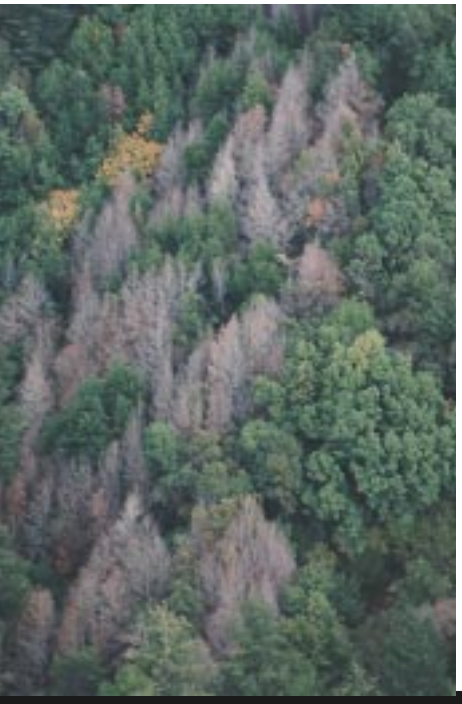
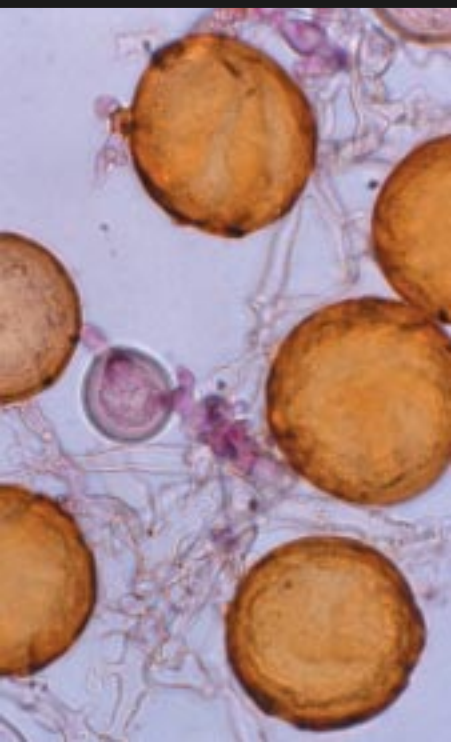
Photo: Keith Parker



How to recognize symptoms of diseases caused by

Phytophthora ramorum



causal agent of Sudden Oak Death

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Photo: Keith Parker

How to recognize symptoms of diseases caused by *Phytophthora ramorum*, causal agent of Sudden Oak Death

Recognizing symptoms of *Phytophthora ramorum*, a newly described microbe and cause of "Sudden Oak Death," can be difficult so we have assembled pictures and information to guide field identification.

Background

Phytophthora ramorum, cause of Sudden Oak Death (SOD), is responsible for widespread tree mortality in Central and Northern California. This common name "Sudden Oak Death" refers to the apparent rapid decline of the entire tree crown; the foliage of trees affected often turns from an apparently healthy green color to brown in only a few weeks. Despite this graphic common name, not all trees affected by the disease incur in this apparent sudden death. There is nothing "sudden" in SOD and it may take several months or years for the pathogen to kill a tree. Researchers are still trying to understand how rapidly the disease may progress; it is expected that disease progression may vary based on different tree individuals, different tree species, different geographic regions and other factors.

Three species of native California oaks (*Quercus* spp.) and a close relative of oaks, called tanoak or tanbark oak (*Lithocarpus densiflorus*), may be killed by the disease. To date, only oaks in the black or red oak group have been found to be susceptible to the disease, white oaks are currently not known to be hosts. At least 11 other unrelated plant species may serve as hosts for *P. ramorum* (Table 1). On many of these hosts, infection by the pathogen may not lead to death of the whole plant, but rather to leaf spots and/or twig and branch dieback. The Latin name of the pathogen means "destructor of branches" reflecting its discovery by plant pathologists in Europe in 1993 as the cause of a new disease of leaves and branches of ornamental rhododendrons. Foliar hosts can at times be killed, especially if host plants are shrubby or small. Progressive dieback of branches can also lead to plant mortality, normally if conditions conducive to the disease persist for several years in a row.

