

# Enlargement of canopy gaps associated with a fungal pathogen in Yosemite Valley, California

D.M. Rizzo, G.W. Slaughter, and J.R. Parmeter, Jr.

**Abstract:** The enlargement of 21 canopy gaps associated with the root pathogen *Heterobasidion annosum* (Fr.:Fr.) Bref. (= *Fomes annosus* (Fr.) Karst.) in the mixed-conifer forest of Yosemite Valley was monitored between 1971 and 1998. Mean expanded gap area was 232 m<sup>2</sup> (range 38–802 m<sup>2</sup>) in 1971 and 1455 m<sup>2</sup> (range 150–4216 m<sup>2</sup>) in 1998. The pathogen is primarily spread among trees via root contacts; therefore, the spread and impact of *H. annosum* from the stump(s) where infection originated (gap makers) were characterized by describing the area of potential root contact as a variable-area plot. The potential root contact zone delineated the area that would be encompassed by mortality if all trees potentially in contact with the original gap makers had died. Of the 21 plots, the actual area of tree mortality exceeded the potential root contact zone in only two gaps. Enlargement rates around gap perimeters were uneven, ranging from 0 to 1.33 m/year. Incense-cedars (*Calocedrus decurrens* (Torr.) Florin) tended to die more slowly than ponderosa pines (*Pinus ponderosa* Dougl.), often 10–30 years after adjacent pines had died. Gap enlargement was not dependent on host density, basal area, or gap-maker diameter, and tended to stabilize over time, probably because of fungal competition in root wood and lack of sufficient suitable host material along gap margins.

**Résumé :** L'expansion de 21 trouées associées au champignon pathogène des racines, *Heterobasidion annosum* (Fr.:Fr.) Bref. (= *Fomes annosus* (Fr.) Karst.), dans la forêt résineuse mixte de la vallée de la Yosemite a été suivie entre 1971 et 1998. L'expansion moyenne des trouées était de 232 m<sup>2</sup> (étendue allant de 38 à 802 m<sup>2</sup>) en 1971 et de 1455 m<sup>2</sup> (étendue allant de 150 à 4216 m<sup>2</sup>) en 1998. Le champignon pathogène s'étend chez les arbres principalement via les contacts racinaires. Par conséquent, la progression et l'impact de *H. annosum* à partir de la souche où avait débuté l'infection (la cause de la trouée) ont été caractérisés en décrivant la zone potentielle de contacts racinaires comme une parcelle de superficie variable. La zone potentielle de contacts racinaires délimitait la superficie où il y aurait de la mortalité si tous les arbres potentiellement en contact avec la souche à l'origine de la trouée avaient été tués. La zone réelle de mortalité des arbres a dépassé la zone potentielle de contacts racinaires dans seulement deux des 21 trouées. Le taux d'expansion autour du périmètre des trouées était variable, allant de 0 à 1,33 m·a<sup>-1</sup>. Les libocèdres à encens (*Calocedrus decurrens* (Torr.) Florin) avaient tendance à mourir plus lentement que les pins ponderosa (*Pinus ponderosa* Dougl.), souvent 10 à 30 ans après que les pins adjacents aient été tués. L'expansion des trouées était reliée ni à la densité des hôtes, ni à la surface terrière, ni au diamètre de la souche où avait débuté l'infection et avait tendance à se stabiliser avec le temps, probablement à cause de la compétition fongique dans le bois des racines et de l'absence d'hôtes convenables en quantité suffisante à la marge des trouées.

[Traduit par la Rédaction]

## Introduction

Most discussions of the temporal patterns of gap dynamics in forest ecosystems have concentrated on closure of stand openings by regeneration or in-growth of border trees. Relatively few studies, however, have been concerned with the enlargement of canopy gaps over time due to the death of gap border trees (e.g., Worrall and Harrington 1988; Lertzman and Krebs 1991; Krasny and Whitmore 1992; Perkins et al. 1992; Runkle 1998). Slowly enlarging canopy gaps will affect light regimes, soil disturbances, and other environmental parameters differently than single acute dis-

turbances, such as windthrow (Krasny and Whitmore 1992). One of the most important causes of enlarging gaps in forest ecosystems is mortality caused by plant pathogens. In particular, fungal root diseases are a major factor associated with the death of mature trees in forest ecosystems. In northern temperate forests, *Heterobasidion annosum* (Fr.:Fr.) Bref. (= *Fomes annosus* (Fr.) Karst.), *Armillaria* spp., and *Phellinus weirii* (Murr.) Gilbn. decay the large woody roots and butts, kill trees directly via killing of sapwood or cambium, or predispose them to other mortality agents such as insects or windthrow (Cobb et al. 1974; Dickman and Cook 1989; Holah et al. 1993, 1997; Lundquist 1995; Hansen and Lewis 1997).

While the importance of spores and other aspects of individual life histories in infection biology varies among these root-rot pathogens, each may be spread through root-to-root contact between infected and uninfected trees. As individual trees die from root disease, gaps open in the forest canopy. Gaps caused by root diseases may continue to enlarge as the pathogen spreads from tree to tree and, therefore, may exert an influence within a forest stand for many years. Many

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root-disease pathogens are also excellent saprobes and once a tree has died the fungus can potentially survive for decades, slowly decaying root wood (Hansen and Lewis 1997). By retaining infective propagules within the gap, root diseases can also influence the type of regeneration that is possible within a gap in a way which acute disturbances cannot. In many disease-caused gaps a range of tree mortality conditions will exist, from older heavily decayed trees on the ground to decayed but still standing snags, to recently killed trees, thus providing a range of habitats for tree mortality associated fauna and flora (McComb and Lindenmayer 1999). While all types of gaps contribute by some degree to stand diversification, the chronic nature of root disease assures a constantly evolving landscape over many decades (Matson and Waring 1984; Dickman and Cook 1989; Gieszler et al. 1980; Holah et al. 1997).

