Background

The effective management of any plant disease is based on an understanding of the disease cycle, including the pathogen’s modes of survival and dissemination, and the role of environmental factors in disease development. The disease cycle, or disease process, consists of several stages, including: production and dispersal of inoculum, establishment of infection (entry into the host), colonization (invasion), symptom development (interaction of the pathogen with the host), and survival. Those stages—except for symptoms, which were discussed in Chapter 2—are discussed below for Phytophthora ramorum Werres, de Cock, and Man in’t Veld.

A diagrammatic disease cycle for P. ramorum (Courtesy N. Ochiai; reproduced with permission from Parke and Lucas 2008) is presented below (Figure 1).

Figure 1. Proposed disease cycle for Phytophthora ramorum in forests (Parke and Lucas 2008). Image: N. Ochiai, Oregon State University.
Although at first glance the *P. ramorum* disease cycle appears complex because of the different habitats and range of hosts involved, it follows the basic steps of any plant disease cycle. In general, *Phytophthora* species that infect aerial parts of plants spread through production of asexual sporangia and zoospores. Survival structures such as chlamydospores, or sometimes sexually-produced oospores, may also have a role in dissemination and spread in those species that produce one or both of these structures. Spores that are disseminated can then initiate infections of host tissue. The new infections can serve as another source of spores to begin the cycle again. Appropriate environmental conditions (temperature and relative humidity), as well as a means of survival of the pathogen (either as spores or mycelia), are necessary for each step of the cycle. Transport of infected host tissue, and/or contaminated growing media or soil, containing the pathogen can introduce the disease to new areas where the pathogen may establish if it can complete the cycle described above (Davidson and Shaw 2003).

### Inoculum Production

This is the stage of the disease cycle where the pathogen produces a structure or structures to initiate infection.

**Types of inoculum:** Like some other *Phytophthora* spp., *P. ramorum* produces asexual sporangia, zoospores and chlamydospores (Figure 2) in culture and in nature (Rizzo and others 2002, Rizzo and others 2005, Parke and others 2002, Werres and others 2001). Sexual structures—oo-gonia, antheridia and oospores—have not yet been found in nature. The production of sexual oospores has only been observed in the laboratory (Werres and Zielke 2003). The pathogen is heterothallic and pairing of opposite mating types is needed for sexual reproduction. In artificial pairings, oospores have been observed in plants and on agar medium (Werres and Zielke 2003, Brasier and Kirk 2004). A recent study (Boutet and Chandelier, unpublished; http://nature.berkeley.edu/IU-FRO2007/phytophthora/abstracts/posters.pdf) suggests that *P. ramorum* oospores produced in culture were the result of selfing rather than hybridization between two mating types. Studies (for example: Ivors and others 2006, Prospero and others 2007) have found no evidence for sexual reproduction in the field.

Moralejo and Descals (2006) and Moralejo and others (2006b) observed that additional multi-hyphal reproductive structures, similar to sporodochia of mitosporic fungi, were produced by *P. ramorum* on fruits and adaxial leaf surfaces following inoculation (isolates or isolate source not
stated) of some woody Mediterranean shrubs (strawberry tree [*Arbutus unedo*], carob bean [*Ceratonia siliqua*], bay laurel [*Laurus nobilis*], mastic [*Pistacia lentiscus*], Italian buckthorn [*Rhamnus alaternus*] and *Viburnum tinus*). Occasionally sporangia and chlamydosori (packed clusters of chlamydospores) were formed on the stromata after emergence through the adaxial leaf surface. Although these structures have not been observed in nature, Moralejo and others (2006b) suggest that the subepidermal position of the stroma initials may protect the pathogen from a dry atmosphere or solar radiation, or that they serve as over-summering survival structures.

**Host impacts on sporulation:** Sporulation can be affected by a variety of biotic and abiotic factors, including host and factors of the environment such as rainfall, temperature, light and humidity.

*Host and production of sporangia and chlamydospores:* The production of sporangia and/or chlamydospores varies with the foliar host; some hosts support rapid and prolific reproduction, whereas other hosts do not (Table 1). Davidson and others (2002b, 2005b) found abundant sporangial formation on moistened leaves of infected California bay laurel (*Umbellularia californica*) and *Rhododendron* spp. within 72 hours in the laboratory. Chlamydospores were also observed on the surface of moistened bay leaves. Under natural conditions, chlamydospores are produced within host tissues and do not appear to be adapted for dispersal.

Parke and others (2002) reported that the production of sporangia, zoospores, and chlamydospores of *P. ramorum* (source of isolates not mentioned) on inoculated leaf disks was greatest and most rapid on California bay laurel compared to other hosts (tanoak [*Lithocarpus densiflorus*], madrone [*Arbutus menziesii*], Pacific rhododendron [*Rhododendron macrophyllum*], evergreen huckleberry [*Vaccinium ovatum*]) and at the time non-hosts (vine maple [*Acer circinatum*], salal [*Gaultheria shallon*], red alder [*Alnus rubra*], Oregon white oak [*Quercus garryana*]) tested; abundant sporangia and active zoospores were released within 24 hours, and zoospores continued to be released for several days (Table 1). The authors found that tanoak also supported production of numerous sporangia and zoospores soon after infection, usually within 24 hours. On madrone leaf disks, only chlamydospores were produced. Sporangial production on other species tested varied from a few to many over the course of several days, with chlamydospore production following. Of the non-host species tested, inoculum production was most abundant on vine maple and salal (Parke and others 2002). These two species have since been reported as rare hosts in nature.

Detached leaf assay tests in the U.K. (Defra 2005c, Defra 2006), using two EU1 European isolates (BBA 16/99 and 1604) and two (NA1) American isolates (0-217 and 1004.1) of *P. ramorum* on ornamental and understory hosts, also found that sporulation potential varies with host species (Table 1). The two NA1 isolates were generally less vigorous in sporulation than the two
EU1 isolates. Chlamydospore production was generally less abundant than sporangial production (Defra 2005c).

In detached leaf assays to determine sporulation on the leaves of European tree species (Denman and others 2006), ash (Fraxinus excelsior) supported consistently high sporulation, while significantly fewer sporangia were observed on horse chestnut (A. hippocastanum) and sessile oak (Q. petraea) (Table 1). In tests on English oak (Q. robur), holm oak (Q. ilex), R. catawabiense and turkey oak (Q. cerris), Q. ilex and R. catawabiense all supported more sporangia than the other two oak species (Q. robur and Q. cerris). Differences among the EU1 and NA1 isolates were not mentioned.