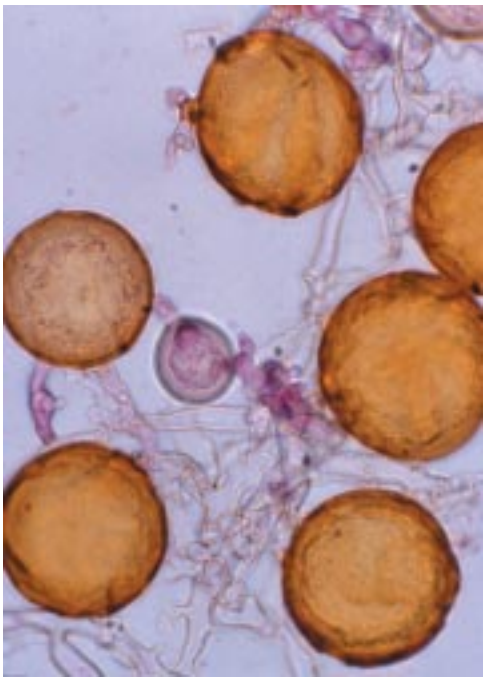
Fig. 1. Sporangia of *P. ramorum*. (Rizzo)

The Pathogen

There are about 60 known species closely related to *P. ramorum*, and only specialists can differentiate amongst them. The reproductive, dispersal and survival structures of the pathogen are only visible under a microscope. Sporangia (Fig. 1) are reproduc-

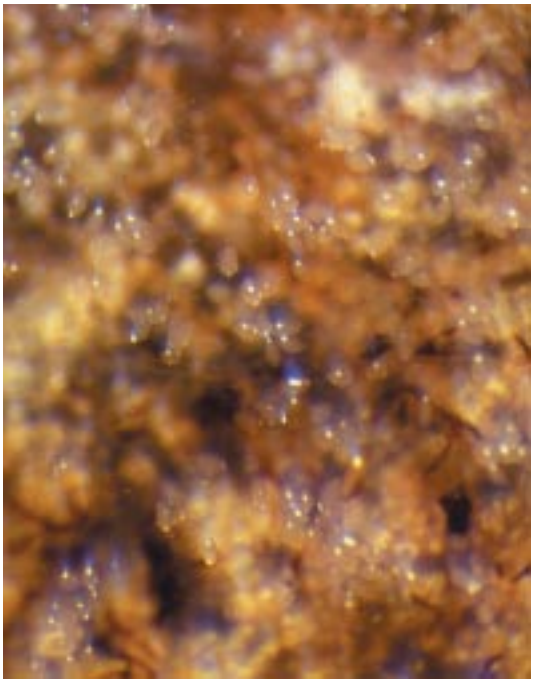
Table 1. Known hosts and symptoms guide of *Phytophthora ramorum* in California.

Host	Common name	Plant part infected	Plant Death	
			Adult trees or plants	Juveniles
Quercus agrifolia (Fagaceae)	Coast live oak	main stem	YES	NO
Q. kellogii (Fagaceae)	California Black oak	main stem	YES	NO
Q. parvula var. *shrevei* (Fagaceae)	Shreve's oak	main stem	YES	NO
Lithocarpus densiflorus (Fagaceae)	Tanoak	main stem, branches, leaves	YES	YES
Arbutus menzesii (Ericaceae)	Madrone	branches, leaves	PROBABLY	YES
Vaccinium ovatum (Ericaceae)	Evergreen huckleberry	main stem, branches, leaves	YES	YES
Arctostaphylos manzanita	Manzanita	branches, leaves	UNKNOWN	UNKNOWN
Rhododendron sp. (Ericaceae) (Ericaceae)	Ornamental rhododendron	branches, leaves	YES	YES
Rhododendron macrophyllum (Ericaceae)	Rhododendron	leaves, branches	YES	YES
Umbellaria californica (Lauraceae)	Bay laurel, Oregon myrtle	leaves	NO	NO
Acer macrophyllum (Aceraceae)	Big leaf maple	leaves	NO	NO
Heteromeles arbutifolia (Rosaeceae)	Toyon	branches, leaves	UNKNOWN	UNKNOWN
Aesculus californica (Hippocastanaceae)	Buckeye	branches, leaves	NO	NO
Rhamnus californica (Rhamnaceae)	Coffeeberry	leaves	UNKNOWN	UNKNOWN
Lonicera hispidula (Caprifoliaceae)	Honeysuckle	leaves	UNKNOWN	UNKNOWN



(left)
Fig. 2.
Chlamydospores of *P. ramorum*. (Rizzo)

(right)
Fig. 3.
Sporangia growing on a rhododendron leaf (Rizzo)



tive structures known to play an important role in spreading the disease. They will release zoospores (propagules with 2 tails), known from other species of *Phytophthora* as key infectious structures. Zoospores will swim in free water and efficiently infect plant tissue. The sporangia of *P. ramorum* may also germinate directly and infect plants. Chlamydospores (Fig. 2) are thick-walled and can probably survive several months.

