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A Reference Manual for Managing Sudden Oak Death in California

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Abstract

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This publication contains background information and guidance for resource management professionals and landowners to understand and manage sudden oak death (SOD) in California forests. The publication is divided into three chapters: Chapter 1 discusses the epidemiology of SOD in California and includes information on biology of the pathogen *Phytophthora ramorum*, host-pathogen interactions, disease spread, and environmental conditions that affect disease development. An understanding of these relationships is needed to choose the most appropriate strategies for managing SOD at a given location. Chapter 2 describes how to develop a plan to manage SOD within a stand and how to identify and prioritize areas that may be suitable for SOD management activities. Options for managing SOD are presented by stage in the disease epidemic: before the SOD pathogen has reached a susceptible forest; during the local epidemic, while disease is active in an area and many hosts are still at risk of becoming diseased; and after SOD has killed so many host trees that forest restoration needs to be considered. Chapter 3 provides descriptions of management techniques.

Keywords: Sudden oak death, *Phytophthora ramorum*, tree disease management, tanoak, coast live oak, mixed-evergreen forests, invasive species.

Contents

1	Chapter 1—Sudden Oak Death in California: An Overview
1	1.1—Diseases Caused by <i>Phytophthora ramorum</i>
2	Box 1-1—Taxonomic Relationships Between Susceptible Trunk Canker Hosts
3	Box 1-2—Native Foliar Hosts of <i>Phytophthora ramorum</i>
4	1.1.1—Pathogen Biology
6	Box 1-3—Host Susceptibility Versus Spore Production
7	1.1.2—Origin and Distribution of <i>Phytophthora ramorum</i> in California
8	Box 1-4—Strains of <i>Phytophthora ramorum</i>
9	1.2—Sudden Oak Death Disease Cycle in California Forests
9	1.2.1—Sudden Oak Death in Tanoak Stands
12	1.2.2—Sudden Oak Death in Oak/California Bay Forests
17	1.3—Factors That Influence Sudden Oak Death Development
17	1.3.1—Environmental Conditions
18	1.3.2—Variation in Host Susceptibility
19	1.3.3—Pathogen Dispersal
19	1.4—Sudden Oak Death Development on Trunk Canker Hosts
19	1.4.1—Symptom Development
28	1.4.2—Patterns of Disease Progression
31	1.4.3—Survival of Trees With Sudden Oak Death
32	Box 1-5—Diagnosing Sudden Oak Death
33	1.5—Stand-Level Sudden Oak Death Impacts
35	Chapter 2—Managing Stands Before, During, and After Sudden Oak Death
35	2.1—Developing a Sudden Oak Death Management Plan
35	2.1.1—Why is Management Needed?
36	Box 2-1—Costs Associated With Sudden Oak Death
36	2.1.2—Why Develop a Management Plan?
37	2.1.3—Elements of a Management Plan
38	Box 2-2—Disease, Damage, and Mortality Related to Other Agents
39	2.2—Management Strategies for Different Stages of the Sudden Oak Death Epidemic
40	Box 2-3—Time Considerations and Sudden Oak Death
40	2.2.1—Before: Reducing Sudden Oak Death Potential in Areas Not Currently Infested
44	2.2.2—During: Managing Disease in Areas Currently Affected by Sudden Oak Death
46	2.2.3—After: Dealing With Impacts of Sudden Oak Death
58	Box 2-4—Restoration in Developed Versus Wildland Settings

58	2.3 Prioritizing Areas for Sudden Oak Death Management Activities
59	2.3.1—Prioritization Categories
62	2.3.2—Developing Overall Priority Rankings
62	Box 2-5—Developing a Numeric Priority Ranking Scale
65	Chapter 3—Technical Management Guidelines
65	3.1—Excluding <i>Phytophthora ramorum</i> From Noninfested Areas
65	3.1.1—Nursery Stock
66	3.1.2—Plant Material—Pruning Waste, Wood
67	3.1.3—Firewood
68	3.1.4—Soil
68	3.1.5—Water
69	Box 3-1—Cleaning Contaminated Tools and Equipment
70	3.2—Reducing Disease Risk in Susceptible Stands
70	3.2.1—Timing of Pruning or Other Wounding
70	3.2.2—Removing California Bay
81	3.2.3—Removing Other Sporulating Hosts
82	3.2.4—Use of Chemical Protectants to Prevent Infection
84	Box 3-2—Selecting and Using Fungicides
95	3.2.5—Nonrecommended Treatments
96	3.3—Monitoring Oaks With Sudden Oak Death to Assess Survival and Failure Potential
96	3.3.1—Overview
100	3.3.2—Guidelines for Assessing Mortality and Failure Risk in Trees Affected by Sudden Oak Death
102	3.4—Assessing Fire Hazard Related to Trees Killed by Sudden Oak Death
102	3.5—Restoring Forests Affected by Sudden Oak Death
109	3.5.1—Species Selection
111	3.5.2—Factors That Constrain Regeneration
114	3.5.3—Natural Regeneration
114	3.5.4—Planting
121	3.5.5—Monitoring Restoration Outcomes
121	Acknowledgments
122	English Equivalents
122	References

Preface

This publication is intended to help resource management professionals and land-owners understand and manage sudden oak death (SOD) in California forests. The publication is divided into three chapters.

Chapter 1 discusses the epidemiology of SOD in California. This includes information on biology of the pathogen *Phytophthora ramorum*, host-pathogen interactions, disease spread, and environmental conditions that affect disease. An understanding of these relationships is needed to choose the most appropriate strategies for managing SOD at a given location.

Chapter 2 describes how to develop a plan to manage SOD within a stand. We discuss how you can identify and prioritize areas that may be suitable for SOD management activities. Options for managing SOD are presented by stage in the disease epidemic:

- Before the SOD pathogen has reached a susceptible forest.
- During the local epidemic, while disease is active in an area and many hosts are still at risk of becoming diseased.
- After SOD has killed so many host trees that forest restoration needs to be considered.

Depending on the size of the landscape, you may need to manage different stages of the epidemic in different parts of your forest.

Chapter 3 provides detailed descriptions of the management techniques that are discussed in Chapter 2.

Our goal in developing this publication was to synthesize what is currently known about SOD into a framework to inform disease management. Readers who are interested in a detailed review of the published research on SOD may consult the literature summary compiled by Kliejunas (2010), which is available at http://www.fs.fed.us/psw/publications/documents/psw_gtr234/. In addition, the California Oak Mortality Task Force (<http://www.suddenoakdeath.org>) Web site contains a comprehensive catalogue of SOD-related publications and information.

Chapter 1—Sudden Oak Death in California: An Overview

1.1—Diseases Caused by *Phytophthora ramorum*

The introduced pathogen *Phytophthora ramorum* Werres, de Cock & Man in't Veld causes a lethal trunk canker disease known as sudden oak death (SOD) (Rizzo and others 2002). Tanoak (*Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S.H. Oh), coast live oak (*Quercus agrifolia* Née), Shreve oak (*Q. parvula* Greene var. *shrevei* (C.H. Mull.) Nixon), California black oak (*Q. kelloggii* Newberry), and canyon live oak (*Q. chrysolepis* Liebm.) are native California trees that are killed by SOD (see box 1-1). In areas invaded by *P. ramorum*, SOD has quickly become the most common cause of mortality of these trees (Swiecki and Bernhardt 2010). Mortality rates are greater than 50 percent in some areas and continue to increase (Maloney and others 2005, Swiecki and Bernhardt 2008a).

Phytophthora ramorum also causes lesions on leaves, twigs, and small stems of many native species, including tanoak, California bay (*Umbellularia californica* (Hook. & Arn.) Nutt.), Pacific madrone (*Arbutus menziesii* Pursh), California huckleberry (*Vaccinium ovatum* Pursh), manzanita (*Arctostaphylos* spp.), toyon (*Heteromeles arbutifolia*) (Lindl.) M. Roem., poison oak (*Toxicodendron diversilobum* (Torr. & A. Gray) Greene), Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*), and coast redwood (*Sequoia sempervirens* (Lamb. ex D. Don) Endl.) (box 1-2). In addition, *P. ramorum* causes foliar and stem diseases in a wide variety of non-native plants, including many ornamentals (box 1-3). This wide host range contributes to the ability of *P. ramorum* to invade and become established in new areas. *Phytophthora ramorum* causes only minor damage on most of these foliar hosts.

Disease can result when *P. ramorum* comes in contact with appropriate tissues of a susceptible host. However, environmental conditions must be favorable for a sufficient length of time for infection to occur. The rest of chapter 1 describes the processes that contribute to disease development. By understanding the SOD disease process, you will be able to determine what steps can be taken to manage this disease at a particular site.

Box 1-1—Taxonomic Relationships Between Susceptible Trunk Canker Hosts

In California, *Phytophthora ramorum* is capable of killing tanoak (*Notholithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), Shreve oak (*Q. parvula* var. *shrevei*), California black oak (*Q. kelloggii*), and canyon live oak (*Q. chrysolepis*). These species are all members of the family Fagaceae.

Tanoak, native to California and southern Oregon, is found only in North America. Until recently, it was considered a member of the genus *Lithocarpus*. In 2008, tanoak was assigned to the newly created genus *Notholithocarpus* as its only member (Manos and others 2008). Under current taxonomy, all *Lithocarpus* species are native to eastern and southeastern Asia. Tanoak is unique among the trunk canker hosts in that it readily develops *P. ramorum* infections on twigs and leaves.

The true oaks, genus *Quercus*, include about 600 species of trees and shrubs. The genus has been divided into two subgenera: *Quercus* and *Cyclobalanopsis*. All North American and European oaks are in subgenus *Quercus*, as are oaks of Africa, South and Central

America, and many oaks of Asia. Oaks in subgenus *Cyclobalanopsis*, which is treated as a separate genus by some taxonomists, are native to eastern and southeastern Asia (Nixon 1993).

The subgenus *Quercus* has been divided into varying numbers of sections. Representatives of three of these sections (table 1-1) occur in California and include at least 21 oak species plus a number of natural hybrids and varieties. Oaks in the red oak section, including species that are native to the Eastern United States, are susceptible to *P. ramorum* trunk cankers. Canyon live oak, the only widespread tree species from the intermediate oaks in California, is also susceptible to lethal trunk cankers. Oaks in the white oak section have not been found infected by this pathogen in the field. Table 1-1 summarizes information on the susceptibility of California's tree species in the Fagaceae to *P. ramorum* trunk cankers. Most of California's shrub oak species occur outside of the known range of SOD and have not been tested for susceptibility.

Table 1-1—Susceptibility of California native tree species in the Fagaceae (oak family) to lethal trunk cankers caused by *Phytophthora ramorum* under field conditions

Group	Species	<i>P. ramorum</i> susceptibility
Non oaks	<i>Notholithocarpus densiflorus</i> (= <i>Lithocarpus densiflorus</i>)—tanoak	Lethal trunk cankers and twig/foiar infections
<i>Quercus</i> section	<i>Q. agrifolia</i> —coast live oak	Lethal trunk cankers ^a
Lobatae	<i>Q. kelloggii</i> —California black oak	Lethal trunk cankers ^a
red (or black) oaks	<i>Q. parvula</i> var. <i>shrevei</i> —Shreve oak	Lethal trunk cankers ^a
	<i>Q. wislizeni</i> —interior live oak	Susceptible in greenhouse tests; disease not yet observed in field
<i>Quercus</i> section	<i>Q. douglasii</i> —blue oak	Not susceptible
Quercus	<i>Q. engelmannii</i> —Engelmann oak	Not susceptible (?) ^b
white oaks	<i>Q. garryana</i> —Oregon white oak	Not susceptible ^c
	<i>Q. lobata</i> —valley oak	Not susceptible
<i>Quercus</i> section	<i>Q. cedrosensis</i> —Cedros Island oak	Unknown
Protobalanus	<i>Q. chrysolepis</i> —canyon live oak	Lethal trunk cankers ^a
intermediate oaks	<i>Q. tomentella</i> —island oak	Unknown

Note: Natural oak hybrids and shrub oak species are not listed.

^a Twig or foliar infections are occasionally observed where shoots are exposed to high spore loads from California bay (Vettraino and others 2008).

^b *Quercus engelmannii* does not occur in areas currently infested by *P. ramorum*. Based on the lack of susceptibility of other related California white oaks (*Q. douglasii*, *Q. garryana*, *Q. lobata*) under field conditions, *Q. engelmannii* is likely not susceptible.

^c *Quercus garryana* develops cankers in laboratory inoculations, but disease has not been observed in the field (Hansen and others 2005).

Box 1-2—Native Foliar Hosts of *Phytophthora ramorum*

As research on *P. ramorum* has continued, the list of plants known to be susceptible to it has expanded. An up-to-date host list is available at <http://www.suddenoakdeath.org>. Table 1-2 lists native trees, shrubs, and herbaceous plants that have been found with *P. ramorum* infections on leaves or twigs under field conditions. These hosts develop minor to significant canopy dieback, but are not killed by *P. ramorum*.

Table 1-2—California native forest species that develop *Phytophthora ramorum* leaf or twig infections (foliar hosts)

Scientific name	Common name
<i>Abies concolor</i> ^a	White fir
<i>Abies grandis</i> ^a	Grand fir
<i>Abies magnifica</i> ^a	Red fir
<i>Acer circinatum</i> ^a	Vine maple
<i>Acer macrophyllum</i>	Bigleaf maple
<i>Adiantum aleuticum</i>	Western maidenhair fern
<i>Adiantum jordanii</i>	California maidenhair fern
<i>Aesculus californica</i>	California buckeye
<i>Arbutus menziesii</i>	Pacific madrone
<i>Arctostaphylos columbiana</i> ^a	Hairy manzanita
<i>Arctostaphylos manzanita</i>	Whiteleaf manzanita
<i>Berberis diversifolia</i> (= <i>Mahonia aquifolium</i>)	Oregon-grape
<i>Calycanthus occidentalis</i> ^a	Spicebush
<i>Ceanothus thyrsiflorus</i> ^a	Blueblossom
<i>Clintonia andrewsiana</i> ^a	Andrew's clintonia bead lily
<i>Corylus cornuta</i> ^a	California hazelnut
<i>Dryopteris arguta</i> ^a	California wood fern
<i>Frangula californica</i> (= <i>Rhamnus californica</i>)	California coffeeberry
<i>Frangula purshiana</i> (= <i>Rhamnus purshiana</i>)	Cascara
<i>Fraxinus latifolia</i> ^a	Oregon ash
<i>Garrya elliptica</i> ^a	Silk tassel tree, coast silktassel
<i>Gaultheria shallon</i> ^a	Salal, Oregon wintergreen
<i>Heteromeles arbutifolia</i>	Toyon
<i>Lonicera hispidula</i>	California honeysuckle
<i>Mahonia nervosa</i> ^a	Creeping Oregon grape
<i>Maianthemum racemosum</i> (= <i>Smilacina racemosa</i>)	False Solomon's seal
<i>Osmorhiza berteroi</i> ^a	Sweet cicely
<i>Phoradendron serotinum</i> subsp. <i>macrophyllum</i> ^b	Colorado Desert mistletoe
<i>Pseudotsuga menziesii</i> var. <i>menziesii</i>	Douglas-fir

Continued on next page

Table 1-2—California native forest species that develop *Phytophthora ramorum* leaf or twig infections (foliar hosts) (continued)

Scientific name	Common name
<i>Rhododendron</i> spp.	Rhododendron
<i>Rosa gymnocarpa</i>	Wood rose
<i>Rubus spectabilis</i> ^a	Salmonberry
<i>Sequoia sempervirens</i>	Coast redwood
<i>Taxus brevifolia</i> ^a	Pacific yew
<i>Torreya californica</i> ^a	California nutmeg
<i>Toxicodendron diversilobum</i> ^a	Poison oak
<i>Trientalis latifolia</i>	Western starflower
<i>Umbellularia californica</i>	California bay, California laurel, pepperwood, Oregon myrtle
<i>Vaccinium ovatum</i>	California huckleberry
<i>Vancouveria planipetala</i> ^a	Redwood ivy, redwood insideout flower

Source: USDA APHIS 2012.

^a Plants have been found naturally infected, and *P. ramorum* has been cultured or detected using polymerase chain reaction, but Koch's postulates (formal proof of pathogenicity) have not been completed as of February 2012.

^b Riley and Chastagner 2011.

1.1.1—Pathogen Biology

Phytophthora ramorum and other *Phytophthora* species are members of the Oomycota, or water molds. Based on DNA analysis, the Oomycota are placed in the kingdom Stramenopila, which also includes diatoms and kelp. Most *Phytophthora* species attack living plants, and many are serious plant pathogens affecting forests, agricultural crops, and horticultural plants worldwide.

The Oomycota were previously grouped with the fungi based on their overall form and life habits. Like the true fungi, *Phytophthora* and related organisms are made up of a network of filaments (referred to as hyphae, or collectively as mycelium). As *P. ramorum* hyphae penetrate and grow into a host plant, they secrete chemicals that kill and degrade plant tissues. The hyphae absorb nutrients released from dead and dying host cells to fuel their continued growth.

Phytophthora ramorum is disseminated via several spore types that are produced by the hyphae. These spores can only be observed with the aid of a microscope. During periods of rainy weather, especially when temperatures are relatively warm (see section 1.3.1, "Environmental Conditions"), sporangia (fig. 1-1) are produced on the surfaces of infected leaves and twigs of some hosts. The sporangia

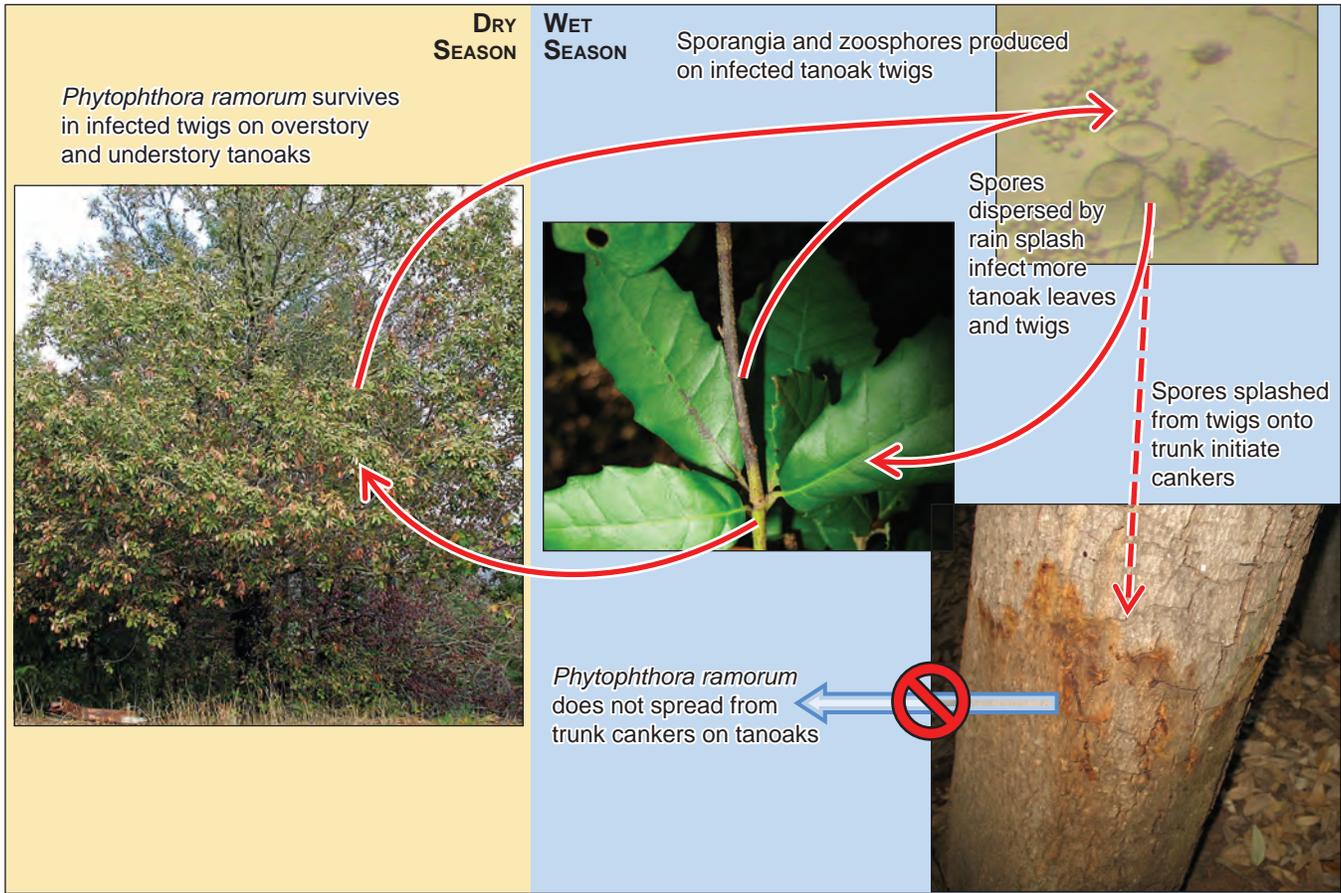


Figure 1-1—Sudden oak death disease cycle in tanoak stands. *Phytophthora ramorum* survives over the dry season primarily in tanoak twig infections. Sporulation occurs during the wet season primarily on infected twigs. Rain-splashed spores initiate new leaf and twig infections within the trees and on nearby tanoaks. Under prolonged wet conditions, high numbers of spores produced in the canopy are splashed onto trunks and initiate trunk cankers (dashed arrow).

detach readily and can be dispersed via splashing water and wind-blown droplets. Relatively few hosts support production of sporangia (see box 1-3).

Sporangia can germinate directly, producing hyphae that can penetrate the host. However, sporangia in water, including water films on plant surfaces, can instead release multiple zoospores, about 30 zoospores from each sporangium (Widmer 2009). Zoospores are motile and can swim small distances, up to about 3 cm, using their threadlike flagella (Duniway 1976). They can be carried large distances by flowing water (at least tens to hundreds of meters, depending on the type of water-course). Zoospores actively swim toward certain chemicals released by host plants, which helps them aggregate on the host. Upon reaching the host, each zoospore transforms into a nonmobile cyst that germinates by producing a single hypha that can penetrate and infect the plant. If zoospores aggregate before they encyst, host tissue may be attacked by many hyphae in a small area. This mass attack increases zoospores' ability to overwhelm host defenses.

Box 1-3—Host Susceptibility Versus Spore Production

Hosts that develop severe foliar symptoms when infected by *Phytophthora ramorum* may not support production of significant numbers of spores. Among native species, Pacific madrone is an example that shows this pattern. In contrast, some species that are not overly susceptible to *P. ramorum* support production of high numbers of spores. Known relationships between host susceptibility and spore production are shown in table 1-3.

Table 1-3—Comparison between susceptibility to *Phytophthora ramorum* foliar infection and *P. ramorum* spore production on various hosts

Amount of sporulation	Susceptibility		
	High	Moderate	Low to resistant
High	<i>Ceratonia siliqua</i> <i>Larix kaempferi</i> ^{a,b} <i>Rhamnus alaternus</i> <i>Rhododendron catawbiense</i> ^a <i>Syringa vulgaris</i> ^a <i>Umbellularia californica</i> ^a	<i>Castanea sativa</i> ^a <i>Cistus salvifolius</i> <i>Lonicera implexa</i> <i>Michelia doltsopa</i> ^a <i>Pistacia lentiscus</i> <i>Pistacia terebinthus</i> <i>Quercus ilex</i> ^a <i>Quercus cerris</i> ^a <i>Rosa sempervirens</i> <i>Vaccinium myrtillus</i>	<i>Acer pseudoplatanus</i> <i>Hedera helix</i> <i>Lonicera periclymenum</i> <i>Quercus petraea</i> <i>Quercus robur</i> <i>Rosa canina</i> <i>Rubus fruticosus</i>
Moderate	<i>Arbutus unedo</i> ^a <i>Camellia japonica</i> ^a <i>Fraxinus excelsior</i> ^a <i>Magnolia sp.</i> ^a <i>Pieris japonica</i> ^a <i>Quercus ilex</i> ^a <i>Rhododendron catawbiense</i> ^a <i>Rhododendron ponticum</i> ^a <i>Viburnum tinus</i> ^a	<i>Aesculus hippocastanum</i> <i>Corylus avellana</i> <i>Malus sylvestris</i> <i>Ulmus glabra</i> <i>Ulmus procera</i>	<i>Viburnum opulus</i> ^a
Low	<i>Arctostaphylos uva-ursi</i> <i>Kalmia latifolia</i> <i>Sambucus nigra</i>		<i>Calluna vulgaris</i> <i>Taxus bacatta</i> ^a <i>Vaccinium × intermedium</i> <i>Vaccinium vitis-idaea</i>
None			<i>Empetrum nigrum</i> <i>Pinus halepensis</i> <i>Quercus pyrenaica</i>

Source: Moralejo and others 2007.

^a Found naturally infected in the United Kingdom.

^b Added to list based on Webber and others 2010.

Zoospores are short-lived, typically swimming for a few hours. Zoospore cysts persist a short while longer, but do not tolerate drying. Sporangia can persist for days, but are relatively thin-walled and do not survive dry conditions. *Phytophthora ramorum* also produces thick-walled, long-lived spores (chlamydospores) within and on various infected plant tissues. Chlamydospores are relatively resistant to environmental degradation and help *P. ramorum* persist through periods unfavorable for growth.

Sporangia, zoospores, and chlamydospores are asexual spores. When these spores germinate, they give rise to new clones (genetically identical copies of the individual that produced the spores). *Phytophthora* species also can reproduce sexually by forming oospores. Oospores give rise to individuals that are genetically distinct from the parental strains. Oospores are thick-walled and can persist in the environment for extended periods. *Phytophthora ramorum* can only form oospores when strains of opposite mating types come in contact with each other (Werres and others 2001). Only a single mating type of the pathogen is currently widespread in California, and sexual reproduction of *P. ramorum* has not yet been observed to occur in California forests (see Box 1-4).

1.1.2—Origin and Distribution of *Phytophthora ramorum* in California

Sudden oak death was first widely recognized in California in 1995, when large numbers of tanoaks began dying in areas of Marin, Santa Cruz, and Monterey Counties (Svihra 2001). It was not until 2000 that *P. ramorum* was diagnosed as the cause of SOD (Rizzo and others 2002).

The pathogen was introduced into California on contaminated nursery stock or other plant materials, possibly in the 1980s. Genetic analyses and other data suggest that *P. ramorum* moved via infested nursery stock to multiple locations in several counties, including Santa Cruz, Marin, and Sonoma, where it spread from nursery-grown plants into the adjacent forests. Eventually, the larger forest infestations served as sources for continued spread of the pathogen to other forests (Croucher and others 2013).

Phytophthora ramorum is now found in coastal California forests from Humboldt to Monterey Counties, and in Curry County in southern Oregon. It is also found in the counties bordering San Francisco Bay. All counties with confirmed *P. ramorum* in natural settings (tabulation below) are under state and federal quarantine. Quarantined counties are subject to regulations regarding the movement and use of susceptible plants. County agricultural commissioners enforce both California and federal regulations related to this pathogen.

Box 1-4—Strains of *Phytophthora ramorum*

Phytophthora ramorum was first detected in Europe in 1993 and has been found in nursery, horticultural, and wild settings in many European countries since then. However, the strains of *P. ramorum* in Europe (EU1 and EU2), are different from the strain originally introduced into California (strain NA1) (Ivors and others 2006). By some measures, the EU1 strain is more aggressive than the NA1 strain. Another unique strain of *P. ramorum* was initially known only from nurseries in Washington state and is designated as NA2. This strain has since been found in California and nurseries in British Columbia (table 1-4). A second European strain (EU2) is present on Japanese larch (*Larix kaempferi* (Lam.) Carrière) in the United Kingdom (including Northern Ireland, southwest England, Wales, and southwest Scotland). The distribution of these four strains and other genetic data indicate that *P. ramorum* is an introduced pathogen in both North America and Europe. The native range of *P. ramorum* is unknown at this time.

In addition to the NA1 strain, both the EU1 and NA2 strains have been found in California nurseries. To date, the EU1 strain has been detected outside of a retail nursery site in Humboldt County. From a disease management standpoint, it is important to avoid widespread introduction of the European strains into California because the European and California strains are of opposite mating types (A1 and A2, respectively). The A2 mating type strain in California forests has been limited to asexual reproduction only. Introduction of the A1 mating type into California forests or nurseries could allow the pathogen to reproduce both sexually and asexually. This could introduce new variability into the *P. ramorum* population, possibly increasing the pathogen’s virulence, host range, or adaptation to a wider range of climatic conditions.

Table 1-4—Summary of currently known *Phytophthora ramorum* strains

<i>P. ramorum</i> strain	Mating type	Description
NA1	A2	Original strain introduced into California and Oregon forests and currently most widely distributed. Also common in U.S. nursery detections.
NA2	A2	Second strain detected in U.S. nurseries, originally in Washington; not closely related to NA1. Also in nurseries in British Columbia, Canada, and California. Has spread from nurseries to waterways in Washington.
EU1	A1	Strain originally found in European nurseries and in landscape introductions in Europe. Found in U.S. nurseries, especially in Washington. Has spread from nurseries to waterways in Washington, also found in a river in Humboldt County.
EU2	A1	Strain identified from Northern Ireland and western Scotland from isolates collected in 2011, with an earliest collection date in 2007. This is the second strain detected from Japanese larch in the United Kingdom.

Sexual reproduction is possible if A1 and A2 strains are crossed, but does not occur if only one mating type is present.

**California counties with confirmed *Phytophthora ramorum*
and under state and federal quarantine (September 2013)**

Alameda	Mendocino	Santa Clara
Contra Costa	Monterey	Santa Cruz
Humboldt	Napa	Solano
Lake	San Francisco	Sonoma
Marin	San Mateo	

County, state, and federal agencies in cooperation with nursery managers have made considerable efforts to eliminate *P. ramorum* from nursery plants. These efforts have greatly reduced the risk of spread of the pathogen in nursery plants. Nonetheless, contaminated nursery stock remains a potential means of pathogen spread within California as well as between states and countries.

The range of *P. ramorum* in California (fig. 1-2) continues to expand. Within infested counties, *P. ramorum* is not uniformly distributed. Stands of uninfected trees commonly occur in counties where SOD is common. The distribution of SOD-affected trees is typically patchy, even within infested stands.

1.2—Sudden Oak Death Disease Cycle in California Forests

The disease cycle of *P. ramorum* in California differs between forests containing tanoak and those containing susceptible oaks. *P. ramorum* sporangia are produced abundantly on tanoak twigs (Davidson and others 2008), so inoculum produced within tanoak canopies is sufficient to initiate trunk cankers on tanoaks (fig. 1-1). Tanoaks can also be infected by spores produced on California bay (Davidson and others 2005, 2008) where these two species occur together.

In California forests that contain SOD-susceptible true oaks, infected California bay leaves typically serve as the primary source of *P. ramorum* spores (fig. 1-3). In oak stands that also contain tanoak, susceptible oaks can be infected by spores produced on tanoak. Because SOD-susceptible oaks are normally infected by spores produced on other hosts, oaks require different disease management strategies than tanoaks.

1.2.1—Sudden Oak Death in Tanoak Stands

Phytophthora ramorum commonly infects leaves and twigs of tanoak, causing twig dieback on both mature trees and understory seedlings and saplings (fig. 1-1). *Phytophthora ramorum* sporangia form readily on tanoak twig cankers (Davidson and others 2008). They can be dispersed by splashing rain to other parts of the

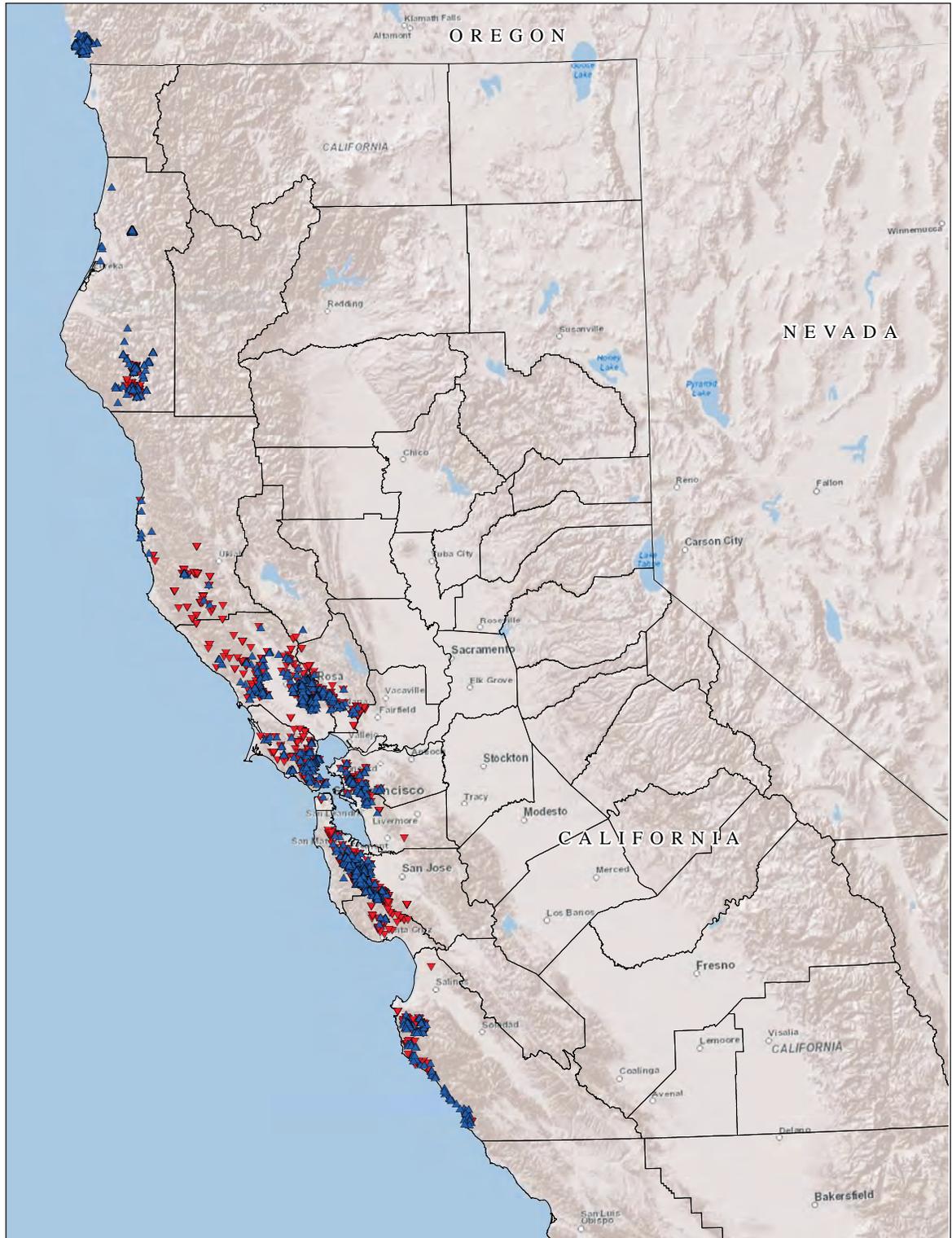


Figure 1-2—Approximate distribution of *Phytophthora ramorum* in California as of July 2012. Note Oregon location, just north of the California border. Upright blue triangles are positive plant and water samples from SODMAP (University of California–Berkeley 2012). Inverted red triangles are officially confirmed records from OakMapper (Kelly and others 2012).