The importance of canopy gaps to forest dynamics has been extensively reported, but individual gaps have rarely been followed over time (Poulson and Platt 1996; Valverde and Silvertown 1997; Runkle 1998; Slaughter and Parmeter 1995). In this paper, we quantify the spatial and temporal patterns of individual gaps associated with a fungal root pathogen, *H. annosum*, over a 27-year period in mixed-conifer forests of Yosemite Valley within Yosemite National Park. As a result of fire suppression policies and meadow draining since the mid-1800s, dense stands of conifers have come to occupy much of the Yosemite Valley floor, replacing significant areas of meadows and oak woodlands (Gibbens and Heady 1964; Heady and Zinke 1978). Tree felling for building projects, scenic vistas, and bark beetle control beginning in the early parts of the 20th century led to the creation of numerous stumps that have served as initiation points for infection by *H. annosum* (Slaughter and Rizzo 1999). We examined two questions: (i) Do gaps associated with *H. annosum* continue to enlarge indefinitely, given a sufficient density of susceptible hosts? and (ii) How does differential mortality of host trees affect the dynamics of gap enlargement and changes in stand structure? Related questions concerning the distribution of canopy gaps associated with root disease across the Yosemite Valley landscape will be explored in future papers.

## Methods

### Study area

Yosemite Valley (37°43'N, 119°37'W) is located in the central Sierra Nevada of California, U.S.A. The Merced River flows in a westward direction through the valley, creating a relatively flat alluvial plain about 11 km long and 0.8 km wide with an elevation of approximately 1220 m (Heady and Zinke 1978). Sheer granite walls up to 1000 m high enclose the valley floor on three sides. The valley has a Mediterranean type climate characterized by hot, dry summers and cool, wet winters. Average monthly temperatures range from 22°C in July to 1°C in January. Annual precipitation is 95 cm, falling mostly as rain from October through May. Snowfalls occur during winter months, but generally melt quickly. The high cliffs of the valley cause some microclimatic differences on the valley floor; e.g., the south side tends to have shadier, cooler conditions during summer months and harder freezing in winter, and the north side tends to have sunnier, drier conditions throughout the year.

Yosemite Valley soils are all derived from similar granitic parent materials, deposited either by the river or by canyon wall deterioration. They range from alluvial deposits near the riverbed, to seldom flooded alluvium some distance from the river, to alluvial fans, apron deposits, and colluvial talus slopes near the canyon walls (Heady and Zinke 1978; USDA 1998). Winter and spring floods are common in the valley, and at each occurrence they leave a layer of fine silt and sand over as much as 50% of the valley floor.

The predominant forest type in Yosemite Valley is currently a mixed-conifer forest containing dense aggregations of ponderosa pine (*Pinus ponderosa* Dougl.) and incense-cedar (*Calocedrus decurrens* (Torr.) Florin), with smaller numbers of white fir (*Abies concolor* (Gord. & Glend.) Lindl.), Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco), black oak (*Quercus kelloggii* Newb.), and other hardwood species (Acree 1994). On the hotter, drier north side of the valley, canyon live oak (*Quercus chrysolepis* Liebm.) is present in the understory below mostly ponderosa pine. On the shady south side of the valley, white fir and Douglas-fir become a significant portion of the stand.

There are several fungal root pathogens native to Yosemite Valley (Slaughter and Rizzo 1999; Rizzo and Slaughter 2000), but *H. annosum* is currently the most important from a management perspective. Two host-specialized forms of *H. annosum* are found in the Sierra Nevada. The "pine-type" primarily infects *Pinus* spp., *C. decurrens*, and *Juniperus* spp.; the "fir-type" is common on *Abies* spp. and *Sequoiadendron giganteum* (Lindl.) Decne. (Harrington et al. 1989; Otrosina et al. 1992). To date, we have identified only the pine-type of *H. annosum* in Yosemite Valley.

### Sampling

In the late 1960s and early 1970s, Yosemite Valley was surveyed for signs and symptoms of root disease, including standing dead and uprooted trees, and stumps with decay and fungal fruiting bodies (Felix et al. 1974; Parmeter et al. 1978, 1979). Samples were taken and the presence of *H. annosum* was confirmed in the laboratory (Parmeter et al. 1978, 1979).

Maps were made of over 100 *H. annosum*-positive gaps and included stumps, fallen logs, standing snags, and a buffer strip of living trees approximately two rows deep around the gap. Data collected included tree species, diameter at breast height (DBH), tree condition (living or dead), and disease symptoms for living trees. Symptoms included dieback, growth reductions, and foliage chlorosis. These gaps were revisited each year between 1971 and 1980, and in 1983, 1984, 1986, and 1989; new tree mortality was recorded on the maps at each visit (Parmeter et al. 1978, 1979; Marosy and Parmeter 1989). Dead and dying trees are often removed by park personnel to avoid hazardous tree situations in campgrounds and other developed areas, so evidence of tree mortality was often only a stump or a pile of chips remaining after stump grinding, where the map indicated a live tree from the preceding visit.

In 1996, 100 of the mapped gaps were revisited and evidence of further mortality was noted. Our intent was to examine gap enlargement over time in an environment as natural and undisturbed as possible; thus, many of the gaps were no longer useful for our purposes because of coalescence with adjacent gaps or extensive anthropogenic disturbance. Maps of 21 gaps were extensively updated in 1996 and 1998 to include all mortality that had occurred since 1989. Year of death was estimated based on the condition of dead trees; e.g., amount of bark retention and extent of wood decay.