The results of detached leaf assays using plants of Mediterranean evergreen oak forest and maquis-type vegetation (Moralejo and Hernández 2002) are presented in Table 1.

Table 1. Host leaves supporting sporulation of P. ramorum.

<table>
<thead>
<tr>
<th>Propagule Type</th>
<th>Host</th>
<th>Sporangia</th>
<th>Chlamydospores</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>California/Oregon hosts</td>
<td>California bay laurel (Umbellularia californica)</td>
<td>+</td>
<td>+</td>
<td>Parke and others 2002, Davidson and others 2002b, Davidson and others 2005b</td>
</tr>
<tr>
<td></td>
<td>tanoak (Lithocarpus densiflorus)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ornamental rhododendron (Rhododendron sp.)</td>
<td>+</td>
<td>-</td>
<td></td>
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<tr>
<td></td>
<td>pacific rhododendron (R. macrophyllum)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>evergreen huckleberry (Vaccinium ova-tum)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>vine maple (Acer circinatum)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>salal (Gaultheria shallon)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>red alder (Alnus rubra)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>Oregon white oak (Quercus garryanna)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>madrone (Arbutus menziesii)</td>
<td>-</td>
<td>+</td>
<td>Maloney and others 2005, Maloney and others 2007</td>
</tr>
<tr>
<td></td>
<td>coast redwood (Sequoia sempervirens)</td>
<td>+</td>
<td>+</td>
<td>Vettraino and others 2008</td>
</tr>
<tr>
<td>Coast live oak (Quercus agrifolia)</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propagule Type</td>
<td>Host</td>
<td>Sporangia</td>
<td>Chlamydomospores</td>
<td>Reference</td>
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<td>---------------</td>
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<td>-----------</td>
</tr>
<tr>
<td>Europe, ornamental and understory hosts</td>
<td>lilac (<em>Syringa vulgaris</em>)</td>
<td>+</td>
<td>+</td>
<td>Defra 2005c, Defra 2006</td>
</tr>
<tr>
<td></td>
<td>Pieris (<em>Pieris japonica</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>mountain laurel (<em>Kalmia latifolia</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Camellia (<em>Camellia japonica</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>California bay laurel (<em>Umbellularia californica</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ponticum rhododendron (<em>R. ponticum</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>dog rose (<em>Rosa canina</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>elder (<em>Sambucus nigra</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ash (<em>Fraxinus excelsior</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>honeysuckle (<em>Lonicera periclymenum</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sycamore maple (<em>Acer pseudoplatanus</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wych elm (<em>Acer pseudoplatanus</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>crabapple (<em>Malus sylvestris</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
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<tr>
<td></td>
<td>English yew (<em>Taxus baccata</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td></td>
<td>bramble blackberry (<em>Rubus fruticosus</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
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<tr>
<td></td>
<td>English ivy (<em>Hedera helix</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
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<tr>
<td></td>
<td>Guelder rose (<em>Viburnum opulus</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>lingonberry (<em>Vaccinium vitis-idaea</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>bearberry (<em>Arctostaphylos uva-ursi</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>European Tree Species</td>
<td>horse chestnut (<em>A. hippocastanum</em>)</td>
<td>+</td>
<td></td>
<td>Denman and others 2006</td>
</tr>
<tr>
<td></td>
<td>Sweet chestnut (<em>Castanea sativa</em>)</td>
<td>+</td>
<td></td>
<td>2006</td>
</tr>
<tr>
<td></td>
<td>ash (<em>F. excelsior</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>turkey oak (<em>Q. cerris</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Holm oak (<em>Q. ilex</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>sessile oak (<em>Q. petraea</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>English oak (<em>Q. robur</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>common lilac (<em>Syringa vulgaris</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>English elm (<em>Ulmus procera</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>large leaf rhododendron (<em>R. catawabiense</em>)</td>
<td>+</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Propagule Type

<table>
<thead>
<tr>
<th>Host</th>
<th>Sporangia</th>
<th>Chlamydospores</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Woody Mediterranean species</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Holm oak (<em>Quercus ilex</em>)</td>
<td>+</td>
<td>+</td>
<td>Moralejo and Hernández 2002</td>
</tr>
<tr>
<td>Italian buckthorn (<em>Rhamnus alaternus</em>)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Strawberry tree (<em>Arbutus unedo</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Laurustinus viburnum (<em>Viburnum tinus</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Mastic tree (<em>Pistacia lentiscus</em>)</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

*Epidemiologically important hosts:* *P. ramorum* apparently does not sporulate on bleeding bole cankers of bark hosts. (Davidson and others 2002b, Defra 2005c, Defra 2006, Garbelotto and others 2003). Sporangia occasionally observed in ooze from bleeding trunk cankers are considered external contaminants. In a study of infected coast live oaks and tanoaks in California (Tjosvold and others 2002a), less than 2 percent of the isolations attempted from the ooze were positive, and no spores were observed in the ooze. All tests for inoculum production on coast live oak cankers (incubation of cankers in growth chambers; pieces of cankered bark in moist chambers), were negative for spore production (Davidson and others 2005b). The authors did not find *P. ramorum* on the surfaces and exudates of cankers of infected coast live oak bark. Because sporulation on oak wood is apparently rare, true oak species have been regarded as non-contagious dead-ends in the life cycle of the pathogen (Davidson and others 2005b, Garbelotto and Rizzo 2004). Tree hosts that are susceptible to trunk infections producing bleeding cankers, but those that are not susceptible to foliar infections, do not provide their own inoculum, and are therefore referred to as terminal tree hosts.

Several tree canker hosts are also foliar hosts. Vettraino and others (2008) have reported *P. ramorum* infecting leaves of several coast live oak saplings in the forest, and the potential of coast live oak foliage to produce sporangia and chlamydospores after inoculation in vitro (Table 1). However, the epidemiological role of those leaf infections in the field is not known. Tanoak is a canker and foliar host that produces inoculum on leaves.