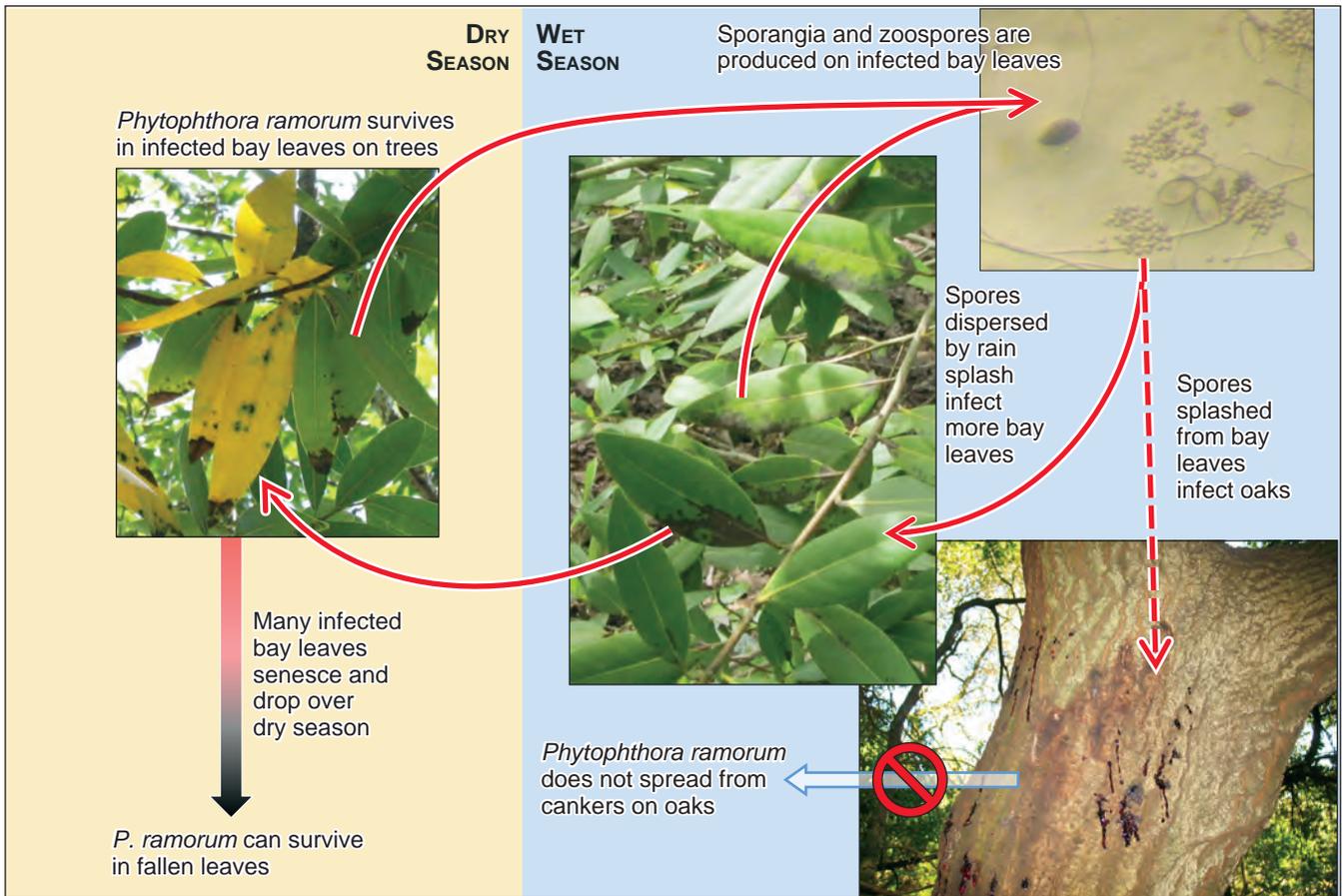


Figure 1-3—Sudden oak death disease cycle in oak-California bay forests. *Phytophthora ramorum* survives over the dry season primarily in infected California bay leaves on trees. Sporulation occurs on bay leaves during the wet season, and rain-splashed spores initiate new leaf infections on nearby California bay foliage. Under prolonged wet conditions, many spores are produced in California bay canopies and are splashed onto nearby oaks (dashed arrow). Cankers develop on infected oak trunks but *P. ramorum* does not sporulate on trunk cankers.

canopy, initiating additional leaf and twig infections. Spores can also be splashed from infected tanoak twigs to canopies of adjacent tanoaks, so *P. ramorum* can spread between trees in a tanoak stand.

Under wet conditions, as more twig cankers form in tanoak canopies, increasingly large numbers of *P. ramorum* spores are produced and dispersed. These spores splash onto tanoak trunks and can also move down the trunk in the rainwater that flows along the stems. These spores can initiate trunk cankers that can kill infected trees. Cankers on the trunk commonly form within 1 to 2 m of the ground but may occur higher on the trunk or on branches. Tanoak trunk cankers do not produce *P. ramorum* spores.

Various studies have shown that in California forests, SOD risk in tanoak is usually greater if California bay is nearby (Davidson and others 2008). When *P. ramorum* initially spreads into tanoak stands, trees located near California bay are commonly among the first to develop lethal trunk cankers. Inoculum from other host species has not been shown to be significant for causing trunk infections in tanoak stands.

1.2.2—Sudden Oak Death in Oak/California Bay Forests

In most California forests, the *P. ramorum* spores that infect susceptible oak species (see Box 1-1) are produced on the leaves of California bay (fig. 1-3) (Davidson and others 2005). However, oaks present in tanoak stands may be infected by spores produced on tanoak twigs.

Phytophthora ramorum causes dark lesions on California bay leaves, typically on downward-hanging leaf tips and leaf edges where water collects (fig. 1-3). The edge of the lesion is usually jagged rather than smooth and has a chlorotic (yellowish) border. Under wet and somewhat warm conditions (see section 1.3.1, “Environmental Conditions”), large quantities of sporangia and chlamydospores can be produced on infected leaves.

During the summer dry season, California bay leaves with *P. ramorum* infections commonly turn yellow (fig. 1-3) and drop earlier than healthy leaves. However, some infected leaves remain attached for at least a year. Consequently, *P. ramorum* can persist over the dry summer in the canopy of California bay.

Typically, the number of infected California bay leaves remaining in the canopy after the summer is relatively low, so few spores are produced during the first rains of the season in the fall and early winter. However, small numbers of spores can initiate new leaf infections on California bay. As the rainy season progresses, spores produced on infected leaves are splashed to other leaves where they start new infections. If rainfall is frequent, the number of foliar infections on California bay can increase explosively (Swiecki and Bernhardt 2008b). Peak *P. ramorum* spore production on California bay typically occurs when frequent rains occur in the spring, after temperatures have warmed up (Davidson and others 2005, 2008) (see section 1.3.1, “Environmental Conditions”).

Sudden oak death in susceptible oaks is largely a byproduct of the *P. ramorum* foliar disease cycle on California bay. When conditions are favorable for foliar disease development in California bay, large numbers of spores from infected leaves are deposited on nearby oaks by dripping and splashing water. Water running down oak stems concentrates spores in the lower trunk area, where *P. ramorum* cankers

typically develop (fig. 1-4). The lower trunk also tends to dry more slowly after rain. This provides an extended period of moisture, which favors *P. ramorum* spore germination and infection.

Sudden oak death is not known to spread from infected oaks to healthy oaks. *Phytophthora ramorum* does not produce spores on infected oak trunks; oaks are a “dead end” host for the pathogen (Davidson and others 2005, but see also section 3.1.3, “Firewood”). Leaf infections caused by *P. ramorum* rarely develop on oaks that are susceptible to trunk cankers (California black, coast live, Shreve, and canyon live oak). Oak foliar infections are only seen in trees exposed to high spore loads from nearby infected California bay foliage. Consequently, infected oak leaves appear to have little or no role in pathogen spread.

California bay–oak distance and disease risk—

Sudden oak death incidence, severity, and mortality rates increase as the distance from oak trunk to California bay foliage decreases (figs. 1-5 and 1-6) (Swiecki and Bernhardt 2007, 2008b). Oaks with California bay foliage directly over or within 1.5 m (5 ft) of the trunk have the highest risk of infection and mortality (fig. 1-5). Disease risk also increases as the total amount of California bay cover within 2.5 to 5 m (8 to 16 ft) of the oak trunk increases.

Proximity of California bay to oaks strongly influences disease risk because few *P. ramorum* spores travel more than 5 m horizontally via rain splash. Spores in water drops splashing from leaves higher in the canopy are more influenced by wind and may travel greater horizontal distances. Spores present on the soil surface and in low foliage (less than about 0.5 m above the ground) are only dispersed about 1 m by rain splash alone (Tjosvold and others 2006).

Other factors may also influence spore deposition on oaks from infected California bays (Swiecki and Bernhardt 2007, 2008b).

- **Height of California bay canopy relative to the oak.** Especially under windy conditions, average spore dispersal distance increases with foliage height. California bay canopy that is very tall or located upslope from oaks can disperse spores greater distances than low understory canopy. In dry locations with widely spaced trees, this effect may be less important because fewer leaf infections are present in the tops of California bay canopies.
- **Prevailing storm wind direction.** Because most spore dispersal occurs during rain events, greater numbers of spores are dispersed downwind. This effect is more important when the spore source is extensive and high (tall canopy or upslope) relative to the target oaks.



Figure 1-4—Bleeding *Phytophthora ramorum* canker at the base of a coast live oak.

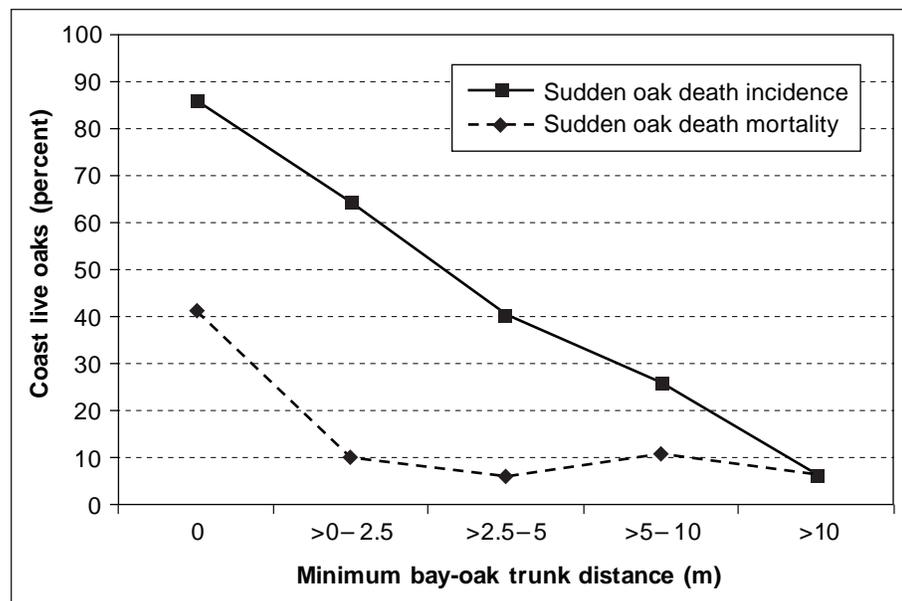


Figure 1-5—Sudden oak death incidence and mortality in coast live oak decreases as the minimum distance between the oak trunk and California bay foliage increases. Minimum California bay-oak trunk distance is the shortest distance between the main stem of an oak and a vertical line dropped from the nearest California bay foliage. Data shown were collected in 2004 from coast live oaks in Marin and Napa Counties (Swiecki and Bernhardt 2007, 2008b).



Figure 1-6—Example of minimum distance between California bay foliage and oak trunk (white arrow).

- **Oak canopy size.** Large oak canopies that have many large branches may serve to collect and concentrate *P. ramorum* spores. Large trees with dominant canopies are typically among the first trees to develop severe SOD infections.
- **Interception (screening) by nonsusceptible species.** If nonsusceptible species, especially those with dense foliage, are located between infected California bay foliage and an oak, many spores will be intercepted and fewer will deposit on the oak. Similarly, the spread of foliar infections throughout a stand containing California bay may be slowed if individual California bays are widely separated from each other by nonsusceptible species.

Other sources of inoculum—

Limited data suggest that poison oak (*Toxicodendron diversilobum*) can serve as a source of *P. ramorum* spores. Oaks that are relatively far from infected California bay (>10 m) have an elevated SOD infection risk if poison oak is climbing in their canopies (fig. 1-7). Climbing poison oak is not associated with SOD unless infected California bay is present in the area. Poison oak that occurs as a small shrub or ground cover is not associated with SOD risk in oaks (Swiecki and Bernhardt 2005, 2007, 2008b).

Other than California bay, tanoak, and possibly poison oak, none of the other known native *P. ramorum* foliar hosts (see box 1-2) have been associated with disease risk in oaks or tanoak in California forests. Infections on many of these other foliar hosts typically develop only under very wet conditions when inoculum levels from California bay and tanoak sources are very high. The small amount of additional inoculum that may be produced on these hosts is likely to have little or no effect on overall spore numbers. Many of these other foliar hosts tend to shed infected leaves readily, which further reduces their ability to produce spores. Finally, spores produced on low-growing understory species rarely move more than a meter horizontally (Tjosvold and others 2006).

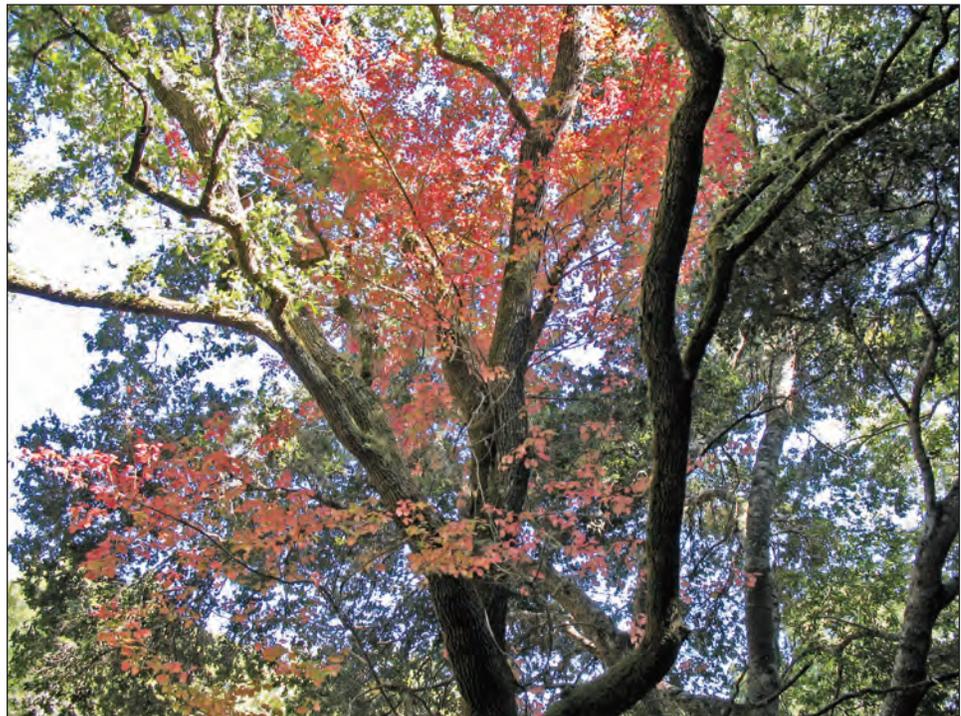


Figure 1-7—Poison oak (reddish foliage in this image taken in September) growing extensively in the canopy of susceptible oaks may serve as a source of *Phytophthora ramorum* inoculum that can initiate trunk cankers on the oak.

Rain washes *P. ramorum* spores into soil and surface water. Splash dispersal from infested soil or watercourses is only likely to transport inoculum up to a meter laterally and a half meter vertically. California bay or tanoak foliage on seedlings and low hanging branches may be infected by splashing of inoculum from soil or streams, but the small amount of spores transported in this way is unlikely to initiate trunk canker infections. Flooding of *P. ramorum*-infested streams has also been associated with the development of foliar infections on inundated hosts. Heavily infested soil or water that is directly deposited on oak trunks by human or animal vectors may initiate trunk infections, especially if the inoculum is introduced into bark wounds. This infection pathway may account for some of the uncommon trunk cankers seen in oaks without nearby California bay trees.

1.3—Factors That Influence Sudden Oak Death Development

1.3.1—Environmental Conditions

Sporangia and zoospores are the most abundant propagules produced by *P. ramorum* that can infect susceptible hosts. These propagules can only develop and disperse under wet conditions when temperatures are suitable. In laboratory tests, California *P. ramorum* isolates formed sporangia at temperatures between 10 and 30 °C (50 and 86 °F), with peak production occurring at temperatures between 16 and 22 °C (61 to 72 °F) (Davidson and others 2005, Englander and others 2006).

Like spore production, the infection process also requires wet conditions. For example, *P. ramorum* requires 6 to 12 hours of continuously wet conditions to infect California bay leaves (Garbelotto and others 2003). Even under wet conditions, infection by *P. ramorum* can be limited by low temperatures. Under laboratory conditions, wet California bay leaves were much less likely to become infected at 12 °C (54 °F) than at 18 °C (65 °F) (Garbelotto and others 2003).

The combination of relatively warm temperatures and extended periods of wetness are especially favorable for *P. ramorum* infection. Infection of oaks and tanoaks is greatest in years with frequent late spring rains, and disease potential may be especially severe when these conditions occur two or more years in a row (Swiecki and Bernhardt 2008a). In contrast, new infections are uncommon after a dry spring, especially in inland areas (Swiecki and Bernhardt 2008a). In coastal areas, fog drip can wet foliage for extended periods, so new infections can develop even in years with low rainfall.

Greater *P. ramorum* infection levels and higher spore production develop on California bay leaves in shady sites with high humidity and prolonged leaf wetness periods. Hence, California bay leaf infections are usually more severe in shaded

drainages and in stands on north- and east-facing slopes. High infection levels also occur more commonly in the lower, shaded leaves of the inner canopy. Leaves in the upper canopy, where lower humidity and more intense sunlight lead to shorter leaf wetness periods, develop fewer foliar *P. ramorum* infections (Swiecki and Bernhardt 2007, 2008b). These microclimate effects are more pronounced in drier climates away from coastal fog.

1.3.2—Variation in Host Susceptibility

Sudden oak death canker hosts differ in their relative susceptibility to *P. ramorum*. Tanoak appears to be more susceptible than the oak species (see section 1.5, “Stand-Level Sudden Oak Death Impacts”). Among oaks, coast live oak appears to be more susceptible than California black oak.

Effects of tree size and condition—

Tanoaks are susceptible to trunk cankers, foliar infections, and twig cankers over their entire lifespan. In contrast, oaks susceptible to SOD rarely develop twig infections (see section 1.2.2, “Sudden Oak Death in Oak/California Bay Forests”) and cankers generally do not develop on trunks less than 10 cm in diameter.

As oak stem diameter increases, so does bark thickness. The bark of tanoak and most susceptible oaks has a thick layer of living phloem tissue. Phloem is the primary tissue that *P. ramorum* invades when it forms trunk cankers (see section 1.4, “Sudden Oak Death Development on Trunk Canker Hosts”). In coast live oak, bark thickness is a better predictor of SOD risk than trunk diameter (Swiecki and Bernhardt 2005). Coast live oaks with thicker bark tend to be infected more readily and show faster rates of disease progress.

Trees with large canopies may also capture higher amounts of inoculum than smaller trees (see “California bay–oak distance and disease risk” in section 1.2.2). This may also contribute to the greater incidence of SOD seen in large trees.

Unlike diseases that preferentially attack stressed trees, *P. ramorum* canker is more likely to affect relatively vigorous, faster growing coast live oaks (Swiecki and Bernhardt 2005). Oaks that are growing slowly because of suppression, water stress, or severe wood decay are less likely to be infected than more dominant, healthier trees. Faster growing coast live oaks develop wide bark fissures with unweathered brown bark as a result of rapid bark expansion. New infections are often associated with these fissures (fig. 1-3), which may be preferred sites for infection by *P. ramorum*. Slow growing coast live oaks that show weathered gray color in almost all bark fissures are less likely to be infected by *P. ramorum* (Swiecki and Bernhardt 2004, 2005).

Genetic resistance—

Genetic differences between individual host plants appears to influence susceptibility to infections caused by *P. ramorum*. If sufficient levels of genetic resistance are present, it may be possible to select oaks and tanoaks with higher levels of resistance to *P. ramorum* for restoration of affected forests. Various researchers are actively investigating this area. Results from this research may have implications for long-term disease management strategies.

As noted above (“Effects of tree size and condition” in section 1.3.2), the age and physiological state of a tree can also influence its resistance to *P. ramorum*. An individual tree located in an infested forest that has no *P. ramorum* symptoms may or may not have genetic resistance to this pathogen. For instance, an individual that shows some resistance to *P. ramorum* infection under low inoculum levels might be fully susceptible under very high inoculum levels.

1.3.3—Pathogen Dispersal

Most of the short-range (mostly less than 5 to 10 m) tree-to-tree movement of inoculum within infested stands is accomplished by rain splash, often assisted by wind (Davidson and others 2005). *Phytophthora ramorum* is spread over larger distances by a number of agents, each of which is capable of acting over various spatial scales (table 1-5). Most of these long-distance transport methods can deliver only low numbers of spores to a given target. Hence, spores transported long distances may initiate foliar infections but are unlikely to be present in numbers required to initiate trunk cankers.

1.4—Sudden Oak Death Development on Trunk Canker Hosts

1.4.1—Symptom Development

Canker location—

In SOD-susceptible oaks and tanoak, *P. ramorum* infects and kills phloem tissue located in the bark of the lower trunk (fig. 1-8). Infections may kill the vascular cambium, the cell layer that produces both phloem and wood (xylem) tissues. The pathogen can also invade the outer xylem (Parke and others 2007, Rizzo and others 2002). Xylem infection appears as dark discoloration that may extend several centimeters into the wood. The pathogen can spread vertically in the xylem and sometimes grows outward from the xylem to form a new canker further up the stem. No new xylem and phloem can develop where the cambium has been killed by *P. ramorum*. However, if the pathogen becomes inactive (see below, “Changes in canker appearance over time”), healthy callus tissue can develop from uninfected live

Table 1-5—Agents that may be involved in long-range dispersal of *Phytophthora ramorum*

Agent	Effective range	Notes
Human activities ^a	Local ^b to many kilometers to global	Many mechanisms are known, ranging from transport of infested soil on shoes and tires, transport of infested plant materials (e.g., California bay leaves) via vehicles, and movement of infected nursery stock in commerce (local to international). Human activities were responsible for the original transport of <i>P. ramorum</i> to California.
Wind	Local to hundreds of meters, rarely kilometers	In some topographic situations, water drops containing sporangia may be transported tens to hundreds of meters (Turner and others 2008). Turbulent airflow may occasionally transport spores greater distances, up to several kilometers (Hansen 2008). Infected leaves or leaf fragments sent aloft by strong winds may be transported tens to perhaps hundreds of meters.
Watercourses	Local to kilometers	During the rainy season, inoculum can enter watercourses directly from overhead foliage or from surface runoff below infected foliage. Inoculum is also produced in both rainy and dry periods on infected bay leaves that fall into watercourses. <i>Phytophthora ramorum</i> has been detected in both small streams and in sizeable rivers, sometimes kilometers downstream from known inoculum sources on land (Davidson and others 2005). Inoculum in watercourses may be splashed directly onto low hanging foliage, moved via animals that visit the water, applied to soil or plants when water is pumped for irrigation, or deposited on foliage during flood events.
Other vectors	Local to kilometers (?)	Possible movement by animal vectors is very difficult to document and may occur only rarely, but could explain anomalous spread occasionally seen in local and longer range (kilometers) situations. Infested soil may be moved on feet or fur of large animals. Birds or rodents may transport inoculum on their bodies or via collected nest construction materials. Insects have not been shown to vector <i>P. ramorum</i> , but snails feeding on infested debris can excrete viable <i>P. ramorum</i> spores (Parke and others, 2008).

^a Transport by humans is well documented. Conclusive data on the other modes of long-distance dispersal are currently lacking, although observations and some data suggest that they are possible.

^b Local range is considered to be up to about 100 m.

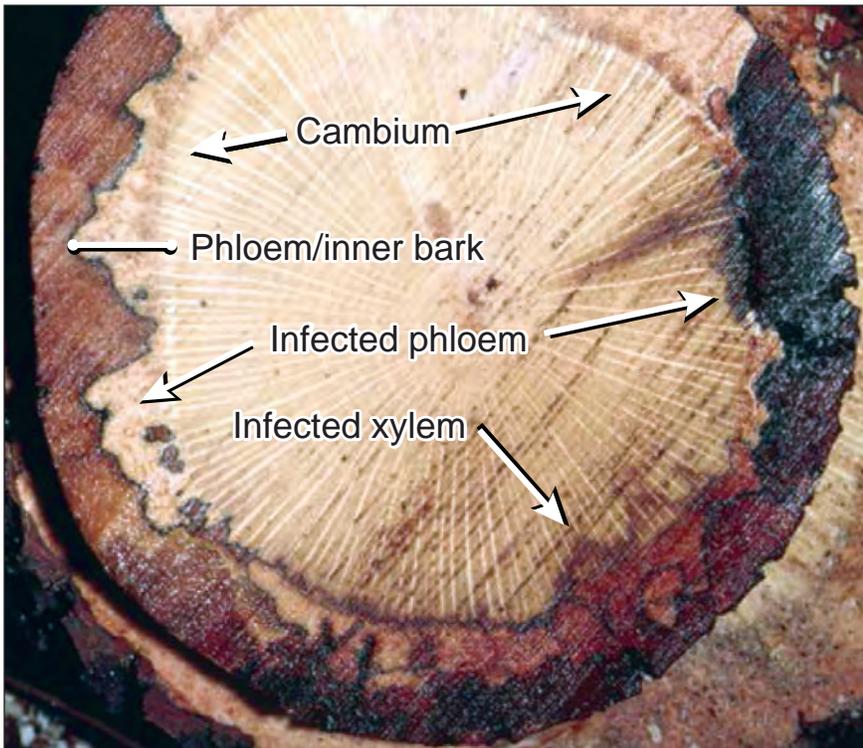


Figure 1-8—Cross section through the trunk of a coast live oak with a *Phytophthora ramorum* canker. Discolored areas in phloem and xylem are infected tissues.

cambium at the edge of the canker. This callus tissue forms new xylem and phloem tissue that grows over the nonexpanding canker.

Cankers normally occur on the lower trunk but occasionally occur on low scaffold branches. In tanoak, cankers sometimes develop many meters up on the main stems. Cankers do not extend more than a few centimeters below the soil line, but can develop on exposed buttress roots.

Early symptoms: bleeding cankers—

In SOD-affected oaks and tanoaks, new and older active cankers commonly bleed a dark, thick, sticky fluid (fig. 1-9). The fluid, which ranges from dark reddish brown to nearly black, oozes from the intact surface of infected bark tissue. The bleeding produced by SOD cankers is nearly odorless, but secondary organisms that invade the cankers can give rise to alcoholic or fermented odors. The ooze may dry and harden over the summer. Rainfall can dissolve the ooze, spreading it out, giving the canker and the area below it a “coffee-stained” appearance (fig. 1-10).

The amount of bleeding can vary greatly. Initially, small cankers may bleed from a single spot, but most large cankers will bleed from multiple areas. Visible bleeding may develop within 6 months of infection or may not develop for 2 years



Figure 1-9—Bleeding *Phytophthora ramorum* canker on coast live oak (top); outer bark has been sliced away in center image to reveal the canker, which has an irregular dark border. This canker failed to expand and became inactive, as shown in bottom photo taken 4 years later.



Figure 1-10—*Phytophthora ramorum* canker on tanoak. In right image, outer bark has been sliced away to show the irregular canker in the inner bark.

or more. Coast live oak, Shreve oak, and California black oak typically develop extensive bleeding, but some trees show little bleeding. Tanoak cankers sometimes do not bleed, especially in small diameter (<20 cm) stems. Cankers in canyon live oak usually show little or no visible bleeding.

When the outer bark is chipped away, *P. ramorum* cankers appear as brownish discolored lesions in the phloem. Cankers typically have a distinct edge, often delimited by a dark line (fig. 1-10). *Phytophthora ramorum* cankers are usually widest near the outer bark surface.

Similar symptoms—Bleeding cankers caused by *P. ramorum* are visually indistinguishable from cankers caused by *P. nemorosa* and *P. pseudosyringae*. *Phytophthora nemorosa* primarily affects tanoaks, whereas *P. pseudosyringae* is more commonly found on oaks. Both of these species appear to be less aggressive than *P. ramorum*; only *P. ramorum* is associated with extensive tree mortality (Wickland and others 2008). Table 1-6 summarizes symptoms associated with agents that cause bleeding in oaks.

Late symptoms: secondary agents—

Secondary agents, including wood-boring beetles and decay fungi, eventually invade most large *P. ramorum* cankers (fig. 1-11). These secondary agents destroy the sapwood, thereby cutting off the water supply to the tree canopy.

Beetles—Ambrosia and bark beetles (including *Monarthrum* and *Pseudopityophthorus* spp.) commonly colonize *P. ramorum* cankers on oaks and tanoaks. These small beetles often attack the stem shortly after bleeding first appears, but

Table 1-6—Common agents other than *Phytophthora ramorum* that may cause bleeding on oak trunks

Agent	Disease/condition	How to distinguish from sudden oak death (SOD) cankers
<i>Phytophthora nemorosa</i> , <i>P. pseudosyringae</i>	Bole cankers	Cankers and bleeding identical to SOD cankers, but generally less widespread. Diagnostic tests (culturing or DNA-based) are required to differentiate from SOD.
<i>Phytophthora cinnamomi</i> , other soil <i>Phytophthora</i> species	Root and crown rot	Cankers and bleeding identical to SOD cankers, but normally low on trunk and extending belowground and associated with root decay. More common in irrigated landscapes or in areas with high soil moisture.
<i>Armillaria mellea</i> (oak root fungus) and other <i>Armillaria</i> species	Root rot and wood decay in lower trunk	Bleeding may be similar but is usually very low on trunk. Affected areas have sapwood decay with white fanlike layers of fungal tissue (mycelium) between bark and wood and a mushroom odor. Decay originates in roots or root crown.
<i>Inonotus andersonii</i> , <i>I.</i> <i>dryophilus</i> , other fungi	Canker rot, wood decay	Ooze is dark but usually thinner. More likely to bleed extensively from one or few spots, often associated with wounds or cavities. Wood decay is commonly found beneath the bleeding area. Cankers are often quite small near the bark surface but become wider as they extend into the inner bark and wood.
Bacteria and associated micro-organisms	Wetwood, alcoholic flux	Ooze is usually watery and not dark; may become slimy where it accumulates on the bark surface. Usually extensive bleeding from a single spot, often associated with wounds or cracks. Ooze may have a sour/rancid smell (wetwood) or may smell yeasty or alcoholic (alcoholic flux). When area is chipped, liquid may stream out under pressure.
Wood-boring insects, including gold-spotted oak borer <i>Agrilus auroguttatus</i>	Insect boring damage	Ooze is typically thin and watery or sometimes foamy and light-colored. Bleeding comes from multiple small holes made by the insects, which may be localized or scattered across the trunk and branches.