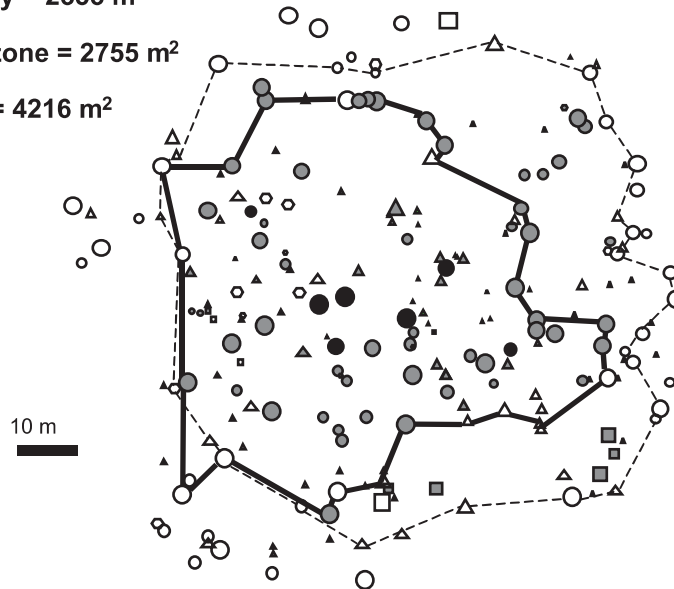
The 21 gaps were selected from the original 100 because (i) they were initiated at stumps or dead trees with confirmed *H. annosum*; (ii) they were discrete, i.e., sufficiently separated from adjacent gaps so that they were not likely to have coalesced with other root-disease-associated gaps; (iii) they were surrounded by sufficient numbers of susceptible trees that they would have the po-

**Fig. 1.** Map of Sentinel Beach gap in Yosemite Valley. Circles are ponderosa pine; triangles, incense-cedar; squares, black oak; and hexagons, Douglas-fir. Size of symbols indicates relative size of trees; the largest symbols represent trees over 100 cm DBH. Open symbols are trees living in 1998, black symbols are trees that died between 1965 and 1970 and were considered gap makers, and gray symbols are trees that died between 1970 and 1998. The dark line represents the area of the potential root contact zone based on the formula given in the text. The broken line delineates the expanded gap.

Area of mortality = 2535 m<sup>2</sup>

Predicted root zone = 2755 m<sup>2</sup>

Expanded gap = 4216 m<sup>2</sup>



tential for future enlargement; and (iv) there appeared to be no proactive management (i.e., trees were only removed after they died, allowing for a natural progression of the disease).

The 21 gaps were located over approximately 600 ha in the ponderosa pine – black oak woodland community types of Yosemite Valley (Acree 1994). Global positioning system equipment (Trimble Corp., Sunnyvale, Calif.) was used in 1996 to record the location of each map center for future reference. To determine soil type for each gap, its location was plotted on a geographic information system map of Yosemite Valley soil types (USDA 1998). All gaps were located on alluvial soils towards the center of the valley. Primary differences in the soil types were in the amount of accumulated organic matter, drainage, and occurrence of flooding (USDA 1998). None of the gaps were found on the talus slopes at the margins of the valley.

In general, we tried to minimize the number of root samples taken from recently dead trees. We did not attempt to determine the actual extent of the fungus, since it would have been necessary to excavate living trees just beyond the margin of recent tree mortality. As these plots were to be monitored over the long term, we decided it was better to allow natural conditions to prevail and to allow tree mortality (if any) to proceed at a natural pace rather than to disturb the sites by digging for root samples. Some mortality in the gaps was attributed to causes other than *H. annosum*, but since all mortality was associated with a discrete gap, it was associated (directly or indirectly) with the *H. annosum* detected in the gap-maker stumps.

In 1996 and 1998, the root collars of a few dying trees at the advancing margin of the gaps were examined to determine the presence of the pathogen. The roots were cut into to look for the white-rot type of wood decay typical of *H. annosum*. Samples were removed and taken to the laboratory for isolations to confirm the presence of the pathogen. Isolations for *H. annosum* were made by standard methods (Worrall and Harrington 1992).

## Analyses

Updated maps of mortality over time were produced and bound-

aries of *H. annosum*-associated gaps were plotted (Fig. 1). Gap areas were measured by overlaying dot grids directly on the maps, counting dots within gap boundaries, and adjusting for map scale. Three methods were used to describe various aspects of living and dead trees present in the gaps: expanded gap, mortality gap, and potential root contact zone. We used the concept of the expanded gap (Runkle 1982) as a description of the canopy opening and to be consistent with usage in other published gap studies. The expanded gap was delineated on the map by connecting the stems of living border trees surrounding the perimeter of tree mortality with straight lines.

The tree mortality gap boundaries were determined by using the stand maps to connect dead trees (considered to have been associated with the gap maker) with straight lines. Trees that had died well outside of the influence of the gap maker were not considered to be part of the same gap. The area of tree mortality was a measure of the approximate extent of the pathogen. Symptomatic trees were not counted as part of gaps for analysis purposes, since relatively few of these were examined in detail.

The 21 gaps recorded in the first survey in 1971 varied considerably in area of enlargement over the 27-year period (Table 1). Because of this gap-size differential, it was difficult to directly compare the effects over time of the root disease on forest stand structure among the different gaps. It was apparent that much information would be lost if we utilized fixed-area plots to analyze the data concerning gap enlargement. Therefore, based on the premise that *H. annosum* is primarily spread among living trees via root-to-root contacts, we characterized the potential spread and impact of the pathogen from the gap makers by describing the area of potential root contact as a variable-area plot. The potential root contact zone delineates the area that would be encompassed by mortality if all trees potentially in direct contact with the gap maker(s) had died.