In mixed evergreen forests of California where coast live oak and California bay laurel are the dominant species, California bay laurel is considered the main supporter of inoculum (Davidson and others 2005b; Figure 3). California bay laurel is significantly represented in the plant species composition of mixed evergreen–coast live oak woodland habitats, present as both an understory and overstory species. In coastal redwood forests, dominated by coast redwood (*Sequoia sempervirens*), tanoak and California bay laurel, sporangia are also produced on infected shoots and leaves of tanoak and redwood. The sporangia produced on tanoak are also a significant source of inoculum in addition to sporangia produced on California bay laurel leaves (Davidson and others...
Pacific rhododendron (*Rhododendron macrophyllum*) is also considered a significant spore-producing host, but it produces lower numbers of sporangia than California bay laurel or tanoak shoots and leaves (Davidson and others 2002b). The pathogen sporulates on new infections on redwood needles and shoots (Maloney and others 2007) (Table 1), but at significantly lower levels than on bay laurel leaves and tanoak leaves and shoots (Garbelotto 2004, Garbelotto and Rizzo 2005).

Studies in California have shown a clear association between the presence of the foliar host California bay laurel and *P. ramorum* infection on true oak species (Kelly and Meentemeyer 2002, Swiecki and Bernhardt 2001), indicating the importance of California bay laurel in the disease cycle of the pathogen. The presence and abundance of California bay laurel within plots was identified as a significant plot-level predictor of disease risk (Swiecki and Bernhardt 2001). Several related variables, including counts and percent canopy cover of California bay trees within the study plot, were significant predictors of *P. ramorum* risk (Swiecki and Bernhardt 2004), showing that disease risk increases with increasing bay laurel density and cover within 8 m of a coast live oak. Disease risk and severity were greatest at California bay laurel foliage-oak trunk distances of 1.5 m or less, and were minimal at a distance of 10 m or more (Swiecki and Bernhardt 2008). Bay laurel cover within 2.5 m of the trunk was a stronger predictor of disease risk and severity than the minimum bay laurel-trunk distance. The results suggest that removing California bay laurel from within 2.5 m of the trunk of a susceptible oak will greatly reduce, but not eliminate, the risk of disease. In addition to the association between the presence of California bay laurel and the risk of *P. ramorum* infection, higher abundance of bay laurel is associated with greater levels of infection (Condeso and Meentemeyer 2007, Maloney and others 2005, Meentemeyer and others 2008).

Although California bay laurel appears to be the main source of inoculum for *P. ramorum* in California forests, the pathogen does become established in tanoak forests with little or no California bay laurel. In plots with few California bay laurel stems, high disease levels were associated with the presence of understory tanoaks (Maloney and others 2005).

Although not a common occurrence in California forests, establishment of *P. ramorum* in tanoak forests without California bay laurel appears to be the normal situation in forests in southwestern Oregon (Rizzo and others 2005). Initial infection and inoculum production in Oregon tanoak...
stands apparently occurs on leaves and twigs in the upper crowns of tanoak trees. Examination of
tanoak trees with bole cankers but with green foliage, and adjacent green trees without bole can-
kers, found *P. ramorum* leaf and twig lesions in upper crowns of 11 of 15 tanoak trees with bole
cankers, and from three of the 12 nearby canker-free trees. Infected rhododendron and huckle-
berry in the understory were found only in the immediate vicinity of cankered trees (Hansen and
others 2006).

In Europe, a parallel situation exists, with *Rhododendron ponticum* serving as the main epide-
miologically important understory plant. Many, but not all, tree stem infections in the U.K. and
The Netherlands have been associated with direct proximity to infected *R. ponticum* plants. In
U.K. outbreaks on established plants in parks, managed gardens and wild sites, *Rhododendron*
(especially *R. ponticum*) appears to be the key sporulating host. Tree hosts with bark cankers
have been directly associated with infected Rhododendron (Brasier and Jung 2006, Brasier and
others 2006, Brown and others 2006) in both the U.K. (principally European beech and individu-
als of several oak species) and The Netherlands (European beech and American northern red oak,
*Quercus rubra*).

**Environmental factors affecting sporulation and germination:** In a laboratory study of four
U.S. isolates (NA1) and three European isolates (EU1) of *P. ramorum*, Englander and others
(2006) found that the pathogen produced spores under a broad range of temperature and light
conditions. Chlamydospores were produced by U.S. isolates at 10 to 28 C° (optimum of 14 to 24
C°, depending on isolate), and by EU isolates at 10 to 26 C° (optimum of 22 to 26 C°), with one
isolate producing chlamydospores at 6 C°. Sporangia were produced at all temperatures tested:
10 to 30°C (optimum of 16 to 22°C, depending on isolate) for U.S. isolates, and 6 to 26°C (op-
timum of 22°C) for EU isolates. The U.S. isolates grew less and produced fewer sporangia and
chlamydospores when exposed to increasing doses of near-UV radiation (50–300 µW/cm2) and
visible radiation (250–1500 µW/cm2). The EU isolates were exposed to 300 µW/cm2 near-UV
only, which significantly reduced growth of one of three isolates, but had no significant effect on
spore production.

In laboratory trials, zoospore production on California bay laurel leaves was influenced by tem-
perature (Davidson and others 2005b). Zoospores were produced at 5, 10, 15, 20 and 25°C, but
not at 30°C, with the highest numbers released at 15 to 20°C. Turner and Jennings (2006) report-
ed that optimum temperature for sporulation and zoospore germination ranged from 20 to 30°C
depending upon experimental conditions.

On agar media (V8, cornmeal with antibiotics [CAR], and water agar plates), the rate of germina-
tion of *P. ramorum* chlamydospores (isolate or isolates source not stated) after 24 hours (0.5 to
10.7 percent) was low and variable compared with published chlamydospor germination rates of other *Phytophthora* species (Smith and Hansen 2008).

Turner and Jennings (2006) found that differences in humidity had the most effect on sporangial production and zoospore germination in vitro, whereas sporangial germination was less sensitive. Maximum levels of sporangial production and zoospore germination occurred at 100 percent humidity.

In California mixed evergreen forests, sporulation and production of inoculum is seasonal, largely dependent on rainfall events and temperature. In California, *P. ramorum* is recovered from rain traps from December into the spring months, peaking during warm rains in May (Davidson and others 2005b). At one study site in a mixed evergreen forest, sporulation of *P. ramorum* was correlated with rain events and air temperature (Davidson and others 2005b). Detectable levels of *P. ramorum* inoculum were present largely during the period of winter rains, and were absent during the hot, dry summer (Davidson and others 2005b). The authors observed a lag period between the start of the rains and the onset of detectable inoculum production at the site, with the highest levels of spor production occurring at the end of the rainy season. Possible explanations include the need for inoculum buildup as infection of bay laurel leaves occurs, or the occurrence of lower air temperatures during the beginning of the rainy season which would inhibit sporangial production (Davidson and others 2005b, Fichtner and others 2006).