Figure 1-11—Beetle boring dust on coast live oak. At right, detail shows small (about 1 mm diameter) exit holes of wood-boring beetles on a tree infected by *Phytophthora ramorum*.

beetle boring may not appear for a year or more after bleeding is seen. Small cankers and cankers in small-diameter oaks and tanoaks may not be attacked by beetles.

Ambrosia and bark beetles produce numerous pinhead-sized holes in the affected area as they bore through the bark (fig. 1-11). In active invasions, copious amounts of fine wood dust are ejected from the boring holes. Boring dust accumulates in bark fissures and at the base of the tree. Accumulated boring dust can wash or blow away after beetles leave the tree. At that point, the tiny beetle holes can be difficult to see in the bark, but evidence of previous beetle activity can be seen in cut or broken stems. Older cankers and branches of SOD-infected trees may be invaded by larger wood-boring beetles (commonly flat-headed borers, family Buprestidae).

Fungi—Wood decay fungi, especially *Annulohypoxylon* (formerly *Hypoxylon*) *thouarsianum* and *Phellinus gilvus* also colonize the wood beneath larger cankers. Wood decay is often quite advanced before external fruiting bodies develop.

Annulohypoxyylon thouarsianum produces hard, black, hemispheric fruiting bodies (fig. 1-12). These may develop within the first year after a canker develops, but more commonly appear later, after bark or ambrosia beetle colonization is evident. *Phellinus gilvus* fruiting bodies (fig. 1-13) typically develop in association with cankers that are at least a year old.

Other fungi can also contribute to the decline of SOD-affected trees. *Armillaria* species commonly colonize the main roots and lower trunk of trees with extensive *P. ramorum* cankers, causing decay of roots and sapwood in the lower trunk. Preexisting infections by wood-decay fungi including species of *Inonotus*, *Phellinus*, *Laetiporus*, and *Ganoderma* may expand more rapidly in trees infected by *P. ramorum*. This accelerated decay hastens tree decline and death, usually by contributing to trunk failure.

Similar symptoms—*Annulohypoxyylon thouarsianum*, *Phellinus gilvus*, ambrosia beetles, and bark beetles are opportunistic invaders that also attack trees that have been compromised by other agents. The presence of these secondary organisms alone does not provide evidence that a tree has been infected by *P. ramorum*. The other wood decay fungi noted above also occur widely in oaks and are not limited to SOD-affected trees.



Figure 1-12—*Annulohypoxyylon thouarsianum* fruiting bodies on the trunk of a coast live oak with late-stage sudden oak death symptoms.



Figure 1-13—*Phellinus gilvus* fruiting bodies on the trunk of a coast live oak with late-stage sudden oak death symptoms.

Patterns of canker development—

Sudden oak death canker development can be grouped into three general categories. Canker development is affected by the initial size of the canker and the host tree's reaction to infection.

Large cankers/susceptible reaction—Highly susceptible host trees develop large, rapidly expanding cankers. These may result from the merging of multiple cankers. Actively expanding cankers commonly show fresh bleeding around the edges. Beetles usually attack these cankers within a year of initial bleeding symptoms. *Annulohyphoxylon thouarsianum* is evident within 1 to 2 years after bleeding is seen.

As the cankers expand, beetles and *A. thouarsianum* may extend beyond the canker margins, especially higher on the stem, into areas that are weakened or killed by the canker's girdling effect.

Small cankers/resistant reaction—Many bleeding cankers (28 to 35 percent in one long-term study) become inactive within a year or two after bleeding is first seen (fig. 1-9). These cankers fail to expand and cease bleeding. They are not colonized by secondary agents and may become undetectable after one to several years. Small cankers (usually less than 10 cm across) are most likely to become inactive. Although individual cankers can become inactive, trees with inactive cankers can develop new cankers on other portions of the trunk if conditions become favorable for establishment of new infections.

Intermediate reaction—Many cankers in the moderate to large size class (>10 cm wide) are invaded by secondary organisms but eventually stop expanding and become largely inactive. Bark over the canker may become sunken and healthy callus tissue may develop at the edges of the canker (fig. 1-14). The dead bark dries out and shrinks, becoming cracked and sunken, and pieces may eventually fall off. Wood-boring insects and decay fungi may eventually degrade the wood beneath the canker. Old infections of this type are difficult to diagnose as SOD cankers.

1.4.2—Patterns of Disease Progression

Several general patterns of tree decline can be seen in oaks with *P. ramorum* cankers. Tanoaks most commonly follow the first pattern of decline (“sudden” death), but sometimes follow the other patterns.

“Sudden” oak death—

Trees develop large cankers and become mostly or entirely girdled within 1 to 2 years. Water transport to the tree canopy is disrupted by the effects of *P. ramorum* infections (Parke and others 2007) and secondary agents on the outer sapwood. This eventually induces severe water stress in the tree canopy. The canopy may appear dull or off-color just before leaves undergo a rapid change from green to brown. These color changes occur within a few weeks to months, especially during periods of warm weather. The tree canopy generally shows no obvious thinning before it turns brown and dies. The name “sudden oak death” is based on this disease pattern, but trees have typically been infected 6 months to several years before they reach this stage.



Figure 1-14—Old *Phytophthora ramorum* canker on California black oak exhibiting an intermediate disease reaction. Note callus tissue at lower right margin of canker and *Annulohyphoxylon thouarsianum* fruiting bodies on dead bark.

Slow decline—

Trees have fewer, less extensive, or slower spreading cankers. After several years, the canopy begins to progressively thin and decline (fig. 1-15). Affected trees may survive for many years, sometimes maintained by only small areas of healthy callus on a mostly girdled trunk. Death occurs either through a “sudden” drying of the remaining canopy as described above or green failure of the trunk (below). Cankers are occasionally colonized by fungi such as *Stereum* species, which primarily decay dead wood and are less aggressive than most of the wood decay fungi noted above (see “Fungi” on page 25).



Figure 1-15—This coast live oak was almost entirely girdled by *Phytophthora ramorum* cankers in 2000. Only a narrow strip of callus tissue (white arrows, top left) connected the tree's canopy to its root system for much of the following decade (top left, 2004; top right, 2009). The canopy thinned and showed significant dieback, but foliage remained green (center left, 2004; center right, 2009). Sapwood decay was extensive for many years, but the tree remained alive with green leaves until it failed in September 2010 (bottom). At the break, the only sound wood was a small strip of sapwood associated with the callused area.

Green failure—

Oaks with large trunk cankers that are extensively decayed by *A. thouarsianum*, *Phellinus gilvus*, or other fungi can fail (break) while the top is green (fig. 1-15). Failure may occur in trees with full canopies or in trees with extensive canopy thinning. Green failures associated with SOD occur more commonly in coast live oak than in tanoak. Major branch and trunk failures typically result in tree death. If failure occurs in one of several trunks or main branches and decay is extensive, the remaining live portions may decline or fail within a few years. Some failed trees resprout vigorously from the stump and may begin to form a new canopy. Factors associated with tree failure are discussed further in “Survival and failure potential of SOD-affected trees” in section 2.2.3, and section 3.3, “Monitoring Oaks With Sudden Oak Death to Assess Survival and Failure Potential.”

1.4.3—Survival of Trees With Sudden Oak Death

The extent to which cankers encircle the trunk can indicate how long a coast live oak will survive (see section 3.3.2, “Guidelines for Assessing Mortality and Failure Risk in Trees Affected by Sudden Oak Death”). Coast live oaks with early symptoms (bleeding cankers only) are unlikely to die unless they are colonized by beetles and/or *A. thouarsianum*. Most coast live oaks with advanced trunk canker symptoms (beetle boring and/or *A. thouarsianum* colonization) that affect more than half of the trunk circumference will die within 1 to 3 years. However, coast live oaks with extensive SOD cankers colonized by secondary organisms have survived for 8 or even 11 years. Trees that live for many years despite extensive canker development tend to be relatively large-diameter trees with no preexisting heartwood decay in the trunk and relatively well-balanced canopy structure. Most tanoaks that develop early *P. ramorum* canker symptoms die within 3 years. Tanoaks with extensive *P. ramorum* cankers sometimes do not develop obvious bleeding. Secondary invasion of the trunk by beetle colonization may be the first sign of disease. Consequently, the visible extent of trunk girdling by *P. ramorum* cankers is not always a reliable indicator of tanoak survival.

Both tanoaks and SOD-susceptible oaks commonly occur as multitrunked trees. If the individual trunks arise from ground level, each trunk generally functions as a separate tree with respect to infection and disease progress. It may be possible to retain healthy trunks of multistemmed trees even if one or more stems have been infected or killed by SOD. However, remaining trunk(s) should be evaluated for structural stability. Wood and root decay associated with killed trunks may compromise the remaining trunks.

Box 1-5—Diagnosing Sudden Oak Death

Within the known range of *Phytophthora ramorum* in California, a preliminary SOD diagnosis can be made based on canker symptoms and the distribution of diseased trees in the field. Key characteristics of SOD are that it:

- Primarily affects healthy, vigorous trees (see “Effects of tree size and condition” in section 1.3.2).
- Typically causes tree mortality within about 2 to 10 years after infection (see section 1.4.2, “Patterns of Disease Progression”).
- Can result in high rates of mortality, especially in tanoak (see section 1.5, “Stand-Level Sudden Oak Death Impacts”).

By comparison, most other native mortality agents primarily affect low-vigor trees; are slow acting, so that trees decline gradually over decades; and typically are associated with lower mortality rates.

Confirmation of SOD in oaks and tanoaks is based on detecting *P. ramorum* in stem cankers. Tissue samples are cut from the edge of the canker below the outer bark surface, typically at the border between healthy and symptomatic tissue. The tissue sample pieces are placed in petri plates containing a selective medium. If the pathogen is still viable in the sampled tissues, it will grow from the pieces into the medium. The resulting cultures can be identified based on characteristics seen under a microscope. Alternatively, molecular identification methods (e.g., polymerase chain reaction (PCR)) can be used to positively identify the DNA of the pathogen in the sample. These and related methods are also used to detect *P. ramorum* in other plant tissues, in soil, and in stream water.

Foliar infections caused by *P. ramorum* on California bay and tanoak may appear years before symptoms develop in trunk canker hosts. Confirmation of *P. ramorum* from California bay leaves or tanoak leaf/twig samples using selective media or PCR provides positive evidence that an area is infested with the pathogen. Many of the initial detections of *P. ramorum* within infested areas in California were made by sampling California bay foliage.

For the purpose of SOD management, you may assume that a site with typical SOD symptoms on foliar or canker hosts is infested if *P. ramorum* presence has been confirmed at nearby sites (up to several kilometers away). If your location is more than a few kilometers from the confirmed range of SOD, you may want to confirm that *P. ramorum* is present before choosing management actions (Chapter 2). In areas well beyond the known range of *P. ramorum*, especially in counties not currently listed as infested, confirmation by California Department of Food and Agriculture (CDFA) or University of California (UC) plant pathologists should be obtained. Your county agriculture department and UC Cooperative Extension offices can provide information about official sampling by CDFA and UC personnel.

1.5—Stand-Level Sudden Oak Death Impacts

Mortality rates have increased among SOD canker hosts since *P. ramorum* was introduced into California forests. Between 2000 and 2012, mortality caused by SOD among coast live oaks (1.15 percent per year) was 50 percent higher than mortality caused by all other agents (0.76 percent per year) (fig. 1-16). Among tanoaks, the SOD mortality rate (2.4 percent per year) was about 3.4 times that for all non-SOD causes (0.7 percent per year). Mortality rates measured in forests without SOD (Swiecki and Bernhardt 2008c) are similar to those attributed to non-SOD causes in figure 1-16.

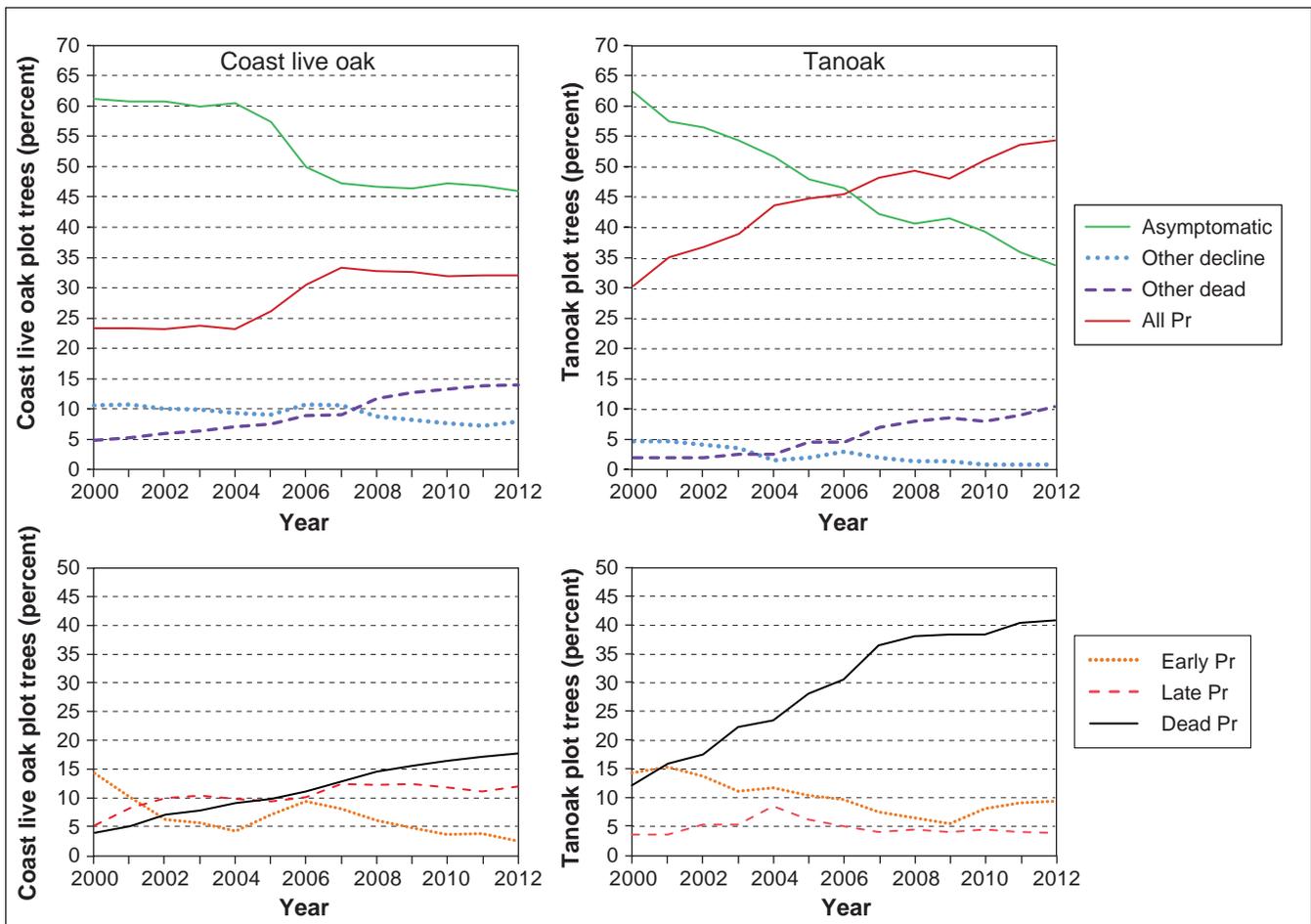


Figure 1-16—Changes in disease status of coast live oak (left) and tanoak (right) from September 2000 through September 2012 in 150 permanent plots in Marin, Sonoma, and Napa Counties. Top graphs: **Asymptomatic** = no evident symptoms of *Phytophthora ramorum* infection or decline resulting from other agents. **Other decline** = tree in severe decline due to agents other than *P. ramorum*. **Other dead** = tree dead due to agents other than *P. ramorum*. **All Pr** = all trees (live or dead) with *P. ramorum* canker symptoms. Bottom graphs: **Early Pr** = live trees with *P. ramorum* cankers only. **Late Pr** = live trees with *P. ramorum* cankers plus beetle boring or *Annulohyphoxylon thourarsianum* fruiting bodies. **Dead Pr** = tree dead due to *P. ramorum* infection.

The trajectory of SOD incidence and mortality varies from stand to stand. At a given time, different stands or portions of a single stand can be at different stages in the disease epidemic based on:

- When *P. ramorum* became established in the area.
- The number of favorable disease development years that have occurred since *P. ramorum* was introduced.
- The density and spatial arrangement of canker hosts and inoculum sources within the area.

Overall, SOD incidence and mortality tend to increase faster and reach higher levels in tanoak stands than in oak stands (fig. 1-16). In oak stands, disease is initially very patchy and is strongly associated with California bay. Even after SOD is well established, oak mortality is not likely to affect all trees in an area. SOD incidence in oak stands tends to increase in pulses over time (fig. 1-16). Large increases in SOD incidence typically develop 1 to 2 years after a wet spring that is favorable for disease development. Few or no new SOD symptoms may be seen following dry years.

If conditions for SOD development are favorable, many of the most highly susceptible trees in a stand may be infected and killed within the first few years after *P. ramorum* becomes established in an area. Subsequent pulses of disease may be less dramatic because fewer highly susceptible trees are left.

Some trees that remain healthy after the initial and subsequent waves of disease may have escaped the disease by chance. These susceptible trees can become infected later if exposed to enough inoculum during a year in which conditions are favorable for disease development. Hence, various disease management actions may be justified even in stands that have already experienced substantial disease.

Chapter 2—Managing Stands Before, During, and After Sudden Oak Death

2.1—Developing a Sudden Oak Death Management Plan

2.1.1—Why is Management Needed?

Most forests affected by sudden oak death are not managed for production of forest products. Typical land uses in affected areas include residential development, park lands, natural reserves, and watershed lands. The forests in these areas are prized for providing wildlife habitat, ecological benefits, recreational uses, or simply a desirable living environment (table 2-1).

Before the introduction of *P. ramorum* to California, SOD-susceptible oaks and tanoaks (*Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S.H. Oh) typically had low mortality rates (see section 1.5, “Stand-Level Sudden Oak Death Impacts”). Little or no active management was necessary to maintain forests dominated by these species. However, SOD has greatly changed the stability and sustainability of these forests. Landowners can no longer assume that the forest will

Table 2-1—Some ecosystem services provided by forests^a

General ecological services	Carbon fixation and storage Air quality protection Erosion protection Water quality protection Moderation of stormwater flows/flood protection Local climate modification Nutrient cycling
Plant and animal habitat	Special status species habitat Locally rare or uncommon species habitat Reserves of native biodiversity Wildlife migration corridors and refuges Special habitat features Suppression of invasive species
Human interactions	Recreational activities Health benefits Historic significance Cultural, aesthetic, spiritual values Property value enhancement Wood products Energy conservation Other forest products—edible fungi, spices Buffering between incompatible land uses Visual impact—views, screening Noise reduction

^a Although most of the general services can be provided by various forest types, the quality of habitat-related services can differ greatly between forest types.

remain stable under the largely passive management that worked in the past. Stands can undergo dramatic changes in canopy cover and vegetation composition within 10 years once SOD becomes established. Active management is needed in many SOD-affected forests to direct forest succession toward desired vegetation types and to restore lost ecosystem services.

Box 2-1—Costs Associated With Sudden Oak Death

The loss of SOD-susceptible species from forests can lead to significant losses in ecosystem services, many of which are unique to SOD-affected species. The loss of acorns provided by oaks and tanoaks affects the traditional cultures of many Native American tribes that utilize this resource. Many wildlife species also depend directly or indirectly on the annual acorn crop. In addition, many birds are dependent on the uniquely rich diversity of insects associated with oaks. Individual trees or groups of trees may also be important for erosion protection, visual screening, or property values. Hence, tree mortality can have widely different impacts in different areas.

Trees killed by SOD can also impose additional costs and risks. Undesirable plant species can invade canopy gaps created by tree mortality, increasing the need for monitoring and suppression activities. The increased failure potential of SOD-affected trees (see section 2.2.3, “After: Dealing with Impacts of Sudden Oak Death”) can put lives, property, and other resources at risk. Fuels from SOD-killed trees can also increase fire hazard. Tree removal and other actions needed to address these issues can be costly and time-consuming.

Appropriate management actions that prevent or reduce SOD can maintain valuable services provided by trees and avoid costs associated with SOD-related mortality. Hence, the costs of SOD management activities need to be considered in comparison to the costs and losses that would be incurred if no actions were taken.

2.1.2—Why Develop a Management Plan?

Resource management plans facilitate the long-term management of forest resources. There are several reasons to consider SOD management actions within the broader context of a resource management plan.

1. Landowners typically manage forest lands for multiple purposes. Planning helps mesh management efforts related to SOD with those for other purposes, thereby minimizing conflicts.
2. Forest ecosystems are complex and dynamic systems that change over time and can vary widely across the landscape. A management tactic that is effective at one time and place may be ineffective under a different scenario. Planning allows consideration of all major ecosystem components and how they are likely to change over time.

3. Management actions are often constrained by economic considerations. Planning allows limited resources to be allocated based on priorities and allows for cost comparisons among alternative management scenarios.

4. Taking no action is a management decision with consequences. Over the short term, a landowner may not incur any costs by taking no action. However, impacts and costs associated with inaction (e.g., allowing SOD to progress without intervention) could be more expensive over the long term than taking simple, well-timed management actions (see box 2-1). A plan can help you assess costs and benefits of management.

5. Trees and forests are long-lived resources whose values develop over a long time-frame, but can be damaged quickly by inappropriate management actions. Developing an adequate plan before executing actions minimizes unintended consequences that may be difficult or costly to correct.

Developing and implementing a resource management plan that addresses SOD can benefit owners of either large or small properties.

2.1.3—Elements of a Management Plan

The process of developing a management plan can be summarized as shown in figure 2-1. The process begins by addressing two basic questions with respect to the resources being considered: “What do you have?” (asses the resources and identify trends) and “What do you want?” (identify needs and set goals). These questions are interdependent and not necessarily answered in order. Landowners’ overall goals will help shape the assessment process. For example, landowners who are not interested in timber harvest would not need specific information related to wood volume.

In the next step (“How do you get what you want?”), specific strategies are selected to achieve goals and objectives. Financial and regulatory constraints on possible strategies also need to be considered. Many management actions need to be carried out over long time periods, and the order and timing of various actions may be critical. Develop a multiyear schedule to show how elements of the plan will be implemented over time.

The final step of this adaptive management process (“Are you getting what you want?”) involves monitoring and analysis. The outcome of various management actions may differ with site conditions or other factors. Because of information gaps, it may be necessary to test management actions on an experimental basis. By tracking management inputs and outcomes over time, you can determine what works and what does not, and alter future plans accordingly.

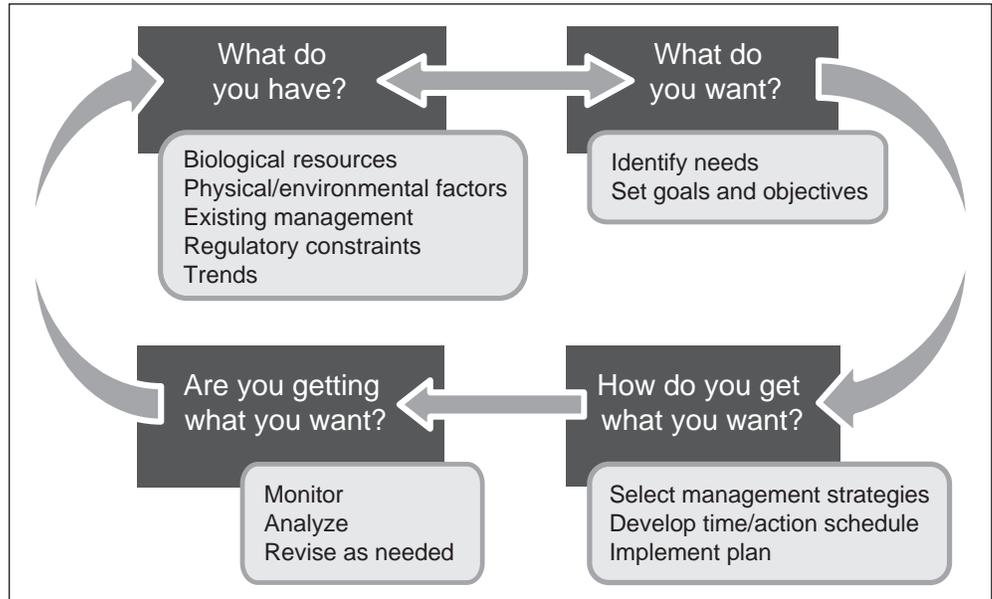


Figure 2-1—Schematic of an adaptive management process for managing forest resources.

Box 2-2—Disease, Damage, and Mortality Related to Other Agents

Since its initial appearance in the 1990s, sudden oak death has become the most important cause of mortality of tanoak and susceptible oaks in coastal California forests. However, other agents and environmental factors can cause tanoaks and oaks in these forests to decline and die. Root diseases and wood decay fungi, especially canker rot fungi, can cause relatively high levels of tree decline and mortality in forests whether SOD is present or not.

Mortality resulting from other agents may also affect trees that are not at risk from *Phytophthora ramorum*. Oaks in the white oak group (table 1-1) are not affected by SOD but are susceptible to most of the same root diseases and decay fungi that affect the SOD-susceptible red oaks. The exotic root rot pathogen *P. cinnamomi* causes decline and death of Pacific madrone (*Arbutus menziesii* Pursh) and California bay (*Umbellularia californica* (Hook. & Arn.) Nutt.) and can kill or cause root disease in a wide variety of other native species, including oaks.

A comprehensive management plan should also consider other invasive insects or diseases that are not currently present at a site but could affect forest health if introduced (e.g., the goldspotted oak borer). For example, management practices that prevent the movement of potentially contaminated materials can reduce the risk that the forest will be affected by a variety of invasive agents.

2.2—Management Strategies for Different Stages of the Sudden Oak Death Epidemic

Stand conditions change after *P. ramorum* invades. Over time, SOD affects progressively higher percentages of the canker hosts in the stand. As a result, SOD management objectives change with the SOD disease stage (table 2-2). The SOD disease stage of a given stand can be considered under the broad categories of **Before**, **During**, and **After** (table 2-2). Specific considerations for managing SOD at these epidemic stages are discussed briefly in the respective sections below.

Table 2-2—Stand characteristics, information needs, and management objectives for forests at different stages of a sudden oak death (SOD) epidemic

	Stage in SOD epidemic		
	Before: No <i>Phytophthora ramorum</i> in foliar or canker hosts	During: SOD disease levels increasing	After: most or all susceptible trees infected or dead
Stand characteristics:			
Percentage of canker hosts with SOD	0	Variable, 0 to over 50 percent	High—50 to 100 percent of at-risk hosts
<i>P. ramorum</i> present in stand	—	+	+
<i>P. ramorum</i> symptoms in foliar hosts such as bay	—	+	+
Primary management information needs:			
Proximity to known <i>P. ramorum</i>	+	—	—
Likely impact if <i>P. ramorum</i> is introduced	+	—	—
Treatment priority (see table 2-7)	+	+	+
Current distribution of SOD across site and location of noninfected canker hosts	—	+	+
SOD-related failure hazard potential	—	+	+
Fuel loading from SOD-killed trees	—	+	+
Restoration objectives	—	—	+
Site capabilities for restoration	—	—	+
Existing regeneration	—	—	+
Factors constraining restoration	—	—	+
Main management objectives: ^a			
Preventing <i>P. ramorum</i> introduction	+++	+ (other strains)	+ (other strains)
Protection of noninfected canker hosts	++	+++	— (higher if noninfected hosts still present)
Minimize <i>P. ramorum</i> inoculum production	++	+++	— (higher if noninfected hosts still present)
Minimize hazards associated with SOD-killed trees	—	+++	+++
Restoration of affected areas	—	+	+++

^a More “+” symbols indicate higher relevance/importance. “—” indicates low priority or not applicable.

The severity of SOD impacts often varies over short distances. It may be useful to divide management units into areas in which the predominant SOD disease stage is relatively uniform. Management in these different areas needs to reflect the current disease impact stage and may need to be adjusted over time as disease impacts change.

Management options differ for stands without SOD and stands that have already been affected by SOD (table 2-3). Detailed technical descriptions of the methods are in Chapter 3. The management options in table 2-3 are based on a considerable body of scientific data and practical experience. They represent the best management practices currently available. However, research into these management practices is ongoing. Check the California Oak Mortality Task Force (COMTF) Web site (<http://www.suddenoakdeath.org/>) for updates on research and management recommendations.

Box 2-3—Time Considerations and Sudden Oak Death

When planning or evaluating SOD management activities, you will need to consider if weather conditions over the previous several years have been favorable for SOD development. As discussed in Chapter 1, the likelihood that *P. ramorum* will initiate many new cankers or spread into new areas varies from year to year based on amount and timing of spring rainfall.

Sudden oak death prevention treatments are most successful when applied to trees that are not infected. Within 1 to 2 years of favorable SOD infection conditions, many infected trees will not yet have external symptoms and will appear healthy. In contrast, after several dry years with low inoculum production, most trees without symptoms are likely to be noninfected.

To evaluate the effectiveness of SOD prevention treatments, you need to monitor both treated areas and similar untreated control areas. During periods of low disease pressure, you will not be able to tell if SOD prevention treatments are working because even untreated trees are unlikely to develop symptoms. Trees need to be monitored for at least several years after one or more seasons of high disease pressure to determine whether treated trees show less disease than untreated controls.

2.2.1—Before: Reducing Sudden Oak Death Potential in Areas Not Currently Infested

In areas that are not currently infested with *P. ramorum*, the main management objective is to prevent introduction of the pathogen. The first question to address for such areas is whether *P. ramorum* could become established, given the climate and vegetation type. If it is likely that *P. ramorum* could invade the area, the next

Table 2-3—Management methods to prevent or reduce sudden oak death (SOD) disease incidence and severity before, during, and after invasion by *Phytophthora ramorum*

Methods	Targets/variants	Management objective ^a	Notes
Exclusion of infested plant material	Nursery stock	Prevent	Also important for exclusion of other soil-borne <i>Phytophthora</i> spp. such as <i>P. cinnamomi</i> .
	Leaves, plant debris	Prevent	Includes both intentional movement (e.g. green waste transport) and inadvertent transport (e.g., debris in equipment such as chippers).
	Firewood	Prevent	Freshly cut wood with attached bark poses the greatest risk.
Exclusion/sanitation	Soil	Prevent	<i>P. ramorum</i> and soil-borne <i>Phytophthora</i> species can be moved on tires, shoes, and equipment, mainly during wet conditions.
	Water	Prevent	<i>P. ramorum</i> spores are common in streams that drain infested areas.
California bay removal	Localized removal	Prevent Minimize Protect	Reduces SOD risk of individual oaks by increasing distance from infected California bay foliage.
	Area-wide removal	Prevent Minimize Protect	More efficient than localized removal if California bay are relatively small and total bay cover is low.
	California bay canopy thinning/raising	Minimize Protect	Reduce California bay foliar disease potential and inoculum production where complete bay removal is not practical.
	Targeted removal of highly susceptible California bay ^b	Minimize	Aim is to slow the seasonal epidemic by removing trees that show most disease carryover and high susceptibility. Removed California bay trees may not be near oaks.
Remove climbing poison oak		Minimize Protect	Primarily done in combination with other disease management treatments.
Avoid pruning or wounding prior to wet season		Protect	Wounds have increased risk of infection for 4 months.
Fungicide application	Individual tree treatment	Protect	Primarily used on selected individual high value trees. For tanoak stands, block treatment may be more effective. Practical levels of efficacy not yet documented in controlled field tests.
	Block treatment ^c	Minimize Protect	Preferred approach for tanoak stands to protect trees and reduce inoculum production in treated block. Practical levels of efficacy not yet documented in controlled field tests.
Removal of understory tanoak		Minimize	Primarily used in conjunction with block treatment of tanoak with phosphite fungicides.

^a Management objectives related to treatment methods:

Prevent *P. ramorum* introduction. For “during” and “after” SOD impact stages, this refers to preventing introduction of additional *P. ramorum* strains.

Minimize potential for *P. ramorum* inoculum production.

Protect noninfected SOD canker hosts.

^b Only possible for “during” and “after” SOD impact stages.

^c Block treatment refers to treating all trees within a stand or in a contiguous area of forest.

question is how soon this could happen. Answers to these questions will indicate the degree of threat posed by *P. ramorum*. As the threat level increases, more intensive precautionary measures may be justified.

Information needs—

Is the area currently free of Phytophthora ramorum?—*Phytophthora ramorum* may be present as a foliar disease on California bay, tanoak, and possibly other hosts (see section 1.2, “Sudden Oak Death Disease Cycle in California Forests”) for years before SOD cankers appear. Inspection, sampling, and testing of California bay leaves or tanoak twigs can be used to help determine whether *P. ramorum* is already established on foliar hosts in the area (see box 1-5 in chapter 1). If infections on foliar hosts are present, proceed to section 2.2.2, “During: Managing Disease in Areas Currently Affected by Sudden Oak Death.”

Phytophthora nemorosa and *P. pseudosyringae* can produce symptoms on California bay leaves and canker hosts that are nearly identical to those caused by *P. ramorum*. To determine whether such symptoms are caused by *P. ramorum*, lab testing is needed to identify the pathogen (see box 1-5 in chapter 1). Sampling should be conducted by persons that have been trained in SOD recognition and sampling protocols. The COMTF Web site (<http://suddenoakdeath.org>) provides a list of individuals who have recently completed a training session.

