk assessment was completed and what is now known. Select sources: **1** - Rizzo et al., 2002; **2** - Werres et al., 2001**; 3** - Fowler et al., 2006**; 4**- Tyler et al., 2006; Govers et al., 2006; **5** - PPQ, 2008; **6** - Davidson et al., 2008; Fichtner et al., 2007; Moralejo et al., 2006a; Prospero et al., 2007; **7** - Mascheretti et al., 2008 ; **8** – Brasier and Webber, 2010; **9** - Davidson et al., 2011; Peterson et al., 2014; **10** - Van Poucke et al., 2012; **11** - Jung et al., 2020; **12** - Grünwald et al., 2016; LeBoldus et al., 2018; **13** - Jung et al., 2021**; 14** - Hamelin et al., 2022.

2. History of *Phytophthora ramorum* in the United States

In the mid-1990s, dying and diseased oaks and tanoaks were first observed in several coastal locations of California (Rizzo et al. 2002). In 1999, the disease was named sudden oak death (Svihra, 1999) to describe the canopies of many infected trees which appear to quickly turn completely brown and die.

By 2000, a better understanding of the cause of *P. ramorum* was beginning to emerge. Werres et al. (2001) described *P. ramorum* on *Rhododendron* and *Viburnum* (later named ramorum blight) in Europe and a year later, Rizzo et al. (2002) published *P. ramorum* as the causal agent of sudden oak death in the United States. In 2001, *P. ramorum* was observed on rhododendron in a nursery in Santa Cruz County (COMTF, 2022b) and recognized to cause another disease—ramorum blight (Tjosvold et al., 2005). This was followed by detections on camellia and viburnum nursery stock, also in California (COMTF, 2022b). In Oregon, *P. ramorum* was recognized as the cause of mortality of tanoaks in Curry County in 2001 (COMTF, 2022a;

[2] Host competency: the ability of a host to transmit the infection to another susceptible host or to a vector (Gervasi et al., 2015). Host competency of many *P. ramorum* hosts has been assessed by measuring sporulation, production of sporangia or zoospores in laboratory and field experiments (See Appendix C).

Hansen et al., 2008) and of ramorum blight on camellias, *Pieris* spp., rhododendrons, and viburnums in 2003 (Parke et al., 2004).

A U.S. federal domestic quarantine for *P. ramorum* was first issued in 2002 (USDA-APHIS, 2023) to address the pathogen in the natural environment in California and Oregon and on rhododendron container plants in a California nursery. Canada issued a quarantine in 2001 and California and Oregon issued emergency orders to prevent the shipment of infected nursery stock. Initially, only a few counties in California and a portion of one county in Oregon were quarantined (USDA-APHIS, 2002). But in 2004, *P. ramorum* was found in a few nurseries—two in Oregon, California, and Washington State—outside of the quarantined areas (USDA-APHIS, 2005; Jones, 2006). Trace-forward investigations indicated these nurseries had sent over two million plants, mainly camellias and rhododendrons, to more than 40 states; this resulted in 176 positive finds of *P. ramorum* in 21 states (Jones, 2006; USDA-APHIS, 2005). Subsequently, Federal orders were issued to regulate the movement of all nursery stock from California, Oregon, and Washington State (USDA-APHIS, 2004). To ship regulated nursery stock interstate[3], nurseries in regulated areas had to be inspected annually for symptoms of *P. ramorum*, and samples of any symptomatic plants had to test negative for *P. ramorum*.

Since the pathogen was first regulated, changes were made that either relieved or imposed regulatory requirements for nurseries, depending on whether those nurseries had tested positive for *P. ramorum* within the previous three years. The change meant substantially fewer nurseries had to be inspected, sampled, and certified to ship host plants interstate.

Primarily due to the movement of nursery stock from California and Oregon since the early 2000s, detections of *P. ramorum* have occurred in multiple states nearly every year (e.g., data from Chastagner et al., 2010; COMTF, 2022a, 2019c; Jeffers et al., 2010; Kurdyla et al., 2014; PPQ, 2019, 2021, see Table 1). Detections are generally divided into three categories according to where the detection occurred: in a nursery, residential/commercial landscape, or stream. When *P. ramorum* is detected in a nursery or residential/commercial landscape, PPQ in cooperation with State Agriculture Departments conducts trace-forward and trace-back investigations to determine if *P. ramorum* is present in any of the supplying or receiving facilities. In addition to these inspections, general surveys for *P. ramorum* are conducted in many parts of the United States by cooperating states. In 2006, the U.S. Forest Service initiated a stream baiting program to check for *P. ramorum* spread and presence in waterways (USFS, 2019) generally close to or downstream from nurseries and high-risk areas in Oregon and California. This later expanded to other states and has resulted in numerous new state records for the pathogen and detections in streams in several states (Table 1). *Phytophthora ramorum* is only known to have been detected once on vegetation adjacent to a stream, nursery, or landscaping. In 2009 it was detected on several salal plants (*Gaultheria shallon*) along the perimeter of an infected nursery in Washington State (Chastagner et al., 2013); it was subsequently eradicated. In 2008 it was also detected on vegetation in Mississippi, outside of a nursery and likely because of flooding that occurred prior to the samples being collected (COMTF, 2022b). In surveys from vegetation later that year in the same location the pathogen was not detected (COMTF, 2022a).

[3] The current list or regulated articles, including hosts, is provided in "*Phytophthora ramorum*, Restricted, regulated, and associated articles; lists of proven hosts and associated plant taxa" (USDA, 2023).

Because infections have not been detected on vegetation in the natural environment outside of California and Oregon, we do not consider *P. ramorum* to be established outside of California and Oregon despite the many finds in streams and the movement of infested nursery plants throughout the United States. Instead, these detections are likely the result of the repeated movement of infected plant material.

To illustrate how frequently *P. ramorum* has been detected throughout the United States, we summarized detections from nursery, residential/commercial landscapes, or streams between 2003 to 2021 (Table 1). Because the data are reported differently, for example, sometimes reporting number of plants, sometimes reporting that the pathogen was detected but no number of plants, or sometimes reporting "multiple *P. ramorum*-positive plant samples", we could not report the precise number of detections in each state for each year. However, using these data we can estimate that from the various locations identified in trace-forwards and number of infected plants, that *P. ramorum* has likely been moved over a thousand times on nursery stock from regulated or unregulated parts of California and Oregon or possible other states across the United States (COMTF, 2022b; PPQ, 2019).

Table 1. *Phytophthora ramorum* detections outside of California and Oregon in nurseries, residential/commercial landscaping, and streams. Detections in nurseries and residential/commercial landscaping are regulatory incidents; the pathogen is not known to be established in any of these sites.

Year	Nursery	Residential / Commercial Landscaping	Stream
2003	WA		
2004	AL, AR, AZ, CO, CT, FL, GA, LA, MD, NC, NJ, NM, NY, OK, PA, SC, TN, TX, VA, WA	NY	
2005	GA, LA, SC, TN, WA		
2006	AL, CT, FL, GA, IN, ME, MS, PA, WA		WA
2007	AL, CT, FL, GA, IN, ME, MS, PA	WA	WA AL*, MS
2008	FL, NC, SC	MS TX, WA	FL, WA AL*, MS***
2009	AL, GA, MS, NC, NJ, SC	WA MD, PA, SC, WA	AL, FL, GA, MS, WA AL*, MS
2010	AL, IA, IL, GA, MS, NC, NY, PA, SC, VA, WA	WA	AL, FL, GA, NC, MS, WA

Year	Nursery	Residential / Commercial Landscaping	Stream
2011	SC, WA	CT	AL, FL, MS, NC, WA
2012	IN, ME, NC, NY, PA, TX, WA	ME, WA	AL, FL, GA, MS, NC, TX, WA
2013	NY, WA		AL, MS, NC, TX, WA
2014	ME, NY, TX, VA, WA		AL, FL, MS, NC, WA
2015	AL, NY, VA, WA	LA, OH, WA**	AL, MS, NC, WA
2016	MD, NY		AL, MS
2017	NY, OK		AL, MS, NC, WA
2018	AL, VA	WA	AL, MS, NC, WA
2019	AR, IA, IL, IN, KS, MD, MI, MO, NE, OH, OK, PA, TN, WA, WI	MI, NE	AL, MS, NC, WA
2020	LA, NC, OK, WA	WA	AL, MS, NC
2021	AR, NH, PA		AL, NY, WA

*Drainage ditch near nursery detection (COMTF, 2022b).
** Botanical garden (COMTF, 2022b).
***On vegetation but likely due to transport of inoculum during flooding that occurred before samples were collected (COMTF, 2022b).
Data sources: Chastagner et al., 2010; COMTF, 2022a, 2022b; Jeffers et al., 2009; Kurdyla et al., 2014; PPQ, 2019, 2021.

3. Biology and Epidemiology

We divide the biology and epidemiology into five sections. In the first section, life cycle, we focus on a description of the various structures of *P. ramorum* that allow survival and spread of the pathogen. In the second section, we focus on the distribution of the different clonal lineages of *P. ramorum*. In the third section, we discuss the modes of reproduction of *P. ramorum*. In the fourth section, we focus on the epidemiology and discuss how host susceptibility and sporulation capacity varies. Finally, in the fifth section we describe how *P. ramorum* responds to different climatic conditions and how these constrain the areas where epidemic disease is likely to develop in the natural environment.

3.1 Life Cycle

Phytophthora ramorum is an oomycete plant pathogen. Oomycetes, also known as "water molds", are similar to fungi in that their main mode of vegetative growth is through long, branching filamentous structures called hyphae (Lamour and Kamoun, 2009). A collection of hyphae is called a mycelium (Agrios, 2005), and is the main "body" of the oomycete. It is the mycelium growing in plant tissue that causes disease (Moralejo et al., 2006b; Parke et al., 2007; Griesbecht et al., 2011).

In general, oomycetes can reproduce both sexually and asexually; however, sexual reproduction (and resulting oospore production) in *P. ramorum* has only been detected once outside of the laboratory, from an isolate from a nursery in Canada (Hamelin et al., 2022) and seldom occurs under laboratory conditions (Davidson et al., 2002; Riedel et al., 2012). Instead, *P. ramorum* reproduces asexually by forming either sporangia which then produce zoospores, or chlamydospores (Grünwald et al., 2012).

Phytophthora ramorum most often follows a multicyclic (multiple generations per year) infection pattern where sporangia and zoospores are the key infection structures (as summarized in Jung et al., 2018). Infection of leaves occurs via stomata[4] or wounds (Florance, 2002; Inman et al., 2005) or possible other leaf structures. Shoots can be infected via lenticels[5] (Florance, 2002) and tree bark via medullary rays[6] (Florance, 2005). Following infection, mycelia may grow in plant tissue (Figure 2). When conditions are favorable, mycelia produce sporangia, which develop in or on the leaves and twigs of hosts (Davidson et al., 2005)[7]. The sporangia detach and wind-driven rains disperse them (Davidson et al., 2005). In many *Phytophthora* species, germination of sporangia can occur directly by forming invasive hyphae (Bassani et al., 2020); however, direct germination of sporangia in the environment is not well documented for *P. ramorum.* Instead, sporangium produces zoospores, (Widmer (2009) estimated an average of 32 motile zoospores on rhododendron) and when temperatures are conducive, zoospores may be released from the sporangium when they are exposed to water (Judelson and Blanco, 2005), such as during rain events, or where water accumulates on leaf surfaces (Widmer, 2009; Davidson et al., 2005). Zoospores are a significant propagule for infection and natural spread (Garbelotto and Hayden, 2012; Widmer, 2009) [8] because they extend the range of the pathogen beyond the sporangia's landing site (Judelson and Blanco, 2005).

[4] Stomata are cell structures, resembling minute pores, in the epidermis of tree leaves and needles that open and close and allow the exchange of carbon dioxide and water between plant leaves and the atmosphere (Grebner et al. 2013).

[5] Lenticels are pores that always remain open and allow the exchange of gasses between plant stems and fruit and the atmosphere. Lenticels are visible on fruit surfaces such as apple, avocado and mango (Yahia and Carrillo-Lopez, 2018).

[6] Medullary rays are strips of cells that link the pith and the cortex (bark) and are used for transport, storage, and defense (Grebner et al. 2013).

[7] Infections also enter through openings in the bark and tree trunks (Davidson, unpublished data: *In* Hansen et al., 2005). However, spores are not formed in the bark (Davidson et al., 2002; Rizzo et al., 2002).

[8] Garbelotto and Hayden (2012) state that "The need for a long-lasting film of water on plant surfaces for abundant infection to occur seems to suggest that most plant infection by *P. ramorum* is not determined by direct sporangium germination but rather by the zoospores that sporangia contain."

Zoospores have flagella that enable swimming (Bassani et al., 2020) on wet leaves or other substrates until they reach an infection site, then they may shed their flagella and attach themselves to the plant surface forming a cyst (as summarized in Fawke et al., 2015). However, because zoospores do not have a cell wall (Judelson and Blanco, 2005), they are prone to desiccation and rarely remain viable for more than a few days (as summarized in Garbelotto and Hayden, 2012).

Chlamydospores are survival spores produced by mycelia in response to environmental stress in many *Phytophthora* species (Judelson and Blanco, 2005). *Phytophthora ramorum* produces chlamydospores in abundance (Davidson et al., 2008; Smith and Hansen, 2008). Although the precise conditions that trigger their production in *P. ramorum* are unknown, chlamydospores are produced—and sustain infection— even under severe conditions including hot summers, cold winters, and drought (Fichtner et al., 2009). Chlamydospores germinate when conditions are conducive (Smith and Hansen, 2008). Chlamydospores, along with living mycelia in plant tissue, are most likely responsible for the long-distance movement of the pathogen outside California and Oregon through infested nursery stock (Shishkoff, 2007) (see section 2).