Recovery of *P. ramorum* from rain traps at five plots in a redwood/tanoak forest type did not show a lag period, with sporangial production beginning soon after the start of the rainy season (Maloney and others 2002, Rizzo and others 2005). Explanations for the earlier sporangial production in redwood/tanoak woodlands than in mixed evergreen forests include less shedding of California bay laurel leaves, and more active lesions on California bay laurel leaves after the hot summer period, because of the wetter cooler conditions in the redwood/tanoak forest type (Davidson and others 2005a).

In contrast, *P. ramorum* inoculum in infested forests of southwest Oregon, most likely dehiscent sporangia, can be produced throughout the year whenever there is water available (Hansen and others, in press). The authors found inoculum on infected tanoak leaves and twigs during periods of leaf wetness, even in extended periods without rain but only fog, anytime during the year.

Spore production varies yearly as well as seasonally. In California forests, relatively high levels of winter/spring rainfall extending into early summer are often followed by high levels of disease (Davidson and others 2005b, Rizzo and others 2005). Epidemic mortality of coast live oak occurred one to two years after increased spring rainfall in 1998 and again in 2005 (El Niño years) (Davidson and others 2005b, Rizzo and others 2005). In Oregon, Kanaskie and others (2008) at-
tributed an unexpected and large amount of disease expansion from eradication sites to two con-
secutive years of unusually wet spring and early summer weather. Hansen and others (in press)
reported that late May rains following a dry April triggered an infection event at two sites, result-
ning in dieback and new growth of tanoak and rhododendron, and numerous new *P. ramorum* bole
infections. These data point to the importance of changes in climate on the establishment and
spread of a largely aerial pathogen such as *P. ramorum* (Rizzo and others 2005).

**Inoculum Dispersal**

*Phytophthora ramorum* inoculum produced on host tissue may be spread or dispersed by vari-
ous means. Local dispersal is by rain splash or wind-driven rain; intermediate dispersal occurs
by turbulent air; and long distance dispersal occurs primarily through the movement of infected
plant material moved in trade, though contaminated soil or watercourses can also result in long
distance dispersal.

**Local dispersal:** Local dispersal occurs primarily through rain splash of sporangia (Davidson
and others 2002b). Rain splash from leaves and soil is the primary means that inoculum is dis-
persed from infected leaves in the shrub canopy to tree stems or from leaves in the overstory to
hosts below. Infection of conifer seedlings and rhododendron placed under infected California
bay laurel occurred only during periods of precipitation and only when the plants were within 4.4
m of the infested overstory (Chastagner and others 2008).

**Wind dispersal:** In California, inoculum has been shown to be dispersed up to 15 m during
wind-driven events (Davidson and others 2005b). The authors (Davidson and others 2005b) sug-
gested that high winds associated with storm events may blow raindrops with spores even further,
thus serving as a means of longer-distance dispersal. In the U.K., *P. ramorum* has been detected
in spore traps at least 50 m from the nearest source of inoculum (Defra 2007c, Turner and others
2008).

In southwest Oregon, most new infections occur within 300 m of infected trees, but rare events
lead to dispersal up to 4 km (Hansen 2008a). Hansen (2008a, 2008b) has suggested that the
observed dispersal over hundreds of meters in Oregon forests could be explained by dry air tur-
bulent dispersal. He hypothesized that clearing weather following spring rains results in over-
night dew and fog, with the lifting of fog as day progresses resulting in lofting of sporangia; the
sporangia are carried by prevailing winds and settle on fog moistened host leaves in the evening
to initiate infection.
Long distance dispersal:

Watercourses: Propagules of *P. ramorum* are dispersed readily in stream water. Davidson and others (2005b) detected inoculum by baiting in stream water 4 to 6 km from an inoculum source in a coast live oak-bay laurel forest. The pathogen is regularly recovered from streams draining infested sites in Oregon, even 5 years after eradication treatment (Goheen and others 2008). The United States Department of Agriculture (USDA) Forest Service (USDA FS) has developed a national *P. ramorum* stream monitoring program for the early detection of *P. ramorum* infestation in the U.S. (Oak and others 2006, Oak and others 2008). In the U.K., *P. ramorum* can be found in watercourses at outbreak sites (Defra 2007c). At an infested site in West Sussex, *P. ramorum* was recovered from the river catchment area originating from the site, but inoculum disappeared within a few km downstream of the outbreak source (Defra 2007c).

The epidemiological significance of inoculum in watercourses is still not fully determined, but contaminated watercourses represent a potential pathway for pathogen spread, especially if water is used for the irrigation of susceptible plants. *Phytophthora ramorum*-positive plant samples have been confirmed outside of a Jackson, Mississippi nursery infested with *P. ramorum*. The samples were associated with a stream receiving runoff from the infested nursery (http://www.suddenoakdeath.org/).

Monitoring of inoculum levels in water suggest that seasonal patterns in amounts of inoculum exist in California and the U.K. In California, the pathogen has been detected in water samples taken from streams during the winter/spring rainy season, but only at reduced levels in the summer months (Davidson and others 2005b). Tjosvold and others (2002b) recovered *P. ramorum* very rarely (on one occasion, in September from one river following a short rain in the river’s drainage) during the dry summer months. In a study using baits placed in California streams within a redwood/tanoak forest (Maloney and others 2002), detection was reported in both spring and summer months, regardless of any rain event. At U.K. study sites, the highest levels of inoculum, as detected by bait tests, generally occurred in winter and spring and the lowest levels in summer (Turner and others 2007; Defra 2007c, 2007d). In another U.K. study, baiting of watercourses within a garden infested with *P. ramorum* generally showed low levels of *P. ramorum* in the summer and winter, but samples taken in the spring, and to a lesser extent in the autumn, were frequently positive for *P. ramorum* (Lockley and others 2008). In contrast, no seasonal effect on recover from streams has been found in Oregon (Sutton and others 2008).