How far away are known Phytophthora ramorum infestations?—If a noninfested management unit is less than about 5 km from an infested area, it has a moderate to high risk of becoming infested in the short term (1 to 5 years, depending on weather). Spread of the pathogen via wind and water will be favored if prevailing storm winds blow, or watercourses drain, from infested areas toward the noninfested area. Stands that include large patches of California bay or tanoak have a higher risk of becoming infested than stands where these species are uncommon or lacking. More intensive management actions are needed to counter the disease threat in stands that have a higher risk of *P. ramorum* infestation (table 2-4).

If the noninfested unit is 5 to 10 km or more from an infested area, it has a low to moderate risk of becoming infested in the short term via natural processes. Watercourses that flow from an infested area may carry inoculum into the unit, where it could be splashed onto susceptible foliar hosts to initiate an infestation. However, human activities pose a greater risk of introducing *P. ramorum* through movement of infested plant material or soil. High risk areas include areas near roads, trails, and buildings.

Maps of *P. ramorum* distribution are available from the OakMapper (Kelly and others 2012) and SODMAP (University of California–Berkeley 2012) Web sites. As shown in figure 1-1, the two sites primarily display different sets of data, although

Table 2-4—Relative importance/usefulness of management actions to prevent new sudden oak death (SOD) infections in areas where *Phytophthora ramorum* does not currently occur

Risk of SOD ^b	Proximity to known <i>P. ramorum</i>	Relative importance of management actions ^a		
		Exclusion	Bay removal	Chemical control
Low	Distant (5 to 10+ km)	+++	—	—
	Midrange	+++	+	—
	Close (<1 km)	++	+++	++
Moderate	Distant (5 to 10+ km)	+++	+	—
	Midrange	+++	++	+
	Close (<1 km)	+	+++	++
High	Distant (5 to 10+ km)	+++	++	—
	Midrange	+++	+++	++
	Close (<1 km)	+	+++	+++

^a Relative importance of management actions: +++ = high, ++ = moderate, + = low, — = very low.

^b Risk of SOD is affected by species mix and climate conditions.

some records occur in both Web sites. At the OakMapper site, only the officially confirmed SOD records (from California Department of Food and Agriculture and other research laboratory records) should be used as indicators of *P. ramorum* distribution. Other information about the distribution of *P. ramorum* in your local area may be available from your local county agricultural commissioner and University of California Cooperative Extension offices.

What is the risk of severe disease if *Phytophthora ramorum* is introduced?—

Introduction of *P. ramorum* to an area does not guarantee that it will become established and cause disease. Disease will not develop if suitable hosts are not present or if the environmental conditions are unfavorable for pathogen survival, reproduction, and infection (see section 1.3, “Factors that Influence Sudden Oak Death Development”).

The density and distribution of California bay and tanoak are good indicators of SOD disease risk. Areas that lack both of these species are at low risk for SOD even if susceptible oak species are present and climate conditions are favorable. In oak stands, SOD risk increases as the amount of California bay in the stand increases (see section 1.2.2, “Sudden Oak Death in Oak/California Bay Forests”). Risk is also higher in stands where California bay is distributed throughout the area so that the pathogen can easily spread from tree to tree. Similarly, SOD risk in tanoak stands is greater where tanoak stands are denser and more continuous. California bay presence within tanoak stands increases overall SOD risk and is commonly associated with earlier and more rapid disease progress. However, SOD risk can be high in tanoak stands with little or no California bay.

For a given stand composition, overall SOD risk increases as the climate and microclimate become more favorable for *P. ramorum* (see section 1.3.1, “Environmental Conditions”). Disease risk is greater in areas that receive higher levels of spring rainfall and have longer wet periods because of high humidity, fog, shading, slope, or aspect. Relative disease risk is lower in drier areas, or where wet conditions occur more sporadically during a given wet season or from year to year.

Maps that predict the risk of *P. ramorum* infection based primarily on hosts and climate variables are shown in Kliejunas (2010). These models generally indicate that the greatest risk of SOD establishment is in the coastal counties from Monterey to Del Norte County. Sudden oak death risk extends eastward into the coast ranges in adjacent counties where suitable hosts are found. These models are best used to predict SOD risk at a regional scale. They may drastically over- or underpredict disease risk at the scale of a specific stand or parcel because the vegetation maps used in the models are generally not detailed enough to reflect the local scale.

Management actions—

The relative importance and usefulness of management actions in noninfested areas vary with the overall SOD threat (table 2-4). In noninfested stands close to an area where *P. ramorum* is present, monitor canker and foliar hosts at least annually for evidence of SOD. Foliar symptoms on California bay or tanoak provide an earlier indication of *P. ramorum* presence than stem cankers or SOD-killed trees. Detailed descriptions of specific management actions are discussed in Chapter 3.

2.2.2—During: Managing Disease in Areas Currently Affected by Sudden Oak Death

Once *P. ramorum* has become established in an area, the main focus of disease management changes from preventing pathogen introduction to limiting disease impacts. In *P. ramorum*-infested areas where SOD impacts are likely to continue to increase over time (the **During** phase), typical management goals are:

1. Minimize *P. ramorum* infection and mortality of SOD canker hosts.
2. Minimize hazards associated with SOD-affected trees.

Management considerations related to the first goal are described in this section; those related to the second goal are described in the **After** section below. Management emphasis may shift from the first to the second goal as the number of SOD-infected trees increases.

Information needs—

What is the incidence of SOD in the affected stand?—More SOD management options are available when disease incidence is still low than when most at-risk trees

have become infected. Only some of the at-risk trees typically become infected during each favorable infection period; little or no new disease may develop in dry years (fig. 1-16). Management actions implemented during lulls in the epidemic can protect asymptomatic trees from becoming infected during later disease pulses.

When assessing SOD impact within stands, note both the presence of cankers and the severity of the infection (percentage of the trunk circumference that is affected, presence of secondary organisms). Sudden oak death may go into remission (see section 1.4.1, “Patterns of Canker Development”) in some oaks that only have one to several small cankers. Such trees may be candidates for future disease suppression activities. The amount of trunk girdling and the presence of secondary invaders (beetles or *Annulohyphoxylon thouarsianum*) also affects the tree’s likelihood of dying or failing. Also, remember to take past weather conditions into account when making SOD assessments (see Box 2-3) to avoid underestimating infection levels.

Management actions—

The main methods for minimizing *P. ramorum* infection are listed in table 2-4 and are described in Chapter 3. Many of these management methods work best when combined in an integrated fashion. For example, chemicals used to protect trees from infection will be more effective if measures are also taken to reduce local inoculum levels.

Selecting individual trees for treatment—Some SOD management treatments are implemented on a tree-by-tree basis. To minimize costs, you may want to limit the most intensive treatments to high value trees that have a high infection risk (table 2-5).

Table 2-5—Factors associated with increased sudden oak death (SOD) risk for individual trees

Category	Factors associated with higher risk
Favorable microclimate conditions for infection	North- or east-facing slopes, shaded drainages, high canopy cover, fog drip
Amount of <i>Phytophthora ramorum</i> spores to which the tree is exposed	Little or no clearance from bay, high local bay or tanoak density, extensive poison oak climbing through canopy ^a
Host tree size and condition	Larger stem diameter, greater bark thickness ^a , high tree vigor ^a , actively expanding (brown) bark fissures ^a , low water stress ^a

^a Correlated with SOD risk for coast live oak.

Monitoring treatment efficacy—By assessing disease symptoms in treated and nontreated areas on an annual basis, you can determine how disease is progressing at a location, how well treatments are working, and whether additional actions may be needed. Select individual trees to monitor so that trends over time can be tracked. Trees can be identified by using permanent tree tags in combination with geographic coordinates or other spatial information.

2.2.3—After: Dealing With Impacts of Sudden Oak Death

As long as noninfected *P. ramorum* canker hosts are present, disease prevention activities of the “During” phase (table 2-3) may still be appropriate. However, once a high proportion of canker hosts have died, management emphasis shifts toward dealing with the aftermath of SOD. This section discusses three main management issues:

1. Survival and failure potential of SOD-affected oaks and tanoaks.
2. Fire hazard and fuel loading due to SOD-induced tree mortality.
3. Restoration of SOD-affected forests.

Survival and failure potential of SOD-affected trees—

Removing SOD-affected oaks has no effect on disease spread in the stand because the pathogen does not sporulate on oaks; they are a “dead end” for the disease. Determining whether or when to remove SOD-affected oaks should be based on management criteria such as potential hazard, cost, and wildlife habitat value.

Information needs—How long will SOD-affected trees survive? Trees with *P. ramorum* canker can follow a wide range of survival trajectories over time (see section 1.4.2, “Patterns of Disease Progression” and section 1.4.3, “Survival of Trees With Sudden Oak Death”). This variation in tolerance can make it more difficult to decide if and when to remove symptomatic trees. Factors correlated with survival are summarized in table 3-3.

What is the failure potential of SOD-affected trees? Oaks and tanoaks with late symptoms of *P. ramorum* canker have an increased risk of failure (breakage) compared to healthy, noninfected trees (table 2-6). Because SOD cankers typically affect the lower trunk, decay associated with SOD cankers often leads to trunk failure. Trees infected by *P. ramorum* often die as the result of large scaffold or trunk failures before the SOD canker actually causes death of the top (fig. 1-15).

Table 2-6—Average annual failure rates (percentage of trees in disease category that failed each year) by disease category among 630 coast live oaks observed over a 10-year period in Marin and Napa County forests affected by sudden oak death (SOD)

Disease category	Average annual failure rate (percent)
Dead from SOD	42
Dead from other causes	20
Late SOD symptoms (beetles or <i>Annulohypoxyton thouarsianum</i> present)	11
Early SOD (bleeding cankers only)	0.4
Healthy; not SOD-infected	0.4

Trees with *P. ramorum* canker have an increased risk of failure because of two main factors:

- Death of the entire tree or large portions of it.
- Increased decay, often in combination with extensive beetle boring.

Dead trees and dead parts of live trees have an elevated likelihood of failure whether or not they are affected by SOD (table 2-6). In SOD-killed trees, small diameter branches and trunks typically decay fairly rapidly and often become heavily colonized by wood-boring insects. If trunk failure does not occur first, branches and scaffolds commonly begin to fail within 1 to 2 years after a tree is killed by SOD (fig. 2-2). Remove dead trees or branches that pose a significant risk to people or property as soon as possible.

Many oaks and tanoaks with *P. ramorum* trunk cankers develop an extensive white rot of the sapwood caused by the decay fungus *A. thouarsianum*. Cankered areas and killed portions of the canopy are typically affected first. Other decay fungi, including *Phellinus gilvus*, may also be present and cause similar decay. In small-diameter trees (less than about 30 cm), sapwood decay associated with these secondary decay fungi can be extensive enough to cause trunk failure (fig. 2-2). Such failures typically occur within one to several years after the top has died.

In larger trees, trunk failure commonly results from a combination of SOD-related sapwood decay and preexisting heartwood decay (fig. 2-3). Many large oaks have extensive internal decay associated with canker rot fungi (especially *Inonotus andersonii*, *I. dryophilus*, and *Phellinus robustus*) or other aggressive wood decay fungi such as *Laetiporus gilbertsonii*. A small amount of SOD-related sapwood decay may trigger failure if it extends to wood already affected by heartwood decay

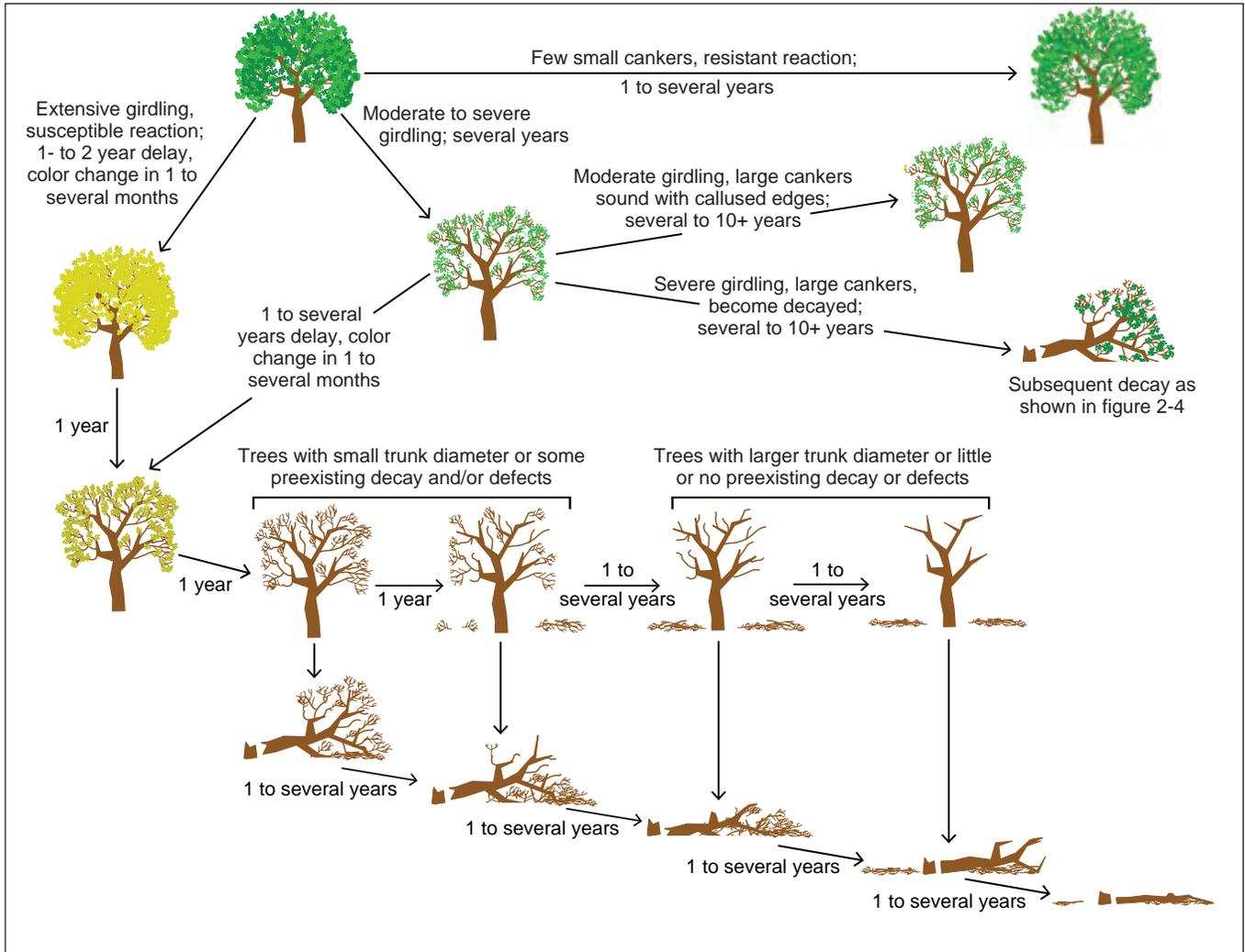


Figure 2-2—Possible paths of sudden oak death progress and eventual tree degradation in coast live oaks that have little or no preexisting wood decay in the trunk. Such trees typically do not fail before the top is killed, and snags may stand for a number of years before the trunk fails. Green tree at upper left represents a newly infected coast live oak with only bleeding cankers.



Figure 2-3—Failure of a large coast live oak killed by *Phytophthora ramorum*. Trunk failure was associated with preexisting heartwood decay and a cavity in the center of the trunk. Fruiting bodies of *Annulohypoxyylon thouarsianum* and *Phellinus gilvus* seen on the bark are associated with sapwood decay that developed after *Phytophthora ramorum* infection.

(fig. 2-3). Failures of live SOD-infected trees (fig. 2-4) commonly develop where sapwood decay approaches or merges with existing heartwood decay. In contrast, large-diameter oaks that are killed by SOD but lack preexisting heartwood decay may stand for 5 to 10 years or more before trunk failure occurs (fig. 2-2).

Management actions—If SOD-affected trees are located where the failed stems may impact persons or property, periodic monitoring is needed to identify and mitigate potential hazards well before failure is imminent. Hazards may be reduced by pruning or removing trees or by keeping people and property outside of the potential impact area (see section 3.3, “Monitoring Oaks With Sudden Oak Death to Assess Survival and Failure Potential”). In areas with many potentially hazardous trees, permanent or temporary closures and posting may be used to reduce the chance that likely impact areas will be occupied when tree failures occur. Trees that have not been affected by SOD can also have a high risk of failure because of decay or structural problems (fig. 2-5), so hazard assessments need to consider all potentially hazardous trees, not only those with SOD.

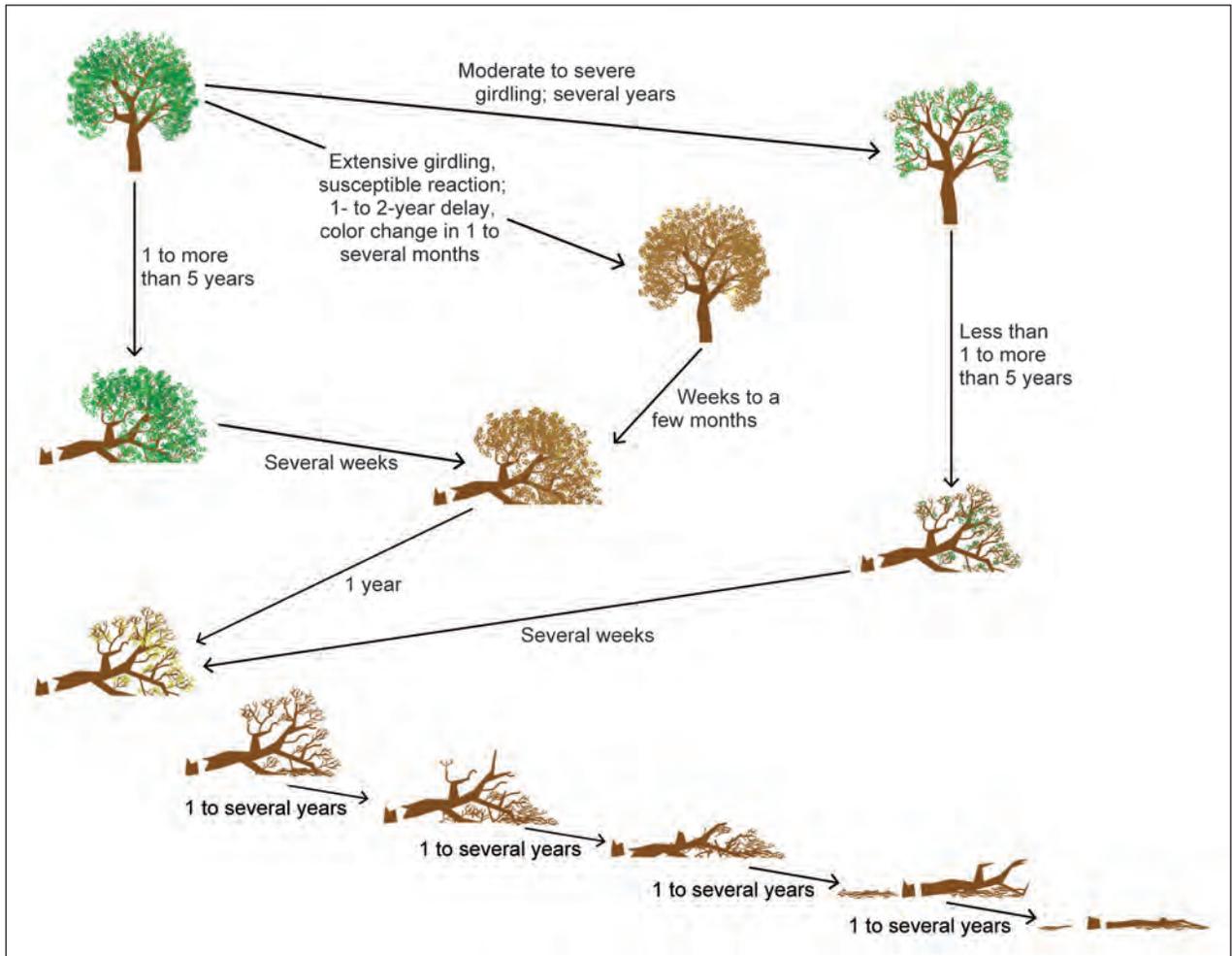


Figure 2-4—Possible paths of sudden oak death disease progress and eventual tree degradation in coast live oaks that fail while the top is still live (green failure) or within a short time after rapid death of the canopy. Trees following this pattern typically have large amounts of preexisting wood decay in the trunk or other severe structural defects, and are extensively colonized by secondary decay fungi and beetles. Green tree at upper left represents a newly infected tree with bleeding cankers only.



Figure 2-5—The trunk of this coast live oak failed because of extensive internal decay. Although the tree was in a location where sudden oak death is common, this tree was not infected with *Phytophthora ramorum*.

In areas where many trees are affected by SOD, it may be necessary to prioritize and phase removals. Trees that are likely to impact persons, structures, utility lines, or access roads typically have a high priority for removal or mitigation. Trees that are likely to fail before the next inspection period should be targeted as the highest priorities. The criteria in tables 3-2, 3-3, and 3-4 (in section 3.3) can be used to help identify trees that are more likely to die or fail in the near future.

SOD-related mortality and fire—

Tree mortality resulting from SOD has the potential to affect fire hazard and behavior by changing the types and distribution of fuels in the forest. Many other factors unrelated to SOD can also influence fuel distribution and fire hazard in forests.

In many stands, the change in fuel distribution from a few scattered SOD-killed trees will not significantly alter fire behavior in the stand. At the other end of the spectrum, high levels of SOD-related mortality seen in some tanoak stands can greatly alter fire hazard for a number of years. Impacts of SOD need to be considered within the context of the entire landscape to determine how much they change fire hazard and which, if any, management actions may be needed.

The presence of SOD-killed trees in a stand can affect fuel amount and distribution as follows:

- During the dry season, dead oaks and tanoaks have low moisture content and are more likely to burn than living trees.
- Recently killed trees have an abundance of dead leaves and twigs. These dry fine fuels can ignite readily. During a fire, they can generate or receive embers that cause spotting (ignitions beyond the fire front).
- Dead leaves and branches from SOD-killed trees can increase the total amount and depth of surface fuels.
- If mortality is common and widespread, dead leaves and branches can form areas of continuous surface fuels in stands that would otherwise have a much more patchy surface fuel distribution.
- Piles of woody debris from dead trees and leaning failed trees can form fuel ladders between surface and aerial fuels.
- SOD-killed trees can contribute a large amount of large-diameter dead woody material to the surface fuel layer. Firefighting operations are complicated when large logs ignite because they burn for extended periods.

Because live trees with late SOD symptoms and standing dead trees have an increased likelihood of failure, these trees can pose a hazard to firefighting personnel during fire suppression and mop-up operations.

Information needs—What kinds of fuels have SOD-killed trees generated?

Fuels generated by SOD-killed trees differ depending on tree species, size, and the length of time between infection and tree death (figs. 2-2 and 2-4). Furthermore, the fuels from killed trees are not static but degrade over time. Once woody materials and leaves decay to the point that they have lost their structural integrity, they typically do not affect fire potential.

Trees killed by SOD begin to degrade before they are dead as they are attacked by beetles and wood decay fungi. Decay and degradation of dead materials proceed faster in wetter sites (high rainfall, frequent fog, deep shade). Degradation patterns and timelines differ between standing dead trees and material that has fallen to the ground because materials on the ground remain moist longer (figs. 2-2 and 2-4). A photo series showing typical fuels associated with decaying SOD-killed coast live oaks is shown in section 3.4, “Assessing Fire Hazards Related to Trees Killed by Sudden Oak Death,” tables 3-5, 3-6, and 3-7. The series is based on data collected primarily in Marin County from 2000 through 2011.

How much additional fuel is present and how is it distributed? Tree mortality caused by SOD affects forests in ways that can alter fire behavior. Sudden oak death can alter the fuel loading and distribution in a forest. Fire intensity and rate of spread depend on the total amount of fuels present, typically expressed as tons of fuel per acre. Higher fuel loads increase the amount of heat that can be produced during a fire. The horizontal and vertical distribution of fuel also affects fire behavior. Fire will spread more uniformly and reliably if a continuous fuel layer is present. Spread will be slower and more unpredictable if fuels are patchy in distribution. Flame length is affected by the height of the fuel bed. Tall piles of debris and dead material that lean up against standing trees can serve as ladder fuels, allowing ground fires to move into the canopy.

Canopy gaps created by SOD-killed overstory trees can open the forest floor to more wind and light. This creates hotter and drier conditions that can increase the rate at which fuels dry as the season progresses. In addition, higher windspeeds at the forest floor can increase the rate of fire spread.

Light penetration into canopy gaps promotes the growth of understory vegetation. Gaps may be invaded by annual grasses and forbs that dry out to form fine fuels that ignite easily. Canopy gaps also support greater growth of native or invasive shrubs, which can further change fuel types and fuel height distribution. Tree regeneration, especially Douglas-fir seedlings and saplings, can also become dense in canopy gaps, forming ladder fuels. Eventually, competition causes dense Douglas-fir clumps to develop extensive lower canopy dieback and mortality, resulting in a more hazardous fuel distribution than existed in the stand prior to SOD.

Management actions—Dead trees: cut or leave? Defensible space typically extends 30.5 m (100 feet) from buildings. Within this zone, trees, shrubs, and other fuel sources should be managed to make it difficult for fire to transfer from one fuel source to another (State Board of Forestry and Fire Protection, California Department of Forestry and Fire Protection, 2006). Within defensible space fuel management areas, SOD-affected trees should be removed as soon as the foliage starts to turn brown. Data from several studies indicate that the water content of foliage of SOD-affected trees does not change appreciably until the tree changes color.

In more extensive forest areas beyond defensible space envelopes, scattered tree mortality occurring over many years will probably not significantly alter fuel distributions at the landscape level. In such sites, leaving trees to degrade and decay in place is often the best option. Where large patches of trees have died within a relatively short period (1 to 3 years), problematic levels of readily ignitable fuels will be present on the ground and in the canopy for at least several years. Canopy

fire hazard can be reduced by felling standing dead trees that still have many dead leaves and fine twigs. Once standing dead trees have lost their leaves and fine twigs, they do not serve as significant aerial fuels. Cutting dead trees at this stage will reduce the potential for tree failure and reduce the fuel ladders that develop when failed trees hang up on adjacent standing trees.

If dead trees are cut to manage fuels, it will be necessary to treat the biomass from these trees to avoid creating excessive amounts of surface fuel. Treatment options include:

- Removal to an acceptable disposal area in accordance with state and federal regulations.
- Piling and burning in accordance with local fire and air quality regulations.
- Chipping, with chips preferably spread out in a thin layer and left onsite.
- Lopping and scattering debris to hasten degradation.

Wood from SOD-killed trees may be used as firewood on the same property (see below, section 3.1.3, “Firewood”), but should not be moved beyond the local area. State and federal regulations generally prohibit the movement of wood from susceptible oaks and tanoak from SOD-infested counties to noninfested counties.

Managing vegetation in canopy gaps. You may need to actively manage vegetation that recruits in canopy gaps to keep hazardous fuel conditions from developing, especially within defensible space. Annual and perennial exotic species that invade canopy gaps should be eradicated or suppressed. It may also be necessary to prune, thin, or selectively remove native species, such as dense Douglas-fir regeneration.

Restoration in SOD-affected forests—

Many of the forests now affected by SOD were previously well stocked with little mortality, so restoration activities were largely unneeded. As SOD-related mortality increases, a landowner or manager may wonder what, if any, actions are needed to restore forest canopy cover. You can identify appropriate options for forest restoration based on stand conditions and management goals by using the adaptive management process discussed above (fig. 2-1).

Information needs—Is stand restoration needed based on the current or projected levels of mortality from SOD and other factors? Although tree mortality from SOD will cause a loss in tree density (trees per unit area), mortality does not always reduce overall canopy cover. If a forest has overlapping tree canopies, scattered tree mortality may not create large canopy gaps. Small canopy gaps may be quickly erased by growth of overstory or understory trees.

Some tree mortality in a dense stand may not be detrimental. Reduced tree density may reduce competition for moisture or light within the stand and improve growing conditions for remaining trees. In contrast, mortality of dominant trees in relatively open stands will typically cause significant changes in canopy cover and forest structure. As canopy cover loss from SOD increases, valuable ecosystem services (table 2-1) are likely to be reduced or lost.

Even small losses of canopy cover can have disproportionately large impacts if they affect critical areas or involve unique resources. For example, acorns produced by oaks and tanoaks can contribute significantly to wildlife habitat value, even if these trees make up a small percentage of the stand. When evaluating the need for restoration in SOD-affected forests, consider both effects on overall canopy cover and ecosystem services that are not strictly related to canopy cover loss.

Assess the size and distribution of canopy gaps created by tree mortality and failure. Canopy gaps represent opportunities for natural or assisted regeneration. Canopy gaps can also provide habitat elements that are absent in closed-canopy forests, which can benefit some wildlife species and increase overall biodiversity.

As noted above (see box 2-2) *P. ramorum* canker is not the only mortality agent that needs to be considered when assessing restoration needs. If other mortality agents are active in SOD-affected forests, mortality can affect a wider range of species and a greater percentage of the total forest canopy. This can further complicate prospects for restoring a functional forest.

Which species are regenerating at the site? Most SOD-affected forests contain understory regeneration (seedlings and saplings) of various tree species. Established understory seedlings and saplings in SOD-caused canopy gaps can be an important source of regeneration. When released from the suppressive effects of overstory competition, these persistent seedlings and saplings commonly show rapid growth (fig. 2-6). Understory regeneration may include both current canopy species and trees not found in the overstory.

In contrast, seedlings of canopy species may be rare or lacking in the understory. For example, California black oak seedlings are often rare under their parent trees (Swiecki and Bernhardt 2008d). Seedlings of coast live oak differ widely in number from year to year and between locations (Swiecki and Bernhardt 2008a).

Gaps may also be colonized by species that do not establish well under canopy. Both shade-intolerant native species and many weedy exotic species may be favored by canopy gaps. Weedy species may suppress natural regeneration of preferred native species by competing for soil moisture and other resources. If aggressive exotics such as broom species (*Cytisus*, *Genista*, *Spartium* spp.) are present in or



Figure 2-6—Dense populations of Douglas-fir seedlings have established in a canopy gap that developed when overstory coast live oaks were killed by sudden oak death in this Marin County site. Douglas-fir overstory was not previously a component of this stand. Douglas-fir can easily outcompete slower growing hardwood species in canopy gaps.

near the site (fig. 2-7), action may be needed to suppress exotics and favor desired native species.

Management actions—After reviewing your information about tree mortality and natural regeneration, your next steps are setting goals and selecting actions for restoring forest structure and function. If SOD impacts are limited, you may not need to take any actions to maintain overall stand conditions. In other situations, you may need to undertake modest interventions in a timely fashion to achieve desired stand conditions. In the most severely affected stands, significant management inputs may be needed to restore lost ecosystem services or prevent undesirable changes in the stand.

Specific management actions needed to achieve restoration goals will vary widely depending on forest types, site conditions, landowner resources and capabilities, and other factors (see below, section 3.5, “Restoring Sudden Oak Death-Affected Forests” and box 2-4). The key to cost-effective restoration is determining what factors are likely to constrain regeneration at the site. Restoration inputs can then be selected to specifically address these site constraints. Monitoring the



Figure 2-7—French broom (*Genista monspessulana*), an invasive exotic shrub, has become well established in the understory of this coast live oak stand. Canopy thinning (seen in coast live oak, center) and tree mortality caused by sudden oak death has favored French broom invasion by reducing competition for light and soil moisture.

results of your restoration efforts will provide additional site-specific data needed to improve and optimize your methods (see below, section 3.5.5, “Monitoring Restoration Outcomes”).

A mosaic of restoration approaches may be applied in different areas and implementation may be phased over a period of many years. Risks associated with unfavorable weather (e.g., drought) can be minimized if the implementation schedule is flexible. Timelines for projects with third-party funding may have less flexibility in the implementation schedule.

Box 2-4—Restoration in Developed Versus Wildland Settings

Developed areas and wildland settings provide distinctly different opportunities and constraints with respect to restoration. In general, more intensive inputs such as irrigation and attention to individual trees may be warranted in developed areas (e.g., near home sites) but are often impractical or unnecessary for wildlands.

Developed areas at the edge of wildlands (the wildland-urban interface) are especially complex. Although it is possible to manage these areas intensively, tree management has the potential to significantly affect ecological processes in the adjacent wildlands. For example, planting nursery stock can introduce soil pathogens into native stands. Planting can also introduce nonlocal genotypes that may alter the genetics of the locally adapted gene pool (see “Genetic considerations” in section 3.5.4). The surrounding forest also has the potential to influence plantings in the interface area. For example, browsing by deer or other wildlife species may damage plantings. In general, plantings in the interface area should follow guidelines established for restoration in wildlands to minimize potential adverse impacts.

2.3 Prioritizing Areas for Sudden Oak Death Management Activities

On small properties with relatively few trees, it may be feasible to reduce disease risk for every tree. On large parcels, economic or logistic constraints will limit the extent to which management actions can be applied. Hence, a key element of the planning process involves setting priorities. Limited resources can be used most effectively by determining:

- **Where:** Which areas, if any, should be treated?
- **How:** What types of treatments should be used?
- **When:** Which areas should be treated first?