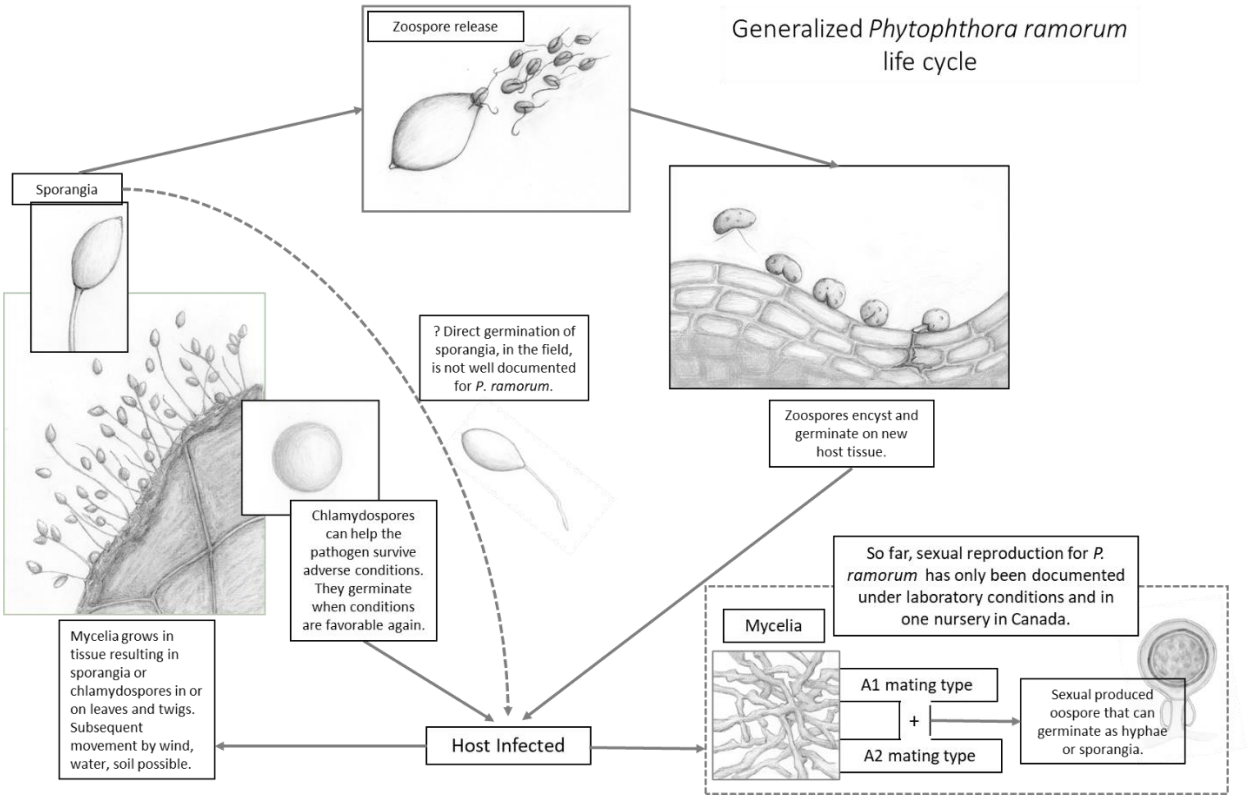


Figure 2. *Phytophthora ramorum* produces asexual sporangia, zoospores, and chlamydospores which contribute to spread and survival. This pathogen rarely produces oospores (dashed box) and their role in the life cycle of *P. ramorum* is poorly understood. So far, sexual reproduction outside the laboratory was only described once in a nursery in Canada (Hamelin et al., 2022).

3.2 Clonal Lineages

Several clonal lineages[9] of *P. ramorum* have been characterized and three have been found in the United States: North America 1 and 2 (NA1 and NA2), and Europe 1 (EU1) (Table 2). Although the pathogen has been detected in different parts of the country in nursery systems or in streams using stream baiting, it is only established in forests in California and Oregon.

Table 2. General information about the various clonal lineages of *P. ramorum* (modified from Grünwald et al., 2019). References are not provided in this table but are throughout the document. The "Hosts" column indicates host on which *P. ramorum* is frequently found and is not meant to be comprehensive. The "First findings" column indicates the year the clonal lineage was first found in a location, not necessarily the year it was first found on a particular host.

Clonal lineage	Mating type	Distribution	System	Hosts	First findings
NA1	A2	United States, Canada	Nursery	*Kalmia, Pieris, Rhododendron, Syringa, Viburnum*	Early-2000s–California
NA1	A2	United States, Canada	Forest	*Umbellularia californica, Notholithocarpus densiflorus, Quercus agrifolia*	Mid-1990s–California
NA2	A2	United States, Canada	Nursery	*Kalmia, Pieris, Rhododendron, Camellia, Viburnum*	2003–British Columbia 2004–Washington Late-2000s–California
NA2	A2	United States, Canada	Forest	*Notholithocarpus densiflorus*, *Gaultheria shallon*	2009–Washington (later eradicated) 2021–Oregon
EU1	A1	United States, Canada, European Union (Continental Europe), United Kingdom	Nursery	*Rhododendron, Viburnum*	1993–Germany 2003–British Columbia 2007 (likely earlier)–California 2010–Washington 2012–Oregon
EU1	A1	United States, Canada, European Union (Continental Europe), United Kingdom	Forest	*Notholithocarpus densiflorus* (Oregon), *Larix decidua*, *L. kaempferi* (United Kingdom, Ireland)	2011–United Kingdom (Northern Ireland and Scotland), Ireland 2016–Oregon

[9] A clonal lineage consists of individuals sharing a common ancestor that have some genetic differences (mutations) but not enough to be considered different species. Clonal lineages accumulate differences over time and among different locations. Genetic changes in clonal lineages can result in differences in virulence (like in *P. ramorum*) or in host specialization resulting in the emergence of *formae speciales* (like in *Fusarium oxysporum*).

Clonal lineage	Mating type	Distribution	System	Hosts	First findings
EU2	A1	United Kingdom, Ireland	Forest	*Larix decidua, L. kaempferi*	2007–United Kingdom (Northern Ireland, western Scotland)
IC1, IC2, IC3, IC4, IC5	A1 & A2	Vietnam	Forest	Not available	2016–Vietnam
NP1, NP2, NP3	A1 & A2	Japan	Forest	Not available	2018–Japan

In California and Oregon forests the predominant lineage is NA1[10]. Based on genetic analysis, NA1 was introduced from nurseries into the environment twice in Oregon (Goheen et al., 2017; Kamvar et al., 2015) and between eight and fourteen times in California (Croucher et al., 2013; Grünwald et al., 2019). However, it is likely that more undetected introductions have occurred.

NA2 has been detected in the environment, in plants neighboring a nursery where *P. ramorum* was detected once, in Washington State in 2009 (Chastagner et al., 2013) but was eradicated. In 2021, NA2 was detected killing tanoaks in an Oregon forest; eradication is currently being pursued (Peterson et al., 2022a).

EU1 has been found in the environment in Oregon (LeBoldus et al., 2018) in close proximity to NA1 (Sudden Oak Death Dashboard, 2023) and in Del Norte County, California in 2020 (COMTF, 2022a). EU1 was also detected in a stream in Humboldt County, CA and in a Humboldt County retail nursery in 2007 (COMTF, 2022b).

NA1, NA2, and EU1 have all been detected in nurseries in the United States (Goss et al., 2011; Hansen et al., 2003; Ivors et al., 2004).

The remaining clonal lineages are: EU2, found in the environment in Northern Ireland and western Scotland (Van Poucke et al., 2012). Additionally, eight more recently identified lineages IC1 to IC5 (Indochina) and NP1 to NP3 (Japan) were published by Jung et al. (2021). The Asian lineages co-occurred in some areas, providing opportunities for sexual recombination. The phenotypic and genetic diversity of the Asian lineages and the absence of severe symptoms on the host species suggest *P. ramorum* is native to laurel forests between eastern Indochina and southwest Japan (Jung et al., 2021).

3.3 Reproduction

The way *P. ramorum* reproduces is of concern because it affects spread and the predictability of the pathogen's behavior. Sexual reproduction allows for the introduction of genetic variation into

[10] NA1 was also detected and eradicated from a botanical garden, Bloedel Reserve in Washington. The following plants were removed: *Mahonia, Rhododendron, Viburnum, Gaultheria, Vinca, Vaccinium, and Camellia* (Elliott et al., 2021b).

a population, which can lead to progeny that are better adapted to new or changing conditions (Sun and Heitman, 2011). For *P. ramorum*, sexual reproduction had not been reported to naturally occur until 2021. This is when variants of *P. ramorum* resulting from the hybridization via sexual recombination between NA2 and EU1 lineages were identified from a nursery in British Columbia[11] (Hamelin et al., 2022). Sexual reproduction could bring larger genetic changes via direct recombination as well as the production of double-walled oospores. Oospores, along with chlamydospores, are considered the most persistent propagules produced by *Phytophthora* species (Erwin and Ribeiro, 1996; Smith, 2007).

The recently recognized sexual recombination event in the Canadian nursery was unexpected. *Phytophthora ramorum* is heterothallic[12] and has two mating types, A1 and A2, required for sexual reproduction (Werres et al., 2001). The A1 isolates are primarily found in the European lineages, whereas A2 isolates are primarily found in the North American lineages (summarized in Grünwald et al., 2019). Thus, the lack of observed sexual reproduction could simply be the result of the geographic distance between different mating types. If that is the case, as the EU1 lineage becomes more widespread in North America, sexual reproduction could occur as the A1 and A2 mating types become more common and if they occur in the same places. However, Garbelotto et al. (2006) suggested that because the two mating types have been isolated for so long, they may have lost compatibility, resulting in a mating system that is no longer "perfectly functional". If true, this would reduce the likelihood of *P. ramorum* successfully reproducing sexually, even if the two mating types are co-located. However, if sexual reproduction of *P. ramorum* were to occur in the United States, it could also bring new genetic traits into the population and change the epidemiology of the pathogen as we know it today.

Asexual reproduction, on the other hand, rapidly creates genetically similar progeny that are adapted to existing conditions but potentially less adaptable to new conditions (Sun and Heitman, 2011). However, there is evidence that even the asexual populations of *P. ramorum* in the United States are evolving to some degree (Dale et al., 2019; Yuzon et al., 2020) and that "host jumps", as observed with EU1 and EU2 jumping to Japanese larch (*Larix kaempferi*) (Brasier and Webber, 2010), can occur in the right environmental conditions.

3.4 Epidemiology

Phytophthora ramorum is a highly adaptable organism: it can live as a pathogen as well as survive in decaying plant tissue in or on the ground and in streams or puddles. However, while it can quickly colonize fresh, recently fallen leaves, it cannot colonize dead leaves (Aram and Rizzo, 2018) and cannot complete its lifecycle without infecting a host[13] (Fry and Grünwald, 2010). Plant tissue infected with *P. ramorum*, both living and decaying, contain mycelia (Widmer, 2009) and almost always chlamydospores (Smith, 2007). These fungal structures are most likely responsible for the movement of the pathogen beyond California and Oregon on nursery stock. *Phytophthora ramorum* also survives in aquatic environments and infection with *P. ramorum* has been demonstrated in submerged tanoak tissue (Morgan, 2017). However, the epidemiological importance of stream to land transmission has not yet been demonstrated in

[11] Hamelin et al. (2022) stated that "the disease was apparently eradicated in that nursery".
[12] Heterothallic: requiring two opposite mating types found in two individuals for sexual reproduction via oospores (as opposed to homothallic species).
[13] *P. ramorum* has been reported to survive up to sixteen weeks in streams (Aram and Rizzo, 2018).

nature and could explain why infections have not occurred in hosts near streams even though *P. ramorum* has frequently been detected in streams.

The disease cycles caused by *P. ramorum* are complicated because it infects many plants and these hosts play different roles depending on their susceptibility and ability to produce inoculum (e.g., data and observations from Conrad et al., 2019; Hansen et al., 2005; Moralejo et al., 2006a; Tooley and Browning, 2009; Tooley et al., 2004). In some forests, combinations of species growing in proximity (host complex) under conducive conditions need to be present for the disease to reach epidemic proportions (Dillon et al., 2014; Garbeletto et al., 2017). Moreover, the host range is becoming broader as researchers continue to discover new hosts (e.g., Webber et al., 2010; Rooney-Latham et al., 2017; Elliott et al., 2021a; Werres et al., 2001, USDA-APHIS, 2023). Additionally, clonal lineages of *P. ramorum* differ in pathogenicity as do isolates of the pathogen (Harris et al., 2021; Manter et al., 2010' Søndreli et al., 2021).

Phytophthora ramorum is established in coastal areas of California and Oregon. Coastal California has a Mediterranean climate, with warm dry summers and wet winters. In the northernmost portion of coastal California and Oregon, the climate is cool and humid, similar to that of the United Kingdom, where *P. ramorum* also causes serious disease. Research on *P. ramorum* completed since our last risk assessment in these areas, allows us to explore which factors related to the pathogen-host relationship and environmental conditions are crucial for disease development.

The development of *P. ramorum* diseases for true oaks (*Quercus* spp.) in California and Oregon forests, likely occurred because highly susceptible dead-end hosts grow near transmissive hosts or transmissive hosts grow near each other (Davidson et al., 2011; DiLeo et al., 2014; Dillon and Meentemeyer, 2019; Garbelotto and Hayden, 2012; Hansen et al., 2008) in a conducive environment. The host complex may consist of transmissive, dead-end, and other hosts. Transmissive (sporulating) hosts drive the epidemic, ensuring the persistence of the disease over time. Dead-end hosts (*Quercus* spp.), which are infected but do not contribute to spread, are killed by the pathogen (Garbelotto et al. 2017). Other hosts may also produce inoculum in California and Oregon, but typically not in large enough quantities to start or sustain an epidemic in forests (Figure 3). In California and Oregon, the transmissive hosts primarily driving the epidemic are California bay laurel (*Umbellularia californica*) and tanoak (*Notholitocarpus densiflorus*), respectively (Rosenthal et al., 2021). These two species occur in forests with highly susceptible true oaks. While foliar infections do not negatively affect *U. californica* (DiLeo et al., 2009), *N. densiflorus* is less tolerant to the disease (Hansen et al., 2005; Hansen et al., 2008) and is the most commonly killed species in Oregon (Goheen et al., 2017). Therefore, although the sporulation capacity observed in the field when *P. ramorum* infects *N. densiflorus* is lower than *U. californica*, the high susceptibility of *N. densiflorus* keeps the infection cycle going (Hansen et al. 2008; Hansen et al., 2005).