Under moist conditions, sporangia, and at times chlamydospores, can be found on the foliage of hosts such as rhododendron and bay (Fig.3). Sporangia formed on leaf surfaces may be rain-splashed and spread aerially to infect new hosts. Thus, foliar hosts of *P*. *ramorum* may allow for the rapid establishment of the disease in an area, and for the natural spread of the pathogen across the landscape.

How to detect *Phytophthora ramorum*

On oaks and tanoaks, *P. ramorum* kills portions of the bark resulting in cankers or lesions. If you encounter oaks or tanoaks with complete crown mortality, especially those with leaves still attached, check nearby oaks that are still green for bleeding. Use a hatchet to shave away the bark in bleeding areas and check for dead (dark-brown to black) patches of bark surrounded by black-zone lines. It is easier to diagnose trees when they are still alive, before their foliage has turned brown, since secondary organisms rapidly colonize dead tissues.

The best way to distinguish *P. ramorum* oak mortality from other causes of oak mortality is to look for symptoms on adjacent known hosts. If you find symptoms on several known hosts adjacent to dead and bleeding oaks, it is likely that infection is due to *P. ramorum*. Laboratory isolation is needed to confirm the presence of this pathogen. Contact your County Agricultural Commissioner or UC Cooperative Extension county office for assistance. *P. ramorum* is a quarantined pathogen. Do not clip samples and take them to a “Garden Center” or plant clinic for diagnosis. Leave all plant materials on site.

Not all oak species are susceptible to the disease, and as stated above, not all host species are affected as seriously. Table 1 summarizes all official hosts and types of symptoms to be expected

on each host species. This list is incomplete; researchers are still studying the host and geographic ranges of the pathogen.

Gallery of symptoms

1-The disease at the landscape level: advanced stage.

Whether in wildlands or at the wildland-urban interface, the advanced stage of the disease results in high levels of mortality of tanoak, coast live oak, and California black oak (Figs. 4 and 5). The entire crown of dead trees will initially turn brown (Fig. 6) and then turn gray as the foliage is lost (Fig. 7). Note that California black oak (a host species) and other non-host species such as Valley oak, appear gray or leafless during the winter because they are deciduous. Tree mortality normally appears in patches. California buckeyes are summer deciduous and naturally drop their leaves in the summer. Other diseases (for example, the root rots *Armillaria mellea, Phytophthora cinnamomi*) can cause patches of dead trees, but their symptoms are quite different. Fires and chemical girdling may also cause patches of dead trees. Look for burnt litter and bark or for chemical injections to differentiate between diseases and other causes of tree mortality.

Fig. 4. SOD in coast live oak in the urban-wildland interface (Keith Parker)

Fig. 5. Dying tanoaks in a redwood-tanoak forest in Marin County (Kent Julin)

Fig. 6. The crown of an understory tanoak killed by *P. ramorum* (Garbelotto lab)

Fig. 7. The gray trees are coast live oaks killed by *P. ramorum* more than a year before the picture was taken (Garbelotto lab)

2-The disease at the landscape level: early stages.

Although not fully understood yet, it is believed that at first the pathogen will colonize leaves of foliar hosts (non oaks) and of tanoaks. The presence of suspicious spots and blotches on leaves of two or more hosts may be an indicator that the disease has arrived in an area and has not yet caused lethal cankers on oaks or tanoak.

3-The disease on various host species.

P. ramorum has been discovered relatively recently; for this reason this list (Table 1, page 4) should not be considered complete or exhaustive. New symptoms and more hosts may be discovered. Due to the limited extent of scientific observations, some of the images shown here are the result not of natural infection, but of artificial inoculations in the laboratory.

Most of the symptoms described below are not exclusively caused by *P. ramorum*, they may be caused by other pathogens, sunburn, frost or other abiotic agents. Beware of look-alikes and look for the clustering of symptoms on several hosts in an area.