A matrix for prioritizing areas for SOD management activities is shown in table 2-7. The matrix provides a framework for prioritizing candidate areas based on the following main categories.

1. **Management opportunities:** Which areas can be treated most easily and effectively?
2. **Disease risk:** What is the likelihood of severe SOD impacts in the area?
3. **Loss of resource benefits:** Which resource benefits, services, and values will be affected by SOD-related tree mortality?
4. **Hazards:** What detrimental or undesirable conditions will develop if the stand is affected by SOD?

You will obtain the greatest benefit per unit cost by focusing on the most important resources that are likely to be affected and are relatively easy to treat.

2.3.1—Prioritization Categories

Management opportunities—

You can readily identify low-priority sites by determining which areas are not feasible to treat. These include areas that are so steep or inaccessible that treatments would be excessively difficult and expensive. Areas that are not practical to treat can be removed from further consideration.

Among sites that could be treated, the ease of treatment and likelihood of obtaining acceptable levels of disease control will be affected by the factors shown in table 2-7. By assessing these factors, you should be able to rank potential sites based on overall SOD management opportunities.

Disease risk—

Another way to prioritize sites for treatment is to assess factors that influence SOD risk. If the risk that SOD will develop in a given stand is small, that stand will have a very low priority for SOD management activities. Disease risk may change over time as conditions change, so priority rankings based on disease risk need to be reevaluated periodically.

Loss of resource benefits—

Sites with at-risk SOD canker hosts can be prioritized by considering the various ecosystem services they provide. Sites that provide the most important benefits and values have the highest priority. Table 2-7 lists a number of potential factors to consider. To develop a list that matches your management objectives and interests, add or delete items from table 2-7 and adjust the definitions of the high- and low-priority categories. Weights applied to various factors can also be shifted.

Table 2-7—Prioritization matrix: factors to consider when prioritizing stands for sudden oak death (SOD) management activities

Category	Factors	High priority	Low priority
Management opportunities	Ease of access for treatment (slope, roads, etc.)	Near level Adjacent to roads	Excessively steep No road access
	Position of treatment site	Site is discrete unit; little or no likely contribution of inoculum from adjacent areas beyond treated zone	Site is adjacent to areas that cannot be treated
	SOD impact stage	No or low percentage of trees with cankers or SOD mortality	High percentage of trees dead or with severe SOD symptoms
	Relative cost of treatment	Low cost per unit treated	High cost per unit treated
	Overall likelihood of success	Likely to be successful because of favorable site or logistical issues	Low probability of success because of difficult site or logistical issues
	California bay presence/density/size class	California bays mostly understory saplings and small trees	California bays are large overstory trees
	California bay continuity	California bays are low percentage of total tree cover	California bays are majority of tree cover
	California bay density	California bay distribution is discontinuous in local area or could be made discontinuous with limited tree removal	Continuous California bay distribution over large area, little or no possibility of creating significant gaps in distribution
	Proximity to known <i>Phytophthora ramorum</i> infestations	Low to moderate density in overstory or understory <i>P. ramorum</i> present in stand	None present within stand
	Risk of pathogen introduction (if not present)	High number of visitors and vehicles	Nearest known <i>P. ramorum</i> at least 10 to 20 km (6 to 12 mi) away Infrequently used site
Disease risk	Climate/microclimate conditions	Wet—high rainfall, foggy, north-facing slope, dense tree canopy	Dry—low rainfall, fog uncommon, south-facing slope, open canopy
	Tree variables	Large, dominant, vigorous trees with thick, expanding bark (bark fissures with brown, unweathered tissue)	Small, overtopped/suppressed, or water-stressed trees with thin bark, slow bark expansion (bark fissures weathered and grayish)

Table 2-7—Prioritization matrix: factors to consider when prioritizing stands for sudden oak death (SOD) management activities (continued)

Category	Factors	High priority	Low priority	
Resource benefits	Stand condition without SOD	Sustainable stand; trees mostly fair to good condition	Stand declining due to other factors	
	Stand uniqueness	Stand unusual because of species composition, age structure	Widespread and common stand type	
	Historical/cultural value	Significant trees or stand	No special significance	
	Aesthetic/amenity/visual values	Significant scenic resource	Stand not within a viewshed	
	Habitat value		Strongly contributes to property value	Low or no specific contribution to property value
			Serves as visual buffer (e.g., screening, privacy)	No visual buffer effects
			Essential or preferred habitat of rare or special status species	Unsuitable habitat for rare or special status species; none present
			Important habitat for other locally significant or desirable species	Poor habitat quality for species of interest
			Special habitat features/areas present (e.g., riparian, wetland, old growth, unique soils)	No special habitat features
			High native species biodiversity	Low native biodiversity (e.g., highly disturbed site)
SOD-related hazards	Soil stability and water quality	Important corridor or connection between different habitats	Does not function as corridor (e.g., isolated forest fragment)	
		SOD-susceptible species in critical location providing slope stability/erosion protection	SOD-susceptible species not important contributors to soil stability at site	
		SOD-susceptible species present along watercourse and providing important bank stabilization	SOD-susceptible species not near watercourse or do not contribute to bank stability	
		Failures would affect occupied structures	Not near structures	
		Failures would affect heavily used roads or trails	Not near roads or trails	
		Failures would affect other built infrastructure (utility lines, drainage structures, etc.)	Not near infrastructure	
	Contribution to fire hazard	Defensible space fire hazard significantly increased by fuels associated with dead standing or down trees	SOD-related mortality poses little or no change to overall fire hazard within or near defensible space	
	Conversion to exotic-dominated or other undesirable vegetation type		Highly susceptible to invasion by undesirable exotics	Invasion by exotics unlikely
			Gaps likely to be converted to undesirable vegetation type	No undesirable changes likely because of species mix and small or no gaps

Hazards—

The presence of many SOD-killed trees on a landscape may pose a number of hazards. Consider the degree to which you can avoid these hazards by preventing or minimizing SOD-related mortality when prioritizing sites for treatment.

2.3.2—Developing Overall Priority Rankings

Sites can be prioritized by comparing their potential management opportunities, loss of resource benefits, SOD risk, and hazards. If you have a limited number of sites, ranking them may only require a quick mental review of these factors. If you have many sites, it may be helpful to use a numeric rating system to develop priority scores for each site. You can then compare sites based on their priority scores. Priority scores for areas can also be displayed on a map for further consideration. It may be more efficient to treat several adjacent sites rather than scattered sites with similar priority scores. A method for creating numeric ratings from the matrix in table 2-7 is described in box 2-5.

Box 2-5—Developing a Numeric Priority Ranking Scale

The prioritization matrix (table 2-7) can be used to develop numeric rankings to aid in comparing a large number of sites. Start by assigning a number to each factor based on the priority rating (e.g., high priority = 3, moderate priority = 2, low priority = 1, not applicable = 0). If you want to weight some factors more than others, multiply the priority rating by a weight factor. For example, a weight factor of 2 doubles the importance of a factor; weighted scores will range from 0 to 6 (table 2-8).

If you simply sum the ranks for each category (with or without weighting), the categories with more factors to consider (e.g., resource values) will have a greater influence on the total score. You can correct for this by calculating the percentage of the maximum score for each category. First, determine the maximum score possible for each category. If you use weighted ratings, sum the maximum weighted scores. Then, divide the sum of ranks for each site by the maximum score for the category. You can multiply this number by 100 to convert it to a percentage.

The final step involves combining the four category scores to obtain an overall priority score. Averaging the four scores is the simplest method. Alternatively, the four scores (expressed as decimals) can be multiplied. This latter method yields a wider spread of values, which may be useful if many sites have similar ratings. Either method yields values that can be expressed as percentages of the maximum possible rating (table 2-9).

When you convert your observations and evaluations to numbers, you typically lose some information. Numeric priority rankings can readily separate the most and least promising sites, but more detailed review of candidate sites may be needed to rank sites with similar numeric ratings.

continues

Box 2-5—Developing a Numeric Priority Ranking Scale (continued)

Table 2-8—Example showing use of prioritization matrix to create numeric priority rankings

Factor	Subfactor	Weight	Priority rating		Weighted rating		Maximum weighted score
			Site 1	Site 2	Site 1	Site 2	
Management opportunities:							
Ease of access for treatment	Slope conditions	2	2	3	4	6	6
	Adjacent to roads	2	3	3	6	6	6
Position of treatment area		1	2	3	2	3	3
Relative cost of treatment		1	2	2	2	2	3
Overall likelihood of success		1	3	3	3	3	3
Existing sudden oak death incidence		1	3	3	3	3	3
California bay presence/density/size classes	Bay mostly understory saplings and small trees	2	3	3	6	6	6
	Bays sparse	2	3	3	6	6	6

Note: Scoring for part of the “Management opportunities” category is shown for two example sites.

Table 2-9—Category totals and overall scores calculated using the averaging method for this example

	Unweighted ratings		Weighted ratings		Maximum weighted score	Percentage of maximum	
	Site 1	Site 2	Site 1	Site 2		Site 1	Site 2
Management opportunities	21	23	32	35	36	89	97
Disease risk	10	10	10	10	12	83	83
Resource benefits	7	23	7	29	42.5	23	58
Hazards	6	8	8.5	10	25.5	33	40
Totals/average percentage of maximum	44	64	57.5	84	116	50	72

Note: Total scores using the multiplication method would be 6 percent for site 1 and 19 percent for site 2. In this example, site 2 has a higher overall priority because of higher ratings in the management opportunity, resource benefits, and hazards categories.

When considering large tracts of at-risk forests, land managers may feel that nothing can be done that will have a significant impact and that SOD should just be allowed to “run its course.” However, costs that will be incurred from SOD-related mortality could outweigh the costs of targeted SOD management activities. Prioritization helps you break down a seemingly impossible task—protecting extensive forest resources from SOD—into viable projects that can be addressed in a logical order.

Chapter 3—Technical Management Guidelines

This chapter provides technical information about (1) the sudden oak death (SOD) disease management methods listed in table 2-3; (2) monitoring and assessing hazards of SOD-affected trees; and (3) restoration of SOD-affected forests. Additional information related to SOD management, including Best Management Practices for various user groups developed by the California Oak Mortality Task Force, is available at <http://www.suddenoakdeath.org>.

3.1—Excluding *Phytophthora ramorum* From Noninfested Areas

Exclusion is the preferred management tactic in susceptible forest types where SOD has not been detected. Exclusion is most likely to be effective if the site is relatively distant from existing infestations and if exclusion can be implemented over a large area, such as an entire watershed or isolated stand. Exclusion is a worthwhile strategy for small parcels, but may be less effective if the land manager cannot exclude *P. ramorum* from adjacent parcels.

Practices related to sanitation and exclusion can also have an important role in *P. ramorum*-infested stands. The distribution of *P. ramorum* can be spotty in the early stages of a local SOD outbreak. Practices that avoid transporting the pathogen throughout the site may slow the rate of *P. ramorum* spread and provide a longer time for implementing control treatments. Sanitation practices also help prevent the spread of *P. ramorum* from an infested location to other noninfested locations.

Exclusion and sanitation measures are also important for preventing the introduction of additional strains of *P. ramorum*. Additional strains introduced via nursery stock or from other forested locations may be more aggressive or better adapted to the site than the existing strain (see box 1-4 in chapter 1). In addition, exclusion and sanitation can reduce the chances that additional exotic pests and pathogens would be introduced into the stand (see box 2-2 in chapter 2).

Phytophthora ramorum may be inadvertently introduced into forests in a variety of ways. Methods for preventing *P. ramorum* introductions are discussed below.

3.1.1—Nursery Stock

Phytophthora ramorum was originally introduced into California forests through movement of contaminated nursery stock. Since that time, federal and state regulations and inspections have been implemented to prevent the movement of *P. ramorum*-infested nursery stock. Nonetheless, contaminated nursery stock remains a pathway through which *P. ramorum* and other exotic *Phytophthora* species can be introduced to a site.

Nursery detections have been made in California and other states in every year since monitoring for *P. ramorum* was initiated. It is difficult to consistently detect nursery stock that is infested with *P. ramorum*, in large part, because:

- A wide variety of species are potential hosts.
- Symptoms in most hosts (small leaf spots) are not distinctive.
- Asymptomatic infections occur in some hosts.
- Fungicide treatment in the nursery can suppress symptom development without eliminating the pathogen.

Nursery-grown plants that are most commonly infested with *P. ramorum* include species and varieties of *Camellia*, *Rhododendron*, *Pieris*, and *Viburnum*. These species in particular should not be planted adjacent to forests containing SOD-susceptible native species. Also avoid using species that are moderately to highly susceptible to *P. ramorum* and support moderate to high levels of sporulation (table 1-3).

Given the potential risks posed by *P. ramorum* and other pathogenic *Phytophthora* species, planting nursery stock in or near native forests should be avoided in general. Nursery-grown plants should not be used where spores produced on the plants could be dispersed into native stands or where contaminated soil or water runoff from the planted area can reach native stands.

3.1.2—Plant Material—Pruning Waste, Wood

Phytophthora ramorum can be transported by moving infected leaves from California bays (*Umbellularia californica* (Hook. & Arn.) Nutt.). Infected leaves can fall into vehicles (truck beds, the area below the windshield) and be moved to new locations. Infected California bay leaves dropped on the ground may produce *P. ramorum* spores that could be splashed to low leaves of susceptible species and initiate a new infestation.

Collect and safely dispose of any hitchhiking leaves discovered in a vehicle. Dispose of potentially contaminated leaves by burning or by placing the leaves in a sealed bag that will be sent to a landfill. Do not add them to a green waste recycling container.

Chippers and trucks used in pruning operations involving SOD hosts, especially California bay or tanoak (*Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S.H. Oh), should not be moved between infested and noninfested areas without being thoroughly cleaned. Clean trucks, chippers, chain saws, and any tools that may contain infested plant fragments before leaving the job site so infested material is not moved to the next work location (see box 3-1).

3.1.3—Firewood

Although cankers on intact oak and tanoak trees do not appear to produce sporangia under normal field conditions, chlamydospores can be formed within infested wood (Parke and others 2007). Under wet conditions, sporangia can be produced on the cut surfaces of wood from trees with *P. ramorum* cankers (Davidson and Shaw 2003, Davidson and others 2008). Therefore, movement of firewood cut from SOD-infested trees poses a risk of transporting *P. ramorum* to noninfested areas. State and federal regulations prohibit movement of firewood cut from SOD hosts out of quarantined infested areas.

Wood from living or very recently killed trees has the greatest risk of being a source of *P. ramorum* spores. Wood from SOD-killed trees that have been dead for many years is unlikely to be a source of viable *P. ramorum* spores. For tanoak, wood of any size has the potential to be infested. For oaks, only the lower portion of the trunk of such trees are likely to have *P. ramorum* cankers. Small-diameter wood from the upper part of the oak canopy is rarely infested.

Storing wood under dry conditions will prevent *P. ramorum* from producing sporangia on firewood. Rapid drying of cut wood also reduces reproduction of various wood-boring beetles. Split and stack firewood in well-aerated piles in a dry, sunny location away from other host trees until it is thoroughly dry.

Wood can also be solarized for several months to speed wood drying and limit beetle reproduction. Cover woodpiles with a clear (not black) UV-resistant polyethylene tarp, 0.15 to 0.25 mm (6 to 10 mil) thick, that is sealed to the ground at the edges. The greenhouse effect created under the clear tarp heats the wood and speeds drying. Heating will be greatest in sunny locations under the long days from late spring through fall. Stacks should be no more than 0.6 to 0.9 m (2 to 3 ft) tall to achieve uniform heating. Angle the sides of the tarp outward so that condensation that forms on the inside of the tarp will drain onto the soil away from the wood.

Burning wood from SOD-affected trees is an effective way to dispose of infested or potentially infested wood because burning destroys *P. ramorum* in the wood. There is no evidence that smoke or embers from infested wood can transport viable *P. ramorum* inoculum.

If firewood is cut from either host or non-host trees within an infested area, *P. ramorum* can also be transported via infested California bay leaves, tanoak leaves and twigs, and soil that may be mixed in with the wood. Do not allow leaves and soil to be moved in or on firewood, equipment, or truck beds. Soil movement can be minimized by cutting and loading firewood when soils are dry. Leaves and twigs of California bay and tanoak, soil, and other potentially contaminated materials that

are accidentally moved to a new location should be collected. This material should be burned or sealed in a bag and sent to a landfill for disposal.

3.1.4—Soil

Moist soil on hiking boots and bicycle tires has been shown to spread SOD.

Vehicles driven on dirt roads that pass through lands infested with *P. ramorum* may also spread contaminated soil, especially when conditions are muddy. Footwear and vehicles should be cleaned before leaving infested areas and before entering new areas (see box 3-1). Dry soil poses a low risk for spreading SOD because dry soil is less apt to stick to surfaces and the amount of viable *P. ramorum* inoculum on the surface of dry soil is very low.

3.1.5—Water

Phytophthora ramorum can survive, and appears to reproduce, in streams and rivers. Watercourses that drain SOD-affected watersheds can contain spores of *P. ramorum*. More spores are typically present in watercourses during the wet season, but spores may be present in some streams year round. Untreated water from potentially infested streams should not be used for irrigation, dust control on roads, or similar purposes. Water can be treated with ultrafiltration, chemicals (chlorine, ozone), or UV radiation to eliminate *Phytophthora* spores.

Box 3-1—Cleaning Contaminated Tools and Equipment

If possible, clean tools, equipment, shoes, vehicles, and other items that may have become contaminated with *P. ramorum* inoculum in soil or debris before leaving the infested location. If cleaning is done at a site that is already infested, inoculum can be brushed or washed from the contaminated surfaces; the contaminated material does not need to be collected and destroyed. Cleaning should be done on a surface that is unlikely to allow cleaned materials to become recontaminated, such as pavement, a plastic tarp, or a continuous layer of gravel.

Simply spraying disinfectants on contaminated surfaces is not effective for killing *P. ramorum* inoculum that is present in large pieces of plant tissue or thick layers of soil. Thorough cleaning of debris and soil from equipment, tires, and shoes is necessary to remove *P. ramorum* propagules (spores or mycelium). Compressed air can be used to help blow debris and soil out of tools and equipment such as chain saws and chippers. Debris and soil can also be brushed or swept from surfaces. Surfaces with clinging mud or damp debris may need to be washed off. Use a brush and clean, uncontaminated water to thoroughly remove soil from surfaces. An appropriate cleaning agent can be used after removing large particles of debris and soil to provide a higher level of protection. Detergent solutions, diluted bleach, and ethyl or isopropyl alcohol (70 percent or higher) can facilitate cleaning of contaminated surfaces and may directly kill exposed spores. Some formulated products, such as Lysol® Disinfectant Spray (79 percent ethyl alcohol), can be used for this purpose. Allow wet surfaces to dry thoroughly to further reduce the chance that any *P. ramorum* propagules will survive.

Because wash water from vehicles may contain heavy metals, hydrocarbons, and other materials, vehicle wash stations should be configured to prevent these residues from contaminating surface or ground water. Contaminated wash water should be contained so it can be treated or disposed of in compliance with local and state regulations.

If it is not possible to completely clean items such as shoes or hand tools at the infested area, these items may be placed in plastic bags for later cleaning at an indoor site such as a utility sink. It is important to make sure that all potentially contaminated material is washed down into the sanitary sewer system (not into stormwater drains) or is sealed in bags that will be sent to a landfill.

Disinfecting with diluted bleach. Standard commercial bleach (5.25 percent sodium hypochlorite) diluted to 10 percent strength (one part bleach, nine parts water) may be used as a disinfectant for hard surfaces. For concentrated (“ultra”) bleach (6.15 percent sodium hypochlorite, EPA Reg. No. 67619-8), the corresponding dilution ratio is about 1 part bleach to 10.5 parts water. Equipment to be disinfected needs to be maintained in contact with the bleach solution for at least a minute to kill *P. ramorum* propagules. Longer contact times and more thorough soaking will be needed if large pieces of debris are present. Use freshly made bleach solutions and change them once they become contaminated. Bleach solutions lose potency over time and are deactivated with use.

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Concentrated bleach and hypochlorite solutions are corrosive and need to be handled with care, using both eye and skin protection. Bleach solutions can also react with various chemicals to release toxic chlorine gas and other hazardous chlorinated gases. Bleach solutions can corrode steel, and are not advisable for equipment such as chain saws.

3.2—Reducing Disease Risk in Susceptible Stands

Once *P. ramorum* has become established in an area, only a few management practices can reduce disease risk to susceptible oaks and tanoak. These practices are primarily preventative, and can also be used in at-risk stands in which the pathogen has not yet been detected. Large-scale field testing of these techniques is still ongoing, so the level of protection provided by these practices under various field conditions is still unknown.

3.2.1—Timing of Pruning or Other Wounding

Although *P. ramorum* readily attacks intact bark, large pruning wounds have an elevated risk of infection for about 4 months (Lee and others 2010). Other wounds that expose inner bark or wood would likely show a similar pattern.

These results suggest that SOD risk may be increased if major pruning or other operations that cause bark wounds are conducted when *P. ramorum* spores are present. To minimize this risk factor, avoid these activities during the rainy season. Complete pruning or activities that cause wounding by the end of summer or early fall if possible.

Many native canker rot and other wood decay fungi that colonize pruning wounds also sporulate during the rainy season. Pruning during the dry season minimizes the risk that pruning wounds will be infected by wood decay fungi.

3.2.2—Removing California Bay

Oak forests—

Removing small understory California bays to reduce SOD risk to canker hosts can be justified in most stands. However, a thorough analysis of stand conditions and management objectives is needed before contemplating extensive removal of overstory California bays. For example, if oaks are already infected but have not yet developed visible symptoms (see box 2-3 in chapter 2), oak mortality could develop after California bay overstory trees are removed. If California bay is the only other canopy tree in the area, the combined canopy loss from SOD-related mortality and California bay removal could be excessive.

Many cities and counties have ordinances that regulate the removal of trees above a given diameter, especially native species such as California bay. Check with your local planning department before considering removal of large-diameter California bay trees.

Before stage—In areas that are not yet infested with *P. ramorum*, reducing or eliminating California bay canopy near oaks can reduce the overall likelihood that the stand will become infested with *P. ramorum*. If the uninfested stand is far from known *P. ramorum* sources, focus initial California bay removal efforts on understory seedlings and saplings and low branches of larger trees. California bay foliage near the ground has the highest risk of being infected by *P. ramorum* spores that may have been introduced via infested soil, plant debris, or water. If SOD-infested areas are close enough that *P. ramorum* spores may reach the area via long-range spore dispersal (table 2-4), more intensive localized or area-wide California bay removal efforts may be justified.

During stage—Once *P. ramorum* is established in an area, reducing or eliminating California bay canopy near susceptible oaks can reduce the amount of inoculum that will be splashed onto the oaks, reducing disease risk. California bay removal can be used alone or in combination with preventive chemical applications to further reduce SOD risk.

Tanoak forests—

Tanoaks near California bays are commonly the first in a stand to become infected and killed by SOD. Hence, California bay removal may be beneficial for delaying SOD establishment in noninfested stands. Once *P. ramorum* is introduced into a tanoak stand, removing California bay near tanoaks may slow the local epidemic. However, California bay removal alone will not eliminate risk in tanoak stands. California bay removal in tanoak stands will help most in the following situations:

- Early in the local SOD outbreak.
- Stands where California bay is a relatively minor component.
- When used in combination with other management activities, such as chemical application.

California bay removal strategies—

California bay removal may be used across a range of spatial scales, from large blocks to small localized patches. Even in areas where extensive removal to protect oaks may not be practical, it may be helpful to remove highly susceptible California bays and those that tend to retain many infected leaves for extended periods. Across a property or landscape, one or more of these strategies may be applied in different

areas, depending on the size and density of California bays and oaks present as well as management goals and priorities.

Methods discussed here are based primarily on relationships that have been observed between SOD risk and California bay in nonmanipulated coast live oak (*Quercus agrifolia* Née) stands. Long-term controlled studies involving California bay removal are under way, but experimental data are not yet available to show to what degree SOD can be prevented by using California bay removal.

Area-wide California bay removal—Area-wide removal involves eliminating all California bay from a relatively large contiguous area containing SOD-susceptible oaks. This method provides the greatest reduction in SOD risk and is preferable where it is practical and consistent with other management objectives. Removing all California bays within an oak stand can be more efficient than localized clearing around individual trees because larger California bays often increase the SOD risk for several nearby oaks. Candidate sites for area-wide California bay removal may encompass an entire stand or only portions of a stand. If the treated area is smaller than about 0.25 ha (0.5 acre), refer to the discussion of localized California bay removal below.

Area-wide removal of California bay is most likely to be feasible and cost effective in the following types of stands:

- Stands in which California bay is a minor component or is limited to localized areas.
- Stands in which most or all of the California bay present consists of seedlings, saplings, or small trees.
- Stands in which California bay removal is consistent with other management goals (e.g., eliminating ladder fuels for fire risk management).

Area-wide removal will be difficult, impractical, or undesirable in stands in which California bay is the dominant canopy species or where much of the California bay canopy consists of large trees.

Remove all California bay size classes, from seedlings to mature trees, from within the treated area if possible. Over the short term, small California bay seedlings (less than 0.5 m tall) that are more than 3 m from a susceptible oak trunk can be left in place. Remove these seedlings before they grow taller than about 1 m. Periodic inspection of the cleared area will be needed to remove California bays that were left in place, have resprouted from cut stems, or have grown from seed. Removing all seed-producing California bays in the treated area will help reduce seedling populations and the need for frequent followup inspections and treatments.

Localized California bay removal—Where California bay makes up a high percentage of the stand or large California bay trees are distributed throughout the stand, area-wide removal may be prohibitively expensive or inconsistent with other management objectives. Removal becomes more difficult and expensive as the size of the tree increases. In addition, it may be difficult to justify removing large healthy California bays in an effort to protect oaks whose disease status is uncertain. In such stands, it may be possible to reduce SOD risk for individual oaks or groups of oaks by increasing oak-California bay clearance in localized areas (fig. 3-1). Increased clearance is achieved through a combination of selective California bay removal and canopy pruning.

In some situations, clearance can be increased by pruning branches or stems from a California bay tree rather than removing the entire tree. As noted above (section 1.3.1, “Environmental Conditions”), low, shaded California bay foliage in the inner canopy typically has higher levels of *P. ramorum* infection than the sun-exposed leaves at the top. Removing low branches (via canopy raising and inner canopy thinning) can reduce the amount of inoculum that will reach a nearby oak even if the clearance from the oak to California bay foliage is less than optimal.

Compared with area-wide removal, localized removal zones may require more frequent inspection and followup treatment to maintain the target oak-California bay clearances. Because fewer California bay trees are removed, localized removal may be less costly and time consuming than area-wide removal, but fewer oaks within the stand will be protected.

For high-value oaks, chemical treatment may be needed in combination with localized California bay removal to reduce the risk of SOD to an acceptable level.



Figure 3-1—Site before (left) and after (right) localized California bay removal around two coast live oaks. California bay foliage that could serve as nearby sources of *Phytophthora ramorum* spores was eliminated by removing one small California bay tree (left side of left image; stump in right image) and several California bay seedlings.

Oak-California bay clearance distance. California bay removal reduces SOD risk by increasing the distance between the spore source (California bay leaves) and the target (a susceptible oak trunk). The number of *P. ramorum* spores that land on an oak depends on:

- The California bay foliage-oak trunk clearance.
- The overall amount of California bay canopy (overstory and understory) that is close enough to serve as a source of inoculum (see sections 1.2.2, “California bay–oak distance and disease risk” and 1.3.1 “Environmental Conditions”).

Increased California bay-oak clearance should be developed only by removal or pruning of California bay. Pruning off oak branches or stems to increase California bay-oak clearance is not recommended. No single prescription has been verified as effective for localized California bay removal. Consider the following general relationships and recommendations when implementing localized California bay removal:

- The risk of SOD decreases as the oak-California bay clearance increases. Removing California bay closest to an oak is associated with the largest reduction in SOD risk. The minimum horizontal clearance from California bay foliage to the oak trunk (fig. 1-6) that is likely to have an effect is about 2.5 m; clearances of 5 m or more are preferable.
- Larger California bay-oak distances are needed as the overall amount of California bay canopy in the oak neighborhood increases. If possible, provide additional clearance in the direction of prevailing storm winds, especially if tall California bay canopy is present in that direction.
- In climates that are more favorable for *P. ramorum* spore production, larger oak-California bay clearance may be needed to achieve adequate risk reduction.
- Removing low inner-canopy California bay branches close to oak trunks may help reduce risk even if foliage in the upper portion of the California bay canopy cannot be cleared to the minimum target distance (2.5 to 5 m).
- California bay seedlings less than 1 m tall should be removed if located within about 3 m from an oak trunk.

When using localized California bay removal, it is often necessary to strike a balance between the maximum risk reduction (greatest clearance possible) and minimizing cost and other impacts (smallest clearance that will provide a substantial risk reduction). Each doubling of the desired clearance distance translates to a quadrupling of the area that needs to be cleared, with a corresponding increase

in effort (fig. 3-2). Furthermore, California bay canopy within the target area may arise from a trunk located outside the clearance distance. Hence, California bays rooted outside of the target clearance distance may also need to be removed or pruned.

Eliminating highly susceptible California bay trees—Individual California bay trees vary in their susceptibility to foliar *P. ramorum* infections (Anacker and others 2008, Meshriy and others 2006). California bay trees also vary with respect to how many infected leaves they retain over the dry season (Swiecki and Bernhardt

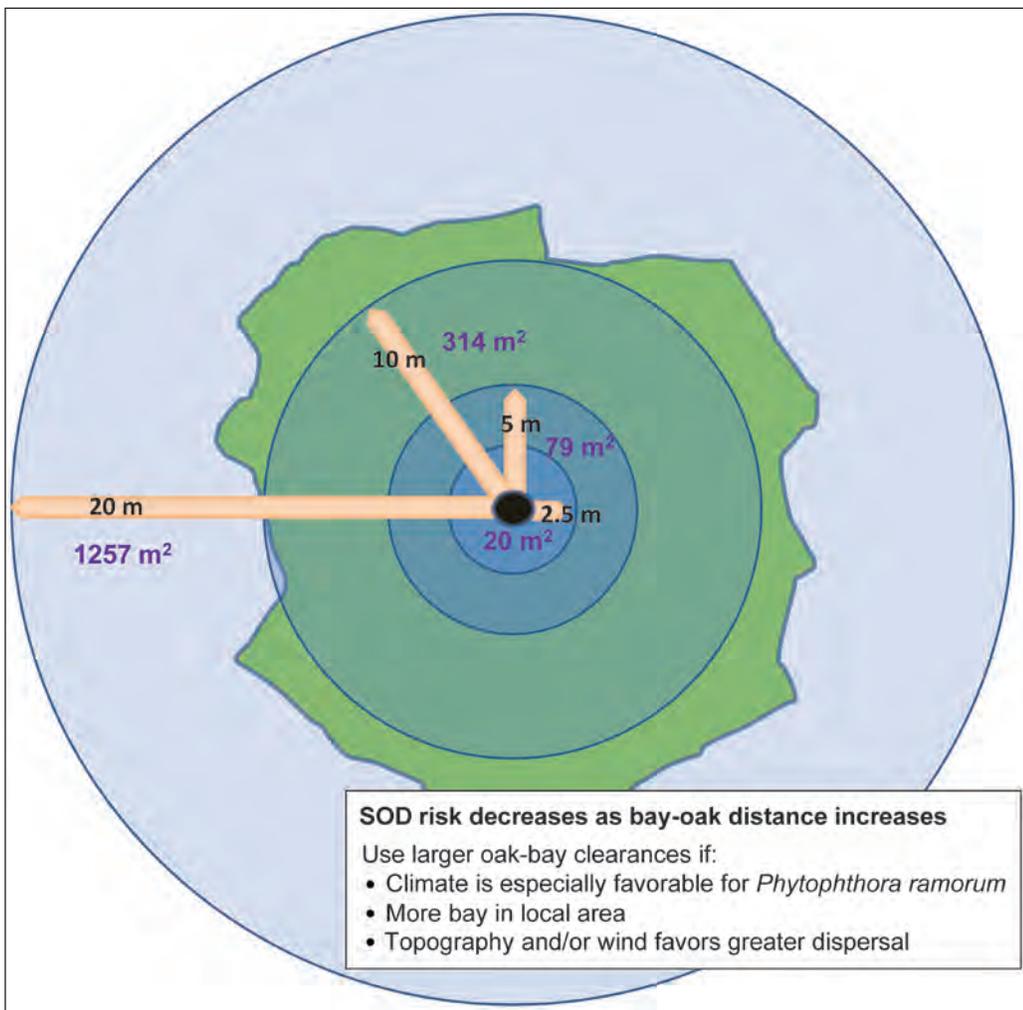


Figure 3-2—Bay removal area (blue circles) for four California bay foliage-oak trunk clearance distances (orange arrows). Green outline represents a large oak canopy (about 22 m canopy diameter) with trunk shown as the central dot. The smallest clearance likely to provide a significant reduction in sudden oak death risk is about 2.5 m. Greater clearances provide more risk reduction, but the area of bay removal quadruples with each doubling of the clearance distance.