Soil: Human movement of soil infested with *P. ramorum* is a means of local as well as longer distance dispersal. Soil attached to hikers’ boots and vehicles has been shown to harbor the pathogen (Figure 4). In the

Figure 4. Recovery of *Phytophthora ramorum* from hiker’s shoes at Henry Cowell State Park, California. Image: Karl Buermeyer, UC Cooperative Extension.
spring rainy periods, the incidence of baiting for *P. ramorum* varied from 40 to 60 percent success rate for trail soil and 40 to 95 percent success rate for soil removed from hikers’ shoes; the pathogen was not recovered from trail soil or shoe soil in the dry summer period (Tjosvold and others 2002c). Davidson and others (2005b) also demonstrated human spread of infested soil during the rainy season. When they baited soil taken from hikers’ shoes after they walked a preserve trail in Sonoma County California, the pathogen was recovered from 7 of 15 samples in one trial and from 5 of 15 samples in a second trial. Cushman and others (2007) showed that hikers have dispersed *P. ramorum* in soil on their shoes to a distance of at least 60 to 100 m into areas of a nature reserve in California that lacked local inoculum sources. If the soil was kept moist, they (Cushman and others 2007) were able to isolate *P. ramorum* from the soil up to 72 hours after the infested soil was picked up by hikers.

In U.K. studies, *Phytophthora* spp. were present in about 30 percent of samples collected from boots prior to disinfection upon leaving woodland outbreak sites. The most commonly occurring species was *Phytophthora citricola*, but 10 to 15 percent of the samples contained *P. ramorum* or *P. kernoviae* (Webber and Rose 2008). There is no direct evidence of new *P. ramorum* infections occurring in forest situations as a result of human movement of infested soil.

*Other Agents:* Although it has been suggested that insects may vector spores or sporangia of the pathogen (McPherson and others 2002), no evidence of insect transmission has been found (Defra 2005c, Kanaskie and others 2002, McPherson and others 2002). Birds as vectors have also been suggested, but as with insects, no evidence of birds serving as vectors of *P. ramorum* has been found.

*Movement of Host Material:* Movement of infected plant material (for example, wood, green material products, and especially nursery stock) is a primary means of long distance spread of many pathogens, including *P. ramorum*. International trade in ornamental plants is postulated as a main driver for the initial emergence of *P. ramorum* (Davidson and Shaw 2003). Local, national, and international trade in plants and plant products is a major pathway by which *P. ramorum* arrives in new areas and spreads once it reaches them. It is the main pathway for long-distance spread of the pathogen, which is carried on foliage and stems and also in the associated growing medium. In the U.S., the dispersal of the pathogen nationwide from one nursery has been documented (Garbelotto and Rizzo 2005, Stokstad 2004), and is discussed in Chapter 2. All three currently identified clades of this pathogen have been identified in some U.S. nurseries (Ivors and others 2006). The finding that some North American nurseries are infested by all three clades is strong evidence of the role played by the commercial nursery trade in unknowingly aiding the movement and maybe even the introduction of this pathogen in the continent (Garbelotto 2004).
Dispersal in Nurseries: Local dispersal of *P. ramorum* in nurseries and garden centers occurs by many of the same means as in the forest. The pathogen may be transmitted through plant to plant contact, rain or overhead irrigation splash dispersal, irrigation and the movement of infested debris, growing media and freestanding water or surface water run-off. In experiments designed to compare relatively long (1 to 4 m) and short (pot to pot) distance dispersal among rhododendrons in simulated nursery conditions, new infections were only detected on plants within a short distance (adjacent and up to 30 cm away) of a centrally-located artificially-infected plant (Tjosvold and others 2006). No infection was detected in the long-distance experiments and no inoculum was detected in rain traps located 1 to 4 m away from the infected plant during rain events. Note however that sporangia have been detected up to 5 m from infested forest canopy (Davidson and others 2002b), demonstrating that height of release influences distance of splash. Werres and others (2007) demonstrated survival of *P. ramorum* in nursery water reservoirs containing contaminated irrigation water during all seasons. Disease symptoms on rhododendron were observed in as little as 7 days after the first irrigation with contaminated water.

Establishment of Infection

In the infection stage, the pathogen germinates, penetrates the host, and initiates its food relationship with the plant.

Environment: *Phytophthora ramorum* is considered a cool temperature organism, with optimal growth between 18°C and 22°C (64.4°F and 71.6°F; Werres and others 2001). Germination of *P. ramorum* sporangia may be direct, by emergence of hyphae through the sporangial wall, or indirect, by formation and release of motile zoospores. With many species of *Phytophthora*, indirect germination predominates at temperatures below about 12°C (53.6°F), and direct germination occurs at higher temperatures (Ribeiro 1983). The infection rate for California bay laurel leaves averaged 92 percent at 18°C, but only 50 percent at 12°C (53.6°F) and 37 percent at 30°C (86°F) (Garbelotto and others 2003).

Free water enhances infection (Figure 5). Garbelotto and others (2003) found that a minimum of 6 to 12 consecutive hours of free water is a prerequisite for the infection of California bay laurel leaves. Infection by zoospores tends to occur on susceptible plant parts where water accumulates, such as at leaf tips.

**Infection courts:** Openings in host tissue—either natural openings such as stomata or lenticels, or wounds—are entry courts for hyphae originating from germinated spores. Using scanning
electron microscopy, Geltz and others (2006) found that the likely sites of initial infection on *Camellia* leaves were stomata on adaxial surfaces.

Studies by Lewis and Parke (2006) suggested that wounded tissue of *Rhododendron* leaves was more susceptible to infection that nonwounded. Using scanning electron microscopy, they observed that germinating cysts were more abundant at wound sites than at stomatal openings. A study in the U.K. (Defra 2005b) found that infection occurred when lower leaf surfaces, where stomata occur, were inoculated, but did not occur when unwounded upper leaf surfaces with no stomata were inoculated. A separate experiment (Defra 2005b) found that some hosts (*Camellia japonica*) required leaf wounding for infection to occur, while others (*R. ponticum, R. catawbiiense, F. excelsior*) did not. Hansen and others (2005) reported many species developed substantial leaf necrosis in leaf dip inoculation, but few or no leaf symptoms from plant dip inoculation, the difference possibly being that—in addition to a higher effective inoculum dose as well as more favorable incubation conditions—the wounds provided on detached leaves.