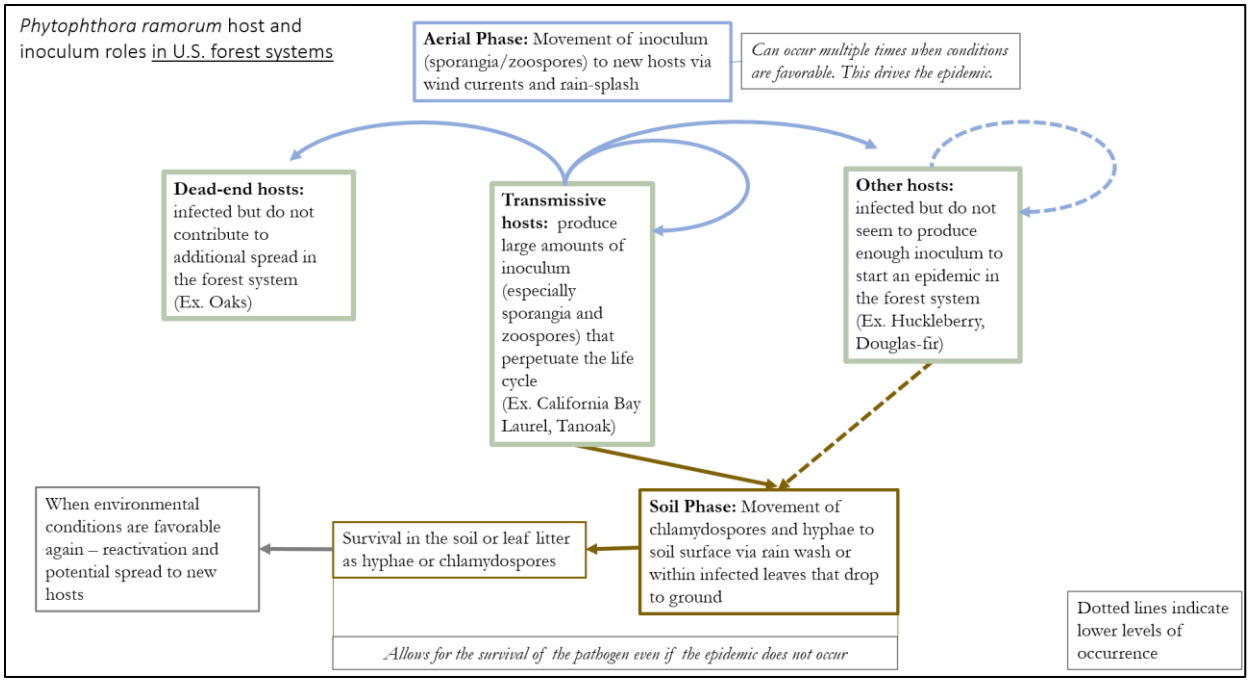


Figure 3. Epidemiological model for *P. ramorum*. In California, transmissive host, *U. californica*, infect dead end hosts, *Quercus spp.*; this host transmission sustains the *P. ramorum* epidemic. In Oregon, *N. densiflorus* is the key transmissive host but is also the most commonly killed host.

3.5 Climatic Suitability

Many studies have investigated the role of ecological and climatic factors in the spread of *P. ramorum*. For example, research from Meentemeyer et al. (2008), DiLeo et al, (2014), and Garbelotto et al. (2017) generally indicates that pathogen spread in California is positively correlated with the presence of *U. californica* and with ample rainfall and mild temperatures in late spring.

Nevertheless, predicting the specific areas where *P. ramorum* is likely to be present and cause severe disease is complicated and has proven difficult. Many early predictive modeling efforts—including APHIS's own—determined areas in the eastern United States were at high risk or highly suitable for *P. ramorum* (Koch and Smith, 2008; Magarey et al., 2008; Venette and Cohen, 2006). However, in forests, no outbreaks or impacts have been reported in the United States outside California and Oregon, indicating these areas are probably not as suitable as the models predicted.

Part of the difficulty in predicting where *P. ramorum* is likely to spread and cause severe disease is that much of what laboratory studies indicate are important environmental thresholds for *P. ramorum* survival and infection cannot be modeled because data does not exist at a useful scale to do so. For example, studies indicate *P. ramorum* zoospores need a film of water on leaves to swim to infection sites. This typically occurs after rain events or in the early morning with condensation that results in dew on leaves (Garbelotto et al., 2003; Garbelotto et al., 2017). However, leaf wetness cannot be modeled reliably because forest vegetation produces microclimates with unique conditions compared to surrounding areas. These microclimates greatly influence the dynamics of *P. ramorum* disease as shown in oak vs. redwood (*Sequoia*

sempervirens) forests of California (Davidson et al., 2011) and larch (*Larix* spp.) in the United Kingdom (Harris, 2014).

Further, the individual effects of temperature, rain, and leaf wetness on *P. ramorum* infection are difficult to separate because in nature they tend to fluctuate simultaneously (e.g., rain events usually result in rapid temperature drops and an increase in leaf wetness). Infection is strongly related to rain events when the temperature is conducive; however, it is difficult to model the appropriate duration of rain events or amount of rain that occurs close to a specific temperature range.

4. Likelihood of *P. ramorum* Spread and Disease Development

Over twenty-five years after the first detection of the disease caused by *P. ramorum* in the United States, California and Oregon remain the only states where *P. ramorum* is established in the natural environment. Even though infected nursery material (Table 1) has brought inoculum to areas previously thought to be high risk or highly suitable for *P. ramorum*, disease has not been observed in these areas. This suggests the risk *P. ramorum* poses to the rest of the United States is much lower than the 2008 risk assessment originally predicted, but the question of why that is the case remains.

Because zoospores are fragile, we assume the pathogen survives long-distance movement in nursery stock as chlamydospores or as hyphal colonies in infected tissue. However, as explained in section 3, infections and epidemics generally occur via the many zoospores that are released from the sporangia. Therefore, for *P. ramorum* epidemics to occur in a new area after nursery stock is moved, there must be favorable conditions to promote new infections. These could occur via chlamydospore germination and the formation of sporangia, or via production of sporangia in the mycelia inhabiting the infected tissue. These newly formed sporangia would then release zoospores and, if susceptible host material is available, these would start a new cycle of pathogen infection.

4.1 Climatic Suitability: Pathogen survival, stress, and infection maps

Our understanding of the range of some climatic conditions required for *P. ramorum* to transition from survival to infection, such as relative humidity and timing of rain events, has improved since our 2008 risk assessment.

Here we used Maxent models to explore what climatic variables explain where *P. ramorum* occurs (Appendix A). We found that temperature was a major driving factor based on the location of infections[14] in California, Oregon, (COMTF, 2019; Navarro, 2019), and Europe (Shamoun et al., 2018), that were tested with 25 climatic variables (see Appendix A). In the most predictive models, the mean diurnal range in temperature had by far the greatest relative importance for both the EU1 (61.6) and NA lineages (81.3). For perspective, the importance of all variables included in the model sums to 100; the next most predictive variable for the EU1 lineage was precipitation of the driest month (22.1) and the next most predictive variable for the NA1 and NA2 lineages was minimum temperature of the driest month (4.6). The response curves indicate the ecological relevance and range of values that are most optimal for infection. These

[14] We did not use data where the pathogen was only found using stream baiting or in nurseries.

show a 20°C difference between the minimum and maximum daily temperatures, or daily amplitude[15]. This is likely found where day and night temperatures do not fluctuate too much, this occurs most often in areas where temperatures tend to be mild. In areas where the temperature amplitude is greater than 20°C (36 °F), infection is unlikely. Understanding the relationship between this 20°C amplitude, as well as the *P. ramorum* temperature thresholds reported from laboratory studies, helps narrow down the areas where infection could occur.

Survival of hyphal colonies and chlamydospores (Figure 4A)

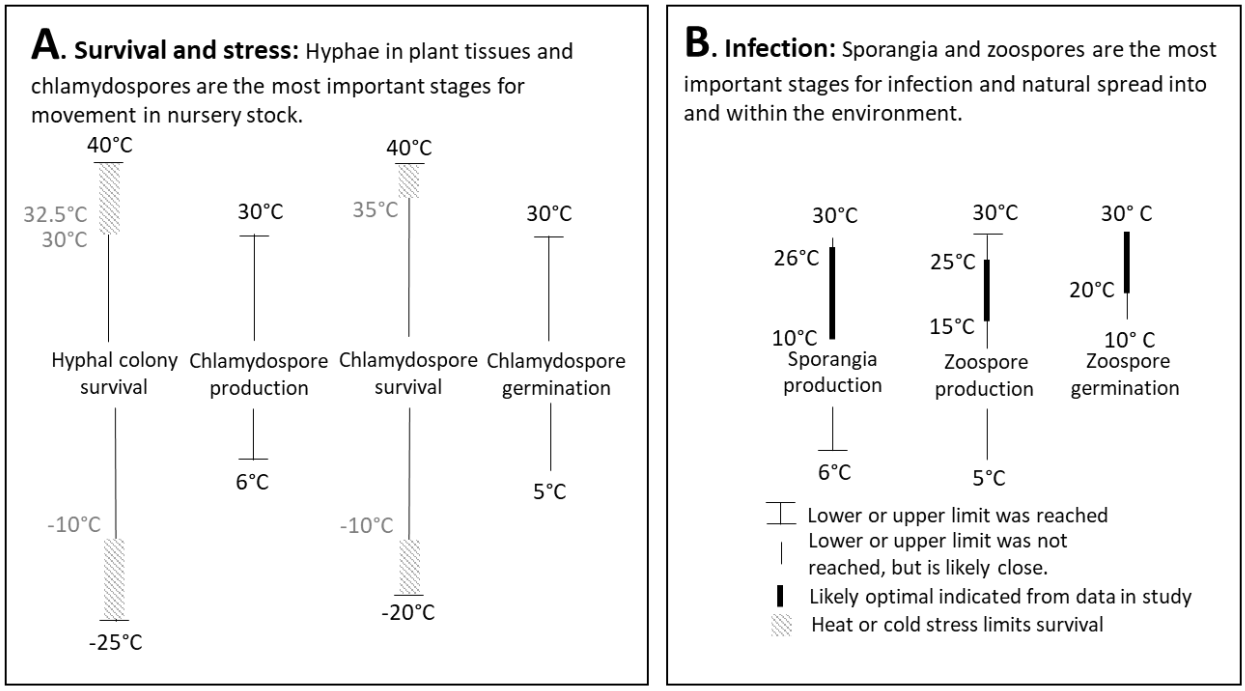
Laboratory studies indicate hyphal colonies and chlamydospores survive a wide range of temperatures and can tolerate cold extremes. *In vitro* studies found hyphal tissue can withstand high temperatures but exposure to 32.5°C and 35°C were stressful and reduced survival after 8 hours (Browning et al., 2008). Temperatures of 37.5–40°C were lethal to *P. ramorum* hyphae within several hours, and temperatures of 42.5–50°C were lethal within minutes (Browning et al., 2008). In northern California forests, the number of days the temperature was above 30°C was correlated with reduced survival of the pathogen in *U. californica*, suggesting high temperatures are less conducive to survival of *P. ramorum* (DiLeo et al., 2014).

Chlamydospores are likely to sustain the pathogen population after climatic stress like hot summers or cold winters (Fichtner et al., 2009). Tooley et al. (2008) found chlamydospores are stressed at 35°C, where they can survive up to four days in infected leaf tissue, and at 40°C, where they can survive for one to two days as free chlamydospores in sand. Tooley et al. (2014a) also found chlamydospores are abundantly formed inside leaf tissue within the boundaries of necrotic lesions and can germinate at rates greater than 50 percent, which is sufficient for *P. ramorum* to survive (Fichtner et al., 2009). Chlamydospore germination occurs at temperatures ranging from 5–30°C (Tooley et al., 2014a).[16]

Sporangia and zoospore production to start an infection (Figure 4B)

Although hyphal colonies and chlamydospores can survive a wide range of temperatures, laboratory studies indicate sporangia and zoospores are produced in a relatively narrow temperature range. Englander et al. (2006) found that although sporangia production occurred at temperatures up to 30°C (the highest tested temperature), more sporangia were produced between 10 and 26°C compared to other temperatures tested, and the optimal range for sporangia production was between 16 and 22°C. Sporangia were not produced at 6°C (Englander et al., 2006). In California forests, Davidson et al. (2005) showed that a relatively low level of zoospore production occurred at 5°C, the lowest temperature tested, but did not occur at 30°C. Their data also indicated more zoospores were produced at 15-25°C than at other temperatures. Zoospore germination occurs over a broad range of temperatures but 10–30°C is optimal depending on the ambient humidity (Turner and Jennings, 2008). *In vitro* experiments showed that humidity levels of 100% were critical for zoospore germination and that optimum temperatures were between 20 and 30°C (Turner and Jennings, 2008).

[15] This is calculated as = ∑ (daily maximum temperature – daily minimum temperature)/ days in the month.
[16] Under laboratory conditions chlamydospore germination can infect nearby roots (Shishkoff, 2007), but these infections are insignificant for driving the epidemic compared to sporangia and zoospore production.



A. Hyphal colony survival: In vitro, did not survive 40°C for more than a few hours. 32.5°C reduced survival after 8 hours. A few colonies survived 24-hour exposure to -25°C. Survival begins to diminish after exposure to -10°C and lower[1]. In plants, fewer days above 30°C was correlated with a greater likelihood of survival [2].
A. Chlamydospore production: In vitro, did not occur at 30° and 6°C [3] .
A. Chlamydospore survival: In vitro, did not survive 1 day of exposure at 40°C and -20°C. Little or no recovery was observed at -10° [4].
A. Chlamydospore germination: In vitro, did not occur at 30°C and only 3 percent germinated at 5°C [5].
B. Sporangia production: In vitro, did not occur at 6°C and tested to only 30°C and production diminished. 10°C to 26°C is an inclusive optimal range indicated in their data [6].
B. Zoospore production: In vitro, did not occur at 30°C and tested to only 5°C, which had low production. 15° to 25°C is an inclusive optimal range indicated in their data [7].
B. Zoospore germination: In vitro, a broad range of temperatures ranging from 10-30 °C support germination; at 100% humidity optimum temperatures ranged between 20-30 °C [8].

Figure 4. Survival and stress (A) and infection (B) temperature thresholds for *P. ramorum* created using temperature threshold data. **1** – Browning et al. (2008); **2** – DiLeo et al. (2014); **3** – Englander et al. (2006); **4**- Tooley et al. (2008); **5** - Tooley et al. (2014a); **6** – Englander et al. (2006); **7** – Davidson et al. (2005); **8** – Turner and Jennings (2008).

Climate model (Figure 5A, 5B)

The maps we present should not be considered predictive of establishment. Instead, they are meant to illustrate the general areas where temperatures are more or less favorable for *P. ramorum* survival and infection, and where temperatures are likely extreme enough to stress *P. ramorum* and hinder its survival. To create these maps, we use temperature to identify areas of the United States where *P. ramorum* is relatively more or less likely to survive, be stressed, or cause infection. There is still too much uncertainty for us to precisely estimate the likelihood disease will develop in areas outside California and Oregon. For example, we still do not understand how climatic conditions affect host susceptibility or where movement from transmissive hosts to dead-end host is likely to occur. In fact, except for some eastern forest understory species (Tooley et al., 2009; Tooley et al., 2013; Tooley et al., 2016; Tooley et al., 2014b), we do not have a good understanding of host transmission and susceptibility for tree and shrub species outside of California and Oregon. For this reason, we do not include hosts in these maps.