The potential root contact zone of all conifers associated with the gap was calculated (in metres) using a formula developed as part of a deterministic model for predicting losses to root disease on a landscape scale (Beukema et al. 1998):



**Table 1.** Expanded gap size (m<sup>2</sup>) in 1965 and 1998 in 21 plots in Yosemite Valley.

Gap	1970	1998
Ahwahnee3	63	563
Ahwahnee11	169	3543
Ahwahnee24	44	500
Cathedral	150	3112
Cathedral2	38	150
Devils Elbow	69	1356
FallsTrail	802	1604
LodgeCreek	101	1546
LowerPines6	238	1156
LowerPines10	269	356
LowerPines102	506	1588
LowerRiver2	94	1350
LowerRiver124	325	1575
NorthPines8	100	898
NorthPines14	400	1244
RockyPoint	206	700
SentinelBeach	173	4216
UpperPines1	53	201
UpperPines7	138	1006
UpperPines8	331	1406
YellowPines	619	2487
Mean	232.8	1455.1
Standard error	44.5	235.5

$$[1] \quad \text{root zone radius (m)} = [\text{tree DBH(cm)/100}]14.26$$

where DBH is in centimetres. As an example, the hypothesized root zone of a 125 cm diameter tree would be approximately 1000 m<sup>2</sup> and that of a 30 cm diameter tree would be approximately 57 m<sup>2</sup>. We projected the hypothetical root zones of the gap makers and surrounding trees onto the maps of 21 gaps. The potential root contact zone of the gap maker was considered to include the root zone of the gap maker plus any overlapping root zones of the surrounding trees (Fig. 1). The potential root contact zone takes into account the size of the gap maker as well as the size of the trees in the surrounding forest stand.

The map data were used to reconstruct the stand composition prior to the introduction of *H. annosum* into the stand. The first surveys were conducted in 1971, and since few trees were believed to have died prior to 1965, this was used as our starting date. Densities (trees/ha) and basal areas (m<sup>2</sup>/ha) of all trees in three diameter size classes (1–30, 31–60, and >60 cm DBH) were determined within the potential root contact zones of each of the gaps for 1965 and 1998. Basal areas on each plot were calculated as cross-sectional area of individual trees at 1.5 m height. A ratio to determine relative enlargement of the mortality area (i.e., spread of the pathogen) was calculated by dividing the actual area of mortality by the area of the potential root contact zone.

## Results

### Expanded gap size

In 1971, the mean expanded gap area of the 21 gaps was 232 m<sup>2</sup> with a range of 38 to 802 m<sup>2</sup> (Table 1). By 1998, the mean expanded gap area of these 21 gaps was 1455 m<sup>2</sup> with a range of 150 to 4216 m<sup>2</sup> (Table 1). All gaps increased in size because of additional mortality of border trees; however, there was no correlation between the size of individual gaps

in 1971 and 1998 ( $r_s = 0.22$ , Spearman's rank correlation). The 21 gaps selected for this long-term study are representative of the range of *H. annosum*-associated gaps found in Yosemite Valley. In a random survey of gaps in the undeveloped west end of the Valley in 1999 (D.M. Rizzo and G.W. Slaughter, in preparation), we detected an additional 21 gaps associated exclusively with *H. annosum*. These additional gaps ranged from 175 to 5075 m<sup>2</sup> with a mean of 1342 m<sup>2</sup>. The mean gap sizes in 1998 for both studies were not significantly different from each other ( $t$  test,  $P < 0.05$ ).

### Mortality factors

All gap makers were confirmed to be colonized by *H. annosum*. A few symptomatic trees and recently dead trees at gap margins were also confirmed to be *H. annosum* positive in 1996–1998. However, it is not certain that all trees within the potential root contact zone of gap makers died of root disease caused by *H. annosum*. Almost all of the pines on the plots were attacked by bark beetles (primarily *Dendroctonus brevicomis* Leconte with occasional attacks by *Dendroctonus ponderosae* Hopkins and *Dendroctonus valens* Leconte) before the root systems had decayed to the point of tree failure. Build-up of bark beetle populations in the root-disease gaps may also have led to small outbreaks that killed pines that were not infected by *H. annosum*. Almost all incense-cedars died following root failure due to *H. annosum* infection; there are no aggressive insects associated with incense-cedar (Furniss and Carolin 1977).

Oaks that died in the gaps were generally not examined in detail, but most apparently died from suppression followed by heart rot and canker rots (e.g., *Inonotus* sp., *Laetiporus sulphureus* (Fr.) Murr.). White fir and Douglas-fir also died from a variety of reasons including bark beetles (*Scolytus ventralis* Leconte and *Dendroctonus pseudotsugae* Hopkins, respectively) and several other root diseases (*Armillaria mellea* (Vahl:Fr.) P. Kumm., and *Phaeolus schweinitzii* (Fr.) Pat.). Other causes of tree death included mechanical damage due to adjacent windthrown trees (primarily root-rotted incense-cedar).

### Potential root contact zones

Potential root contact zones centered around the gap makers of the 21 plots ranged from 244 to 3109 m<sup>2</sup> with a mean of 1093 m<sup>2</sup> (median = 918 m<sup>2</sup>) (Table 2). There was a significant positive correlation ( $r_s = 0.69$ ,  $P < 0.01$ ) between the area of the potential root contact zone and the actual area of mortality (Fig. 2). However, after 27 years, the actual area of mortality exceeded the potential root contact zone in only two gaps (1.37 and 2.65 times the root contact zone) and only 8 of 21 gaps enlarged to greater than 50% of the root contact zone (mean 59%) (Fig. 2). Recent mortality associated with gap makers indicates that the fungus remains active in all gaps.