*Phytophthora ramorum* zoospores are capable of penetrating bark of mature trees directly. Brown and others (2006) reported that *P. ramorum* zoospores were able to penetrate intact European beech (*F. sylvatica*) bark. Wounding of mature logs of beech (*F. sylvatica*), red oak (*Quercus rubra*), sweet chestnut (*C. sativa*), English oak (*Q. robur*), Sitka spruce (*P. sitchensis*) and Douglas-fir (*P. menziesii*) was not necessary for the infection of logs from mature trees by zoospores (Webber 2004). Inoculations resulted in infection and phloem necrosis development two weeks following inoculation of intact bark in the absence of any wound. Infection occurred most frequently in the thin-barked species (red oak, sweet chestnut, beech).

Bark of saplings of some tree hosts is less susceptible to direct bark infection than bark of mature trees (Defra 2005c). Three-year old saplings of various species (Douglas-fir [*Pseudotsuga menziesii*], sweet chestnut [*Castanea sativa*], red oak [*Quercus rubra*], English oak [*Quercus robur*], Sitka spruce [*Picea sitchensis*], wild cherry [*Prunus avium*], beech [*Fagus sylvatica*], horse chestnut [*Aesculus hippocastanum*], small leaved lime [*Tilia cordata*], ash [*Fraxinus excelsior*], sycamore [*Acer pseudoplatanus*], and hornbeam [*Carpinus betula*]) were only infected when wounded, and trees were more susceptible in the summer than in the winter, with Sitka spruce, horse chestnut and hornbeam not developing symptoms following winter inoculations (Defra 2005c).

Swiecki and Bernhardt (2006) reported that bark thickness and unweathered brown tissue within bark fissures of coast live oak were positively correlated with *P. ramorum* disease risk. The authors suggested that those areas of bark expansion tissue may represent relatively rapidly expanding regions of the outer bark in fast-growing trees that may be more easily breached by *P. ramorum* zoospores. Alternative suggestions include that the outer periderm in these areas may
be so thin that plant substances can diffuse from them when the bark surface is wet, which may attract *P. ramorum* zoospores, increasing the chance of infection, or that the bark fissures may be wetter longer than other areas of the bark which would also favor infection.

Although *P. ramorum* has not been reported to cause root infection on mature trees, several reports of root infection by the pathogen occur in the literature. Root infection of tanoak seedlings in the forest has been reported (Parke and others 2006). Lewis and others (2004) and Parke and Lewis (2007) reported root infection of three-year old rhododendron following replanting of the cuttings into potting soil containing inoculum of *P. ramorum*, indicating transmission of *P. ramorum* from infested potting media to stems via infected, symptomless root tissue. Isolation of *P. ramorum* from healthy appearing rhododendron roots, 4 weeks after inoculation of soils with chlamydospores, was reported by Colburn and others (2005). Bienapfl and others (2005) isolated *P. ramorum* from asymptomatic roots of commercially grown rhododendron obtained from a commercial nursery.

**Host etiology:**

*Host leaf age:* Host leaf age affects susceptibility (De Dobbelaeere and others 2008, Hansen and others 2005). In detached leaf dip assays, tanoak and myrtlewood (California bay laurel) young leaves were more susceptible than mature (Hansen and others 2005). In a test of 80 different *Rhododendron* species and cultivars, De Dobblelaere and others (2008) found that leaf age affected susceptibility to *P. ramorum*. When using wounded leaves, young leaves of all cultivars tested showed a higher level of susceptibility than mature leaves. However, when using non-wounded leaves, young leaves of some cultivars were less susceptible than older leaves. This effect was correlated with the presence of hairs on the young leaves of those cultivars, which probably form a barrier to the zoospores and prevent tissue penetration.

*Temperature exposure:* In the same studies, the authors (De Dobblelaere and others 2008) found that exposing *Rhododendron* plants to high temperatures for several hours before collection of the leaves had a negative effect on susceptibility in most experiments. They suggest that the increase in temperature may have resulted in stomatal closure, which may have reduced penetration capacity.

*Season of year:* Based on log inoculations, seasonal variability in host susceptibility occurs. Brasier and Kirk (2002) concluded that the trees they tested were immune in April and most susceptible in late summer and fall. Hansen and others (2005) reported smaller lesions following inoculation on tanoak logs harvested in January than on tanoak logs harvested in March, September, or November, while lesions on Douglas-fir inoculated in January were larger than at other times of the year.
**Inoculum pressure:** The amount of inoculum (inoculum pressure) affects infection potential. Differences in susceptibility with different levels of *P. ramorum* inoculum have been reported (Defra 2005b, Hansen and others 2005). For example, at low inoculum levels (1.3 x 10^3 zoospores/ml), common elder (*Sambucus nigra*), lilac (*S. vulgaris*), California bay laurel (*U. californica*) and honeysuckle (*Lonicera periclymenum*) were not infected in detached leaf assays with unwounded leaves. However, when the amount of inoculum level was increased (2.3 x 10^5 zoospores/ml), 67 to 100 percent of the leaves inoculated became infected (Defra 2005a). The same study (Defra 2005b) found that unwounded leaves of *C. japonica* remained uninfected at both low and high inoculum pressures, but 33 percent of wounded leaves were infected at the low inoculum pressure and 100 percent at the high inoculum pressure. Hansen and others (2005) reported that inoculum dose affected susceptibility in detached leaf assays, with higher zoospore concentrations generally resulting in more symptoms.

**Colonization (Invasion)**

The invasion stage occurs when the pathogen grows on or in the host and obtains nutrients from it.

Following entry through the bark of woody hosts, hyphae of *P. ramorum* grow within the phloem and cambial tissues, and then into xylem tissues (Rizzo and others 2002, Brown and Brasier 2007, Parke and others 2007). Xylem colonization is the norm. Rizzo and others (2002) noted that infection on *Quercus* spp. and tanoak appeared to begin in the outer bark, progress to the cambium and eventually reach the xylem. Discoloration was generally more extensive in the cambium and secondary phloem tissues than in the xylem; typically black discoloration extended less than 1 mm into xylem tissues, but was occasionally observed up to 3 cm into the xylem. Brown and Brasier (2007) found that *P. ramorum* commonly occupies xylem beneath phloem lesions, that the pathogen can penetrate xylem tissue, and that it spreads in xylem tissue ahead of phloem lesions. Parke and others (2007) demonstrated the presence of *P. ramorum* in the sapwood of mature, naturally infected tanoak. Hyphae were abundant in the xylem vessels, ray parenchyma, and fiber tracheids; chlamydospores were observed in the vessels. The authors found reduced sap flux and specific conductivity in infected tissues compared to non-infected, and suggested that the reduction may result from increased embolism caused by *P. ramorum* infection, the presence of fungal structures, and the increased abundance of tyloses present in the vessels. They further suggest that the reduced stem water transport may contribute to crown mortality associated with *P. ramorum* infection.