TANOAK (*Lithocarpus densiflorus*): Tanoak is highly susceptible to *P. ramorum* infection through a combination of foliar blight, branch dieback, and stem cankers. Plants of all sizes may be killed. Early symptoms of the disease include wilting of apical shoots (“Shepard’s crook ”)

Fig. 8. Wilting of tanoak shoot (Pavel Svihra)

Fig. 9. Tanoak leaves showing initial foliar symptoms of SOD (Garbelotto lab)

(Fig. 8), light brown leaf spots and blotches (Fig. 9), and dead leaves. As the disease progresses, branches die back due to formation of branch cankers. Stem cankers develop at various heights from the ground, most commonly just above the soil line.

Cankers appear as brown water-soaked lesions in the bark and the cambium of infected trees, often progressing into the outer part of the xylem (Fig. 10). Black lines are often, but not always, seen at the canker margins (Fig.11). Seeping of viscous sap, black to amber in color, can at times be seen, creating the typical SOD symptom referred to as “bleeding” (Fig.12).

Stem cankers may girdle the tree causing the entire tree crown to turn yellow then brown. Crown decline is generally rapid after a period of months when the stem is dying. Infected trees will often resprout at the base but eventually all, or most of these basal sprouts, die as well.

Once the tree is seriously compromised by advanced infection of *P. ramorum*, opportunistic organisms may become established. These include the sapwood decay fungus *Hypoxylon thouarsianum* (Fig. 13); this fungus produces

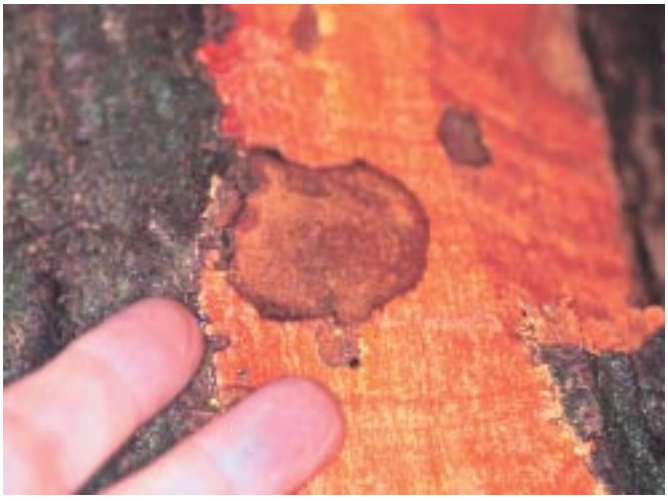


Fig. 10. Cankers appear as brown water-soaked lesions in the bark. (Garbelotto lab)

Fig. 11. Close up of a black line between infected tissues (right) and healthy tissue (Rizzo)

(left)
Fig. 12.
Seeping through bark of an infected oak tree (Garbelotto lab)

(right)
Fig. 13.
Fruiting bodies of *Hypoxylon*

charcoal-black globose fruiting bodies on the bark. Ambrosia and bark beetles, tunneling into the sapwood and bark respectively, will produce sawdust expelled from the beetle galleries. It should be noted that these secondary organisms are not necessarily associated with SOD, but can be seen when trees decline and die because of a variety of reasons.

COAST LIVE OAK, CALIFORNIA BLACK OAK, SHREVE'S OAK (*Quercus agrifolia, Quercus kelloggii, and Quercus parvula var. shrevei*). These three oak species can effectively be killed by stem cankers caused by *P. ramorum.* Leaves and branches generally do not appear to be susceptible. Cankers are mostly seen on the lower part of the stem just above the root collar, but do not apparently extend into the roots. This feature differentiates *P. ramorum* cankers from those caused by other Phytophthoras (e.g., *P. cinnamomi*). Cankers can also develop high on the stem, but they are rarely seen on portions of the stem smaller than 10 cm. Infection of seedlings is unreported in nature, and infection of saplings appears to be extremely rare. Cankers are water-soaked regions of dead bark often extending into the outer portion of the xylem. They are generally demarcated by visible black zone lines (Fig. 11). Seeping is commonly associated with cankers. Seeping ooze is generally black to amber in color and viscous. At the early stages of the canker, seeping will occur through the intact bark without any noticeable physical wounding. In later stages, the bark can fracture and seeping keeps occurring both through broken and intact bark. Often seeps dry out and can be identified by their brownish tinge on the darker bark (Fig. 14).

Fig. 14. Brown dried seep on the bark of a coast live oak (Garbelotto lab)

Once trees are girdled by the pathogen they will eventually die. In general, the foliage of infected trees will change from green to brown over a period of several weeks; however, this browning may occur from several months to more than a year after the pathogen has effectively girdled the whole stem. It is not uncommon to observe the loss of a significant number of leaves after cankers have girdled the tree but before the whole crown browns.