2007, 2008a). Because *P. ramorum* does not appear to affect the survival of infected California bays, these more disease-prone trees are not eliminated from the population by natural selection. If California bays that consistently show high levels of infection or retain high numbers of infected leaves throughout the dry season are identified, they can be targeted for removal. Reducing the density of these California bays in the landscape should help slow the buildup of *P. ramorum* spores on leaves during the wet season, reducing SOD risk to oaks in the area.

In low rainfall years that are unfavorable for *P. ramorum* sporulation, few California bays develop severe foliar symptoms. California bays that have significant numbers of infected leaves by late summer in such years are logical candidates for removal. Some of these California bays may have high persistent infection levels because of microclimate effects (e.g., being located in shaded drainages) rather than high genetic susceptibility. Removing such trees may not help select for more resistant California bay genotypes, but should still help slow the seasonal buildup of *P. ramorum* in the stand.

In years that are favorable for disease, most California bays may be heavily infected and highly susceptible genotypes cannot be identified. It may be possible to identify California bays that are somewhat resistant to *P. ramorum* infection in favorable disease years. Resistant trees should show especially low infection levels when surrounded by California bays with a high level of foliar disease.

Selective elimination of highly susceptible California bays is more likely to be effective in areas where conditions are less favorable for *P. ramorum* sporulation, such as drier, more inland areas near the edge of the current *P. ramorum* range. This tactic is also more likely to be useful in areas with relatively low California bay density.

Bay removal procedures—

Timing of removal operations—In areas where *P. ramorum* is present, avoid cutting down or pruning California bay during wet conditions, especially in the spring rainy season, because:

- *Phytophthora ramorum* spores may be splashed directly onto susceptible oak stems during felling operations if foliage is wet.
- *Phytophthora ramorum* is more likely to be spread around and beyond the area in wet soil and California bay litter.
- Fresh wounds on oak stems made during clearing operations may provide favorable sites for infection.
- Cut California bay foliage on the ground will dry slowly and may serve as a potential source of inoculum for an extended period.

If you need to remove California bay in winter or spring, do it after an extended period of dry weather and observe stringent sanitation procedures.

The optimal time for California bay removal operations is between late spring, after rains have stopped and the surface soil has dried out, and late fall, before the soil has become wet. During these periods, it is unlikely that viable spores would be dispersed and cut foliage will dry out relatively quickly.

In areas where *P. ramorum* is not present, it is still preferable to avoid California bay removal during wet conditions or when soil is muddy. This will reduce the risk of introducing *P. ramorum* or other *Phytophthora* species to the site. Follow strict sanitation protocols (see section 3.1, “Excluding *Phytophthora ramorum* From Noninfested Areas”) to ensure that all vehicles and equipment are free of contamination before entering the site.

Mechanical removal—Small California bays in the understory are relatively easy to remove with hand tools. Seedlings up to about 2.5 cm (about 1 inch) diameter at breast height can be pulled out using a brush/seedling removal tool (e.g., Weed Wrench™, fig. 3-3). Small seedlings (<1 cm basal diameter) can often be pulled by hand or can be chopped out below the soil surface with a mattock. Removal is easier if the soil is somewhat moist. Small seedlings may also be cut off with loppers or a brush cutter. Cut seedlings may resprout, so repeated cutting may be necessary.

California bay wood is relatively light, so small stems are relatively easy to cut and handle using small chain saws or hand saws. In contrast, removing large California bay trees requires much greater effort and is more hazardous. Felling large California bay trees requires trained and experienced personnel with proper equipment and safety gear. Crews will need chain saws, powered or manual pole pruners, and ropes. In dense stands, cut California bays may hang up in the canopies of adjacent trees. Care must be taken to avoid breaking oak branches when felling California bays. Oak branches or stems with substantial decay can fail if they are hit by felled branches or trees. In addition, decayed oak branches or stems may fail during or after removal of a supporting California bay.

Dealing with cut California bay—Leave cut California bay onsite if possible to minimize the risk of spreading *P. ramorum*. The main options for disposing of cut California bay are noted below.

Lop and scatter. This is the most practical method in locations well away from roads or structures, and may be used in other areas if the amount of cut material is not excessive. The main stem and branches are cut into sections that lie low to the ground and are spread out to avoid stacking. California bay debris treated in this manner dries out quickly and decays substantially within 1 to 2 years. As a



Figure 3-3—Brush pullers (Weed Wrench™ shown) can be used to extract bay seedlings that are too large to pull by hand.

precaution, keep cut California bay foliage at least 2 m (6.5 ft) away from trunks of SOD-susceptible oaks. This is especially important if damp weather will prevent drying and allow foliage to serve as a potential inoculum source for many days or weeks.

Chipping. In areas where it is undesirable to leave cut material on the ground, branches can be chipped. Preferably, both chipping and disposal should occur on site to avoid possible spread of inoculum to other areas. Set the chipper so that chipped material is not deposited on trunks of susceptible oaks. Clean all potentially infested debris out of the chipper and truck before they are moved from an infested area (see box 3-1).

Pile and burn. In some sites, it may be possible to pile cut California bay branches for later burning. Local fire and air quality regulations should be checked before contemplating this option. Set burn piles well away from retained trees to avoid scorching tree canopies or damaging shallow roots that are present beneath the duff layer. Burn piles can cause excessive heating of the soil, which can have long-term negative effects on soil properties, soil microbes, and the soil seed bank. Keep burn piles relatively small and include only small-diameter branches to help minimize soil heating and associated negative impacts.

Sprout regrowth/retreatment intervals—California bay typically resprouts from cut stumps. The amount of regrowth that occurs within a season varies with climate and stump size. In Humboldt County, California bay resprouts can grow to a height of 2 to 3 m (6 to 10 ft) in a year, whereas regrowth in drier sites in Sonoma, Solano, and San Mateo Counties has been less than 1 m per year. Where sprout growth rates are relatively low, browsing by deer or other animals can be sufficient to completely suppress sprout regrowth (fig. 3-4).

Monitor sprout regrowth after cutting to determine when sprouts should be cut. As long as sprouts remain short (1 m or less), spores produced on infected leaves are unlikely to be dispersed more than about 2 m. In addition, sprouts that develop during the dry season will normally remain free of *P. ramorum* lesions at least until the next substantial infection period. Thus, California bay sprouts that are well away from oak stems may not serve as significant sources of inoculum for at least several years.

If stump sprout removal is done while shoots are still small, sprouts can be removed using various hand tools and the amount of material to dispose of will be minimal. Repeatedly removing sprouts will eventually deplete stored carbohydrates in the root system, leading to weaker sprout regrowth and eventual death of the stump. This depletion will occur faster if sprouts are not allowed to regrow substantially before recutting.

As discussed below, sprout regrowth can be suppressed by treating freshly cut stems with appropriate herbicides. In sites where the survival of oaks is questionable and where California bay would be acceptable as a replacement canopy species, it may be preferable to suppress resprouting by cutting rather than herbicides. This allows the possibility of recruiting California bay resprouts as replacement canopy if needed.

Herbicides—Trees that are too difficult to cut can be killed using herbicides, although this method may not be appropriate in many situations. Herbicide-treated California bays may not die for an extended period of time (months to over a year) and may continue to produce *P. ramorum* spores during this time. Also, because trees are left to die in place, management concerns related to visual appearance, fire hazard, and tree failure can limit where herbicide treatments can be used. Direct removal is preferable if an herbicide-treated tree will eventually need to be cut because of hazard issues.

The most common method for applying herbicides to kill large standing trees is the “hack and squirt” or frill method. A hatchet is used to make downward angled cuts through the bark around the circumference of the tree. Herbicide (usually full-strength concentrate to quarter-strength dilution depending on the product;



Figure 3-4—Bay stump sprouts 1 year after tree felling. (A) Strong sprout growth from a nonbrowsed, untreated bay stump. (B) Untreated stump with sprout growth kept in check by deer browsing. (C) Glyphosate-treated stump showing no sprouts.

follow label directions) is immediately applied into the open wounds. Alternatively, a specialized hatchet- or lance-like herbicide injection device can be used to simultaneously cut the bark and apply herbicide. Some herbicides (e.g., glyphosate, triclopyr) will not be effective on large California bay trees unless they are applied to overlapping cuts around the entire trunk circumference. Herbicide combinations (e.g., imazapyr plus glyphosate) may be more effective than individual herbicides.

Timing of herbicide application can make a large difference in efficacy. In a San Mateo County study, California bays treated with 20.5 percent glyphosate (hack and squirt method) in March died within a year, but trees in the same stand treated in July showed almost no effect of the application. Additional observations suggest that glyphosate applications made during the wet season from late fall through early spring are most effective, at least in the San Francisco Bay area.

Stump sprouts from cut California bays may also be suppressed through the use of systemic herbicides such as glyphosate. Apply the herbicide to the cambial area (bark/wood junction) of freshly cut stumps. Appropriate herbicides may also be applied to sprouts after they develop on cut stumps, but such applications will typically require greater amounts of spray solution and pose a greater hazard to nontarget plants from overspray and drift. In San Mateo County research plots, applications of 20.5 percent glyphosate to freshly cut stumps significantly suppressed resprouting (fig. 3-4).

In one site, glyphosate applied to freshly cut stumps in November translocated through root grafts and killed California bays many meters away. At another site treated in September, well before the start of the rainy season, no evidence of glyphosate translocation from treated stumps to nearby California bay was seen.

3.2.3—Removing Other Sporulating Hosts

Climbing poison oak—

Observations indicate that poison oak vines that climb into oak canopies may sometimes serve as an important source of *P. ramorum* spores (see “Other sources of inoculum” in section 1.2.2). Kill climbing poison oak vines when SOD management treatments are being used to protect susceptible trees. Climbing poison oak vines can be killed by cutting their stems near the soil line. Cut stems may immediately be treated with an herbicide to prevent regrowth. Once stems are severed, vines in the canopy die quickly and can be left in place. Poison oak can cause a highly irritating rash on many people, so wear protective clothing and take precautions to avoid contact with the plant.

Other species—

No California native plants species other than California bay, tanoak, and poison oak are clearly associated with SOD risk in susceptible oaks. However, it is possible that some other native foliar host species could be significant sources of *P. ramorum* spores in some situations. If further research shows that other native hosts can serve as sources of *P. ramorum* inoculum, localized removal, as discussed for California bay above, should help reduce SOD risk. Check <http://www.suddenoakdeath.org> for current information.

In the United Kingdom, other species have been shown to be associated with infection of canker hosts in the field. Infected rhododendrons, particularly *Rhododendron ponticum* L. and Japanese larch (*Larix kaempferi* (Lam.) Carrière) produce enough *P. ramorum* spores to initiate trunk infections. Based on this information, SOD risk may be minimized by keeping horticultural plants that can support moderate to high levels of *P. ramorum* sporulation (table 1-3) at least 5 to 10 m from susceptible oaks. As a general rule, keep the area within 2.5 to 5 m of an oak trunk free of both plants and irrigation to minimize the risk of other oak diseases, such as Armillaria root disease.

3.2.4—Use of Chemical Protectants to Prevent Infection

Although *Phytophthora* species are no longer considered to be fungi, chemicals that are used against diseases caused by *Phytophthora* are still classified as fungicides. Most fungicides do not kill fungi directly; they inhibit fungal growth (see box 3.2). Hence, fungicides generally work best when disease pressure has also been reduced to the maximum degree possible by using other management methods.

Some fungicides have shown promise against SOD in greenhouse tests and assays on mature trees. However, field studies to determine if fungicides can reduce SOD incidence or severity in mature trees are still ongoing. Results available to date have been mixed and no studies have shown that oaks or tanoaks can be completely protected from SOD by fungicides alone.

Systemic fungicides (box 3-2) have the highest potential to be useful for SOD management because infections may occur over a long time period through the winter and spring. To be effective, systemic fungicides need to be absorbed by living plant tissues and accumulate in areas that are subject to infection. Areas already affected by cankers will not effectively absorb or translocate fungicides, so applications will be most effective when applied before infection occurs. Even if applied well before infection occurs, systemic fungicides may not provide useful prevention of SOD if poor uptake or translocation patterns result in low fungicide concentrations in the bark of the lower trunk.

Chemical application is relatively expensive and needs to be repeated on an ongoing basis to provide continued protection against SOD. Therefore, fungicides are best suited for use on limited numbers of high-value trees in areas where SOD risk is high. Consider the overall disease potential at the site, including the abundance of California bay, and the significance of at-risk trees when deciding whether chemical applications may be appropriate.

Fungicide use considerations differ for tanoak, which can produce *P. ramorum* spores, and oaks, which are normally infected by spores produced on California bay.

Fungicide treatment of tanoak—

Systemic fungicide treatments may have the potential to inhibit both *P. ramorum* trunk cankers and twig infections. If fungicide-treated tanoaks develop fewer or smaller twig infections, spore production will be reduced, lessening the likelihood of trunk infections. To benefit from the synergistic effects of reduced spore production and increased resistance to trunk infections, contiguous blocks of tanoaks need to be treated. If untreated tanoaks are left within a stand, spores produced on these trees could splash onto treated trees, which may impede disease control.

In well-stocked tanoak stands, it may be more efficient and cost-effective to remove small understory tanoak seedlings and saplings instead of treating them with fungicides. Because spores from small (<1 m) understory tanoak regeneration or stump sprouts are unlikely to splash more than about 2 m, these small shoots only need to be removed if they are within about 2 to 3 m of treated tanoaks.

Fungicide treatment of oaks—

Because California bay is the primary source of *P. ramorum* inoculum in oak stands, fungicide treatment of all oaks within a block does not provide any advantage over selective treatment of individual oaks. For susceptible oaks, use of fungicides should always be combined with California bay removal, if possible, to reduce disease pressure. Where California bays are too large or significant to remove, fungicide use may be warranted to help protect high-value oaks that are closest to California bay trees. However, fungicides may not be sufficient to prevent SOD in trees that are exposed to large numbers of *P. ramorum* spores.

Use of phosphites (phosphonates) to manage SOD—

Phosphites (also known as phosphonates) are a group of fungicides which have potential for use in managing SOD. Phosphites are selective, systemic fungicides that have been used to manage *Phytophthora* diseases for a number of years. They have a high level of environmental safety and very low nontarget toxicity. The most widely used material in this fungicide group is potassium phosphite (also known as

Box 3-2—Selecting and Using Fungicides

Controlled studies are needed to determine how well any fungicide will work for a given host/pathogen combination and what dosage is required. To date, only phosphite fungicides have been studied extensively for their ability to protect trees from sudden oak death (SOD). It is not yet clear whether phosphites can provide useful levels of protection against *Phytophthora ramorum* in forests. Other fungicides that are active against *Phytophthora* species may be evaluated for potential use in SOD management in the future. The following factors should be considered when selecting and using fungicides as a SOD management tool.

Type of activity: Most fungicides work best when applied as protectants (before infection has occurred), rather than as eradicants or curatives (after the plant has been infected). Using fungicides as eradicants increases the chance of selecting for fungicide resistance in the pathogen population.

Contact or surface fungicides are not absorbed by plants and act mainly to inhibit germination and growth of spores on plant surfaces. Contact fungicides need to be applied as a complete coating over the susceptible plant and must be reapplied as the fungicide residues break down, are washed away, or spread out as the plant parts expand.

Systemic fungicides are absorbed by plant tissues and are translocated within the plant. Fungicides transported in the water-conducting xylem tissues can move upward only, whereas those transported in the phloem (stem tissue that transports carbohydrates) can move downward as well. Because systemic fungicides move within the plant, it is less critical to obtain complete plant coverage. Systemic fungicides typically have longer residual activity than contact fungicides and are reapplied less frequently. Materials that need to be reapplied more than once each year may not be practical for protecting large numbers of trees from SOD.

Spectrum of activity: Most systemic fungicides are selective. Many fungicides that are effective against true fungi have little or no activity against *Phytophthora* species, and vice versa. Many contact fungicides (e.g., copper salts) are relatively nonselective and are active against a wide variety of fungi as well as *Phytophthora* species.

Application methods: Fungicides are applied by a variety of methods (e.g., spray, injection, soil application), which are specific to the fungicide/disease/plant combination. The application method can affect cost and practicality of treatment, absorption and distribution of the chemical in and on the plant and the risk of nontarget exposure. Application methods that may be practical for treating small numbers of trees may be too expensive to use for entire forest stands.

Phytotoxicity: Some fungicides can cause damage to plant tissues (phytotoxicity) under certain conditions. Phytotoxicity can be influenced by factors such as plant species and variety, plant maturity, temperature, application method, or interactions with other applied materials. Fungicides need to be effective at a dose that is well below levels that cause serious phytotoxicity.

Effects on nontarget organisms: Most fungicides and other pesticides work by interfering with vital metabolic processes in the target pathogen. Fungicides also have the potential to interfere with metabolic processes of other nontarget microorganisms, invertebrates, and vertebrates. Fungicides used in settings such as forests need to have little or no negative effects on the wide variety of nontarget species found in these environments.

Toxicology: Humans are very important nontarget organisms to consider when selecting a fungicide. Potential effects of fungicides on human health are related to the toxicity of the material (its potential to cause acute or chronic health effects) as well as the amount and routes of exposure to the material. Persons who mix and apply fungicides to trees have the greatest likelihood of exposure to these chemicals. Dietary exposure to fungicide residues used for SOD prevention may occur if acorns are used for food or if animals that consume acorns (e.g., livestock, deer, wild turkey) are eaten.

Applicator safety: Proper safety procedures and personal protective equipment (PPE) should be followed to minimize exposure to applied chemicals. Appropriate PPE for applicators, such as gloves, face shields, respirators, and protective clothing, depends upon both the type of chemical used and the application method. The fungicide label indicates types of required safety equipment and other necessary precautions.

continued on next page

Box 3-2—Selecting and Using Fungicides (continued)

Persons applying pesticides for hire in California are required to have a Qualified Applicator's License or Certificate issued by the California Department of Pesticide Regulation (CDPR). Qualified Applicators must pass tests related to pesticide application and safety and take continuing education classes to maintain certification.

Regulatory issues: Chemicals or natural products that are sold expressly for the purpose of controlling a plant disease, such as SOD, need to be registered as pesticides by the U.S. Environmental Protection Agency (EPA). In addition, pesticides used in California must be registered by CDPR. Registered pesticides that are classified as general use pesticides may be applied by general users, including homeowners. More hazardous pesticides are classified as restricted use pesticides and may only be applied by CDPR-certified applicators. Fungicides or other products that are used to manage SOD must be registered for that use and applied in a manner consistent with the label, observing all label precautions.

potassium phosphonate, or mono- and di-potassium salts of phosphorous acid). This material is sold under a number of trade names.

At least two general modes of action have been identified for phosphites (Guest and Grant 1991). Potassium phosphite moves systemically, in both the xylem and phloem. At high concentrations in the plant, phosphite is directly toxic to *Phytophthora* species. At lower concentrations, phosphites act indirectly by increasing a plant's natural resistance response to *Phytophthora* infection. At these lower concentrations, phosphite appears to interfere with *Phytophthora* metabolism, causing the release of compounds that trigger host defense responses. Phosphites are primarily effective against *Phytophthora* species and have little or no activity against most diseases caused by true fungi.

Phosphite is most likely to be effective when applied as a preventative, before trees are infected. In field tests, phosphite has not been effective against cankers in previously-infected tanoaks, even if external cankers symptoms were not visible at time of application.

Most studies indicate that phosphite does not prevent infection by *P. ramorum*, but may reduce the rate at which *P. ramorum* cankers expand. If disease pressure is high, phosphite may not suppress disease progress enough to provide practical levels of disease control. In a recent controlled study in San Mateo County, involving over 470 mature tanoaks, 3 years of preventative annual phosphite bark spray applications did not reduce disease incidence or mortality when SOD subsequently spread into the stand. Phosphite applications were started well before *P. ramorum* was detected in or near this stand.

Phosphite solutions can be phytotoxic to some species if applied at sufficiently high concentrations. Leaves of many species, including tanoak, may develop extensive browning (necrosis) if they are sprayed with the highly-concentrated phosphite solution used in trunk-spray applications. Mosses, which are common on oak and tanoak trunks in some locations, are also killed by the spray. Injected phosphite solutions can also cause necrosis in the bark and sapwood. Phosphite should be tested for potential to cause phytotoxicity whenever it is used on a new plant or if a modified-application method is used.

Environmental and user safety—Phosphites are considered by the EPA to be biopesticides because these salts are closely related to common, widely occurring substances. However, phosphonate salts are not naturally occurring mineral substances. Information on toxicity and environmental fate of this material was summarized in an EPA fact sheet (USEPA 1998):

ECOLOGICAL RISK ASSESSMENT

A potential for exposure exists to nontarget insects, fish, and other wildlife with foliar spray applications. However, test results indicate that the compound is practically nontoxic to birds and freshwater fish, and, at most, slightly toxic to aquatic invertebrates. Low toxicity, the proposed rate of application, and mitigating label language present minimal to nonexistent risk to wildlife.

Phosphorous acid and its ammonium, sodium, and potassium salts are also exempt from food tolerance, for both crop and postharvest uses (USEPA 2006). In exempting phosphites from food tolerance, the EPA cited both the low toxicity of phosphites and the fact that phosphites had already been used widely as fertilizers. In a preliminary study, acorns from tanoak trees treated with phosphite showed no substantial differences in nutritional quality when compared with nontreated trees (Meyers and others 2007).

Although phosphites are relatively safe materials, phosphite solutions, particularly in concentrated form, may be harmful if inhaled, ingested, or absorbed through the skin and must be handled safely to avoid exposing applicators or others. Formulations of potassium phosphite typically have the “Caution” signal word on the label. Applicators and others handling the material for mixing and loading must wear protective eyewear, long pants and a long-sleeved shirt, waterproof gloves, shoes, and socks. Depending on the application method, additional personal protective equipment may be necessary.

Potassium phosphite is corrosive to many metals, especially brass, which is commonly used in sprayers and injection equipment. Sprayer components that are exposed to potassium phosphite solution should be made of plastic or stainless steel. To minimize the potential for corrosion, thoroughly rinse application equipment with water immediately after use.

Comparison of application methods—Phosphite can be applied to trees via stem injection, bark or foliar spray, and soil drench. In tests conducted to date, soil drench applications have not been effective against *P. ramorum* canker (Garbelotto and others 2007). Foliar applications, which are widely used to control root-rotting *Phytophthora* species, have shown relatively short-lived activity against *P. ramorum* canker in greenhouse tests on oaks. Foliar applications are also difficult to execute on large trees and may require high volumes of spray. Low volume aerial application of phosphite by helicopter to mature tanoaks has been tested in Oregon (Kanaskie and others 2010). Although these tests showed that aerial phosphite application suppressed *P. ramorum* in treated trees to some degree, infections were not completely prevented. This study did not provide conclusive results on aerial phosphite application for controlling SOD, but results were not overly promising.

Phosphite applied via stem injection or bark spray application reduced *P. ramorum* canker size on potted coast live oaks that were artificially inoculated (Garbelotto and others 2007, Garbelotto and Schmidt 2009). The effect appeared to last at least 18 months after treatment. In addition, branches cut from coast live oaks after treatment with a bark spray application developed smaller lesions than branches from nontreated trees when inoculated in the laboratory with *P. ramorum* (Garbelotto and Schmidt 2009). Based on these results, injection and bark spray application are currently the preferred methods for applying phosphite to oaks and tanoaks. Each method has advantages and disadvantages (table 3-1). In general, injection is more suited for treating a small numbers of trees.

Injection—Stem injection involves introducing a chemical into the water-conducting tissues of a tree (xylem) via holes drilled into the outer sapwood. To speed uptake, the solution is applied under slight to moderate pressure. Some injectors apply the chemical to the cambial region, which may allow some of the chemical to be directly absorbed by the phloem. Current recommendations for injecting with potassium phosphite products are discussed below. These practices are subject to change based on the outcome of ongoing studies.

Timing: Trees should be injected at least 4 to 6 weeks before the onset of conditions favorable for disease because of the time lag between injection and subsequent

Table 3-1—Comparison of injection and bark spray methods for phosphite application

	Stem injection	Bark spray application
Equipment	Need injection equipment. Most injector types also require a drill motor and bit. Cost varies, depending on type of injection equipment used. Having more injectors can reduce application time, but increases equipment cost.	Need sprayer, wand, and nozzles. Cost varies depending on equipment used (backpack vs. tank sprayer).
Material required	Lower amount of phosphite required—minimum of about 1.75 ml formulated product (45.8 percent potassium phosphite) per inch stem diameter.	Higher amount of phosphite required—minimum of 31 ml of formulated product per centimeter stem diameter. Surfactant (Pentrabark®) also required.
Efficiency of uptake	Except for leakage from injectors, all of applied product is introduced into tree.	Significant losses of material via overspray and runoff during application process. A relatively small percentage of the material deposited on the trunk is absorbed by the tree.
Translocation	Most or all of the chemical introduced into the tree's xylem. Phosphite moves up to the tree canopy before it is translocated back to the bark of the lower trunk. Some injectors apply chemical to the cambial zone, where direct uptake into the bark is possible.	Material is absorbed directly through bark into the phloem and can move upward and downward.
Application speed	Varies with injection equipment and tree size, but slower than spraying, especially if many trees are being treated. More suitable for small numbers of trees.	Relatively fast. Because of fixed setup and cleanup time, generally more efficient to treat multiple trees at a time. Suitable for treating large numbers of trees.
Frequency of application	Current recommendation is a 1.5- to 2-year retreatment interval.	Current recommendation is two treatments in first year and annually thereafter.
Accidental inoculation via contaminated equipment	Pathogen could be accidentally introduced into trees via drilling and injectors (depending on type used) when moving from asymptomatic infected trees to noninfected trees unless equipment is cleaned and disinfested.	Negligible risk of cross-contaminating trees via spraying process.
Nontarget effects	Wounds created on trunk may provide entry point for insects and pathogens, including <i>Phytophthora ramorum</i> . Multiple treatments will result in a large number of injection wounds. Injected solutions can kill phloem and xylem tissues if too concentrated. Injection sites may ooze for up to several years.	Spray solution kills mosses on bark surface. Overspray can cause browning (necrosis) of wetted foliage of some species.

translocation of phosphite throughout the plant. Repeat injections at 1.5- to 2-year intervals, depending on the timing of the previous treatment. For instance, if the end of the 1.5-year interval falls in the summer, injection may be delayed until fall.

For low pressure injectors, trees must be actively transpiring in order to take up injected phosphite solution. Deciduous trees such as California black oak should be injected when expanded leaves are present. The fastest uptake of injected solution occurs under sunny conditions. Uptake may be slow to negligible during rainy, foggy, or heavily overcast conditions. Uptake may also be slow late in the day, especially in dense, shaded stands. In very dry sites, uptake may be better with light cloud cover and cooler temperatures than under very hot, dry conditions.

Injection holes in the bark have an elevated susceptibility to *P. ramorum* infection, so trees should not be injected in late winter or spring. Injection is not recommended for trees under severe water stress because phytotoxicity may develop in the canopy. Foliar phytotoxicity may also develop if trees are injected when temperatures are above 32 °C (90 °F).

Dose: Approximately 0.71 ml of nondiluted potassium phosphite formulation (45.8 percent potassium phosphite) per cm of stem diameter is a common label rate for tree injection. This dosage may not be adequate for large diameter trees. Different methods of calculating the applied dose provided on some labels can generate dosages up to about 10 times this level.

Concentrated potassium phosphite solutions (45.8 percent) must be diluted with water before injection, although dilution ratios have differed with the injector type. Dilutions of concentrated phosphite solution and water ranging from 1:2 to 1:5 (15.3 to 7.6 percent potassium phosphite) have caused significant phytotoxicity in injected tanoaks and coast live oaks. Injection holes exhibited prolonged bleeding (up to several years) and necrosis in the phloem and xylem (fig. 3-5). Studies are underway to determine the dilution rate needed to avoid phytotoxicity. The dose needed to provide efficacy may also need to be adjusted for more dilute injection solutions.

Injection points: To ensure that injected material is evenly distributed throughout the tree canopy, injection points need to be spaced at relatively close intervals. Injectors are typically spaced 15 to 20 cm (6 to 8 inches) apart, but some injector manufacturers recommend wider spacing, up to 23 to 25 cm (9 to 10 inches). If injectors are spaced too widely, phosphite will not be evenly distributed throughout the canopy. The optimum injector spacing needed to ensure even distribution of phosphite in the lower trunk is currently unknown.

Injection holes should be at least 0.3 m above ground level to minimize contamination of the holes with soil, but the actual height of the injection holes is not critical. If bark is too thick to allow proper placement of the injectors (see below),



Figure 3-5—Necrosis in phloem and xylem (dark brown discoloration) associated with injections of 15.3 percent potassium phosphite (10 ml per injection). Injections were made 2 and 4 years before the tree was cut. Outer bark has been chipped away around injection points.

insert injectors higher on the trunk where the bark is thinner. Drill holes level horizontally or angled with the opening pointing slightly downward so that water running down the stem will not collect in the holes.

Follow the injector manufacturer's instructions to select the appropriate diameter of the drill bit. Use a sharp drill bit to obtain a cleanly cut hole and use the bit to clear shavings from the hole before injection. For most injector types, holes should extend through the bark to the outer sapwood. If possible, clean the drill bit with isopropyl or ethyl alcohol (70 percent or higher) between each hole to prevent cross-contamination. At a minimum, clean the drill bit when moving from tree to tree. Clean injectors with 70 percent alcohol prior to use in another tree.

Ensure that the injector tip fits tightly into the hole to prevent leakage. For injectors that are designed to apply material into the sapwood, holes should extend about 1 cm into the sapwood. Some injectors allow phosphite solution to be injected into the inner bark and cambium area, but this has been associated with phloem necrosis at the dilutions noted above. Some injection systems employ a plastic plug with a septum that is inserted into the drill hole and remains in the tree. These plugs must be seated at the depth specified by the injector manufacturer.

Drill holes into healthy areas of the trunk. Avoid areas with dead bark, decayed wood, cracks, sunken areas, branch stubs, or callus tissue. Stagger the injection height, include a vertical offset of about 10 to 20 cm (4 to 8 inches) for adjacent holes to avoid placing all injection points in a single line around the tree's circumference. Drill new holes for injectors when retreating trees; do not reuse holes made for a previous injection. Offset new holes horizontally as far as possible from the previous set(s) of holes and use different vertical heights.

Applicator safety: Some injectors contain solution under pressure after filling. Take precautions while carrying or handling injectors and while making injections to ensure the solution is not unintentionally squirted at the applicator. A full face shield is preferable to goggles for persons handling injectors.

Bark spray application (basal bark spray)—Standard spray application equipment can be used to apply phosphite to the bark. A high rate of an organosilicate surfactant (trade name Pentrabark[®]) is mixed with the phosphite solution to enhance absorption through the bark. The efficacy of the bark spray applications are still being tested in the field. In several field trials, preventative bark spray applications made as specified below have failed to lower SOD incidence and mortality rates in treated tanoak trees compared to nontreated trees.

Timing: Spray trees at least 4 to 6 weeks before the onset of favorable disease conditions to allow for uptake and translocation. Two applications are recommended in the first year (fall-spring or spring-fall), followed by annual applications thereafter. Based on results of field studies noted above, this regime may not be adequate to protect large tanoaks.

If the bark is saturated by rainfall at the time of application, spray will run off sooner, which may lead to reduced absorption. Otherwise, phosphite uptake is not adversely affected by rainfall. Light to moderate rainfall after application can rewet residues on the bark surface and may favor additional bark uptake. Phosphite that washes off the trunk into the soil is inactivated by soil micro-organisms within a few weeks.

Dose: For the bark spray application, potassium phosphite (45.8 percent potassium phosphite) is diluted 1:1 with water. Pentrabark[®] surfactant is added to attain a final surfactant concentration of 2.3 percent by volume in the spray mixture. This surfactant concentration can cause excessive foaming and may be higher than is necessary to obtain uptake. The resulting diluted spray solution contains 22.4 percent potassium phosphite or 302 g potassium phosphite/L solution (2.52 lb/gal).

Various stem characteristics and the spray equipment used can affect the point where the spray begins to run off the stem. These include:

- Bark roughness: rougher bark will retain more spray than smooth bark.
- Stem angle: spray will begin to drip off strongly sloping stems or branches sooner than it will from a nearly vertical stem.
- Spray application rate: a high spray flow rate may flood the bark surface so quickly that little adsorption occurs and runoff begins almost immediately.

Because of these variables, it is not possible to precisely calculate the amount of spray needed to treat any tree from the label instructions alone. Figure 3-6 shows the approximate amount of diluted spray needed to treat trees of varying diameters.

When phosphite is applied following label instructions, the amount of spray applied per tree will increase as the trunk diameter (and bark surface area) increases. In addition, larger trunks receive more spray solution per diameter unit because of their rougher bark. For stems less than about 30 cm (12 in) diameter, about 31 ml of spray solution is applied per cm of trunk diameter when the lower stem is sprayed to runoff. For a 75 cm (30 in) diameter stem, the amount increases to 62 ml per cm of trunk diameter. It is necessary to increase the amount of spray per unit stem diameter (ml/cm) as trunk diameter increases to maintain a more constant spray amount per unit trunk volume (ml/cm³).