Several studies have examined *P. ramorum* colonization of *Rhododendron*. Pogoda and Werres (2004) found that *P. ramorum* colonized the cortex, phloem, xylem and pith of the necrotic zone.
The authors also found hyphae in the cortex and pith of healthy looking material about 1 cm below visible discoloration; chlamydospores were only observed in the necrotic zone where they developed mainly in the cortical parenchyma. This study also showed that *P. ramorum* can grow both intra- and intercellularly but chlamydospores were only observed in the intercellular spaces.

Using epifluorescence microscopy, Riedel and others (2008) found *P. ramorum* hyphae in the cortex and pith of infected *Rhododendron* plants when discoloration was present. In healthy looking stem and root segments located adjacent to the discolored areas, hyphae were found most often in the secondary xylem. With one exception, chlamydospores were only located in discolored parts of infected *Rhododendron* plants, being present in the cortex (stem) and palisade mesophyll (leaf). Less frequently chlamydospores were observed in the pith of necrotic stem segments. Using the same epifluorescence microscopy technique, Parke and Lewis (2007) observed attraction of zoospores to wounds and root primordia of *Rhododendron* tissue culture plantlets in vitro, and colonization of the cortex and vascular tissues of roots and stems, including the xylem.

**Survival**

The disease potential for a pathogen, including *P. ramorum*, is partially dependent on its ability to survive during conditions unfavorable for growth and reproduction. Like most species of *Phytophthora*, *P. ramorum* survives during conditions unfavorable for growth—such as hot, dry summer months in California—in host tissues or in various non-host substrates (such as organic matter, soil, and potting media) as hyphae or as asexual structures. For other species of *Phytophthora*, chlamydospores are the typical survival structures in the disease cycle. The role of chlamydospores in the survival of *P. ramorum* is unknown.

**Survival in vitro:** In moist conditions, zoospores and chlamydospores of *P. ramorum* remain viable for at least 1 month, with chlamydospores probably able to survive much longer (Davidson and others 2002b). Zoospores and chlamydospores did not germinate on selective medium after 30 minutes on dry filter paper. After 30 minutes on moist filter paper, 41 percent of chlamydospores and 20 percent of zoospores germinated. In de-ionized water, 75 percent of chlamydospores and 20 percent of zoospores germinated on selective medium after 30 days (Davidson and others 2002b).

A U.K. laboratory study (Defra 2005c) found that sporangia and chlamydospores germinated on agar after exposure to -2 °C for 24 hours. Chlamydospores were not capable of germinating after exposure to 55 °C for one hour. No chlamydospores germinated following exposure to 40 °C for 24 hours or -25 °C for just 4 hours. Sporangia did not germinate after a 2-hour exposure to these temperatures. Sporangia germinated after 6 hours, but not 24 hours, at room temperature in dry
conditions. In a study to determine the effects of temperature and composting treatments on the viability of *P. ramorum* (Swain and others 2006), the pathogen was not recovered from compost by plating on selective medium or by baiting with pears following temperatures of 55°C for 1 hour or 40°C for 24 hours. The pathogen was also not detected using PCR, suggesting that *P. ramorum* was absent from the treated compost and just suppressed or dormant. Similar results were reported by Turner and Jennings (2006); chlamydospores germinated after two months storage in vitro at 0, 5, 15 and 30°C, but not at -25°C or 40°C.

**Survival in host material:** One important means of *P. ramorum* survival over the hot, dry summer months in California is as infections in California bay laurel leaves (Davidson and others 2005b, Rizzo 2006). Survival in attached leaves was found to be higher than in abscised leaves (Davidson and others 2002a, Davidson and others 2003a). Survival (as assessed by cultural isolation) in attached bay leaves declined from approximately 90 percent in June to 50 percent in August, but persisted throughout the summer. Survival in abscised leaves collected from leaf litter in June, July, and August was nearly zero. The same study determined that infected bay leaves were significantly more likely to abscise in drier mixed-evergreen forest as opposed to tanoak-redwood forest. The authors surmised that, given that survival of *P. ramorum* occurs in attached rather than abscised leaves, the observed differences in abscission rates of *P. ramorum* infected bay leaves in coast live oak versus tanoak-redwood forests may be one factor contributing to differences in the onset of detectable inoculum production in these two forest types during the winter rainy season.

Fichtner and others (2008) compared summer survival of *P. ramorum* associated with California bay laurel leaves in redwood-tanoak and mixed-evergreen forests. Pathogen isolation from attached leaves ranged from 40 to 100 percent at each site in May and declined to a range of 0 to 40 percent in August, with higher isolation recovery observed in redwood-tanoak forests than in mixed-evergreen forests.

In leaf-debris survival experiments in Oregon (McLaughlin and others 2006), *Rhododendron* and tanoak leaves inoculated with *P. ramorum* were placed in the field; the leaves were shaded or unshaded, and placed above ground or buried. Pathogen survival was higher (89 percent) in buried leaves after 8 weeks than from those on the soil surface in shade (66 percent survival) or on the soil surface exposed to the sun (26 percent recovery).

Shishkoff and Tooley (2004) reported survival of *P. ramorum* in infected *Rhododendron* leaf tissue containing chlamydospores buried in mesh bags in pots containing nursery stock for up to 155 days after burial (Shishkoff and Tooley 2004). In additional experiments, Shishkoff (2007) recovered *P. ramorum* from moist potting mix or sand for many months, whether buried as infected plant leaf tissue or as mycelium bearing chlamydospores. The author found no significant
difference in recovery over time among treatments (sand or potting mix; infected plant tissue or mycelium); after approximately a year, colonies could be recovered at 0.8 to 14.3 percent.