Seeping can be caused by other pathogens such as *Armillaria* or other *Phytophthora* spp. or by physical wounding caused by people and insects. Wetwood, a bacterial infection often initiated by major wounding and tree aging, can also result in streaks of blackish seeps on the bark surface. Wetwood seeps are always dark, not viscous, and often associated with a physical opening in the bark to surface. Wetwood seeps also have a foul odor as compared to a wine smell of seeping caused by *P. ramorum* infection. Common secondary organisms are the same as those described for tanoaks.

RHODODENDRON. *Rhododendron* spp. Not all species and ornamental varieties are equally susceptible, but precise information on host range is still unavailable. Both California native species (*R. macrophyllum* and *R. occidentale*) can succumb to the pathogen. Leaves are readily infected by *P. ramorum*, and brown-black lesions often develop on the leaf portion where water accumulates. These lesions generally have "fuzzy" borders while lesions caused by sun damage typically have clear black contour lines (Fig. 15). At times, though, *P. ramorum* concentric ring growth pattern is evident when examining the lesions. *P. ramorum* can also infect and kill branches, and may progress to kill entire plants. The pathogen readily sporulates on the leaf surface of *Rhododendron* spp. (Fig. 3).

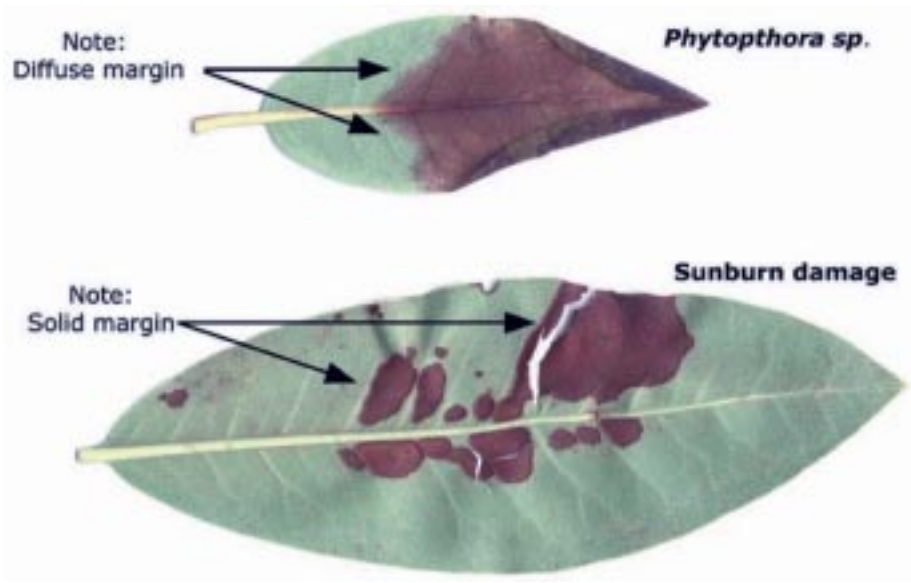


Fig. 15. Comparative damage caused by sunburn and by *P. ramorum* on rhododendron leaf (Tim Tidwell)

Fig. 16. Branch dieback of a huckleberry plant. (Davidson)

HUCKLEBERRY. The California or Pacific huckleberry (*Vaccinium ovatum*) can be infected by *P. ramorum*. Although individual leaf spots have been observed, the most common symptom includes branch dieback. Lesions can be observed on the stems (canes), and the whole branch upward from the lesion will die and brown. Although entire

Fig. 17. Necrotic tips and spots on bay laurel (Garbelotto lab)

Fig. 18. Dead branches on bay trees attributed to *P. ramorum* (Garbelotto lab)

Fig. 19. Madrone leaf with spots caused by *P. ramorum* (Garbelotto lab)

plants can be killed, it is more common to observe patches of dead and live branches (Fig. 16).

CALIFORNIA BAY LAUREL (*Umbellularia californica*). *P. ramorum* damage on this plant is virtually undistinguishable from symptoms caused by another common disease known as bay anthracnose. For both diseases, the most striking symptom consists of a black tip, turning gray in time. It is often, but not always, delimited by a chlorotic (e.g., yellow) zone (Fig. 17). Lesions are normally on the tip since water is most likely to accumulate there. On leaves that are flat or carried with the tip upwards, lesion can develop in the middle section, especially around the edges or at the base of the petiole. Often infection results in a number of clearly distinct black spots that are visible on the green portion of the leaf. At times, only the black spots may be visible without the presence of an extensive blotch. Some branch dieback has been observed in trees infected by *P. ramorum*, but it is still uncertain whether *P. ramorum* is responsible or not for the development of this symptom (Fig. 18). Mortality of bay laurel due to infection by *P. ramorum* has never been reported.