### Changes in tree density and basal area

In 1965, ponderosa pine accounted for 46%, incense-cedar for 48%, and other tree species for 6% of the stems within the potential root contact zones (Table 2). Ponderosa pine had the highest stem densities on 12 of the 21 plots and highest basal areas on 18 of the 21 plots. On seven of the plots, only pine and incense-cedar were present. Of the other

tree species present on the plots, black oak was the most common species followed by white fir and Douglas-fir.

A total of 569 trees died between 1965 and 1998 within the borders of the potential root contact zones. This represents a mean reduction in total living stems of 53% (range 12 to 83%). During 1965–1998, 62% of pines, 45% of incense-cedars, and 39% of other tree species died within the potential root contact zones (Table 2). On individual plots, loss of pines ranged from 0 to 100% (mean 57.3%) and loss of cedars ranged from 0 to 100% (mean 48.9%). In 1998, incense-cedar accounted for 56% and pine for 36% of the total number of living stems; in contrast with the 1965 data, incense-cedar had higher stem numbers on 13 of the 21 plots.

Over the course of the study, relative basal area decreased overall by 32% within the borders of the 21 potential root contact zones (range 86% reduction to 4% increase). Relative basal area increased on only 3, 10, and 8 plots for pine, incense-cedar, and other species, respectively. In 1998, pine had higher basal areas than incense-cedar on 15 of the 21 plots.

Densities and basal areas of incense-cedars, ponderosa pines, and all conifers combined within the potential root contact zone were not significantly correlated ( $r_s < 0.20$  for all comparisons) with any of the measures of gap size, including the expanded gap, area of mortality, or the actual/potential gap ratio. The densities and basal areas of individual size classes of each tree species were also not significantly correlated with any of the measures of gap size.

### Tree mortality over time

Cumulative mortality within the potential root contact zone was plotted over time for three size classes of pine and incense-cedar (Fig. 3). During the first 5 to 10 years after gap initiation (1965–1975), mortality of pine trees <60 cm DBH was significantly greater than that of all other size classes of pine and incense-cedar ( $t$  test,  $P < 0.01$ ) (Fig. 3). From 1975 to 1980, mean mortality of pines >60 cm DBH and incense-cedar <30 cm DBH increased to a point that was significantly different ( $P < 0.05$ ) than the two larger size classes of incense-cedar (Fig. 3). This sampling period coincided with a severe drought in Yosemite during 1975–1977 (Smith et al. 1994); the increased mortality rates of pines were also associated with an increase in bark beetle populations. Even with the drought, overall mortality of the largest cedars (>30 cm DBH) lagged behind the pines until 1985 (Fig. 3). A second drought during 1987–1992 did not result in accelerated mortality rates, as judged by changes in the slope of the graph. This may be due to less competition for resources between residual trees following nearly 30 years of stand thinning due to tree mortality.

Over the course of the study, the greatest mortality was noted in the 1–30 and 31–60 cm DBH size classes of pine and the 1–30 cm DBH size class in incense-cedar (Fig. 3); in 1998, these differences were significantly different from the mean mortality of the other size classes ( $P < 0.01$ ). While the mean mortality of large pines and cedars across the 21 plots was significantly less than that of the smaller size classes, mortality of larger trees within potential root contact zones on some individual plots was very high. Particularly

for pines, mortality of all size classes was over 70% within the potential root contact zone on 9 of the 21 plots (Table 2).

The graph of cumulative mortality suggested that while both pine and incense-cedar were susceptible to *H. annosum*, incense-cedars died more slowly (Fig. 3). We further quantified the differential mortality between pine and incense-cedar by comparing adjacent trees for time of death. All pairs of pine and incense-cedar within 5 m of each other for trees >30 cm on the 21 plots were considered. There were 170 pairs of trees in which one of the members of the pair had died. The only spatial consideration was the 5-m limit; the relative position to the plot center was not considered even though we would expect trees closer to the plot center to have been infected first. Of the 170 pairs, the pine died first in 126 cases (74.1%), the cedar died first in 31 cases (18.2%), and the two trees died in the same year in 13 cases (7.7%). This is significantly different ( $\chi^2 = 64.60$ ,  $P < 0.001$ ) than what would be expected if pine and incense-cedar were equally likely to die from the pathogen. The data were grouped into two categories. In the first category, both trees of the pair had died since data had been collected (1989); incense-cedars died a mean of  $3.03 \pm 1.4$  ( $\pm 1$  SE,  $n = 77$ ) years after the adjacent pine had died regardless of spatial position. In the second category, one of the members of the pair was still alive in 1998. Incense-cedars were still living an average of  $12.8 \pm 1.6$  ( $n = 93$ ) years after adjacent pines had died. Combining the data sets, incense-cedars lived an average of  $8.48 \pm 1.2$  years after an adjacent pine had died. In 36 of the cedar–pine pairs, the cedar was still alive 20 or more years after an adjacent pine had died. Most of these living cedars had crown symptoms consistent with infection by *H. annosum* and the disease was confirmed in the roots of several of the trees. In contrast, in only 9 of the cedar–pine pairs did a pine survive greater than 20 years following the death of an adjacent cedar.

### Gap enlargement

The trees that were still alive in 1998 were not spatially dispersed throughout the potential root contact zone; these remaining trees constituted the border trees of the expanded gaps (Table 1). The leveling of mortality rates across all size classes during the 1990s (Fig. 3) therefore reflects a slowing of the spread of the pathogen at the margins of the potential root contact zones.