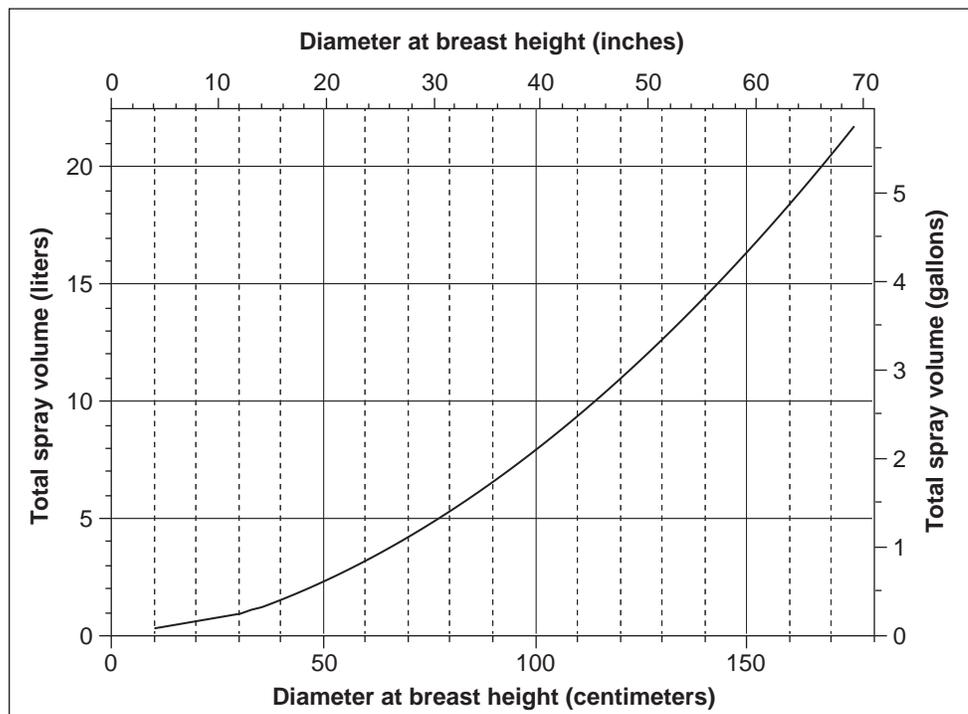


Figure 3-6—Approximate amount of diluted potassium phosphite spray solution (1:1 dilution of 45.8 percent concentrate with water plus Pentrabark[®] surfactant) used to treat oak and tanoak trunks. The applied amount varies with trunk diameter at breast height (DBH) = diameter measured 137 cm (4.5 ft) above the soil grade. For trees less than 30 cm (12 in) DBH, a linear function is used to calculate spray volume from trunk diameter. For stems 30 cm DBH or larger, the function includes a quadratic term, which causes the line to curve upward.

Application procedures: The applied phosphite/surfactant solution must penetrate dead outer bark tissues and be absorbed by living cells in the bark to be effective. To facilitate uptake, direct the spray to areas where the outer bark is thinner, such as bark fissures. Spray is less likely to be absorbed if applied to thick areas of dead outer bark.

Dense layers of moss on the bark surface can also decrease phosphite absorption. Target spray away from dense moss patches. Where this is not possible, use a larger spray droplet size and hold the spray nozzle close to the bark surface to spray beneath leafy mosses that extend outward from the bark. For appressed mosses, apply enough spray to wet the bark under the moss. Moss on the bark surface is killed by the phosphite/surfactant spray solution, so moss presents less of a problem after the initial phosphite application.

Brushes with stiff bristles can be used to remove moss from the portion of the stem that will be sprayed. This is very labor-intensive and may not be practical for treating large numbers of trees. Vigorous brushing can cause small wounds by chipping off bark fragments, so brushing or other mechanical moss removal should be completed several months before the rainy season.

Phosphite uptake may also be reduced if the bark surface is dusty. For dust-covered stems, apply after early rains have rinsed off the dust or use water to hose off excess dust before application. Allow the bark surface to dry after washing so that the applied spray solution will not run off immediately.

Select nozzle type and spray pressure to maximize spray deposition and minimize spray drift. Drift can be minimized by using nozzles that produce uniformly large spray droplet sizes, such as air-induction nozzles. Low spray pressure (near the nozzle's minimum pressure rating) will help minimize the production of fine spray droplets that drift and will also reduce the tendency of larger spray drops to bounce off the bark surface.

Spray is commonly applied to the lower 2 m (6 ft) of the trunk to just before the point of runoff. The spray volumes shown in figure 3-6 can be used to determine the amount of spray to be applied if the treated zone is shifted upward. Applying the spray higher on the stem provides several potential advantages:

- Material is applied where the outer dead bark is thinner and more permeable.
- Higher portions of the trunk may have less moss cover.
- When spray residues high on the trunk are rewetted by rain and washed farther down the stem, additional uptake of phosphite is possible on lower portions of the stem. Phosphite applied low on the stem is more quickly washed into the soil where it becomes inactivated.

Spray can be applied high on the trunk (up to about 6 m [20 ft]) by attaching spray nozzles to a telescoping pole (fig. 3-7). Compared with basal spraying (to about 2 m in height), higher spraying requires some specialized equipment and more effort. High bole application also has greater potential for overspray, so the applicator should wear appropriate PPE to avoid exposure. The relative efficacy of high bole application versus basal application is currently under study.

Applicator safety: Most spray equipment generates fine droplets that can be inhaled, so appropriate respiratory protection is required. All sides of the stem need to be sprayed, so it may not be possible for the applicator to rely on wind to avoid spray mist. When applying at heights at or above head level, wear a waterproof hat



Figure 3-7—A telescoping pole with nozzles mounted at the end can be used to spray potassium phosphite solution high on the trunk.

and full face shield to prevent exposure to spray mist and solution that bounces or drips off the stem. Coveralls or waterproof outer clothing are also recommended, especially if large numbers of trees are to be treated.

3.2.5—Nonrecommended Treatments

Cultural techniques for managing disease are not recommended unless they have been scientifically tested to show both efficacy and a lack of adverse effects. Many oaks and tanoaks infected by *P. ramorum* have symptoms that fail to progress for multiple years or may go into complete remission (see “Patterns of canker development” in section 1.4.1). Because symptom remission can occur with or without treatment, anecdotal or case-study reports of treated trees do not provide proof that SOD treatments are effective. To document a real effect, treatments need to be tested under experimental protocols that include an adequate number of appropriately matched untreated controls. Because of the slow progress of SOD in many trees, and years during which few or no new infections occur, studies typically need to last at least 5 years to produce meaningful results.

Enhancing tree vigor—

Arborists recommend various cultural practices to enhance or maintain oak tree vigor as a general prescription to maintain tree health or counteract decline. These practices may include:

- Root zone mulching.
- Fertilization.
- Light irrigation in the outer portion of the root zone.
- Radial trenching or other manipulations to reduce soil compaction.
- Application of mycorrhizal inoculants or other soil amendments.
- Light pruning.
- Removal of competing vegetation under the canopy.

If appropriately applied, some of these practices have the potential to stimulate new shoot growth and improve tree appearance, at least over the short term. However, there is no evidence that any of these practices provide any protection against SOD or enhance survival in infected trees.

Furthermore, high tree vigor is associated with an increased risk of developing SOD in coast live oak (see “Effects of tree size and condition” in section 1.3.2, and table 2-5). Because measures intended to increase tree vigor have the potential to increase disease risk, they are not recommended for managing trees at risk of developing SOD.

Soil pH modification—

One marketed tree vigor prescription involves the use of a liming material (Azomite[®] Soil Sweetener—40 to 50 percent limestone [calcium carbonate]) applied to the soil around affected trees to raise soil pH. The method also calls for coating the lower portion of the trunk with a lime suspension (Arbriht[®] lime-wash—26.8 percent calcium hydroxide), a material “intended for topical use to prevent sunburn,” according to its label. No epidemiological or biological data suggests that there is any relationship between soil pH and *P. ramorum* canker. Furthermore, in controlled studies with both potted and field grown coast live oaks, this treatment provided no measurable protection against *P. ramorum*, whereas phosphite significantly reduced disease susceptibility (Garbelotto and Schmidt 2009).

3.3—Monitoring Oaks With Sudden Oak Death to Assess Survival and Failure Potential

3.3.1—Overview

The hazard posed by a tree that may fail (break or topple over) is related to three factors:

- The likelihood that the tree or a part of it will fail in a given time period.
- The ability of the failing part to cause damage (because of its size and the distance that it may fall).
- The presence of a valuable target within the failure’s impact zone.

The USDA Forest Service, International Society of Arboriculture, and other organizations have developed standardized numerical tree hazard rating systems based on evaluation of these factors (see below “Sources of information on tree hazard assessment methods”).

Although many trees on a given property may have an elevated likelihood of failure, only those trees that have the potential to cause personal injury, death, or property damage need to be included in most tree hazard surveys. If a target zone is rarely occupied (for instance, a low-use trail), it is not likely to be occupied at the moment of tree failure. Hence, the risk of damage can be low even if the tree is likely to fail. In contrast, if the target area is continuously occupied, any failure is likely to cause injury or loss. In such sites, even trees with a moderate failure potential need to be considered for possible hazard reduction.

As discussed in Chapter 2 (see “What is the failure potential of SOD-affected trees?” in section 2.2.3), trees with late-stage SOD symptoms have an elevated likelihood of failure overall compared to similar trees lacking these symptoms. Specific factors related to failure risk in SOD-affected trees are shown in tables 3-2 and 3-3.

Table 3-2—Guidelines for assessing mortality risk and hazard potential in wildland coast live oaks based on *Phytophthora ramorum* canker symptoms (see also table 3-3)

Risk factor	Factor level	Effect on mortality risk	Contribution to failure potential within 1 year	Additional considerations and interactions
Early <i>P. ramorum</i> canker symptoms (bleeding cankers only)		Low risk of mortality within the next year	None	Symptom remission is most likely in trees with small cankers (<20 percent of circumference girdled). Trees with extensive girdling (>80 percent of circumference) are likely to die within 2 to 4 years, but cankers normally become invaded by secondary organisms before mortality or failure. Mortality risk may be underestimated if cankers are larger than indicated by bleeding. Monitor trees at least annually for symptom progression.
Late <i>P. ramorum</i> canker symptoms (cankers plus beetle boring and/or <i>Annulohyphoxylon thouarsianum</i> sporulation)		Varies with amount of trunk affected	Moderate to high	Mortality risk and failure potential increase with increasing colonization by secondary organisms. Failure potential is also influenced by the presence of other types of decay and defects present (table 3-3).
Fruiting of <i>A. thouarsianum</i> or <i>Phellinus gilvus</i>	2.5 to 50 percent of trunk circumference with sporulation	Moderate risk of mortality within 1 to 2 years	Moderate	Mortality and failure risk of live trees increase with percentage of trunk circumference affected. Failure risk interacts in an additive fashion with other decay columns present in tree.
	50 percent or more of trunk circumference with sporulation	High risk of mortality within 1 to 2 years	High	Decay occurs before fruiting bodies develop, so sapwood decay may be extensive even if fruiting bodies are not seen. Also, old fruiting bodies can fall off or degrade.
Beetle boring	2.5 to 50 percent of trunk circumference with exit holes	Moderate risk of mortality within 1 to 2 years	Moderate	Mortality and failure risk of live trees increases with the percentage of trunk circumference affected. Beetle boring is usually associated with decay. Beetle boring seldom penetrates more than 10 cm into the wood, so beetles damage without extensive decay poses less risk to large diameter trees. Inactive galleries may be difficult to detect if boring dust has washed off.
	50 percent or more of trunk circumference with exit holes	High risk of mortality within 1 to 2 years	High	

Table 3-3—Additional factors related to hazard potential in trees affected by sudden oak death (SOD)^a

Risk factor	Factor level	Contribution to failure potential	Additional considerations and interactions
Decay and related factors:			
Tree dead or partly dead	Present	High	Small-diameter stems tend to fail sooner than larger stems.
Decay	<25 percent of the stem cross section affected	Low	Failure potential increases with increasing decay. Decay in structurally critical areas (e.g., branch junctions) can greatly increase failure potential. Decay assessment methods (drilling, etc.) can be used to assess amounts of decay in standing trees. In the absence of direct assessments, use other indicators of decay (fruiting bodies, canker rot symptoms, decline symptoms, beetles) to estimate decay extent.
	25 to 50 percent of the stem cross section affected	Moderate	Presence of fruiting bodies indicates decay is present. This decay interacts in an additive fashion with decay caused by <i>A. thourarsianum</i> . Primary decay fungi raise failure risk more than secondary decay fungi, with some differences between species in each group. In the absence of a positive identification, consider any fruiting body emerging from or through bark to be important.
Fruiting of wood decay fungi other than <i>Annulohyphoxylon thourarsianum</i>	<i>Inonotus</i> spp., <i>Phellinus</i> spp., <i>Laetiporus gilbertsonii</i> and other primary decay fungi <i>Stereum</i> spp., <i>Trametes</i> spp. and other secondary decay fungi	High	
Cavities	>50 percent of stem cross sectional area affected	Moderate to high	Risk increases with the percentage of cross sectional area affected. Cavities increase failure potential primarily if decay and other risk factors are also present.
Canker rot symptoms	Symptoms present, but no fruiting bodies	Moderate	In the absence of fruiting bodies, canker rot symptoms provide an indication that decay columns are present. Decay from canker rot fungi interacts in an additive fashion with decay caused by <i>A. thourarsianum</i> and <i>Phellinus gilvus</i> .
Decline resulting from other agents	Tree in severe decline, but no fruiting bodies	Moderate	In the absence of fruiting bodies, symptoms of severe decline may indicate that extensive decay columns or root disease are present.
Old failures, large decayed stubs	Present	Low to moderate	Can serve as point of weakness where subsequent failure is likely to occur. May also serve as indicators of internal decay.

Table 3-3—Additional factors related to hazard potential in trees affected by sudden oak death (SOD)^a (continued)

Risk factor	Factor level	Contribution to failure potential	Additional considerations and interactions
Tree structure factors:			
Number of stems from ground	Multiple stems from ground	Low to moderate	Mainly increases failure potential in trees with dead stems, decay, and other substantial risk factors.
Multiple branches from one point	Present (especially if crowded)	Low	Mainly increases branch failure potential in trees with dead stems, decay and other substantial risk factors.
One-sided canopy	Present	Low	Mainly increases bole failure potential in trees with dead stems, decay and other substantial risk factors. Severe trunk lean can have a similar effect.
Stand factors:			
Sky-exposed canopy rating	<50 percent of canopy exposed to overhead sunlight	Low	Failure potential increases as sky exposure decreases (i.e., higher in overtopped trees). Trees with dead stems, decay, and other substantial risk factors are most likely to show increased failure risk due to this factor.
Tree neighborhood altered	Other dead or failed trees present within 2 to 3 canopy widths of tree	Low	This factor may primarily serve as an indicator of a <i>Phytophthora ramorum</i> disease cluster. Increased exposure to wind may increase failure risk in trees with dead stems, decay and other substantial risk factors.

^a Most of these factors are not strongly related to mortality risk in the near term, see also table 3-2.

These risk factors should be incorporated into the tree hazard evaluation process in areas where SOD is present and should be considered in combination with other tree failure risk factors.

Monitoring. As SOD symptoms develop over time, the likelihood of failure also changes. Monitor SOD-infected trees so they can be removed in a timely manner to minimize hazards.

A 1-year reinspection interval is a useful starting point in many situations. Of all the factors that contribute to failure risk (tables 3-2, 3-3), factors related to *P. ramorum* infection tend to change the most rapidly over time. If you collect detailed observations on individual trees in an initial baseline survey, changes in condition should be apparent when follow-up inspections are made (table 3-4).

Sources of information on tree hazard assessment methods—

Johnson, D.W. 1981. Tree hazards: recognition and reduction in recreation sites.

Tech. Rep. R2-1. U.S. Department of Agriculture, Forest Service, Rocky Mountain Region, State and Private Forestry. <http://www.na.fs.fed.us/spfo/pubs/hazardtrees/treehazards/thazards.pdf>. (27 August 2012).

Pokorney, J.D.; O'Brien, J.; Hauer, R.; Johnson, G.; Albers, J.; Bedker, P.; Mielke, M. 2003. Urban tree risk management: a community guide to program design and implementation. NA-TP-03-03. St.Paul, MN: U.S. Department of Agriculture, Forest Service, Northeastern Area, State and Private Forestry. 194 p. <http://www.na.fs.fed.us/spfo/pubs/uf/utrm/>. (27 August 2012).

National Park Service hazard tree guidelines. http://na.fs.fed.us/fhp/hazard_tree/pubs/misc/nps.htm. (27 August 2012).

Other publications on tree risk assessment are available for purchase from the International Society of Arboriculture (<http://secure.isa-arbor.com/webstore>).

3.3.2—Guidelines for Assessing Mortality and Failure Risk in Trees Affected by Sudden Oak Death

The guidelines in tables 3-2 and 3-3 can be used to augment existing tree hazard rating systems to account for additional failure risks associated with SOD. The likelihood that a tree will be killed by SOD needs to be considered because SOD-killed trees have a high risk of failure. Table 3-4 lists specific assessment methods to monitor the progression of SOD symptoms that predict survival and hazard potential.

The guidelines were developed from data on coast live oak branch failures (≥ 20 cm in diameter) and bole failures (≥ 15 cm in diameter) in SOD-affected stands in

Table 3-4—Methods for assessing factors related to mortality and failure potential in trees affected by sudden oak death (SOD)

Factor	Assessment methods	Notes
<i>Phytophthora ramorum</i> canker activity/expansion	<ul style="list-style-type: none"> • Note presence of current-year bleeding • Estimate percentage of trunk circumference affected • Note which sides of trunk are affected using compass directions (N, NE, E, etc.) • Mark edges of canker using paint or lumber crayon • Document with digital photos • Note if callus is developing along edge of older cankers 	Comparisons between annual assessments will indicate whether cankers are expanding or inactive.
Trunk decay	<ul style="list-style-type: none"> • Types of fruiting bodies present (photograph if not identified) • Estimate percentage of trunk circumference with <i>Annulohyphoxylon thouarsianum</i> or other fruiting bodies • Note location and extent of cavities, decay zones 	Decay can be extensive before fruiting bodies appear. Intensive decay-detection methods (drilling, imaging) can provide more detail, but are expensive and time-consuming. Decay fungi present can be identified via DNA testing of wood shavings from drilled holes.
Beetle colonization of trunk	<ul style="list-style-type: none"> • Estimate percentage of trunk circumference affected • Note which sides of trunk are affected using compass directions (N, NE, E, etc.) • Note presence of fresh boring dust 	Bark and ambrosia beetle boring is easiest to rate when fresh boring dust is present. Beetle holes may be difficult to see when dust has washed away, so the extent of colonization may appear to decline over time.

Marin and Napa Counties (Swiecki and Bernhardt 2003, 2010; Swiecki and others 2006). These data are from a limited range of forest types. The importance of some factors may vary geographically. Additional local observations may be needed to determine if other factors are important.

Additional risk factors are important for trees in urban areas that are subject to root disturbances, altered moisture regimes, or other conditions. For example, oaks in developed areas commonly show a much higher incidence of root failure than those in undisturbed forests. Factors associated with root failure potential should be given greater attention in areas where roots may have been compromised by excavation, construction activities, or summer irrigation.

3.4—Assessing Fire Hazard Related to Trees Killed by Sudden Oak Death

The types of fuels that develop from SOD-killed trees are influenced by multiple factors. These include how rapidly a tree has died, whether failures occurred before or after tree death, and the length of time since tree death (see also figs. 2-2 and 2-4, and “SOD-related mortality and fire” in section 2.2.3) as well as local environmental conditions. Tables 3-5 and 3-6 show examples of ground and aerial fuels that form over time in coast live oaks killed by SOD.

Table 3-7 shows example time sequences for degradation of three different trees that were killed by SOD. Degradation of trees that are dead and defoliated before failure is about 1 to 2 years advanced compared with green failures. Degradation rates are similar if the date of tree death is used as the starting time. In tables 3-5, 3-6, and 3-7, icons representing tree condition are the same as used in figures 2-2 and 2-4.

Note that all standing dead trees have an elevated risk of failure and in particular can pose a hazard to firefighting personnel.

3.5—Restoring Forests Affected by Sudden Oak Death

When developing a restoration plan, start by assessing your planting needs and existing regeneration (see “Restoration in SOD-affected forests” in section 2.2.3). Although SOD can significantly affect forests within a few years, site restoration may take many decades. First, address critical needs such as soil stabilization. Next, plan for a transition to a forest structure that provides a wider set of services and resource values.

In many sites, specific restoration goals can be achieved using various methods. Consider the pros and cons of various alternatives before deciding on restoration methods. Some factors to consider include:

Short- and long-term costs: High cost alternatives do not necessarily provide the greatest benefit or highest likelihood of success. Using existing natural regeneration can cost little and is often superior to planting. Consider costs of followup maintenance (e.g., for thinning or weed suppression) when comparing alternatives.

Long-term sustainability: Areas that will not be actively managed are best suited to techniques that do not require frequent attention. In areas that will be managed more intensively (near structures, roads, etc.), methods that require periodic inputs may be viable.

Timeframe: Critical needs (e.g., erosion protection) that need to be addressed in the short term may require the use of more intensive methods. Less intensive methods that take longer to achieve results may be preferable if conditions are fairly stable.

Table 3-5—Changes in aerial fuels characteristics of standing dead coast live oaks killed by sudden oak death

Factor	Level	Icon	Photo
Fuel stage	Dead1. Standing dead with leaves		
Time period	1 to 2 years after tree death		
Primary fuel type(s)	Aerial		
Fire effects	Canopy readily ignited, will serve as source of embers		
Fuel stage	Dead2. Standing dead without leaves with fine branches		
Time period	2 to 3+ years after tree death		
Primary fuel type(s)	Aerial		
Fire effects	Canopy may ignite if sufficiently low and dense. Failure risk is high		
Fuel stage	Dead3. Standing dead with branch failures, some fine branches present		
Time period	2 to 4+ years after tree death		
Primary fuel type(s)	Surface, aerial		
Fire effects	Piles of failed branches (failed2 to failed3) increase depth of ground fuel layer, can form fuel ladders to connect ground and aerial fuels. Aerial fuels may be too sparse to ignite readily but are highly prone to failure		

Table 3-5—Changes in aerial fuels characteristics of standing dead coast live oaks killed by sudden oak death (continued)

Factor	Level	Icon	Photo
Fuel stage	Dead4. Snag, large-diameter stems failed		
Time period	4 to 6+ years after tree death		
Primary fuel type(s)	Surface		
Fire effects	Large standing stems do not easily ignite, but are prone to fail. Debris piles are fuel stage B3 to B4, dominated by large diameter branches that may be mostly decayed. Fine fuels are decayed and in or near duff layer		

Table 3-6—Changes in ground fuel characteristics of failed coast live oaks killed by sudden oak death

Factor	Level	Icon	Photo
Fuel stage	Failed1. Failed tree or branch with leaves		
Time period	1 to 2 years after tree death or green failure		
Primary fuel type(s)	Surface		
Fire effects	Very readily ignited, can serve as fuel ladder to carry surface fire to canopy		
Fuel stage	Failed2. Large-diameter stems, fine branches, few to no leaves		
Time period	2 to 4 years after tree death or green failure		
Primary fuel type(s)	Surface		
Fire effects	Readily ignited, can serve as ground-to-canopy fuel ladder		
Fuel stage	Failed3. Primarily large-diameter branches, fine fuels mostly rotted and in or near duff layer		
Time period	3 to 8+ years after tree death or green failure		
Primary fuel type(s)	Surface		
Fire effects	Increases total ground fuels, but debris piles are generally low, typically no more than 1 m tall		
Fuel stage	Failed4. Decayed large-diameter stems, all small branches decayed		
Time period	6 to 10+ years after tree death or green failure		
Primary fuel type(s)	Surface		
Fire effects	Minimal contribution to ground fuels		

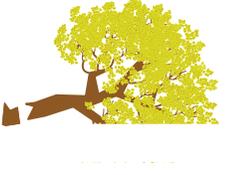
Table 3-7—Time sequences of degradation of aerial and ground fuels in three coast live oaks killed by sudden oak death

Icon	Photo	Description
		<p>Tree crown in year of tree death. Fuel stage Dead1.</p>
		<p>One year later. Fuel stage is intermediate between Dead1 and Dead2. Several large branches have failed.</p>
		<p>Two years later. By 2 years after death, this tree has progressed to fuel stage Dead3. (Marin County)</p>

Table 3-7—Time sequences of degradation of aerial and ground fuels in three coast live oaks killed by sudden oak death (continued)

Icon	Photo	Description
		<p>Tree died and was defoliated before the trunk failed 2 years later. Fuel stage Failed2.</p>
		<p>One year later. Fuel stage intermediate between Failed2 and Failed3</p>
		<p>Three years later. Fuel stage Failed3. (Marin County)</p>

Table 3-7—Time sequences of degradation of aerial and ground fuels in three coast live oaks killed by sudden oak death (continued)

Icon	Photo	Description
		<p>Large tree about 1 to 2 months after live failure. Fuel stage Failed1.</p>
		<p>Three years after failure. Fuel stage Failed3. Note sprout growth from stump.</p>
		<p>Eight years after failure. Fuel stage still Failed3. (Marin County)</p>

Potential for nontarget effects: When more intensive methods are used, unintended effects may be possible. For example, soil disturbance associated with clearing or grading may increase erosion potential and promote weed establishment.

3.5.1—Species Selection

The term “restoration” implies a return to a previous condition. Because forest composition changes over time, various forest types may have occupied a site in the past. Forest stands in most of California have been substantially altered by human activity over the past two centuries. Because of tree cutting, changes in fire frequency, grazing, and other cultural practices, current stand composition and structure may differ from conditions that existed prior to European/American settlement. For example, tanoak-dominated stands in some areas developed after clearcutting of redwood/Douglas-fir forests in the mid-20th century.

Reliable information about the former forest cover can tell you what once grew in an area. Also consider how site conditions may have changed since that forest type was established. In addition to the presence of *P. ramorum*, sites may be substantially changed in other ways, including:

- Increased soil erosion from clearing and burning.
- Litter and debris accumulation from tree mortality.
- Increased growth of competitive, nonnative weedy species.
- Hotter, drier microclimate resulting from the loss of overstory species.
- Increased browsing by herbivores (deer, rodents, livestock).

Such changes may inhibit the establishment of species that once occupied the site. Some of these factors can be addressed by management actions. For instance, surface litter can be removed or exclosures can be used to reduce herbivory. These interventions typically increase costs. Other constraints that cannot be eliminated need to be dealt with through appropriate species selection. For example, warmer, drier conditions associated with microclimate change may render sites unsuitable for species that were only marginally suited to the area in the past.

Use of SOD canker hosts for restoration—

Under some conditions, it may be feasible to replace SOD-killed oaks with the same species. If the stand will be managed to eliminate California bay and any other significant sources of *P. ramorum* spores, SOD-killed oaks may be replaced with susceptible species such as coast live oak, California black oak, and canyon live oak.

Although tanoaks vary in their susceptibility to *P. ramorum*, highly SOD-resistant tanoaks have not yet been identified. Until tanoaks with useful levels of SOD resistance can be identified, regenerated tanoak stands will continue to be at

risk. Consider replacing tanoaks lost to SOD with other species. It may be possible to include tanoak in regenerated stands as widely separated trees, which have a reduced risk of sustaining a lethal *P. ramorum* infection.

Choosing replacement species—

In SOD-affected stands where California bay is abundant, killed SOD canker hosts (tanoak and susceptible oaks) need to be replaced with species that are not affected by *P. ramorum*. Replacement species that will dominate the stand must be well adapted to the site and stocked at an overall density that is sustainable over time.

Both canopy cover and tree species characteristics need to be considered when designing restoration projects to specific restoration goals (table 3-8). Visual considerations may be especially important when choosing trees to plant around homes. For example, replacing shorter oaks with tall conifers may obstruct views or sunlight. Where visual screening is desired, evergreen species will be more effective than deciduous trees. Deciduous blue or valley oaks provide many of the same ecosystem services as SOD-susceptible coast live oaks, but do not provide the same level of visual screening during the winter. Various combinations of species and canopy cover levels can be used to meet multiple goals, but a single forest structure is not likely to fulfill all goals.

Growing conditions can change over relatively short distances because of factors such as soil type and depth, topographic position, slope, and aspect. Deep soil at the base of a slope near a seasonal creek provides a different growing environment than an adjacent west-facing slope with shallow soils. Some SOD canker hosts, such as coast live oak, tolerate a broad range of site conditions. Multiple replacement species may be needed to fill the sites previously occupied by a single SOD-killed species.

Integrating restoration with other land management goals and practices—

Organizations that manage large areas of forest and other natural habitats (e.g., park and open space districts, land trusts, water districts) typically have overall land management goals that apply across their holdings. When setting goals for restoration of SOD-affected sites, resource managers need to consider how the restored sites will function in the context of broader land management goals.

Individual private landowners commonly manage smaller parcels. These parcels occur within the context of a wider landscape that can influence restoration project outcomes. For example, invasive exotic plants may be more difficult to suppress if adjoining lands are heavily infested. Where possible, coordinate management efforts with adjacent landowners.

Table 3-8—Levels of canopy cover and tree characteristics needed to achieve various restoration goals

Restoration goal	Canopy cover	Tree characteristics ^a
Soil stabilization	High	Fast establishment, wide fibrous root system, evergreen
Moderation of stormwater flows	High	Evergreen or deciduous with high leaf surface area during rainy season
Maintenance or improvement of wildlife habitat and biodiversity	Varies, diversity of cover levels may be important	Provides food (e.g., acorns, insects), cover, nesting sites
Maintenance or improvement of native plant habitat and biodiversity	Varies, diversity of canopy cover densities may be important	More open canopies allow for better development of many understory plants
Suppression of undesirable invasive species	High	Dense canopy or allelopathic (suppressive) properties
Visual screening	Varies	Evergreen trees with high canopy density at desired height range
Aesthetics/property value enhancement	Varies	Varies
Shading for energy conservation	High in areas near structures	Deciduous trees with relatively early leaf drop and late leaf emergence
Minimize fire hazard in defensible space	Low in firebreak area; low to moderate in reduced fuel zone	Relatively low flammability, high canopy with clear lower trunk, low litter production

^a Understory species (shrubs, herbaceous plants) may also be consistent with specific goals, but will not restore forest canopy cover.

Most small-scale restoration projects do not involve activities that are addressed by various federal, state, and local environmental and land use regulations. However, larger projects that involve intensive inputs such as land clearing, grading, or burning may require agency permits or approvals. Landowners should check with appropriate local and state agencies before undertaking these activities. To comply with state and local regulations and guidelines related to fire hazard, vegetation in defensible space areas must be managed appropriately. Proper plant selection, placement, and maintenance will help minimize fire hazard in these areas.

3.5.2—Factors That Constrain Regeneration

Various factors can constrain regeneration of oaks and other species in SOD-affected forests (table 3-9). Restoration inputs are designed to overcome these constraints. You can minimize cost and effort by using only those management inputs needed to address the site’s constraints.

Table 3-9—Factors that limit regeneration of tree species and possible remedies

Factor	Constraints on regeneration	Possible remedies
Seed production	<ul style="list-style-type: none"> • Seed production varies widely between individual trees and years. • Oaks and other outcrossing, wind-pollinated species may not set seed reliably if trees are widely separated. • Oak and tanoak acorns do not store well and must be planted within a few months of collection. 	<ul style="list-style-type: none"> • Plan to collect and plant seed in multiple years. • Expand area of seed collection as needed to find adequate numbers of seed trees.
Seed dispersal	<ul style="list-style-type: none"> • For heavy seeds such as acorns, dispersal is primarily by gravity and most seed is deposited near the parent tree. Many species with small seeds have adaptations that allow seed to be dispersed longer distances via wind. • Animals can move seeds relatively long distances, but resulting dispersal patterns are nonuniform. 	<ul style="list-style-type: none"> • Plant if needed to obtain desired species mix.
Seedbed conditions	<ul style="list-style-type: none"> • Acorns lose viability upon drying, so lack of organic debris on the soil surface may greatly reduce germination and seedling establishment. • Some species with small seeds are adapted to germinate on bare mineral soils and will not tolerate deep duff layers. • Soil compaction can reduce root growth, leading to poor establishment in many species. 	<ul style="list-style-type: none"> • Adjust seedbed (e.g., increase or decrease litter layer) as needed to favor target species. • Till soil in areas where compaction is excessive.
Damage from herbivores	<ul style="list-style-type: none"> • Livestock, deer, rodents, and insects can damage seeds and seedlings. • In areas with high herbivore pressure, regeneration of preferred palatable species can be completely suppressed while reproduction of less palatable species is favored. • Herbivore damage varies seasonally and from year to year because of changes in herbivore populations, alternative food availability, and plant maturity. • Impacts of herbivory are most serious on small seedlings and saplings. 	<ul style="list-style-type: none"> • Use fencing or individual plant protection (wire cages, tree shelters, etc.) to exclude herbivores through the plant establishment period. • Modify or suspend livestock access to restoration site until tops of plants are well above browse line (the height that grazing animals can reach). • Modify habitat to disfavor damaging agents and increase populations of natural enemies (e.g., mowing to increase predation of rodents by raptors).
Diseases	<ul style="list-style-type: none"> • <i>Phytophthora ramorum</i> has potential to reduce seedling growth and survival for species that are subject to foliar and twig blighting (e.g., tanoak). • Soilborne plant pathogens and some foliar diseases have the potential to reduce seedling growth and survival. 	<ul style="list-style-type: none"> • Avoid using species that are susceptible to diseases present on site. • Use only nursery stock that is certified or tested to be free of <i>Phytophthora</i> spp. and other significant pathogens. • Increase species diversity to minimize overall losses caused by a single pathogen.

