Substantial Decay in Pacific Silver Fir Caused by *Hericium abietis*

GREGORY M. FILIP, Plant Pathologist, and ALAN M. KANASKIE and SUSAN J. FRANKEL, Biological Technicians, Forest Pest Management, Pacific Northwest Region, USDA Forest Service, Portland, OR 97208

ABSTRACT

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Fifty-three Pacific silver firs (*Abies amabilis*) were felled and examined for external indicators and extent and cause of advanced decay on the Olympic Peninsula of Washington. Total volume of decayed wood was 3.1% (m^3) and 10.4% (board-foot). Most of the decay was caused by *Hericium abietis* (34.4%) and *Echinodontium tinctorium* (20.5%). *H. abietis* in Pacific silver fir has never been reported to cause this high a proportion of total decay. Decay caused by *H. abietis* was not associated with typical decay indicators (ie, conks, wounds, or top damage).

Only one study done in British Columbia (4) provides information concerning long-pocket rot caused by *Hericium abietis* (Weir: Hubert) K. Harrison in Pacific silver fir (*Abies amabilis* (Doug.) Forb.). In that study, the occurrence of and damage caused by *H. abietis* in Pacific silver fir was minimal—3.5% of the total infections and 4.8% of the total decay volume. *Echinodontium tinctorium* (Ev. & Ell.) was the most damaging heartrot fungus in the same study, causing 64.9% of the decay. *H. abietis* was not found in the Upper Fraser River region of British Columbia (2). Recent reports by timber cruisers and scalers of extensive amounts of long-pocket rot in Pacific silver fir on the Olympic Peninsula prompted this study to determine the frequency of infection and amount of decay, especially that caused by *H. abietis*.

MATERIALS AND METHODS

In August 1982, a 5-ha area on the Quinault Ranger District, Olympic National Forest, WA, was examined. Trees in this area had been felled and bucked by a purchaser and were ideal for sampling. All Pacific silver firs on the site were examined.

Stumps were examined for basal wounds and, when present, the size (length × width) and age (by dissection) of each wound were determined. Tree age was calculated by ring count and diameter by measuring 1.4 m above the ground.

Log length, diameter inside bark (d.i.b.) at both ends, and diameter of advanced decay columns at the log ends were measured. The positions of conks, wounds, and broken, forked, or multiple tops on each log were recorded and drawn to scale. Wound size and age were also recorded. Advanced decay originating in the sapwood after trees were felled was not recorded. Cubic log and decay volumes were calculated by Smalian's formula, board-foot (Scribner) log volumes by Knouf's formula, and board-foot decay volumes by the squared defect method (3). Merchantable top d.i.b. of sample trees was 10 cm.

Decay was examined by first removing a disk 3 cm thick from each log end to expose a fresh surface. Disks were then split several times to expose longitudinal sections because decay caused by *H. abietis* is difficult to detect from log ends. Only advanced decay, as determined by ease of penetration of a knife point, was measured. If no advanced decay was observed in freshly exposed log ends or in dissected disks, no further cutting was done. If small amounts of advanced decay were observed, depth of decay was estimated and no further cutting was done. If moderate to severe amounts of advanced decay were observed, especially if present at both ends of the log, logs were further dissected at 2-m intervals to determine if columns were continuous. When conks or wounds were present, logs were bucked at conks or wounds to determine diameter and length of associated decay columns.

For all advanced decay columns, the causal organism was tentatively identified in the field and a sample of decayed wood was returned to the laboratory for culture and identification of the fungi causing decay. In the laboratory, samples were split, and 10 chips (5 × 5 × 15 mm) were removed asceptically with a gouge from each sample. Each chip was placed in a culture tube containing 2% malt agar with 1 μg/g of benomyl. Cultures were incubated for 6 wk at room temperatures in the dark, then attempts were made by the Center for Forest Mycology Research, Madison, WI, to identify all basidiomycetes to species.

RESULTS AND DISCUSSION

Fifty-three Pacific silver firs were examined for external indicators and extent and cause of advanced decay (Table 1). Total volumes of decayed wood were 3.1% (m^3) and 10.4% (board-foot). Board-foot units are used commonly for measuring manufactured products and thus are better related to economic loss than cubic volume. Within the total volume of decayed wood, the percentage of decay by each causal agent was nearly identical between cubic and board-foot volumes. The greatest proportion of decay (34.4%) was caused by *H. abietis* and was found in six of 30 decayed trees (Table 2). *E. tinctorium* accounted for 20.5% of the decay (four of 30 decayed trees) and was associated with conks about half of the time. Brown cubical rots accounted for 29.3% of the decay (nine of 30 decayed trees), some of which was caused by *Laetiporus sulphureus* (Bull.: Fr.) Bond. et Sing. Other fungi causing brown cubical rot could not be identified. Decay caused by *Heterobasidion annosum* (Fr.) Bref. accounted for 10.5% of the decay (two of 30 decayed trees) and was always associated with wounds.