Gaps did not enlarge in a circular pattern around the gap maker (Fig. 1). To describe the uneven nature of gap enlargement around various parts of the gap perimeter, gaps were divided into octants with 8 partitions radiating outward in cardinal directions from the map center. The maximum distance from map center to the most recently killed tree was measured. Linear enlargement of gaps from the original map center within these octants ranged from 0 to 45.3 m with a mean of 13.5 m. Dividing by the number of years during which the gap was studied, we found a mean enlargement rate of 0.45 m/year with a range of 0 to 1.33 m/year. The largest gap (Fig. 1) enlarged in all directions with a mean linear distance of 33.17 m and a mean enlargement rate of 0.98 m/year.

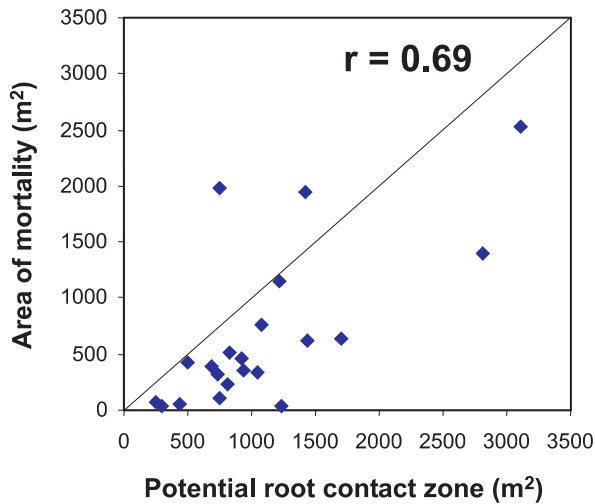
There was no correlation between soil type associated with the plots and enlargement of gaps. Gaps separated by 10 to 20 m were found on the same soil type and differed

**Table 2.** Area, density, and basal area (BA) of potential root contact zones centered around gap makers in 21 Yosemite Valley plots.

Gap	Potential root contact zone (m <sup>2</sup> )	1965						1998					
		<i>Pinus ponderosa</i>		<i>Calocedrus decurrens</i>		Other <sup>a</sup>		<i>Pinus ponderosa</i>		<i>Calocedrus decurrens</i>		Other <sup>a</sup>	
		Density (trees/ha)	BA (m <sup>2</sup> /ha)	Density (trees/ha)	BA (m <sup>2</sup> /ha)	Density (trees/ha)	BA (m <sup>2</sup> /ha)	Density (trees/ha)	BA (m <sup>2</sup> /ha)	Density (trees/ha)	BA (m <sup>2</sup> /ha)	Density (trees/ha)	BA (m <sup>2</sup> /ha)
Ahwahnee3	756	79	57.9	198	48.9	39	3.2	79	61.2	93	216.3	13	3.7
Ahwahnee11	750	267	140.9	200	50.7	0	0	27	22.3	67	22.8	0	0
Ahwahnee24	294	102	55.1	408	67	0	0	102	81.6	136	34.3	0	0
Cathedral	1419	423	71.9	226	38.3	7	0.1	70	16.9	113	24.3	7	0.1
Cathedral2	438	183	34.5	685	74.9	159	8.5	46	40.6	388	111.2	114	8.9
DevilsElbow	1437	77	39.5	376	43.8	28	0.5	14	5.1	84	19.9	28	2.4
FallsTrail	1217	312	80.4	288	33.4	24	1.1	58	26.9	107	16.5	8	0.2
LodgeCreek	918	349	171	22	0.2	0	0	163	105.7	22	0.3	0	0
LowerPine6	681	176	101.8	29	8.7	15	0.9	132	94.6	0	0	0	0
LowerPine10	1238	113	41.3	57	25.9	32	6.2	97	41.2	48	31.3	32	6.7
LowerPine102	1706	240	84.1	94	16.1	29	1.4	141	75.2	29	5.3	29	1.7
LowerRiver2	831	205	104.1	156	10.8	48	1.6	60	27.3	36	10	12	1.6
LowerRiver124	738	176	132.3	298	19.1	0	0	0	0	81	21	0	0
NorthPine8	806	360	122.3	149	13.1	0	0	199	108.1	74	15.4	0	0
NorthPine14	944	138	53	148	22	0	0	64	40.5	127	26.7	0	0
RockyPoint	1050	267	167.8	381	44.4	28	3.6	124	124.8	295	58.9	28	5.1
SentinelBeach	3109	193	101	183	24.8	49	2.5	19	18.6	93	17.1	42	6.2
UpperPines1	244	779	134.8	984	35.2	164	2.9	287	72.5	902	56.2	82	4.9
UpperPines7	506	257	31	296	15.4	20	3.6	59	16.4	178	22.3	0	0
UpperPines8	1075	242	53	186	14.1	0	0	140	40.6	102	9.4	0	0
YellowPine	2806	257	88.6	132	13.9	18	1.4	100	54.2	71	15.7	7	0.4
Mean	1093.5	247.4	88.7	261.7	29.5	31.4	1.8	94.3	51.2	145.0	25.9	19.1	1.9
Standard error	158.1	33.4	9.4	49.0	4.3	10.1	0.5	14.7	7.9	42.5	5.4	6.5	0.6

<sup>a</sup>Other tree species include *Quercus kelloggii*, *Abies concolor*, and *Pseudotsuga menziesii*.

**Fig. 2.** Correlation between the area of tree mortality and that of the potential root contact zone for 21 root-disease gaps. The line in the center represents the point where the area of tree mortality is exactly the same as the predicted root contact zone.



dramatically in total size and relative enlargement (e.g., the two Cathedral plots, Table 1). The three largest gaps were each located on different soil types.