To create the survival and stress (Figure 5A), and infection (Figure 5B) maps for *P. ramorum*, we used the Spatial Analytic Framework for Advanced Risk Information Systems (SAFARIS, 2020) and Parameter-elevation Regressions on Independent Slopes Model (PRISM) daily temperature data at 4-km resolution from 2010 to 2019 (PRISM, 2021). For each year from 2010 to 2019, we calculated the number of days the following criteria were met:

- **Survival:** The minimum temperature never went below −10°C and the maximum temperature never went above 30°C on the same day (Figure 5A).
- **Stress:** The daily minimum temperature was below −10°C or the maximum temperature was above 30°C (Figure 5A).
    - We used 30°C as the upper transition between survival and stress because this is the temperature at which chlamydospore production ended, survival on *U. californica* began diminishing, and heat stress began, leading to mortality after at least a few days.
    - We used −10°C as the lower transition between survival and stress because this is the temperature at which cold stress began for hyphal colonies and chlamydospores.
- **Infection:** The minimum temperature never went below 6°C and the maximum temperature never went above 26°C on the same day (Figure 5B).
    - We used a 20°C range in minimum and maximum temperatures, which was identified as a key variable and value in the Maxent analysis using data on the location of infections.
    - We selected 26°C as the upper limit because that was the likely upper optimal temperature for sporangia production and 6°C as the lower limit because sporangia production did not occur at or below that temperature.
    - To accommodate the 20°C range, we preferred using a colder lower limit rather than a hotter upper limit because exposure to hot temperatures likely prevents sporangia germination and reduces the number of infections (e.g., DiLeo et al., 2014).

We then averaged the number of days that met the criteria and presented the data at monthly intervals over the 10-year period. For the *P. ramorum* stress map, we averaged the number of days that would result in cold or heat stress and then summed cold and heat stress days (Figure 5A,B, for larger maps that include Alaska, Hawaii, and Puerto Rico see Appendix B).

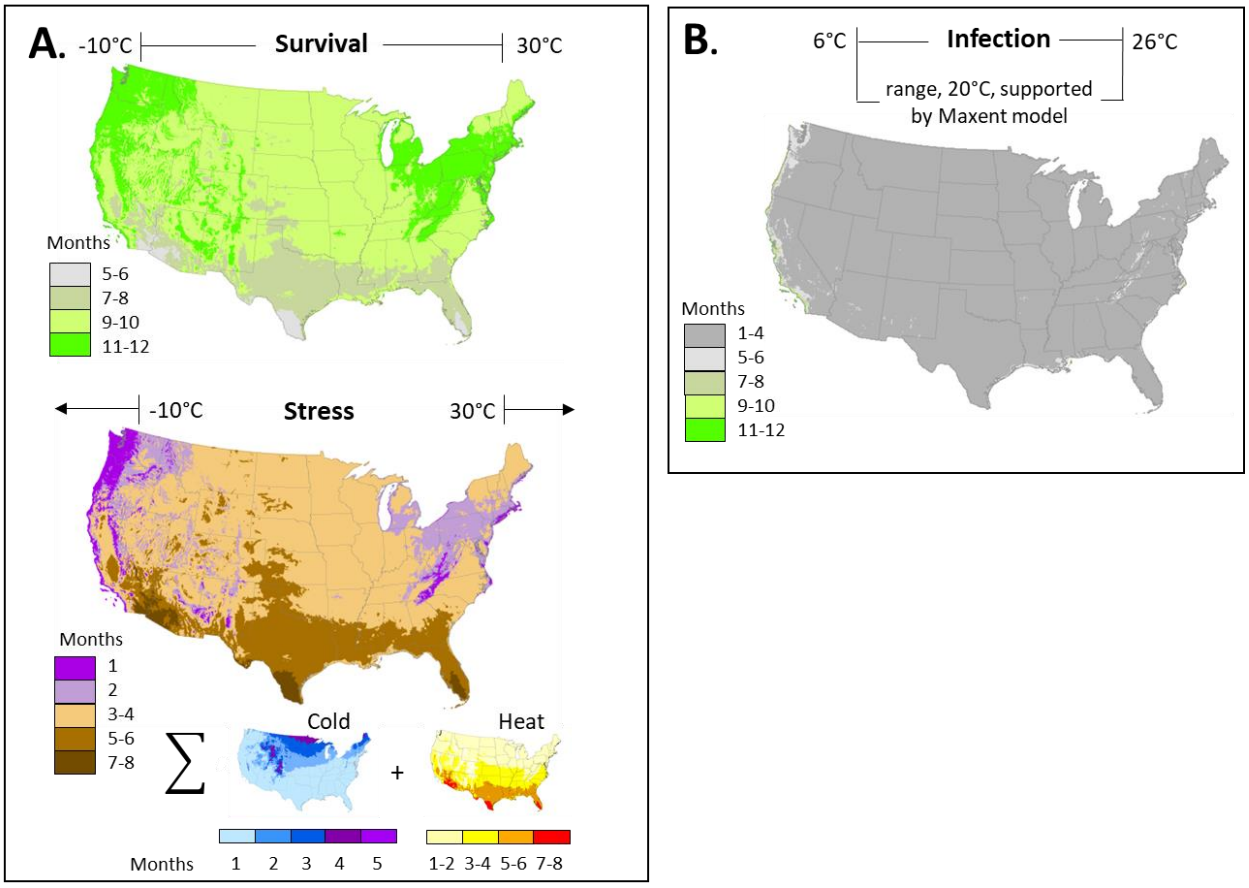


Figure 5. . Survival and stress (A) and infection (B) maps for *P. ramorum* created using temperature threshold data. For larger maps that include Alaska, Hawaii, and Puerto Rico see Appendix B.

Our climatic model suggests temperatures suitable for survival of *P. ramorum* in areas outside the west coast of the United States are common (Figure 5A). In contrast, temperatures suitable for infection are rarer (Figure 5B). Periodic and frequent environmental stresses are likely to knock back inoculum production in these areas. Since the amount of inoculum is a determining factor of disease severity (review by Garbelotto and Hayden, 2012) and sporangia and zoospores are the most significant inoculum for epidemic infection (e.g., Widmer, 2009), it is unlikely that there would be enough inoculum produced for *P. ramorum* to persist in the environment long enough (outside of the west coast) to reach a receptive host for an infection to occur. Another factor contributing to this is that zoospores, which are abundantly produced from sporangia (Widmer, 2009), dry out easily and would only be viable for a few days even when the temperature is favorable. We hypothesize this is the reason diseases caused by *P. ramorum* have not reached significant levels in the environment outside of Oregon and California (Figure 6), despite many finds in nurseries, residential locations, and waterways over the past several years[17].

[17] NA2 was detected in the environment in plants neighboring a nursery in Washington State in 2009 (Chastagner et al., 2013). This was likely a spillover from the nursery and was eradicated.

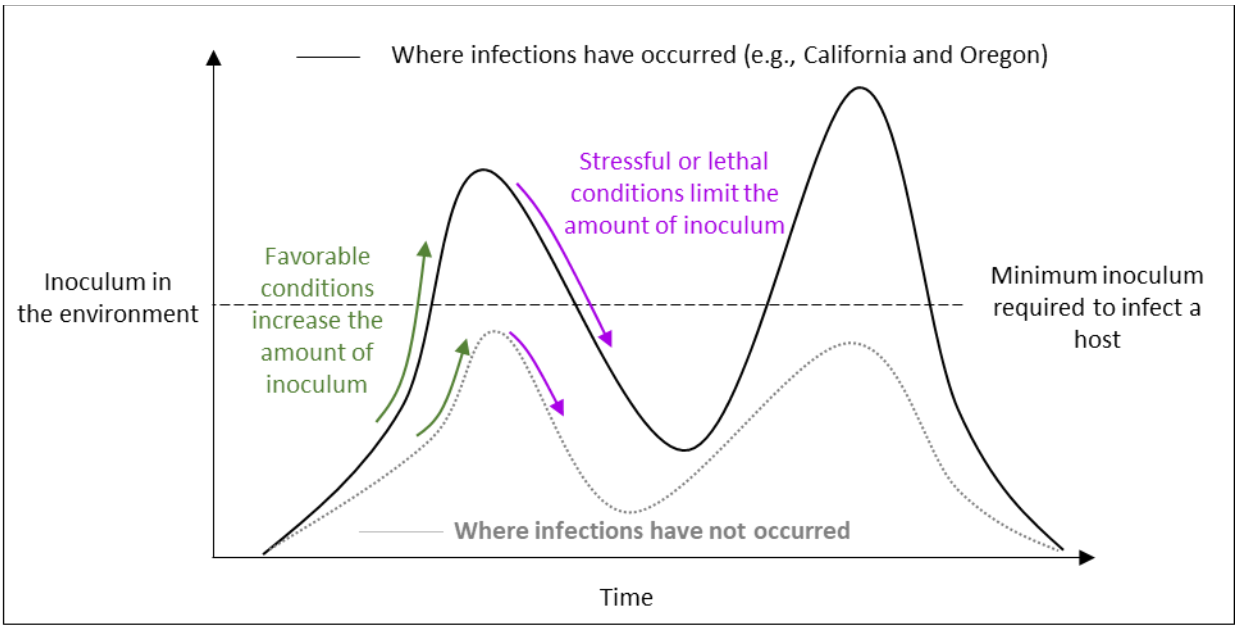


Figure 6. Conceptual diagram of *P. ramorum* environmental inoculum in areas where incursions have occurred but the disease has not been observed. It is likely the amount of inoculum in the environment (dotted gray line) has never reached the minimal inoculum load required to infect a host (dashed black line) because of lethal environmental conditions (magenta arrow). The amount of inoculum in the environment in California and Oregon (solid black line) has passed the minimum inoculum load required to infect a host (dashed black line) because conditions are favorable (green arrow).

4.2 Host Susceptibility and Competency

Our climatic suitability model does not include hosts because we do not have enough information to model susceptibility and competency throughout the United States and therefore the model is not restricted by host presence or absence in any given area. Host susceptibility can vary with phenology (Harris and Webber, 2016), nutritional status, and factors such as drought or herbivory that stress the plant. For *P. ramorum* to occur in new areas, inoculum must reach the host at a time when it is receptive to infection.

Studies have focused mostly on the biology of the pathogen; only a few studies address the susceptibility of the host to *P. ramorum* and even fewer address the competency of the host in producing *P. ramorum* spores (Appendix C). Most of these have been laboratory studies focused on either identifying potential hosts or determining the environmental ranges under which those hosts are infected or produce inoculum. In the field, host receptivity to infection may be more limiting than the inherent susceptibility estimated in a greenhouse or laboratory study. In other words, a host may be susceptible to *P. ramorum*, but its phenology or physiological status may cause it to be unreceptive when inoculum is available.

Furthermore, it is not the individual species performance per se in producing inoculum or being susceptible to infection, but the forest species composition and structure that lead to disease development. For example, in many of the forests where disease outbreaks have been most severe, transmissive hosts occur near susceptible dead-end hosts, allowing the “spill over” of the pathogen from host to host. Sporangia and zoospores carried by wind-blown rain or dripping, splashing, and running down rainwater from the taller canopy of transmissive hosts to reach the trunks of dead-end hosts (Dillon and Meentemeyer, 2019; Rizzo et al., 2005) perpetuate the

disease cycle and promote spread and impacts. Where inoculum is present in enough quantity in the environment at the right time, for example from *U. californica*, nearby susceptible dead-end hosts are infected and trees may weaken and die, for example *Quercus agrifolia* . Dead-end hosts have the most severe disease symptoms. However, *P. ramorum* does not typically produce infectious propagules on dead-end hosts, and when it does, production is not sufficient to be epidemiologically significant. Consequently, both transmissive hosts, which produce infectious propagules, and dead-end hosts are typically required for the infection to perpetuate *and* produce large-scale tree mortality. In the United States, *Notholitocarpus densiflorus* is an exception because it is a transmissive host that also dies of *P. ramorum* infections; because of its high susceptibility and host competency, it is the species with the highest mortality rate in Oregon forests (Goheen et al., 2017).

Forest structure, more specifically the presence of infected and competent overstory hosts, also determines pathogen spread and *P. ramorum* disease expression. In Oregon forests, infection of understory species usually occurs within close proximity to infested *N. densiflorus* trees in the overstory, which are the main source of inoculum in areas where tree mortality is observed (Peterson et al., 2014). Infection of the forest understory with *P. ramorum* allows the pathogen to persist but does not result in significant infection of other neighboring species (Peterson et al., 2014), likely due to absence of downward rain splash dispersal of the inoculum, or lower inoculum levels due to lower host competency or, a combination of the two.

Host species and forest composition in the eastern United States and Washington State differ from that in California and Oregon. For example, the natural range of the key transmissive host, *U. californica* in California, and *N. densiflorus* in Oregon, are only in those two states (Kartesz, 2015). While eastern forests have qualities that seem conducive to the development of *P. ramorum* disease, it is still unclear if eastern hosts could produce *P. ramorum* inoculum under field conditions in sufficient amounts to infect other host species and if this would occur at times when hosts would be susceptible. On the east coast, evergreen hosts like *Rhododendron* sp. and *Kalmia* sp. are shrub hosts of *P. ramorum* that often occupy the forest understory, but they can reach heights comparable to *U. californica* and *N. densiflorus*. When this happens, this could potentially support vertical and lateral dispersal of inoculum similar to how *Rhododendron ponticum* functions in *P. ramorum* epidemics in much of the UK (DEFRA, 2007). Additionally, *Rhododendron sp.* and *Kalmia sp.* as well as *Magnolia sp.* are evergreen, so infected leaves could potentially produce inoculum throughout the year whenever favorable conditions occur. Last, these three eastern hosts, also occur in mixed forests with oak species, however disease outbreaks have not been seen outside of California and Oregon. It is possible that transmissive hosts are not producing enough inoculum, that eastern hosts are not as susceptible, or a combination of both.

While forests outside the west coast do have *P. ramorum* hosts, studies on the susceptibility and competency of these hosts to *P. ramorum* have been restricted to greenhouse or laboratory studies in seedlings or detached plant tissue. Comparisons among these studies are also challenging because they use different clonal lineages, methodologies and fungal structures for inoculation, host tissue, and environmental conditions. For instance, Tooley at al. (2014b) first reported on the susceptibility of three eastern tree species but the experiment did not compare their susceptibility with western hosts. Consequently, we cannot yet make comparisons on

competency and susceptibility between western and eastern hosts and there is too much uncertainty about eastern hosts to evaluate the influence they would have on establishment and impact.