Advanced decay caused by *H. abietis* appeared initially as small (5–15 mm) oblong pockets filled with yellow fibers. As decay progressed, pockets coalesced. Decay often contributed to trunk breakage when trees with decay were felled.

There were no apparent correlations between amount of decay and tree diameter or age, probably because of the small sample sizes for some diameter classes (Table 1). The study in British Columbia (4), using a larger sample, showed relationships between amount of decay and both tree diameter and age.

More than half of the trees with decay also had external indicators (Table 1). Furthermore, none of the trees with *H. abietis* had typical external indicators. External indicators associated most consistently with decay were conks of *E. tinctorium* and broken, forked, or multiple tops. Four of 14 trunk wounds were not associated with decay, but all of

Present address of second author: Weyerhaeuser Co., Technology Center, 32902 32nd Dr. S., Federal Way, WA 98003.

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Table 1. Frequency of decay and external indicators in Pacific silver fir by tree diameter and age class (Olympic Peninsula, WA)

Tree diameter class (cm)	No. of trees	Cubic volume decayed (%)	Board-foot volume decayed (%)	Percentage of trees with			
				Decay and indicators[a]	Decay, no indicators	Indicators, no decay	No decay, no indicators
38–50	5	0.1	0.9	0	20	20	60
51–63	2	0.6	3.3	50	0	0	50
64–76	11	3.1	10.2	9	27	18	46
77–89	13	0.6	2.2	8	23	15	54
90–102	8	3.0	10.6	62	13	0	25
103–115	8	7.6	24.7	50	12	25	12
116–128	5	0.1	0.2	20	20	0	60
129–141	1	4.9	13.9	100	0	0	0
Total or average	53	3.1	10.4	26	19	13	42
Tree age class (yr)							
00–151	1	0.0	0.0	0	0	0	100
151–200	9	1.0	3.4	22	22	22	33
201–250	17	3.1	10.9	41	6	18	35
251–300	13	2.7	10.9	15	31	15	39
301–350	2	3.2	17.1	0	50	0	50
351–400	5	5.0	15.2	40	20	0	40
401–450	2	0.1	0.4	0	50	0	50
450+	4	4.2	10.9	25	0	0	75
Total or average	53	3.1	10.4	26	19	13	42

[a] Indicators include wounds, conks, and broken, forked, or multiple tops.

Table 2. Frequency of decay and external indicators in Pacific silver fir by causal agent and indicator (Olympic Peninsula, WA)

Causal agent	No. of trees[a]	Cubic volume decayed (%)	Board-foot volume decayed (%)	Trees with indicators and decay (%)
Hericium abietis	6	34.4	35.5	0
Echinodontium tinctorium	4	20.5	24.1	50
Heterobasidion annosum	2	10.5	7.4	100
Brown cubical rot fungi[b]	9	29.3	27.7	22
Other	9	5.3	5.3	89
External indicators				**Trees with indicators, no decay (%)**
Conks	2	19.1	25.7	0
Wounds	14	16.9	14.5	29
Broken, forked, or multiple tops	2	2.7	3.2	0
None	14	61.4	59.5	···

[a] Some trees had more than one causal agent or external indicator.
[b] Includes decay caused by *Laetiporus sulphureus*.

these wounds were either very recent or small.

Much of the decay caused by *H. abietis* was not associated with wounds. This contrasts with grand fir (*A. grandis* (Doug.: D. Don) Lindl.), in which decay by *H. abietis* usually is associated with wounds, especially basal wounds (1). Perhaps, the biology of *H. abietis* in Pacific silver fir resembles that of *E. tinctorium*. Dormant infections of *E. tinctorium* occur primarily in suppressed advanced regeneration and may be activated by trunk wounds or formation of branch stubs (1,5). *H. abietis* has been isolated from healthy-appearing wood in suppressed grand fir (G. Filip and P. Aho, *unpublished*). Many of the trees in our sample had been suppressed since establishment, but we did not sample healthy-appearing tissues. Also, we observed many large branch stubs on both sound and decayed logs. If dormant infections by *H. abietis* were once present, they may have been activated by the formation of large branch stubs.

The relatively high frequency of *H. abietis* in Pacific silver fir may be unique to the area we examined. Additional sampling would be necessary to determine if *H. abietis* is an important decay pathogen elsewhere in the range of Pacific silver fir.

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