## Discussion

### Gap enlargement and stabilization

By examining individual root-disease gaps in Yosemite Valley over a 27-year period, complex patterns of gap enlargement became apparent. Gaps did not simply enlarge by gradual mortality in a circular pattern from a centrally located gap maker. In general, gaps that were initiated at large gap makers, or in stands with many large trees, enlarged to a much greater size than those initiated at small gap makers. This would be expected; large trees have more extensive root systems that would cover larger areas and offer greater opportunities for the pathogen to spread. However, some gaps that initiated in large trees, and in stands with high densities of large trees, failed to enlarge, while others that were initiated in small trees enlarged greatly. Some gaps enlarged in only a single direction even though susceptible trees completely surrounded the gap maker. Stabilization of *H. annosum*-associated gaps has also been shown to occur throughout pine-juniper forests on the eastside of the Sierra Nevada in California; gaps in these forests, however, tended to be much smaller (mean area of 209 m<sup>2</sup>) (Slaughter and Parmeter 1995).

Based on the potential root contact zones, a majority of gaps will, at most, enlarge to the area occupied by the root system of the gap maker and the stems of trees whose roots are directly in contact with the roots of the gap maker. Only rarely did the fungus appear to spread from the gap maker into trees beyond the margin of the potential root contact zone. This would require the fungus to colonize the roots of trees directly in contact with the gap maker, then spread into the bole and back out the portions of the root system distal to the gap. Cumulative mortality for all the gaps as a whole shows that the greatest effects of root disease are on smaller

trees and that the largest trees tend to be fairly tolerant of the disease on the temporal scale of our study. However, there was considerable variation among the individual gaps; in some gaps, all trees, regardless of size, were eliminated.

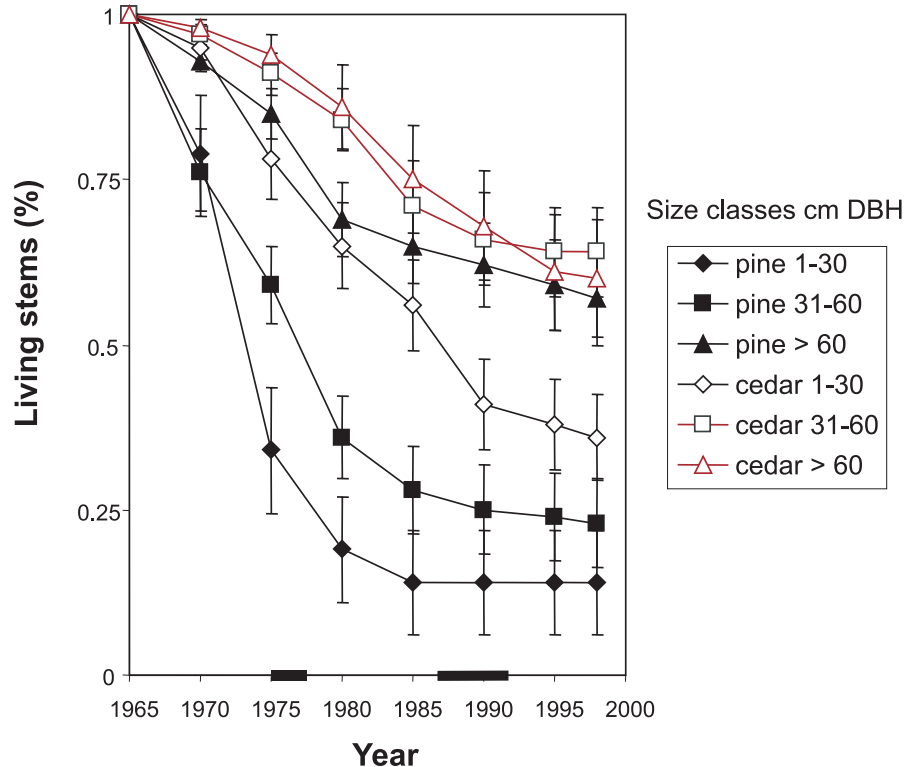
Why do some gaps enlarge greatly and others do not? What are the major factors involved in enlargement of gaps associated with root disease? Population variation in pathogen virulence and host susceptibility are two obvious influences on the spread of the pathogen. However, these attributes are difficult to test with *H. annosum* in mature trees and in the few studies that have been conducted, great differences in virulence or susceptibility have not been noted (Garbelotto et al. 1997; Delatour et al. 1998). It is not clear whether living trees associated with the root-disease gaps have survived because of resistance to the pathogen or simply by escape (e.g., lack of root contacts). Spread of pathogens in many plant-pathogen systems is strongly density-dependent (Burdon and Chilvers 1982). However, mortality of trees and gap enlargement did not proceed in a density dependent manner within our study gaps. Stem density per se is not important in the initial infection process; all that is needed is a suitable substrate. Subsequent spread of the pathogen does require root contacts between infected and noninfected trees; therefore, gap enlargement requires a sufficient density of hosts around a gap maker. At all of our sites, however, stem densities were sufficiently great to allow pathogen transmission and it was not a surprise when stand density did not correlate with enlargement of canopy gaps.

Soil conditions have been reported to be very important in contributing to the spread of *H. annosum*. Most severe disease and spread has generally been associated with soils with high pH, low organic matter content, and a high sand content (Stenlid and Redfern 1998). Although there are a number of different soil types in Yosemite Valley, all are derived from recently deposited alluvium and have high sand contents, relatively high pH (6.0 to 6.5), and limited organic layers (USDA 1998). Most Yosemite Valley soils would fall into the high risk category, and therefore, soil does not appear to account for variation of enlargement rates within Yosemite Valley.