4.3 Likelihood of Introduction: Conclusions

Based on our evaluation of recent literature on *P. ramorum* and mapping pathogen survival, stress, and infection, we determined that there appears to be three interrelated factors (Figure 7) that make the occurrence of disease caused by *P. ramorum* unlikely outside of California and Oregon. These factors are likely causing an asynchrony between the susceptible stage of the host complex and the infective stage of *P. ramorum* when environmental conditions are favorable for infection and disease development. These factors are:

1. Environmental stress, such as heat stress, decreases inoculum survival of *P. ramorum.*
2. Inoculum does not build up in sufficient amounts to produce significant disease.
3. The infectious stage of *P. ramorum* does not overlap with the susceptible stage of hosts in the host complex for enough time, reducing the likelihood of infection and disease development.

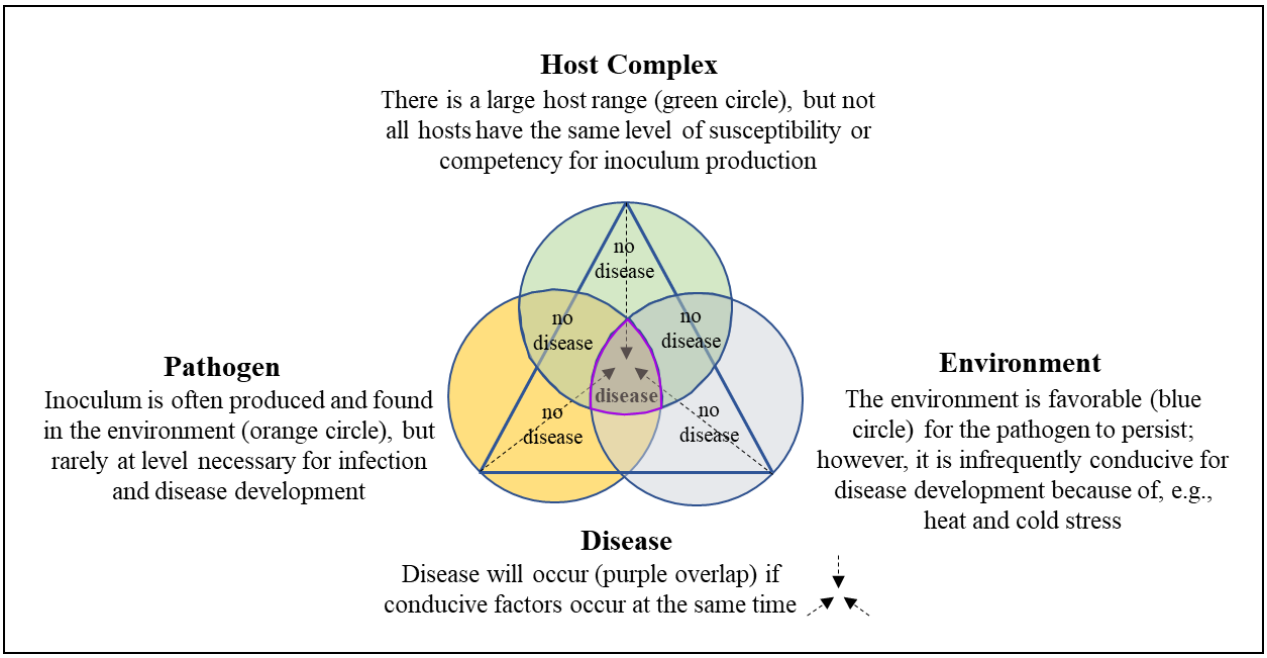


Figure 7. Likely factors that over time cause the asynchrony between the susceptible stage of the host complex and the infective stage of *P. ramorum* when environmental conditions are favorable for infection and disease development.

P. ramorum is also unlikely to transition from surviving in decomposing litter in water systems to producing sporangia and zoospores that can infect living host tissue. This conclusion is supported by environmental sampling, such as stream baiting, indicating *P. ramorum* is found

across a wide range of conditions in decaying leaves and streams but infections do not occur in surrounding vegetation[18].

More research would be helpful to improve our understanding of spread and disease development outside of Oregon and California and help reduce the uncertainty surrounding *P. ramorum*. This includes research to determine the:

- Susceptibility and competency of the various host combinations in the host complex:
    - Comparative field or greenhouse studies of western and eastern hosts.
    - Studies on how phenology affects susceptibility of western and eastern hosts.
- Timing of inoculum production of the various host combinations in the potential host complexes, understanding when the greatest amount of inoculum is likely to be produced and if that overlaps with when hosts would be susceptible.
- Timing and duration, for example the number of consecutive days, temperatures or other climatic factors are favorable for infection, allowing inoculum to increase in a plant.

However, our understanding of the environmental conditions necessary for infection to develop at this point in time leads us to conclude the likelihood of introduction is much lower than we initially determined in 2008.

5. Consequences of Introduction

5.1 Nurseries

The consequences of *P. ramorum* and ramorum blight in nurseries are not well documented and are difficult to quantify. In the scientific literature, we identified only one study describing the consequences after *P. ramorum* was detected in a retail nursery and this publication only presented the market price of plants that were destroyed (e.g., data from Dart and Chastagner, 2007). There have been many other instances where individual plants or entire blocks of plants have been destroyed from production or retail nurseries (COMTF, 2022b). Qualitatively, if left unmitigated in a nursery, *P. ramorum* is likely to damage the leaves of hosts and cause shoot dieback (as summarized in Tjosvold et al., 2005) and can cause damping off (death) of plants in early stages of propagation. The extent of the infestation would vary according to the management practices.

Though the consequences are difficult to quantify, there is a good understanding of the best management practices that are needed to mitigate *P. ramorum* and ramorum blight. Many of these practices have been developed since *P. ramorum* was introduced in California and Oregon. For example, though *P. ramorum* is primarily a foliar pathogen, it can persist in nursery soils (Peterson et al., 2022b). In which case steaming (Schweigkofler et al., 2014) or solarization (Yakabe and MacDonald, 2010) can be effective mitigations to sanitize the nursery and help prevent spread within the nursery and the repeated movement of infected material from the same nursey. Other practices include (Parke et al., 2003; Tjosvold et al., 2005; Tjosvold 2015):

- Use of clean containers with a clean potting mix; ensure that nursery beds have well drained materials, such as gravel, below them; and clean tools frequently.

[18] In 2008 *P. ramorum* was also detected on vegetation in Mississippi in a riparian area likely because of flooding that occurred prior to the samples being collected (COMTF, 2022b). In surveys from vegetation later that year in the same location the pathogen was not detected (COMTF, 2022a).

- Irrigate early in the day or use drip irrigation to minimize how long leaves are wet; disinfect irrigation when water sources are recirculated; do not over water.
- Apply fungicides to protect plants.
- Isolate new incoming plant material for observation and quarantine; remove infected plants, plant parts, and plants adjacent to known infected plants.

These practices are effective for other *Phytophthora* spp. and downy mildews that are common, often damaging, and widespread throughout the United States. These practices are described in extension publications for *Phytophthora* spp. (e.g., Beckerman and Creswell, 2020, Concklin, 2011; Griesbach et al., 2012; Parke, 2011), including the root rot complex (Concklin, 2011; Moorman, 2014) and downy mildews (e.g., Beckerman 2022).

Most best management practices to exclude *P. ramorum* and *Phytophthora* spp. also include (CANGC-HRI, 2008; Griesbach et al., 2012; Kliejunas, 2010; Suslow, 2007):

- Training – When growers and nursery managers are trained it enhances prompt disease recognition and the application of management practices.
- Pest scouting and audits – Regularly inspect plants in and around nurseries to ensure early detection.
- Recordkeeping and traceability – Record incoming and outgoing plants to facilitate trace-backs and trace-forwards.
- Documentation – Record evidence that best management practices are being implemented to help identify the cause of infestations.

There is little doubt that years of experience with *P. ramorum* in California and Oregon nurseries has led to improved management practices. For example, the Safe Procurement and Production Manual: A Systems Approach for the Production of Healthy Nursery Stock (Griesbach et al., 2012) focuses on the nursery as a system and describes practices to mitigate soil pathogens or prevent quarantine pests from entering. This, along with a Systems Approach to Nursery Certification (SANC, 2023) have become important sources for better management and certification to improve the quality of nursery stock. These tools and management practices that are already applied for other *Phytophthora* spp. or downy mildews, are likely to mitigate the impacts of *P. ramorum* in nurseries. What is uncertain, however, is how many or the extent to which nurseries already routinely apply these practices. If a nursery is not applying these practices, detecting *P. ramorum* in their nursery would likely require an update to their management practices and they would incur some additional costs.

5.2 Environment

The consequences of *P. ramorum* in the environment are well documented. In California, many oak species, *Q. agrifolia*, *Q. kelloggii*, *Q. parvula* var. *shrevei*, and *Q. chrysolepis*, are extremely susceptible to the disease (Swiecki and Bernhardt, 2013) and millions trees have been killed by the disease. Cobb et al. (2020) estimated that over 40 million trees greater than 1 cm diameter at breast height have died because of infection between 2012 and 2019. *N. densiflorus* is the most susceptible species in California and Oregon (Frankel and Palmieri, 2014) and entire local *N. densiflorus* populations have declined significantly (Cobb et al. 2012; Cobb et al., 2020) and their decline is changing the nature of those forests and can lead to increased fire risks. For instance, when infections first occur, dead branches and trees increase the fuel loads on the forest

floor and their absence from the canopy allows more sunlight to reach the forest floor, which becomes drier. This relationship is demonstrated for redwoods in coastal California forests, whose mortality increased when the pathogen and fire were present even though redwoods are generally resistant to the pathogen and fire individually (Metz et al., 2013). After the initial infections when *P. ramorum* kills only the aboveground tissues, disease-killed trees may resprout prolifically resulting in dense stands dominated by small stems that remain at risk of infection and increase fire risk (Quiroga et al., 2023). Finally, wildlife that depends on *N. densiflorus* and oaks is likely to be locally affected; impacts have been documented for some smaller vertebrates and birds (Monahan and Koenig, 2006; Swei et al., 2011).

Other reported hosts in California and Oregon, include redwood, Douglas fir, grand fir (*Abies grandis*), red fir (*Abies magnifica*), and western hemlock (*Tsuga heterophylla*). Symptoms of disease and impacts on these hosts are limited. For instance, observations in infested forests show most infection in redwoods are foliar or twig blight that are non-lethal (Davidson et al., 2008)[19]. Newer reports from Meentemeyer et al. (2015) and Garbelotto et al. (2020), supported by citizen science efforts, have confirmed impacts of ramorum blight on six species of manzanita (*Archtostaphylos*) with branch cankers and canopy mortality. This is an important discovery, because California is the center of diversity for the genus, and 59 out of the 105 species that inhabit the state are rare or endangered species (Kauffman et al. 2015; Schmid, 2002).

In 2009, *P. ramorum* was confirmed to be the cause of extensive dieback and mortality in plantations of Japanese larch (*Larix kaempferi*) in southwest England and Wales (Webber et al., 2010). This marked the first widespread, lethal damage caused by *P. ramorum* to a commercially important conifer species. More recently, the EU1 lineage was found in Oregon infecting grand fir and Douglas fir (LeBoldus et al., 2018). The range of individual clonal lineages has also expanded. NA2 was reported for the first time causing disease in *N. densiflorus* in Oregon (Peterson et al., 2022a) and EU1 has also expanded in California wildlands with a recent detection in *N. densiflorus* in Del Norte County (Garbelotto et al., 2021). These discoveries are concerning because 1) the EU1 genotype is characterized as a prolific sporulator in the natural environment (Harris, 2014) and experiments show it has the potential to be more aggressive (Søndreli et al., 2021) and 2) it introduces the possibility for genetic recombination with NA1 or NA2 lineages, which occurred in a Canadian nursery (Hamelin et al., 2022).

Estimating consequences of introduction outside Oregon and California is difficult because the susceptibility of eastern species remains unclear. The jump of *P. ramorum* to larch in Europe and the recent co-occurrence of EU1 and NA lineages in the environment in the United States introduces uncertainty. First, it is possible that the EU1 lineage could affect U.S. conifers, in which case the consequences of introduction would worsen. Second, sexual recombination between the NA lineages and EU1 lineage could occur, as has been observed in a nursery in British Columbia, Canada (Hamelin et al., 2022). Though pathogenicity tests of the NA1and EU1 progeny (oospores) had variable outcomes (Xavier et al., 2010), sexual recombination could increase *P. ramorum*'s adaptability and potentially alter the consequences of introduction.

[19] Chastagner et al. (2013) tested containerized saplings against *P. ramorum* and found Douglas fir, Sitka spruce (*Picea sitchensis*), and western hemlock develop lesions, but only larch samplings were reported to be killed. The authors tested four species of larch, western larch (*Larix occidentalis*), Japanese larch (*Larix kaempferi*), eastern larch (*Larix laricina*), and European larch (*Larix decidua*), but report mortality for all larch species together.

6. Conclusions

Outside of California and Oregon, there appears to be three interrelated factors that cause an asynchrony between the infective stage of *P. ramorum* and the susceptible stage of the host complex that make the occurrence of disease caused by *P. ramorum* unlikely. First, environmental stress decreases inoculum survival of *P. ramorum.* Second, inoculum does not build up in sufficient amounts to produce significant disease. Third, the infectious stage does not overlap with the susceptible stage of the host complex for enough time, reducing the likelihood of infection and disease development. While *P. ramorum* may survive in the environment, the necessary conditions for the development of ramorum blight, ramorum shoot dieback, and sudden oak death are not occurring, and consequently hosts are not symptomatic—at least not on a noticeable scale. In forests, disease and impacts have only been reported for California and Oregon (Figure 8). Therefore, unless conditions change, we conclude that *P. ramorum* does not pose a high risk to the rest of the United States.

One important caveat is that repeated incursions of the pathogen outside of California and Oregon could potentially increase inoculum pressure in the system. Under the right environmental conditions, the inoculum could surpass the threshold for infection of susceptible hosts, resulting in infections, disease, and impacts. Twenty years of experience with this pathogen and numerous movements of infected plant material outside of California and Oregon, however, leads us to conclude that this is unlikely.