PACIFIC MADRONE. Leaf spots, leaf death, and branch dieback are commonly associated with infection by *P. ramorum* on this species. A precise analysis of the effects of this disease on pacific madrone is complicated by its high susceptibility to other common pathogens that produce similar symptoms. *P. ramorum*'s destructive effect on madrone is suggested by the observation that in areas infested by *P. ramorum*, levels of tree dieback and mortality among the regenerating madrone/juveniles and seedling madrone are higher than in comparable sites where *P. ramorum* is not present. Discrete leaf spotting, often with a purplish tinge, is one of the first symptoms of infection by *P. ramorum* (Fig. 19). Spots expand into larger botches that often turn to gray and brown. Lesions also may develop on the stems of small plants and on branches. Although not always a reliable trait, infection by other foliar and branch pathogens may lead to the formation of small black reproductive structures barely visible by the naked eye. At times though, several pathogens may be present on the same leaf and diagnosis must be made by a professional.

Fig. 20 & 21. Different stages of infection by *P. ramorum* on buckeye (Garbelotto lab)

CALIFORNIA BUCKEYE. *P. ramorum* can infect the leaves, the petioles and the twigs of California buckeye trees. Early symptoms start as rounded individual spots that tend to coalesce later in the season (Figs. 20 and 21). The symptoms are very similar to those caused by the buckeye anthracnose pathogen, *Guignardia aesculi*. Darkened lesions on petioles and twigs (Fig. 22) indicate presence of *P. ramorum*. Early foliar senescence (buckeyes are deciduous trees and drop the foliage unusually early in hot and dry areas) causes leaf distortions similar to those caused by *P. ramorum*.

BIG LEAF MAPLE. In this tree species, *P. ramorum* infection appears more like a scorch, normally starting from the edges of the leaf (Fig. 23). In general, the scorching will have irregular borders that do not precisely follow leaf contour. The discoloration can be variable in color with hues ranging from orange to brown (Fig. 24). Once the leaves senesce and yellow, the discoloration should still be visible for some time. Branch dieback has been observed in areas infested by *P. ramorum*, but a

Figs. 21.

Fig. 22. Petiole infection by *P. ramorum* on buckeye. (Garbelotto lab)

Fig. 23 & 24. Big leaf maple leaf showing scorch-like lesions caused by *P. ramorum* (Garbelotto lab)

Fig. 25. A naturally infected leaf of toyon (Garbelotto lab)

Fig. 26. Toyon leaves inoculated with *P. ramorum* (Rizzo)

Fig. 27. Manzanita stem inoculated with *P. ramorum*. Note the dark lesion and death of the two leaves at the inoculation point. (Rizzo)

clear cause-effect relationship has not been determined yet.

TOYON, HONEYSUCKLE, MANZANITA and COFFEEBERRY are less understood hosts for *P.ramorum*. Dark foliar spots, at times demarked by a thick black line, may be one of the symptoms caused by *P. ramorum* on Toyon, a poorly understood host (Figs. 25 & 26). Less prominent foliar spots, branch lesion and unusual death of entire plants have been reported. In general, lesions appear where water accumulates (e.g., on the tip of a leaf), they are dark in color and have "fuzzy" margins. Lesions with discernible concentric rings have been reported in honeysuckle. The water-soaked appearance of the cankers and the absence of black fungal reproductive structures (pycnidia), may help to differentiate *P. ramorum* symptoms on manzanita from those caused by *Botryosphaeria* (Fig. 27).

Fig. 28. Viburnum in Germany infected with *P. ramorum* (Sabine Werres)

VIBURNUM (Viburnum x bodnantense). *P. ramorum* was isolated from wilting Viburnum nursery plants in Europe. The pathogen has never been found on Viburnum in California or Oregon. Unlike other shrub species, which typically suffer branch dieback, infection on Viburnum starts at the base of the stem, the outer stem tissues are killed and eventually the entire plant wilts. (Figs. 28 & 29)

Fig. 29. Basal lesion on Viburnum due to *P. ramorum* (Sabine Werres)

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List of Figures