Although susceptible hosts, sufficient tree densities, and conducive soil conditions are necessary for pathogen spread to occur, they do not guarantee it. Reasons for stabilization of root-disease gaps may include a combination of factors acting along gap perimeters. Roots extend in various directions from the original gap maker and each root constitutes a separate set of probabilities for growth of the pathogen; the fungus may or may not grow along individual roots far enough to infect adjacent trees, there may be no adjacent trees, or adjacent trees (e.g., oaks or white fir) may not be susceptible to infection by *H. annosum*. Studies have shown that *H. annosum* rarely moves distally through small-diameter roots (<9 cm diameter) in *Abies concolor* (Garbelotto et al. 1997). While this has not been demonstrated in pines and cedars, this may account for the limited spread of the fungus at a distance from the original gap maker in our study. Competing root-decay fungi (other pathogens and saprobes), may also effectively prevent spread of *H. annosum* along large and small roots (e.g., Morrison and Johnson 1978). While *H. annosum* can readily colonize living trees or stump tops of very recently cut trees, other



**Fig. 3.** Mean cumulative mortality (% of living stems  $\pm$  1 SE) within the potential root contact zone for 21 gaps between 1965 and 1998. Different symbols represent size classes of ponderosa pine and incense-cedar. The heavy lines on the *x*-axis represent drought years (1975–1977 and 1987–1992).



saprobic fungi may potentially outcompete and dominate host trees under certain circumstances (Holdenrieder and Greig 1998). For example, pines are often killed quickly by bark beetles before *H. annosum* has colonized the entire root system. While the pathogen may continue growing through the remainder of the root system, competing saprobic decay fungi may colonize portions of the dead root system and possibly limit growth of *H. annosum*. Bark beetle attack of uninfected trees at the gap perimeter may prevent enlargement of the gaps by providing a substrate for saprobic fungi to the complete exclusion of *H. annosum*.

### Gap structure and composition

Mortality of dominant and codominant trees has opened canopy gaps of varying sizes across the Yosemite Valley landscape. Our surveys have shown that approximately 30% of the forest land in Yosemite Valley is occupied by gaps associated with root disease (Rizzo and Slaughter 2000). At the scale of individual gaps associated with *H. annosum*, spread of the pathogen has resulted in a sequence of differential mortality of pines and incense-cedar. Ponderosa pines tend to die quickly within a few years of root infection, while incense-cedars, with nonaggressive bark beetle associates, slowly decline. Cedars often remain alive over 10 years after adjacent pines have died. The consequence of this is an initial conversion of impacted areas to an overstory of incense-cedar. This increase in dominance of incense-cedar was also documented by Sherman and Warren (1988), who utilized earlier vegetation data of Gibbens and Heady (1964). Sherman and Warren (1988), however, did not dis-

cuss mortality of dominant and codominant incense-cedar in their paper. Given a longer time horizon, many incense-cedars also die and leave openings in the forest canopy that are not occupied by trees.

Although many gaps in Yosemite Valley are not rapidly enlarging, some mortality is still occurring on the gap perimeter and in trees that have been left in stand openings due to the death of surrounding trees. In addition, many border trees have thin crowns and wood decay consistent with infection by *H. annosum*. Trees remaining within stand openings and border trees were primarily incense-cedar. Because incense-cedars may be able to tolerate infection for decades, this allows more time for *H. annosum* to occupy a large portion of the living root system and remain in a pathogenic mode for longer periods rather than relying on saprobic survival. Thus, while we may not see extensive lateral spread of *H. annosum* beyond the trees in contact with the gap maker, the fungus may remain active in a particular spot.

We have only done a qualitative examination of regeneration within gaps in Yosemite Valley. After 27 years, however, none of the gaps we have surveyed have returned to a closed forest canopy. In no instance did regeneration in the gaps reach higher than ca. 8% of the surrounding canopy height. In only one case did conifer regeneration reach heights of 3–4 m. In one gap (Cathedral Picnic), a cluster of ponderosa pine regeneration occupied less than 300 m<sup>2</sup> of a 3112-m<sup>2</sup> gap. Some of these sapling-size pines had signs and symptoms consistent with infection by *H. annosum* and the presence of the pathogen was confirmed by laboratory isolations. Based upon the years of death of the trees that



originally occupied the exact location of the regeneration, residual root wood was still providing *H. annosum* inoculum after 20 years. Our observations of the 21 gaps in this study and an additional 71 gaps in a larger survey indicate that most of these gaps may remain open for decades after they have begun to enlarge.

It is not clear what direction the species composition of the Yosemite Valley gaps will take. If conifers do eventually recolonize the gaps, will they be susceptible to infection by *H. annosum*? Our observations of infection of conifer regeneration in the Cathedral gap suggest that the answer is probably yes. Large gaps formed in pure *Tsuga mertensiana* (Bong.) (mountain hemlock) stands due to *Phellinus weirii* infection are colonized by a variety of conifers, including mountain hemlock (Dickman and Cook 1989). New mortality due to *P. weirii* within these filled-in gaps may not take place for 80–120 years (Dickman and Cook 1989). Present stand densities have only been a part of the Yosemite Valley ecosystem for about 100 years, so our observations may be only the beginning of the *H. annosum* root-disease cycle in these forests.

As Yosemite National Park resource managers work toward the integration of ecosystem-based management techniques (e.g., use of prescribed burns) with the impact of 4 million visitors per year (National Park Service 1997), we believe that the presence of root disease in conifers will play an important role in determining the direction of vegetation management in Yosemite Valley. A critical decision in a National Park is whether to manage forest diseases or allow natural processes to take precedence (National Park Service 1997). An understanding of the temporal aspects of gap dynamics will be important in implementing vegetation management with minimal impact to the valley ecosystem. For example, one proposed method of control for *H. annosum* is the felling of trees surrounding a root-disease gap to remove potential hosts thereby preventing spread of the pathogen into the surrounding stand via root contacts (West 1989). The fact that gaps associated with *H. annosum* will not enlarge indefinitely increases the predictive capabilities of Yosemite managers to the point where decisions can be made whether mitigation of root disease is actually necessary in specific areas of the valley.

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