Chlamydospores of *P. ramorum* in lilac (*S. vulgaris*) and rhododendron leaves, either as surface leaf litter or buried in soil at a depth of 5 cm, survived the winter season in experiments conducted in northern England and Scotland (Defra 2005c). Pathogen survival gradually decreased over time, but *P. ramorum* could be recovered from at least 50 percent of leaves in all treatments. Survival was slightly higher on the host (rhododendron) that had thicker and more durable leaves compared to the host (lilac) which had more fragile and less durable leaves. Survival was highest on rhododendron leaves that had been buried 5 cm below the soil surface, with over 80 percent of leaves still yielding the pathogen after 4 months. Tests carried out in Scotland in a parallel experiment showed similar survival under ambient conditions over the same period.

The pathogen has been recovered from host tissues at eradication sites. At Oregon eradication sites, *P. ramorum* was recovered from tanoak sprouts around 88 percent of the tanoak stumps sampled (Hansen and Sutton, 2006). Initial sprouts were destroyed, and subsequent exams found no infection on later establishing sprouts or seedlings (Hansen, personal communication). At one eradication site in the U.K., the pathogen was observed on new shoots emerging from the stumps of cut rhododendron (Defra, 2007d).

Shelly and others (2006) isolated *P. ramorum* from 1 of 30 specimens of tanoak and coast live oak logs that had air-dried for 6 months, demonstrating that *P. ramorum* could survive on firewood for at least 6 months.

**Survival in soil and potting media:** The pathogen is capable of survival for extended periods of time without the presence of a host.

Davidson and others (2002b, 2005b) did not recover *P. ramorum* from soil and litter surrounding infected oaks within a coast live oak woodland in the summer months. The failure to detect the pathogen in soil during the summer coincided with a drop in mean soil water content to less than 15 percent, indicating that seasonal drying is sufficient to reduce viability of spores in these substrates. Maloney and others (2002) also found that recovery of *P. ramorum* from soils dropped to zero during the summer months, with frequency of recovery from soil during the rainy season approximately 20 percent, but summer soil collections showing no *P. ramorum* recovery.

In contrast, Fichtner and others (2006, 2007) demonstrated that *P. ramorum* survives and produces chlamydospores in forest soils over summer, providing a possible inoculum reservoir at the onset of the fall disease cycle. Although Davidson and others (2002a, 2005b) could not detect *P. ramorum* in soils associated with oaks within approximately 1 month of the last spring rain event,
Fichtner and others (2006, 2007) recovered the pathogen from soils up to 3 months after the last rain event. Fichtner and others (2007) reported that recovery frequency of *P. ramorum* was highest under California bay laurel, intermediate under tanoak, with only occasional recovery under redwood. Recovery of the pathogen from infected rhododendron leaf discs after 8 weeks was highest in the soil (80 percent), intermediate in the litter/soil interface (60 percent) and poor (1 percent) on the leaf litter surface. The differences between the two studies may be explained by the fact that Davidson and others (2002a, 2005b) monitored soil inoculum in a mixed-evergreen forest using pear baits, whereas Fichtner and others (2006, 2007) monitored soils in a redwood-tanoak forest using rhododendron leaf baits.

Lockley and others (2008) reported variable results with survival of *P. ramorum* at a site in the U.K. At one site, where the infected host (*Rhododendron*) was removed and composted mulch applied to the soil surface, the pathogen could not be detected in soil samples. At a second site, where host removal was impractical and the only treatment was removal of lower branches of the rhododendrons, detection of *P. ramorum* in soil samples gradually declined. In The Netherlands, Aveskamp and others (2006) found that the pathogen remained viable for at least 1 year in sandy soil. The authors reported that the pathogen was more abundant at a depth of 20 cm than at the soil surface.

*Phytophthora ramorum* has also been found at depths of up to 15 cm in soil in areas of severe plant infection (Turner and others 2008). Monitoring at sites where eradication attempts were made early in the disease epidemic has shown that, in the absence of inoculum sources, residual contamination in soil declined slowly over time and in some cases declined to below thresholds of detection (J. Turner, personal communication, as cited in the Defra datasheet). At five eradication sites in Oregon, *P. ramorum* was recovered over a 4 year period from soil at the base of selected stumps at three sites, only in the initial sample at one site, and never recovered from soil at one site (Goheen and others 2008).

*Phytophthora ramorum* has a high potential for infesting and remaining viable in potting media. Linderman and Davis (2006a, 2006b) reported survival of *P. ramorum* in potting media or soil for up to 6 months when the pathogen was introduced to the media as sporangia, and 12 months when introduced as chlamydospores. Potted *Rhododendron* became infected following inoculation of potting mix with *P. ramorum* (Grünwald and others 2008). Colburn and others (2005) found no decline in chlamydospore populations after 4 months in sand, potting soil mix or forest soil held at 4°C, but the population decline in forest soil at 22°C. Jeffers (2005) reported recovery of *P. ramorum* from a composite sample container mix (containing plants shipped from California to South Carolina retail nurseries) after storage for 6, 8, and 12 weeks at 4°C, but not from another sample stored for 10 weeks at room temperature.
In a study designed to help define treatments capable of inactivating chlamydospores in soil substrates, Tooley and others (2008), investigated germination potential over a 7 day temperature treatment by incubating chlamydospores in sand at low (0, -10, -20 °C) and high (30, 35, 40 °C) temperature treatments. Near 100 percent germination was observed at temperatures of 0°C for up to 7 days in the low temperature treatments, but almost no germination occurred at -10 or -20°C over the 7 day period. For the high temperature treatments, high levels of chlamydospore germination were observed over the 7 day period at 30°C, while no growth was observed at 40°C. At 35°C, high levels of chlamydospore germination were observed at day 1, but recovery (growth on selective agar medium) declined steadily and was zero by 7 days.

The role of *P. ramorum* oospores as survival structures is unknown, as they have not been observed in the field. In other species of *Phytophthora*, the thick-walled oospores typically serve as survival structures.

**Summary**

In order for any pathogen to successfully complete its disease cycle, a suitable environment and the presence of a susceptible host that will support sporulation are key. The exotic, introduced organism *P. ramorum* has found a favorable environment and suitable hosts in the mixed evergreen/redwood-tanoak forests of coastal California and southwest Oregon, in gardens, woodlands and parks in the U.K. (Defra 2004), and in nurseries.

Knowledge of the disease cycle and the environmental requirements of the pathogen to complete each stage of the cycle are essential for effective management of any disease. Some examples of potential management actions based on the disease cycle of *P. ramorum* are summarized in Rizzo and others (2005). For instance, the findings that *P. ramorum* inoculum survives in soil has led to recommendations to avoid movement of soil from infested to uninfested areas.

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