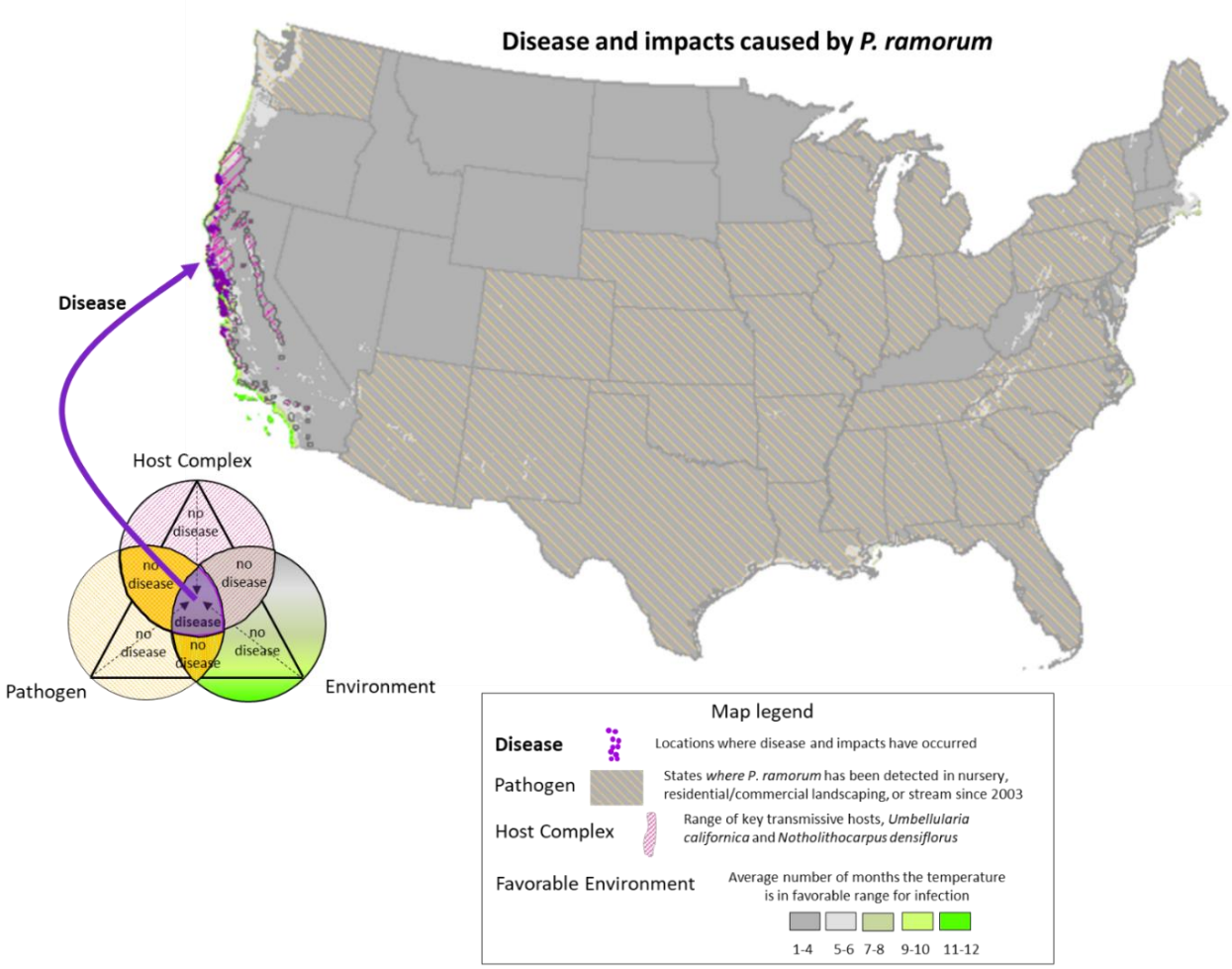


Figure 8. Locations where disease and impacts have been caused by *P. ramorum* in coastal California and Oregon forests. In other states, asynchrony or other factors have thus far resulted in no observable disease or impacts on forest vegetation.

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Appendix A. Exploratory Maxent model of the *Phytophthora ramorum* North American and European Clonal Lineages

We developed a Maxent ecological niche model (Phillips et al., 2017; Phillips et al., 2006) for the North American (NA1 and NA2) and European (EU1) clonal lineages of *P. ramorum* to understand what climatic variables explain where *P. ramorum* occurs but not map or predict where *P. ramorum* is likely to occur. The Maxent model integrates occurrence data with environmental variables and it can be used without species abundance or absence data (Evangelista et al., 2008; Kumar et al., 2009) to determine the variables correlated with where species occur.

Maxent modeling approach

Maxent is a self-contained Java application for modeling species distributions from occurrence records and environmental data (Phillips et al., 2017; Phillips et al., 2006). It extracts a sample of background locations from the landscape that it contrasts against the presence locations. It transforms environmental variables into a set of feature types and uses a regularization multiplier (RM) to reduce the number of parameters to control model complexity (Elith et al., 2011; Phillips and Dudik, 2008). The default RM value is 1; a smaller value of RM (≤1) may potentially overfit the model and produce more restricted distributions, whereas a higher value (>1) results in simpler models with less discriminating power and broader potential species distributions (Kumar et al., 2016).

We obtained 7,478 occurrence records for *P. ramorum* NA1 and NA2 (hereafter referred to as the NA lineage) for California and Oregon from the California Oak Mortality Task Force and Sarah Navarro (COMTF, 2019; Navarro, 2019). NA1 and NA2 data were collected between 2001 and 2018, though approximately 1/3 of the records did not have a year associated with them. We also obtained 245 *P. ramorum* EU1 records from Dr. Simon Shamoun and colleagues (Shamoun et al., 2018), published records (e.g., O'Hanlon et al., 2018), and Sarah Navarro (Navarro, 2019) (Figure A1). There was no date associated with the EU1 records. Both datasets, to the best of our knowledge, were collected from observations in forests or trees in natural settings with the exception of three EU1 detections that were recorded in “nurseries and cities” in Croatia where *P. ramorum* was first noticed in 2007 (EPPO, 2023). Though some of these data were collected from areas under eradication, they represent infections and spread of *P. ramorum.*

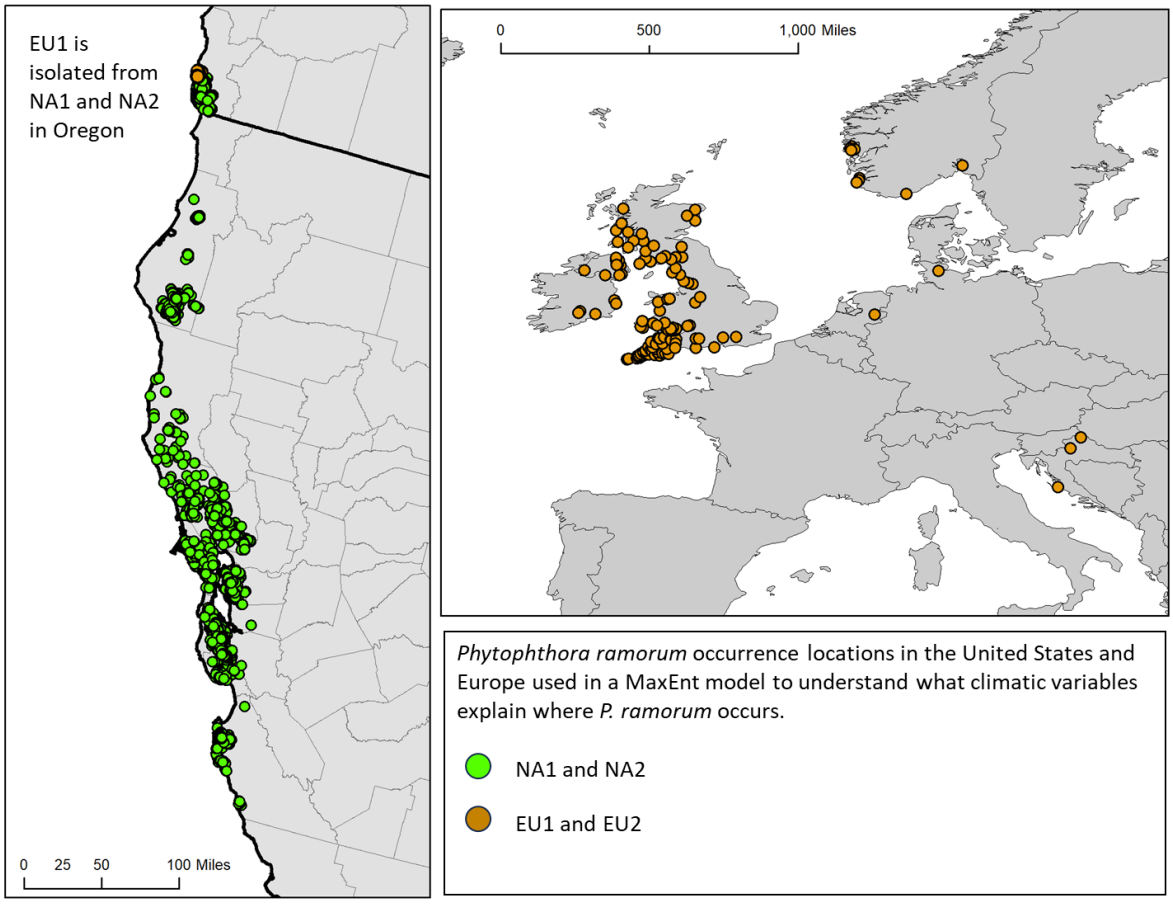

Figure 1A. Occurrences of *P. ramorum* in the United States and Europe used in the Maxent model to understand what climatic variables explain where *P. ramorum* occurs.

Climatic suitability for *P. ramorum* has been mapped using different ecological niche models that predicted varied *P. ramorum* potential distributions in the United States (e.g., Kluza et al., 2007; Václavík et al., 2010; Václavík and Meentemeyer, 2012; Ireland et al., 2013; Shamoun et al., 2018). Our approach duplicates the approach used by Vaclavik et al. (2012) and Shamoun et al. (2018) but substitutes current *P. ramorum* occurrence data and recent advances in Maxent modeling (Merow et al., 2013; Phillips et al., 2017; Warren et al., 2014), with the following features:

1. Spatial filtering (i.e., reducing density of occurrences) to reduce spatial autocorrelation effects and using a bias layer to minimize sampling bias towards areas that have greater density of occurrence points.
2. Not extrapolating the model beyond the predictive variables' values in the areas where the species currently occurs.
3. Selecting model with optimal complexity and biologically plausible shape of fitted response curves, and
4. Including biologically relevant predictor variables such as spring (March-May) relative humidity.

We removed duplicate records (>1 presence point within a grid cell) and reduced spatial autocorrelation using spatial filtering" (i.e., reducing density of occurrences) using SDMToolbox (Brown, 2014; Kumar et al., 2016). This reduced occurrences to 757 for the NA1 lineage and 127 for the EU1 lineage. We then calculated a Gaussian kernel density layer of occurrence data of both lineages in ArcMap using SDMToolbox, which we used to account for potential sampling bias in occurrence data.

Results

We fitted multiple models with different RM values and feature types and then selected the best model with the optimal level of complexity for each *P. ramorum* clonal lineage. We evaluated model performance using the area under the receiver operating characteristic (ROC) curve (AUC[20]; Phillips et al., 2006). We used the ten-fold cross-validation procedure and reported averaged test AUC values across the 10 replicates.

Then, we extracted 25 climatic variables (Table A1) to correlate with the species locations as follows:

- Nineteen bioclimatic variables of average monthly temperature, precipitation, seasonal variables, and climatic extreme indices data from 1979-2013 (Hijmans et al., 2005; Karger et al., 2017).
- Average annual relative humidity and spring relative humidity (March-May) based on PRISM climate data from 1999-2018, using SAFARIS (SAFARIS, 2020).
- Vapor pressure deficit (minimum, mean, and maximum) and dew point temperature data from PRISM (30-year normals, 1981-2010; PRISM, 2019).

All 25 variables were examined for cross-correlation (Pearson correlation coefficient, r), and highly correlated variables ($|r| > 0.80$) were excluded to reduce multicollinearity. The decision to exclude or include a variable was based on its biological relevance to *P. ramorum* and its relative predictive power in the model.

Maxent models for the *P. ramorum* NA1 and EU1 lineages performed very well with the test AUC values of 0.975 (±0.002) for the NA lineage model and 0.963 (±0.021) for the EU1 lineage model. Both models included eight predictor variables (Table A1) and correctly predicted all *P. ramorum* occurrences. Mean diurnal range in temperature (Bio2) was the most important variable for both *P. ramorum* clonal lineages, contributing 81 percent to the NA1 model and 62 percent to the EU1 model (Table A1). Fitted response curves for the best predictors for both lineages were biologically plausible.

[20] AUC values vary from 0 to 1. A value of 0.5 indicates the model performance is equivalent to random. A value less than 0.5 indicates performance worse than random, from 0.5–0.7 indicates poor performance, from 0.7–0.9 indicates reasonable/moderate performance, and greater than 0.9 indicates strong performance (Peterson et al., 2011).

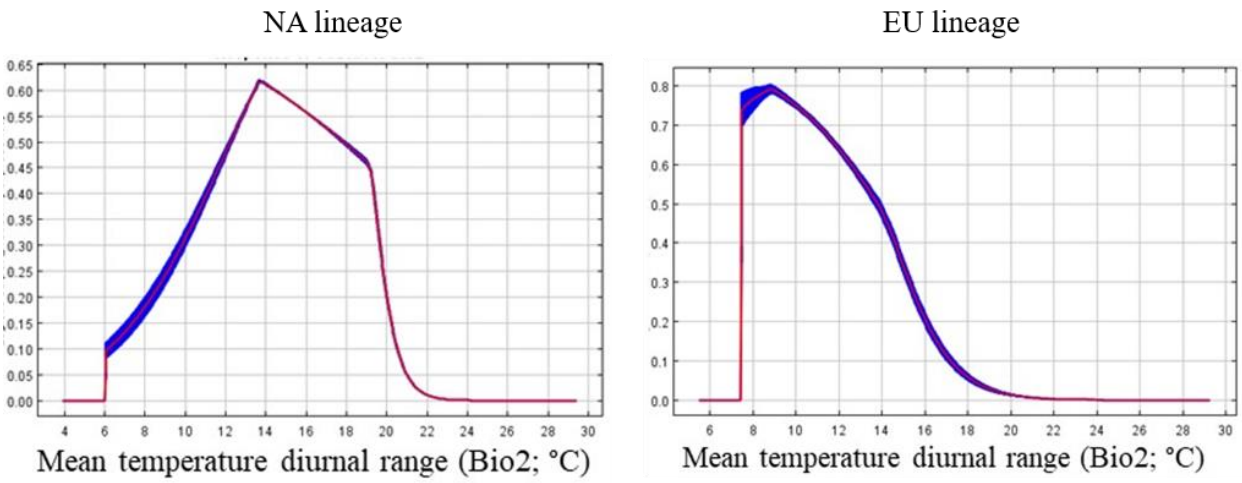

Figure A2. Response curves for the mean diurnal temperature range for the NA and EU1 lineage models in Maxent.

Table A1. Relative importance of 25 climatic variables considered in the *P. ramorum* NA and EU1 models. Bold shows the variable was used in the Maxent model; other variables were dropped because of high cross-correlations or lower predictive power in the model. Values are averages for 10 replicate runs.