Table 3-9—Factors that limit regeneration of tree species and possible remedies (continued)

Factor	Constraints on regeneration	Possible remedies
Soil moisture	<ul style="list-style-type: none"> • Levels of soil moisture, especially in late summer, limit species survival. • Species that are relatively tolerant of low soil moisture when mature (e.g., many oaks) may be killed by drought during the first few seasons of establishment. • Drought cycles or successive years of heavy precipitation may strongly influence plant establishment. • High plant densities increase competition for limited amounts of soil moisture. • The total amount of plant-available water in soils varies by soil type. Available water is lowest in coarse or sandy soils, greatest in loam and clay loam soils. • Available water increases with increasing soil depth (to bedrock or impermeable layers) and increasing organic matter content. • Shallow water tables or surface flows can increase plant available water. • Organic mulch on the soil surface helps retard evaporative water loss. 	<ul style="list-style-type: none"> • Match species' moisture needs to the local soil properties. • Plan to restock over multiple years to take advantage of wetter seasons and avoid drought periods. • Control weeds to maximize available soil moisture. • Thin to conserve soil moisture. • If needed, maintain an organic mulch/litter layer to conserve soil moisture.
Soil erosion	<ul style="list-style-type: none"> • Soils prone to erosion may be too unstable to establish slow-growing species. • Excessive topsoil loss reduces soil water-holding capacity and available plant nutrients. Severe erosion may render the site unsuitable for species that previously occupied the site. 	<ul style="list-style-type: none"> • Use temporary vegetation, mulch, and other soil stabilization methods to minimize erosion while target species are becoming established. • Modify species selection to match species tolerance to current site conditions.
Solar radiation	<ul style="list-style-type: none"> • The amount of light needed for seedling establishment and growth differ by species. Some species establish better under partial to complete canopy cover, others require full sunlight. • Species' shade tolerance can vary with age. Many species tolerate less shade as they mature. • Shade reduces water use, and may partly compensate for limited soil moisture. • Solar radiation is lowest on north-facing slopes and greatest on south- and west-facing slopes. 	<ul style="list-style-type: none"> • Select species with light tolerance that matches site conditions. • Plan for successional changes in vegetation as tree growth over time changes available light. • Start restoration activities before all tree canopies are killed to provide shade for seedling establishment. • Thin or prune as needed to maintain appropriate light levels for target species.

3.5.3—Natural Regeneration

Most sites support natural regeneration of various herbaceous and woody species. Natural regeneration may be so plentiful that thinning is needed to achieve target plant densities. If natural regeneration of preferred species is sparse, you may need to favor it through cultural inputs, such as protection from browsing animals or reducing competition from fast-growing exotic or native plants (fig. 3-8).

Potential regeneration may be present as dormant seed in the soil or as incoming seed transported to the site by wind, water, or animals. Canopy gaps caused by SOD-related mortality can have an altered environment (light, temperature, soil moisture) that favors germination of dormant or incoming seeds. Consequently, species that recruit in gaps from these seeds may have been absent before the gaps developed.

Existing seedlings and saplings are a common source of natural regeneration. Oaks and many other forest trees have short-lived seeds. These seeds germinate within a year, and seedlings develop to a limited degree. Seedlings remain small because overstory trees limit their access to light and soil moisture. These persistent seedlings (also known as advance regeneration or a seedling bank) may grow rapidly when overstory trees die or are removed.

Stump sprouts are another potential source of natural regeneration. Many species found in SOD-affected forests, including most oaks, tanoaks, California bay, and coast redwood, commonly resprout from stumps. SOD-killed oaks and tanoaks may produce basal sprouts that can give rise to a new tree. However, such sprouts are clones of the susceptible parent and therefore SOD-susceptible. The resulting trees are likely to develop SOD cankers eventually if a source of *P. ramorum* spores remains in the area. Sprout-origin oaks can also have poor structural properties. Decay associated with the old stump increases failure potential and reduces the useful life of these trees. However, stump sprouts grow fast because of their established root systems. In some situations, stump sprouts can provide fast, if temporary, replacement tree canopy.

3.5.4—Planting

Where existing regeneration is insufficient or does not include the preferred species to meet restoration goals, planting may be necessary. The optimum type of planting material varies with species and site conditions. The USDA Forest Service and other organizations have developed a wide variety of materials related to forest tree planting and silviculture (see below “Sources of information on tree planting and silviculture”). For California forest species, more information is available for commercially utilized conifers than for noncommercial hardwoods such as native oaks.



Figure 3-8—Madrone and Douglas-fir regeneration in a canopy gap caused by sudden oak death (SOD) shown in September 2008 (top) and October 2010 (bottom). The gap formed when a large coast live was killed by SOD in 2002 and failed 2 years later. Without management, the faster growing Douglas-fir regeneration is likely to outcompete the madrone.

Direct seeding—

For large-seeded species such as oaks, direct planting of seed at the restoration site can be effective and cost-efficient. Direct seeding avoids various problems that may be associated with nursery stock (table 3-10). Timing of seed collection, storage conditions, and planting methods differ by species.

Transplanting natural seedlings—

For small-scale projects, it may be possible to locate and transplant naturally occurring seedlings during the late fall or winter. This requires a plentiful supply of nearby seedlings growing in soil that is not excessively rocky. Seedlings must be small enough that the root system can be easily dug up and moved. Species differ in their ability to survive transplanting. It is possible to transplant large saplings using a tree spade (a truck-mounted device that can move a sapling tree or shrub along with a relatively large volume of soil), but this method is expensive and typically limited to use in developed areas.

Locally collected natural seedling transplants have the advantage of being from the local plant population. They are normally colonized by local species of mycorrhizal fungi, which are important for plant growth and survival. However, locating and transplanting natural seedlings can be time consuming and will disturb the seedling source site. Deleterious organisms, such as root disease pathogens, may also be moved with transplants. If the source site is very close to the restoration site, the risk of introducing new pathogens may be low. Nonetheless, seedling source sites should be inspected for evidence of disease problems before they are selected. Testing of plants or soil may be appropriate to determine if soilborne *Phytophthora* species or other pathogens are present before transplanting natural seedlings.

Nursery-grown transplant stock—

Nursery-grown planting stock is commonly used in forestry and environmental restoration. However, nursery stock can pose a high risk of introducing exotic pathogens, especially *Phytophthora* species, into forested areas.

- The initial introduction and spread of *P. ramorum* in California was associated with movement of contaminated nursery stock (Mascheretti and others 2008).
- The exotic pathogen *Phytophthora lateralis*, which causes a lethal root rot of Port-Orford-cedar (*Chamaecyparis lawsoniana* (A. Murray bis) Parl.), was apparently introduced into forests of Oregon and California via contaminated nursery stock (Hansen and others 2000).

Table 3-10—Comparisons between direct seeding and nursery-grown container transplants for restocking

Factor	Seed	Transplants
Relative cost	Low. Local seed reduces transportation and handling costs.	High. Production requires seed collection and seedling maintenance until outplanting.
Storage	Low space requirements. Short-lived seed such as acorns can typically be stored for only a few months. Other seed may remain viable for many years under proper storage conditions.	High space requirement because of the size of the containers. Plants may be held for multiple years under good growing conditions, but more space and larger containers will be needed as plants grow.
Planting	Relatively fast and easy to plant large number of sites.	Greater effort required for planting, especially if many sites are involved or larger stock is used.
Postplanting success	Varies by species and site conditions. Overplanting can be used to obtain adequate survival. For species such as oaks, high rates of germination and establishment can be obtained.	Varies with stock quality and site conditions. Transplants are often highly susceptible to dry conditions after planting and irrigation may be needed to ensure establishment. High rates of success can be obtained with many commercial forest species with proper site preparation.
Root structure	Root structure adapts to soil conditions as seed germinates and grows. Taproot development typically good.	Root system is modified strongly by container and is more restricted, requiring adaptation after transplanting. Taproot structure of species with strong taproots (e.g., many oaks) is distorted or eliminated. Root form problems may develop in the container or as a result of poor planting technique.
Shoot structure	Shoots typically develop in proper balance with roots and show adaptations to site conditions.	Shoot-to-root ratio may be artificially high, leading to water stress after planting. Shoots may be thin and mechanically weak if plants are crowded in the nursery.
Local germplasm	Can be obtained by collecting seed in and near restoration site.	Seed source is unlikely to be local unless custom propagation is done, requiring adequate lead time for growing stock.
Introduction of pathogens or insect pests	Typically not an issue for locally collected seed.	Can pose high risk for introduction of soil diseases such as <i>Phytophthora</i> spp. and movement of some insect pests.

- In Bavaria (Germany), nursery stock used for reforestation was the source of a now-widespread *Phytophthora* root and crown rot of alders in reforested stands (Jung and Blaschke 2004).
- The soil-borne root pathogen *Phytophthora cinnamomi* is a serious forest disease in various parts of the world. In recent years, *P. cinnamomi* and some other exotic root-infecting *Phytophthora* species have been associated with mortality of forest trees and native shrubs at numerous northern California locations. Contaminated nursery plants appear to be the likely source of these pathogens at a number of the infested wildland sites. *Phytophthora cinnamomi* and other root-rotting *Phytophthora* species are common in nursery stock produced in native plant nurseries and other commercial nurseries. (Fichtner and others 2010, Swiecki and others 2011).

From these examples, it is clear that nursery stock infested with any *Phytophthora* species should not be planted in or adjacent to natural areas. Unfortunately, most nurseries, including native plant nurseries, have not taken necessary steps to ensure that stock they produce is free of pathogenic *Phytophthora* species.

The U.S. Nursery Certification Program (USNCP, see http://www.aphis.usda.gov/plant_health/acns/certification.shtml) is a pilot, voluntary phytosanitary certification program for U.S. nurseries that ship nursery stock to Canada. The phytosanitary standards include “practical freedom from nonregulated plant pests,” which would include many *Phytophthora* species. The *Safe Procurement and Production Manual* (Griesbach and others 2012) provides checklists, best management practices (BMPs), and information related to obtaining USNCP certification. Nurseries that adhere to the BMPs listed in this manual are highly likely to produce nursery stock that is free of *Phytophthora* species and other serious pests and diseases.

If a nursery does not have USNCP certification, an inspection of the nursery production facilities can show whether the cultural practices are sufficient to produce clean stock. To produce plants free from *P. cinnamomi* and other *Phytophthora* species, nurseries need to follow phytosanitary procedures throughout the entire propagation cycle. These principles apply equally to large and small nurseries, including backyard propagators.

Clean nursery production is possible only if pathogen-free soil, disease-free propagation stock, and clean containers are used. Nursery-grown plants may become infested with *Phytophthora* species through the use of recirculated irrigation water and movement of infested soil via hoses, tools, pots, hands, shoes, and vehicles. To avoid contamination, do not place pots and flats directly on soil. Routinely disinfest benches, pots, tools, and other items used for propagation prior to each use.

Unless a nursery follows a systems approach to prevent contamination through the entire production cycle, planting stock can eventually become infested. Older, larger container planting stock is more likely to be infested with *Phytophthora* species than younger, smaller container stock. Liner stock grown in the ground (e.g., bareroot stock) may be infested at any age if the production field is contaminated.

The presence of *Phytophthora* infection can be masked in fungicide-treated plants. Fungicide treatment can prevent disease symptoms but will not free plants and container soil of *Phytophthora* spores. *Phytophthora* activity increases and symptoms develop as fungicide residues decline. Nursery plants should be set aside, prior to planting, without fungicide treatment for at least 3 months to reveal symptoms of any existing *Phytophthora* infestations.

Genetic considerations—

If onsite natural regeneration is inadequate for accomplishing restoration goals, the next best source of planting material is locally collected seed or planting stock derived from it (table 3-11). Local germplasm represents the result of natural selection under local soil and climate conditions. Trees growing from locally collected seed should be adapted to site conditions and are likely to perform well over time (Millar and Libby 1991).

It is important to realize that selection pressure can be high at the seedling stage under natural conditions. High rates of seedling mortality are common in nature. Seedling mortality can exert selection pressure for characteristics that are important for early survival. Such characteristics are likely to be important for long-term stand sustainability. If intensive inputs are used to ensure high rates of seedling survival, this type of adaptive selection is minimized or avoided. Consider overplanting with inexpensive planting material (e.g., seed) and allowing natural selection to function rather than using intensive inputs to ensure the survival of large expensive stock.

Sources of information on tree planting and silviculture—

McCreary, D. 2009. Regenerating rangeland oaks in California. Publication 21601e.

Oakland, CA: University of California, Agriculture and Natural Resources Publication Services. 62 p. <http://anrcatalog.ucdavis.edu/Items/21601e.aspx>. (7 December 2012).

Burns, R.M.; Honkala, B.H., tech, coords. 1990. Silvics of North America, 1.

Conifers. 2. Hardwoods. Agric. Handb. 654. Washington, DC: U.S. Department of Agriculture, Forest Service. 877 p. http://www.na.fs.fed.us/spfo/pubs/silvics_manual/table_of_contents.htm. (27 August 2012).

Table 3-11—Recommendations for the management of genetic resources in forest restoration projects

Recommendation	Notes
Use existing regeneration	Conserve existing seedling regeneration that has been exposed to selective pressures.
Use local seed	For trees such as oaks and tanoaks, trees within the same watershed or located within a few to about 10 km of the restoration site may be considered to be within the local seed zone. Wider local zones may apply for species that disperse seed and pollen more widely.
Match seed source and planting site characteristics	When collecting seed within or outside of the local seed zone, match the microclimate (e.g., north slope) and soil characteristics (type and depth) of the seed source site to that of the target restoration site to the degree possible. It may be desirable to collect seed from sites that are hotter and drier than the target site to provide a potentially wider range of climate adaptation variation.
Ensure genetic diversity in seed collections	Seed collected for planting should be from a large number of individuals spread widely over the local seed zone. Because of variation in seed production between individuals from year to year, seed collection and planting should be conducted over a series of years.
Avoid genetic pollution	Avoid collecting seed from trees likely to be pollinated by horticultural specimens of unknown origin. Do not plant stock (nursery-grown or transplanted) of non-local or unknown origin within about 1 to 3 km of existing native stands in areas to prevent spread of pollen and seed from nonlocal genotypes into native stands.
Let natural selection function	Allow for attrition resulting from natural selective pressures in restoration plantings. Plant seeds or the smallest planting stock size available and plan for relatively high rates of seedling mortality so that survival will be largely limited to the best-adapted genotypes.

3.5.5—Monitoring Restoration Outcomes

Restoration of forests affected by SOD is largely experimental at this time. Through careful monitoring of your restoration projects, you can compile the data needed to determine the success of various techniques under the range of conditions that occur at your site. The locations of various treatments applied in each year (planting, weed control, etc.) can be documented using maps, geographic coordinates, or permanent field markers (e.g., metal tags or stakes). Photos taken from specific locations (photo points) are also useful for documenting locations and changes over time.

Include information on factors that limit growth or survival in your monitoring data. These include soil conditions (texture, moisture, mulch), weather (precipitation, temperatures), shading, plant competition, browsing by livestock or wildlife, diseases, insect injury, and other damage agents (e.g., fire). Some of these constraints can be addressed by cultural inputs (e.g., fencing to exclude herbivores, thinning, weed control). If factors such as an extended drought or fire are limiting, it may be necessary to switch to species that will better tolerate these adverse conditions.

Initial seedling and sapling survival rates are commonly the focus of restoration project monitoring. However, high seedling survival in the first few years of a project does not guarantee long-term survival. Continued monitoring is needed to determine whether the restoration project is succeeding, and if not, what additional steps are needed.

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English Equivalentents

When you know:	Multiply by:	To find
Centimeters (cm)	0.394	Inches
Meters (m)	3.28	Feet
Millimeters (mm)	0.0394	Inches
Kilometers (km)	0.6215	Miles
Square meters (m ²)	10.76	Square Feet (ft ²)
Grams (g)	0.352	Ounces
Pounds per gallon	1.717	Kilograms per liter
Degrees Celsius (°C)	1.8 and add 32	Degrees Fahrenheit

References

- Anacker, B.L.; Rank, N.E.; Hüberli, D.; Garbelotto, M.; Gordon, S.; Harnik, T.; Whitkus, R.; Meentemeyer, R. 2008.** Susceptibility to *Phytophthora ramorum* in a key infectious host: landscape variation in host genotype, host phenotype, and environmental factors. *New Phytologist*. 177: 756–766.
- Croucher, P.J.P.; Mascheretti, S.; Garbelotto, M. 2013.** Combining field epidemiological information and genetic data to comprehensively reconstruct the invasion history and the microevolution of the sudden oak death agent *Phytophthora ramorum* (Stramenopila: Oomycetes) in California. *Biological Invasions*. 2013: 1–17.
- Davidson, J.M.; Patterson, H.A.; Rizzo, D.M. 2008.** Sources of inoculum for *Phytophthora ramorum* in a redwood forest. *Phytopathology*. 98: 860–866.
- Davidson, J.M.; Shaw, C.G. 2003.** Pathways of movement for *Phytophthora ramorum*, the causal agent of sudden oak death. Sudden oak death online symposium. DOI: 10.1094/SOD-2003-TS.
- Davidson, J.M.; Wickland, A.C.; Patterson, H.A.; Falk, K.R.; Rizzo, D.M. 2005.** Transmission of *Phytophthora ramorum* in mixed evergreen forest in California. *Phytopathology*. 95: 587–596.
- Duniway, J.M. 1976.** Movement of zoospores of *Phytophthora cryptogea* in soils of various textures and matric potentials. *Phytopathology*. 66: 877–882.
- Englander, L.; Browning, M.; Tooley, P.W. 2006.** Growth and sporulation of *Phytophthora ramorum* *in vitro* in response to temperature and light. *Mycologia*. 98: 365–373.

- Fichtner, E.J.; Rizzo, D.M.; Swiecki, T.J.; Bernhardt, E.A. 2010.** Emergence of *Phytophthora cinnamomi* in a sudden oak death-impacted forest. In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M., tech. coords. Proceedings of the sudden oak death fourth science symposium. Gen. Tech. Rep. PSW-GTR-229. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 320–321.
- Garbelotto, M.; Davidson, J.M.; Ivors, K.; Maloney, P.E.; Hüberli, D.; Koike, S.T.; Rizzo, D.M. 2003.** Nonoak native plants are main hosts for sudden oak death pathogen in California. California Agriculture. 57: 18–23. <http://nature.berkeley.edu/garbelotto/downloads/garbelotto2003a.pdf>. (7 December 2012).
- Garbelotto, M.; Schmidt, D.J. 2009.** Phosphonate controls sudden oak death pathogen for up to 2 years. California Agriculture. 66: 10–17.
- Garbelotto, M.; Schmidt, D.J.; Harnik, T.Y. 2007.** Phosphite injections and bark application of phosphite + Pentrabark™ control sudden oak death in coast live oak. Arboriculture & Urban Forestry. 33: 309–17.
- Griesbach, J.A.; Parke, J.L.; Chastagner, G.A.; Grünwald, N.J.; Aguirre, J. 2012.** Safe procurement and production manual: a systems approach for the production of healthy nursery stock. Wilsonville, OR: Oregon Association of Nurseries. 98 p. <http://oan.org/associations/4440/files/pdf/SafeProduction.pdf>. (7 December 2012).
- Guest, D.; Grant, B. 1991.** The complex action of phosphonates as antifungal agents. Biological Review. 66: 159–187.
- Hansen, E. 2008.** Rethinking *Phytophthora*—research opportunities and management. In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M., tech. coords. Proceedings of the sudden oak death third science symposium. Gen. Tech. Rep. PSW-GTR-214. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 5–14. <http://www.treesearch.fs.fed.us/pubs/29767>. (7 December 2012).
- Hansen, E.M.; Goheen, D.J.; Jules, E.S.; Ullian, B. 2000.** Managing Port-Orford-cedar and the introduced pathogen *Phytophthora lateralis*. Plant Disease. 84: 4–14.

- Hansen, E.M.; Parke, J.L.; Sutton, W. 2005.** Susceptibility of Oregon forest trees and shrubs to *Phytophthora ramorum*: a comparison of artificial inoculation and natural infection. *Plant Disease*. 89: 63–70.
- Ivors, K.; Garbelotto, M.; DeVries, I.; Ruyter-Spira, C.; Te Hekkert, B.; Rosenzweig, N.; Bonants, P. 2006.** Microsatellite markers identify three lineages of *Phytophthora ramorum* in US nurseries, yet single lineages in US forest and European nursery populations. *Molecular Ecology*. 15: 1493–1505.
- Jung, T.; Blaschke, M. 2004.** *Phytophthora* root and collar rot of alders in Bavaria: distribution, modes of spread and possible management strategies. *Plant Pathology*. 53: 197–208.
- Kanaskie, A.; Hansen, E.; Sutton, W.; Reeser, P.; Choquette, C. 2010.** Aerial application of Agri-Fos[®] to prevent sudden oak death in Oregon tanoak forests. In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M. Proceedings of the sudden oak death fourth science symposium. Gen. Tech. Rep. PSW-GTR-229. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 225–232.
- Kelly, M.; Tuxen, K.A.; Kearns, F.R. 2012.** OakMapper.
<http://www.oakmapper.org>. (13 July 2012).
- Kliejunas, J.T. 2010.** Sudden oak death and *Phytophthora ramorum*: a summary of the literature. Gen. Tech. Rep. PSW-GTR-234. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station. 181 p.
- Lee, C.; Valachovic, Y.; Garbelotto, M. 2010.** Protecting trees from sudden oak death before infection. Publication 8426. Berkeley, CA: University of California, Agriculture and Natural Resources. 14p.
- Maloney, P.E.; Lynch, S.C.; Kane, S.F.; Jensen, C.E.; Rizzo, D.M. 2005.** Establishment of an emerging generalist pathogen in redwood forest communities. *Journal of Ecology*. 93(5): 899–905.
- Manos, P.S.; Cannon, H.C.; Oh, S-H. 2008.** Phylogenetic relationships and taxonomic status of the paleoendemic Fagaceae of Western North America: recognition of a new genus, *Notholithocarpus*. *Madroño*. 55(3): 181–190.
- Mascheretti, S.; Croucher, P.; Vettraino, A.; Prospero, S.; Garbelotto, M. 2008.** Reconstruction of the sudden oak death epidemic in California through microsatellite analysis of the pathogen *Phytophthora ramorum*. *Molecular Ecology*. 17: 2755–2768.

- Meshriy, M.; Hüberli, D.; Harnik, T.; Miles, L.; Reuther, K.; Garbelotto, M. 2006.** Variation in susceptibility of *Umbellularia californica* (bay laurel) to *Phytophthora ramorum*. In: Frankel, S.J.; Shea, P.J.; Haverty, M.I., tech. coords. Proceedings of the sudden oak death second science symposium: the state of our knowledge. Gen. Tech. Rep. PSW-GTR-196. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 87–89.
- Meyers, K.J.; Swiecki, T.J.; Mitchell, A.E. 2007.** An exploratory study of the nutritional composition of tanoak (*Lithocarpus densiflorus*) acorns after potassium phosphonate treatment. *Journal of Agricultural and Food Chemistry*. 55: 6186–6190.
- Millar, C.I.; Libby, W.J. 1991.** Tree planting: not a simple solution. *The Environmental Professional*. 13: 289–290.
- Moralejo, E.; Denman, S.; Beales, P.; Webber, J. 2007.** The ability of key tree and non-tree hosts to support inoculum production of *P. ramorum*. RAPRA Deliverable Report 27. <http://rapra.csl.gov.uk>. (14 March 2012).
- Nixon, K.C. 1993.** Infrageneric classification of *Quercus* (Fagaceae) and typification of sectional names. *Annals of Forest Science*. 50: 25s–34s.
- Parke, J.L.; Oh, E.; Voelker, S.; Hansen, E.M.; Buckles, G.; Lachenbruch, B. 2007.** *Phytophthora ramorum* colonizes tanoak xylem and is associated with reduced stem water transport. *Phytopathology*. 97: 1558–1567.
- Parke, J.L.; Stamm, E.; Oguchi, A.; Fichtner, E.; Rizzo, D.M. 2008.** Viability of *Phytophthora ramorum* after passage through slugs. *Phytopathology*. 98: S121.
- Riley, K.L.; Chastagner, G.A. 2011.** First report of *Phytophthora ramorum* infecting mistletoe in California. *Plant Health Progress*. DOI:10.1094/PHP-2011-0209-02-BR. <http://www.fs.fed.us/psw/publications/uesd/psw.2011.riley.PhytophthoraRamorumInfectingMistletoeCA.PHP.pdf>. (11 April 2012).
- Rizzo D.M.; Garbelotto, M.; Davidson, J.M.; Slaughter, G.W.; Koike, S.T. 2002.** *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and *Lithocarpus densiflorus* in California. *Plant Disease*. 86: 205–214.
- State Board of Forestry and Fire Protection, California Department of Forestry and Fire Protection. 2006.** General guidelines for creating defensible space. http://www.fire.ca.gov/cdfbofdb/PDFS/4291finalguidelines2_23_06.pdf. (14 March 2012).

- Svihra, P. 2001.** Diagnosis of SOD: case study of a scientific process. California Agriculture. 55(1): 12–13.
- Swiecki, T.J.; Bernhardt, E. 2003.** Relationships between *Phytophthora ramorum* canker (sudden oak death) and failure potential in coast live oak. Vacaville, CA: Phytosphere Research. 57 p. http://phytosphere.com/publications/P_ramorum_coast_live_oak_failure_report_Oct_2002.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E.A. 2004.** *Phytophthora ramorum* canker (sudden oak death) in coast live oak and tanoak: factors affecting disease risk, disease progression, and failure potential. 2003–2004 contract year annual report. Vacaville, CA: Phytosphere Research. 35 p. [http://phytosphere.com/publications/2003–2004_Case-control_Pramorum_annual_report.pdf](http://phytosphere.com/publications/2003-2004_Case-control_Pramorum_annual_report.pdf). (27 August 2012).
- Swiecki, T.J.; Bernhardt, E.A. 2005.** *Phytophthora ramorum* canker (sudden oak death) in coast live oak and tanoak, 2000–2004: factors affecting disease risk, disease progression, and failure potential. 2004–2005 contract year annual report. Vacaville, CA: Phytosphere Research. 41 p. http://phytosphere.com/publications/2004-2005_Case-control_Pramorum_annual_report.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E.A. 2007.** Influence of local California bay distribution on the risk of *Phytophthora ramorum* canker (sudden oak death) in coast live oak. Vacaville, CA: Phytosphere Research. 30 p. http://phytosphere.com/publications/Influence_bay_distribution_SOD_coast_live_oak.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E. 2008a.** *Phytophthora ramorum* canker (sudden oak death) in coast live oak and tanoak, 2000–2007: factors affecting disease risk, disease progression, and failure potential. 2007–2008 contract year annual report. Vacaville, CA: Phytosphere Research. 40 p. http://phytosphere.com/publications/Phytophthora_case-control2007-2008.htm. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E. 2008b.** Increasing distance from California bay reduces the risk and severity of *Phytophthora ramorum* canker in coast live oak. In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M., tech. coords. Proceedings of the sudden oak death third science symposium. Gen. Tech. Rep. PSW-GTR-214. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 181–194. http://www.fs.fed.us/psw/publications/documents/psw_gtr214/psw_gtr214_181-194_swiecki.pdf. (27 August 2012).

- Swiecki, T.J.; Bernhardt, E. 2008c.** Impacts of *Phytophthora ramorum* canker and other agents in Sonoma County forests. In: Merenlender, A.; McCreary, D.; Purcell, K.L., tech. eds. Proceedings of the sixth symposium on oak woodlands: today's challenges, tomorrow's opportunities. Gen. Tech. Rep. PSW-GTR-217. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 209–233. http://www.fs.fed.us/psw/publications/documents/psw_gtr217/psw_gtr217_209.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E. 2008d.** Regeneration of oaks and tanoak in *Phytophthora ramorum*-affected forests. In: Merenlender, A.; McCreary, D.; Purcell, K.L., tech. eds. Proceedings of the sixth symposium on oak woodlands: today's challenges, tomorrow's opportunities. Gen. Tech. Rep. PSW-GTR-217. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 357–370. http://www.fs.fed.us/psw/publications/documents/psw_gtr217/psw_gtr217_357.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E. 2010.** Long-term trends in coast live oak and tanoak stands affected by *Phytophthora ramorum* canker (sudden oak death). In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M., tech. coords. Proceedings of the sudden oak death fourth science symposium. Gen. Tech. Rep. PSW-GTR-229. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 207–209. http://www.fs.fed.us/psw/publications/documents/psw_gtr229/psw_gtr229_207.pdf. (8 December 2012).
- Swiecki, T.J.; Bernhardt, E.; Drake, C.; Costello, L.R. 2006.** Relationships between *Phytophthora ramorum* canker (sudden oak death) and failure potential in coast live oak. In: Frankel, S.J.; Shea, P.J.; Haverty, M.I., tech. coords. Proceedings of the sudden oak death second science symposium: the state of our knowledge. Gen. Tech. Rep. PSW-GTR-196. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 427–453. http://www.fs.fed.us/psw/publications/documents/psw_gtr196/psw_gtr196_007_064Swiecki.pdf. (27 August 2012).
- Swiecki, T.J.; Bernhardt, E.; Garbelotto, M.; Fichtner, E. 2011.** The exotic plant pathogen *Phytophthora cinnamomi*: A major threat to rare *Arctostaphylos* and much more. In: Willoughby, J.W.; Orr, B.K.; Schierenbeck, K.A.; Jensen, N.J., eds. Proceedings of the CNPS 2009 conservation conference: strategies and solutions. Sacramento, CA: California Native Plant Society: 367–371.

- Tjosvold, S.A.; Chambers, D.L.; Koike, S.; Fitchner, E. 2006.** Epidemiology of *Phytophthora ramorum* infecting rhododendrons under simulated nursery conditions. In: Frankel, S.J.; Shea, P.J.; Haverty, M.I., tech. coords. Proceedings of the sudden oak death second science symposium: the state of our knowledge. Gen. Tech. Rep. PSW-GTR-196. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 459–461. http://www.fs.fed.us/psw/publications/documents/psw_gtr196/psw_gtr196_007_068Tjosvold.pdf. (8 December 2012).
- Turner, J.; Jennings, P.; Humphries, G.; Parker, S.; McDonough, S.; Stonehouse, J.; Lockley, D.; Slawson, D. 2008.** Natural outbreaks of *Phytophthora ramorum* in the UK—current status and monitoring update. In: Frankel, S.J.; Kliejunas, J.T.; Palmieri, K.M.; tech. coords. Proceedings of the sudden oak death third science symposium. Gen. Tech. Rep. PSW-GTR-214. Albany, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 43–48.
- University of California–Berkeley. 2012.** SODMAP project. Forest Pathology and Mycology Laboratory. <http://sodmap.org>. (13 July 2012).
- U.S. Department of Agriculture, Animal and Plant Health Inspection Service [USDA APHIS]. 2012.** APHIS list of regulated hosts and plants proven or associated with *Phytophthora ramorum*. http://www.aphis.usda.gov/plant_health/plant_pest_info/pram/downloads/pdf_files/usdaprlist.pdf. (16 March 2012).
- U.S. Environmental Protection Agency [USEPA]. 1998.** Mono- and di-potassium salts of phosphorous acid (076416) fact sheet. http://www.epa.gov/opp00001/chem_search/reg_actions/registration/fs_PC-076416_1-Oct-98.pdf. (23 January 2013).
- U. S. Environmental Protection Agency [USEPA]. 2006.** Phosphorous acid and its ammonium, sodium, and potassium salts (076002) mono- and di-potassium salts of phosphorous acid (076416) Federal Register notices. http://www.epa.gov/oppbppd1/biopesticides/ingredients_keep/fr_notices/frnotices_076002.htm. (28 January 2013).
- Vettraino, A.M.; Huberli, D.; Garbelotto, M. 2008.** *Phytophthora ramorum* infection of coast live oak leaves in Californian forests and its capacity to sporulate in vitro. *Australasian Plant Pathology*. 37: 72–73.

- Webber, J.F.; Mullett, M.; Brasier, C.M. 2010.** Dieback and mortality of plantation Japanese larch (*Larix kaempferi*) associated with infection by *Phytophthora ramorum*. *New Disease Reports*. 22: 19.
- Werres, S.; Marwitz, R.; Man In't Veld, W.A.; de Cock, A.W.A.M.; Bonants, P.J.M.; de Weerd, M.; Themann, K.; Ilieva, E.; Baayen, R.P. 2001.** *Phytophthora ramorum* sp. nov., a new pathogen on *Rhododendron* and *Viburnum*. *Mycological Research*. 105: 1155–1165.
- Wickland, A.C.; Jensen, C.E.; Rizzo, D.M. 2008.** Geographic distribution, disease symptoms, and pathogenicity of *Phytophthora nemorosa* and *Phytophthora pseudosyringae* in California, USA. *Forest Pathology*. 38: 288–298.
- Widmer, T.L. 2009.** Infective potential of sporangia and zoospores of *Phytophthora ramorum*. *Plant Disease*. 93: 30–35.

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