Variable	*P. ramorum* NA (percent contribution)	*P. ramorum* EU1 (percent contribution)
Mean diurnal range in temperature (Bio2; °C)	**81.3**	**61.6**
Minimum temperature of coldest month (Bio6; °C)	**4.6**	**1.0**
Precipitation of coldest quarter (Bio19; mm)	**4.1**	**1.2**
Precipitation of driest month (Bio14; mm)	**3.8**	**22.1**
Precipitation of warmest quarter (Bio18; mm)	**3.2**	-
Average relative humidity (spring) (PRISM)	**1.8**	-
Isothermality (Bio3)	**0.7**	-
Mean temperature of wettest quarter (Bio8; °C)	**0.6**	-
Temperature seasonality (SD x 100) (Bio4)	-	**7.6**
Precipitation seasonality (CV) (Bio15)	-	**3.4**
Mean temperature of driest quarter (Bio9; °C)	-	**1.7**
Maximum temperature of warmest month (Bio5; °C)	-	**1.2**
Annual mean temperature (Bio1; °C)	-	-
Temperature annual range (Bio7; °C)	-	-
Mean temperature of warmest quarter (Bio10; °C)	-	-
Mean temperature of coldest quarter (Bio11; °C)	-	-
Mean annual precipitation (Bio12; mm)	-	-
Precipitation of wettest month (Bio13; mm)	-	-
Precipitation of wettest quarter (Bio16; mm)	-	-
Precipitation of driest quarter (Bio17; mm)	-	-
Average relative humidity (annual) (PRISM)	-	-
Maximum vapor pressure deficit (VPD) (PRISM)	-	-
Minimum vapor pressure deficit (VPD) (PRISM)	-	-
Mean vapor pressure deficit (VPD) (PRISM)	-	-
Mean dew point temperature (PRISM)	-	-

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Caveats and uncertainties

We expected some sampling bias in occurrence data because some of the data were not collected based on pre-designed surveys, for example data from United Kingdom. We corrected this bias by including a bias surface in the Maxent model (similar to Brown, 2014; Kumar et al., 2016). In addition, decisions made during model calibration may affect ecological niche model results. For example, these models can be affected by selection of predictor variables, multicollinearity among predictor variables, spatial accuracy of species occurrences, or spatial autocorrelation in occurrence data (e.g., Jarnevich et al., 2015). We performed additional analyses such as spatial filtering and ten-fold cross-validation to mitigate any potential issues.

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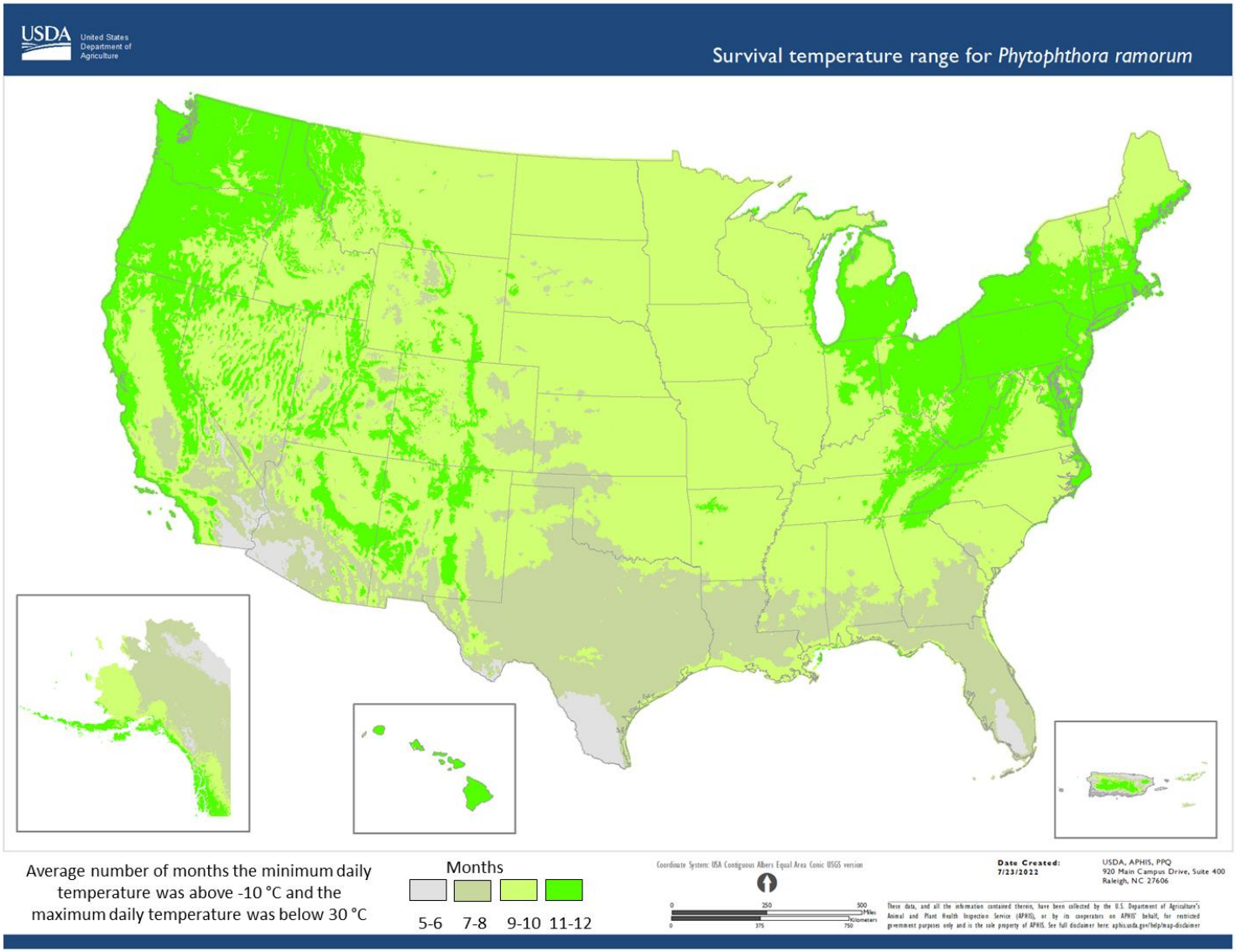
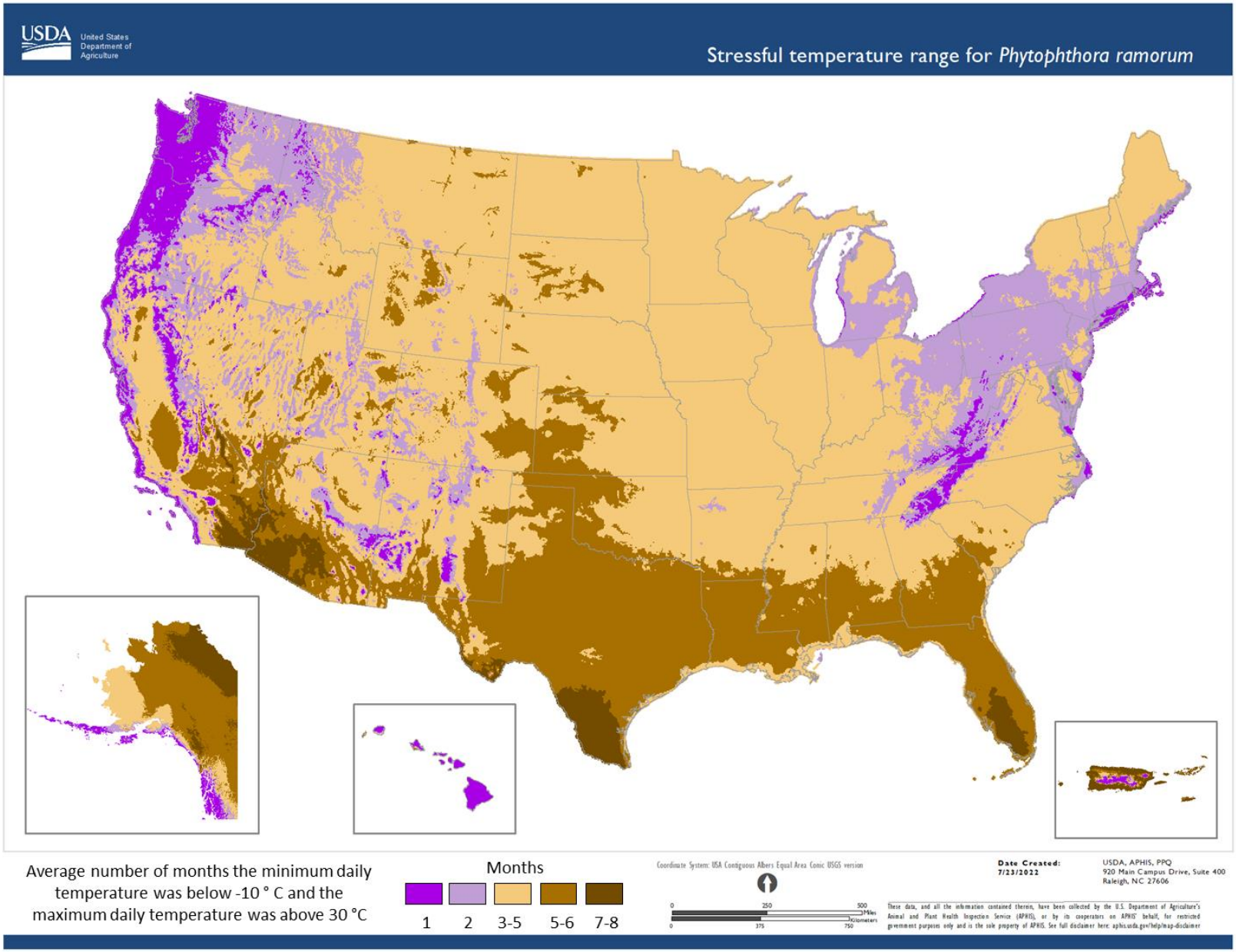
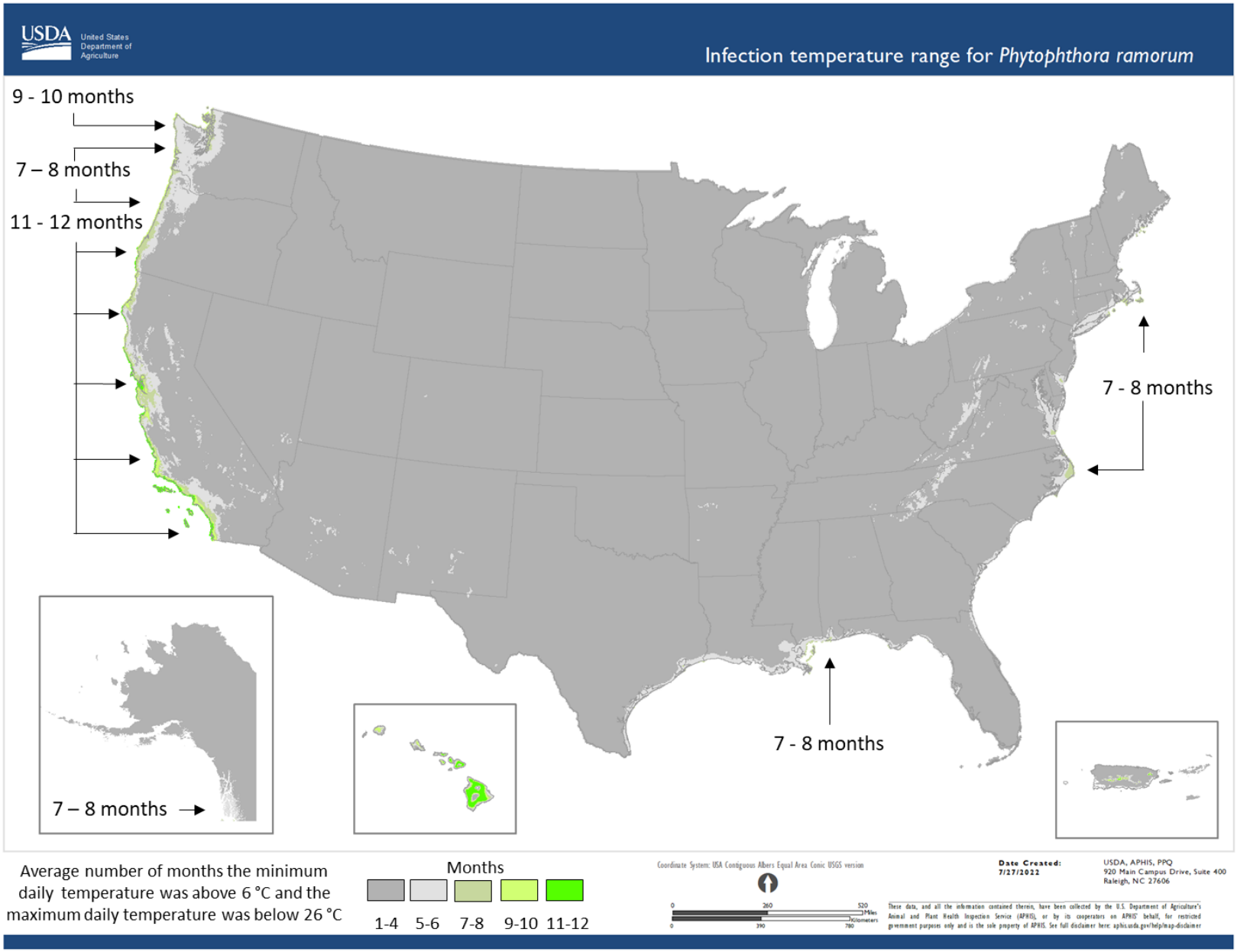
Appendix B. Survival, stress, and infection maps of *P. ramorum*.




USDA United States Department of Agriculture
Stressful temperature range for *Phytophthora ramorum*
Average number of months the minimum daily temperature was below -10 ° C and the maximum daily temperature was above 30 °C
Months
1 2 3-5 5-6 7-8
Coordinate System: USA Contiguous Albers Equal Area Conic USGS version
Date Created: 7/23/2022
USDA, APHIS, PPQ
920 Main Campus Drive, Suite 400
Raleigh, NC 27606
These data, and all the information contained therein, have been collected by the U.S. Department of Agriculture's Animal and Plant Health Inspection Service (APHIS), or by its cooperators on APHIS' behalf, for restricted government purposes only and is the sole property of APHIS. See full disclaimer here: aphis.usda.gov/help/map-disclaimer


USDA United States Department of Agriculture
Infection temperature range for *Phytophthora ramorum*
9 - 10 months
7 – 8 months
11 - 12 months
7 - 8 months
7 - 8 months
7 – 8 months
Average number of months the minimum daily temperature was above 6 °C and the maximum daily temperature was below 26 °C
Months
1-4 5-6 7-8 9-10 11-12
Coordinate System: USA Contiguous Albers Equal Area Conic USGS version
Date Created: 7/27/2022
USDA, APHIS, PPQ
920 Main Campus Drive, Suite 400
Raleigh, NC 27606
0 260 520 Miles
0 390 780 Kilometers
These data, and all the information contained therein, have been collected by the U.S. Department of Agriculture's Animal and Plant Health Inspection Service (APHIS), or by its cooperators on APHIS' behalf, for restricted government purposes only and is the sole property of APHIS. See full disclaimer here: aphis.usda.gov/help/map-disclaimer

Appendix C. *Phytophthora ramorum* host susceptibility and host competency studies.

Table 1. *Phytophthora ramorum* host susceptibility studies have mostly focused on identifying potential hosts or determining the environmental ranges conducive for infection. Since the methodologies used for inoculation and inoculum concentrations are different, the results are not comparable among studies. The hosts studied in each experiment are listed below the table. Host competency is the ability of a host to transmit the infection to another susceptible host or to a vector (Gervasi et al., 2015).

Susceptibility									
Experiment setting	**Incubation temperature**	**Light / dark cycle**	**Experiment duration (days)**	**Clonal lineage**	**Fungal structure and mode of inoculation**	**Inoculum concentration**	**Tissue inoculated**	**Was the host tissue surface disinfested?**	**Reference**
Incubator	15 °C	Not reported	21	EU2	Sporangia suspension	2 - 5 x 10^5 sporangia ml^{-1}	Pre-wounded (5 mm) tree shoots (1 cm basal diameter)	Not reported	Dun et al., 2022
Growth chamber	20 °C (day), 18 °C (night)	Yes, 18-hour phyto-period	7	NA1, EU1	Mycelia agar plugs	N/A	Seedlings	Not reported	Søndreli et al., 2019
Incubator	Not reported	Not reported	10	EU2	Mycelia agar plugs	Not reported	Pre-wounded detached leaves/needles	No	Shamoun et al., 2017
Moist boxes	20 °C	Not reported	7	Not reported	Droplets of zoospore suspension placed between midvein and leaf margin	4 - 5 x 10^5 spores ml^{-1}	Pre-wounded detached leaves	No	Johnston et al., 2015

Susceptibility									
Experiment setting	**Incubation temperature**	**Light / dark cycle**	**Experiment duration (days)**	**Clonal lineage**	**Fungal structure and mode of inoculation**	**Inoculum concentration**	**Tissue inoculated**	**Was the host tissue surface disinfested?**	**Reference**
Greenhouse	20 °C	Not reported	60	NA1, EU1	Local application of sporangia suspension with a paintbrush	5×10^2, 1×10^3 and 3×10^3 sporangia ml^{-1}	Wounded and unwounded seedlings	Not reported	Tooley et al., 2014
Growth chamber	19 - 20 °C	24-hour light	8 - 55	NA1, NA2, EU1	Zoospore suspension	1×10^5 zoospores ml^{-1}	Seedlings	Not reported	Chastagner et al., 2013
Humid chambers	15, 19, 23 or 28 °C	Ambient light	14	NA1	Zoospore suspension	1×10^2, 1×10^3, 1×10^4, and 2.7×10^4 zoospores ml^{-1}	Detached leaves and leaves attached to a branch in water	Yes	Hüberli et al., 2012
Moist boxes	20 °C	Dark	14	NA2, EU1	Sporangia suspension or mycelia agar plugs	930 sporangia ml^{-1}, 1050 sporangia ml^{-1}, or mycelium plugs	Pre-wounded detached leaves	No	Linderman et al., 2007
Moist chambers	20 °C	Yes, 8-hour day	6	EU1, NA2	Zoospore suspension	$2 - 4 \times 10^5$ zoospores ml^{-1}	Unwounded and pre-wounded shoots with needles or	No	Denman et al., 2005

Susceptibility									
Experiment setting	**Incubation temperature**	**Light / dark cycle**	**Experiment duration (days)**	**Clonal lineage**	**Fungal structure and mode of inoculation**	**Inoculum concentration**	**Tissue inoculated**	**Was the host tissue surface disinfested?**	**Reference**
							detached leaves		
Moist boxes or bags	17 - 20 °C	Yes, 12-hour day	7 - 35	Not reported, but likely NA2 based on paper year and collection location.	Leaf dip in zoospore suspension and immersion of terminal portion of seedlings in zoospore suspension	2.4×10^3, 1.2×10^4, or 6.0×10^4 and 8×10^4 zoospores ml^{-1}	Detached leaves, stem-wounded seedlings and logs	No	Hansen et al., 2005
Dew chamber	20 ºC	No, in darkness	7	Not reported, but likely EU1, NA2 based on paper year and collection location.	Plant limb dip in sporangia suspension	5×10^3 sporangia ml^{-1}	Unwounded plants	No	Tooley et al., 2004

Hosts tested in each study:

1) Dun et al., 2022. *Larix kaempferi.*

2) Søndreli et al., 2020. *Notholithocarpus densiflorus.*

3) Shamoun et al., 2017. *Abies balsamea, Abies grandis, Acer macrophyllum, Acer saccharum, Alnus rubra, Arbutus menziesii, Arctostaphylos* spp.*, Betula alleghaniensis, Betula papyrifera, Camellia japonica, Cornus nuttallii, Fraxinus americana, Gaultheria procumbens, Gaultheria shallon, Larix occidentalis, Mahonia nervosa, Picea glauca, Picea sitchensis, Pinus contorta, Populus trichocarpa, Pseudotsuga menziesii, Quercus garryana, Quercus rubra, Rhododendron caucasicum x ponticum, Rhus typhina, Ribes* spp.*, Rubus discolor, Rubus idaeus, Thuja plicata, Tsuga heterophylla, Umbellularia californica, Vaccinium corymbosum, Vitis vinifera.*

4) Johnston et al., 2015. *Umbellularia californica*

5) Tooley et al., 2014. *Quercus rubra, Q. prinus, Acer rubrum.*

6) Chastagner et al., 2013. *Larix decidua, L. kaempferi, L. laricina, L. occidentalis, Picea sitchensis, Pseudotsuga menziesii, Tsuga canadensis, Tsuga heterophylla.*

7) Hüberli et al., 2012. *Umbellularia californica.*

8) Linderman et al., 2007. *Acer rubrum, Alnus serrulata, Amorpha fruticosa, Asimina triloba, Betula nigra, Calycanthus fertilis, Carpinus caroliniana, Carya cordiformis, Carya lacinosa* 'Fayette,' *Carya ovata, Castanea pumila, Cephalanthus occidentalis, Chionanthus virginicus, Cornus amomum, C. foemina, C. florida, Corylus americana, Euonymus americanus, E. atropurpureus, Fraxinus americana, F. pennsylvanica, Halesia carolina, Juglans nigra, Ligustrum vulgare, Lindera benzoin, Liquidambar styraciflua, Liriodendron tulipifera, Nyssa sylvatica, Ostrya virginiana, Oxydendrum arboreum, Prunus serotina, Rhamnus caroliniana, Rhododendron cumberlandense, R. maximum, Rhus copallinum, R. glabra, R. typhina, Robinia hispida, Rosa setigera, R. palustris, Rubus occidentalis, Sambucus canadensis, Staphylea trifolia, Symphoricarpos orbiculatus, Tilia americana.*

9) Denman et al., 2005. *Abies procera, Acer pseudoplatanus, Aesculus hippocastanum, Alnus glutinosa, Betula pendula, Carpinus betulus, Castanea sativa, Chamaecyparis lawsoniana, Corylus avellana, Eucalyptus gunnii, Fagus sylvatica, Fraxinus excelsior, Ilex aquifolium, Picea abies, Picea sitchensis, Pinus contorta, Pinus nigra* var. *maritima, Pinus sylvestris, Populus tremula, Prunus avium, Pseudotsuga menziesii, Quercus cerris, Quercus ilex, Quercus petraea, Quercus robur, Quercus rubra, Quercus suber, Rhododendron catawbiense, Sequoia sempervirens, Taxus bacata, Tilia cordata, Tsuga heterophylla, Ulmus procera, Umbellularia californica.*

10) Hansen et al., 2005b. *Abies concolor, Abies grandis, Abies magnifica, Abies procera, Acer circinatum, Acer macrophyllum, Alnus rhombifolia, Alnus rubra, Arbutus menziesii, Arctostaphylos uva-ursi, Castanea dentata, Castanopsis chrysophylla, Chamaecyparis lawsoniana, Corylus cornuta, Cornus nuttallii, Fraxinus latifolia, Larix occidentalis, Libocedrus decurrens, Lithocarpus densiflorus, Picea sitchensis, Pinus contorta* var. *contorta, Pinus lambertiana, Pinus monticola, Pinus ponderosa, Populus tremuloides, Populus trichocarpa, Populus trichocarpa × P. deltoides, Prunus emarginata, Pseudotsuga menziesii, Quercus chrysolepis, Quercus garryana, Quercus*

kelloggii, Quercus rubra, Quercus palustris, Rhamnus purshiana, Rhododendron macrophyllum, Rhus diversiloba, Rubus spectabilis, Salix hookeriana, Salix lasiandra, Sequoia sempervirens, Sequoia gigantea, Thuja plicata, Tsuga heterophylla, Taxus brevifolia, Umbellularia californica, Vaccinium membranaceum, Vaccinium ovatum, Vaccinium parvifolium.

11) Tooley et al., 2004. *Arctostaphylos uva-ursi, Gaultheria procumbens, Gaylussacia baccata, Gaylussacia frondosa, Kalmia angustifolia, K. latifolia* 'Madeline,' *K. latifolia* 'Minuet,' *K. latifolia* 'Olympic Wedding,' *Leucothoe axillaris* 'Greensprite,' *L. fontanesiana, Pieris floribunda, P. japonica, Rhododendron* 'Aglo,' *R. arborescens, R. calendulaceum, R. carolinianum, R. catawbiense, Rhododendron* 'Chinoides,' *Rhododendron* 'Cunningham's White' (*R. caucasicum* × *ponticum* var. *album*), *R. dauricum* PJM type, *Rhododendron* 'Delaware Valley White' (*R. mucronatum* hybrid), *Rhododendron* 'Exbury hybrid,' *Rhododendron* 'Girard's Fuchsia,' *Rhododendron* 'Girard's Rose,' *Rhododendron* 'Glacier,' *Rhododendron* 'Gloria,' *Rhododendron* 'Hino Crimson,' *R. indicum* 'Macrantha,' *Rhododendron* 'Inga,' *R. macrosepalum, Rhododendron* 'Marilee,' *R. maximum, R. micranthum, R. minus, Rhododendron* 'Nova Zembla,' *Rhododendron* 'PJM,' *Rhododendron* 'Purple Gem,' *Rhododendron* 'Purple Splendor,' *Rhododendron* 'Roseum Elegans,' *R. vaseyi, R. viscosum, R. yakushimanum* 'Ken Janeck,' *R. yedoense* var. *poukhanense, Umbellularia californica, Vaccinium angustifolium, V. corymbosum* 'Bluecrop,' *V. corymbosum* 'Duke,' *V. corymbosum* 'Weymouth,' *V. macrocarpon* 'Crowley,' *V. macrocarpon* 'Stevens,' *Zenobia pulverulenta*.

12) Søndreli et al., 2019. *Notholithocarpus densiflorus.*

13) LeBoldus and Søndreli, 2020. *Notholithocarpus densiflorus, Pseudotsuga menziesii, Tsuga heterophylla, Larix occidentalis, Picea sitchensis.*

14) Tooley and Browning, 2009. *Acer rubrum, Amelanchier canadensis, Asimina triloba, Cercis canadensis, Cornus amomum, C. florida, C. racemosa, C. stolonifera, Ilex verticillata, Kalmia latifolia* 'Hoffman's K,' *K. latifolia* 'Hoffman's Pink,' *Lindera benzoin, Lonicera japonica, L. sempervirens, Myrica pennsylvannica, Prunus serotina, Rhododendron* 'Cunningham's White,' *Rhus typhina, Robinia pseudoacacia, Rosa multiflora, Rubus allegheniensis, Sambucus canadensis, Sassafras albidium, Smilax rotundifolia, Syringa vulgaris, Viburnum dentatum.*

Table 2. Few studies have characterized *Phytophthora ramorum* host susceptibility and competency. Since the methodologies used for inoculation and inoculum concentrations are different, the results are not comparable among studies. The hosts studied are listed below the table; sporulation values are not consistently reported and not included in this table. Host competency is the ability of a host to transmit the infection to another susceptible host or to a vector (Gervasi et al., 2015).

					Competency and Susceptibility				
Experiment setting	**Incubation temperature**	**Light / dark cycle**	**Experiment duration (days)**	**Clonal lineage**	**Fungal structure and mode of inoculation**	**Inoculum concentration**	**Tissue inoculated**	**Was the host tissue surface disinfested?**	**Reference**
Field and greenhouse	Not reported	Not reported	Not reported	NA1, EU1	Not reported	Not reported	Not reported	Not reported	Søndreli et al., 2020
Field	Not reported	Not reported	Not reported	NA1, EU1	Not reported	Not reported	Not reported	Not reported	Søndreli et al., 2021
Dew chamber	20 °C	No, in darkness	5	Not reported	Sporangia suspension	4×10^3 sporangia ml^{-1}	Seedlings	No	Tooley and Browning, 2009

Hosts tested and sporulation values reported in each study:

1) Søndreli et al., 2019. *Notholithocarpus densiflorus.* Sporulation values were reported qualitatively. The authors found a ten-fold increase in sporulation in the EU1 isolates compared to NA1 isolates on tanoak.

2) Søndreli, K. L., A. Kanaskie, S. M. Navarro, P. Reeser, and J. M. LeBoldus. 2021. Characterizing the variation in aggressiveness and sporulation of the NA1 and EU1 lineages of *Phytophthora ramorum* in Oregon. Plant Pathology 70(6):1342-1353.

3) Tooley and Browning, 2009. *Acer rubrum, Amelanchier canadensis, Asimina triloba, Cercis canadensis, Cornus amomum, C. florida, C. racemosa, C. stolonifera, Ilex verticillata, Kalmia latifolia* 'Hoffman's K,' *K. latifolia* 'Hoffman's Pink,' *Lindera benzoin, Lonicera japonica, L. sempervirens, Myrica pennsylvannica, Prunus serotina, Rhododendron* 'Cunningham's White,' *Rhus typhina, Robinia pseudoacacia, Rosa multiflora, Rubus allegheniensis, Sambucus canadensis, Sassafras albidium, Smilax rotundifolia, Syringa vulgaris, Viburnum dentatum.* Sporulation potential for each host was reported as sporangia per cm^2 lesion area and sporangia per leaf/leaflet.

References to Appendix C

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