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Susceptibility of Pine Populations to Western Gall Rust—Central Idaho

R. J. Hoff¹

ABSTRACT

Differences among provenances of ponderosa pine were observed in response to infection by western gall rust. The average level of resistance was 21 percent and varied from 5 to 45 percent. Differences appeared to vary at random within the geographic study area. Several host responses were visible, and these are discussed in terms of their possible role as defense mechanisms.

KEYWORDS: western gall rust, host responses, provenances

Rust fungi are among the most destructive pathogens of cultivated plants. Nearly every agronomic breeding program includes resistance to one or more rusts. Without continued vigilance to maintain, improve, or develop new rust-resistant varieties, crop losses would be prohibitive.

Most, if not all, of the pines of the world are susceptible to one or several rust fungi of the genera *Cronartium*, *Endocronartium*, or *Peridermium*. In situations where the host trees grow naturally, destruction by these fungi is minimal and economically unimportant. But, as in agronomic crops, tree cultivation causes a disturbance of natural ecosystems which alters the habitat and in many cases causes an increase in damage by tree rusts. The classic case in forestry is the *Cronartium fusiforme*:*Pinus taeda*, *Pinus elliottii* interaction that has caused serious losses (Siggers and Lindgren 1947). Fusiform rust resistance has become an important and expensive requirement in intensive management programs of these important timber species.

Western gall rust caused by *Endocronartium harknessii* is potentially a serious disease of many pines throughout most of North America (Peterson 1967). Two important timber species in the Northern Rockies, lodgepole pine (*Pinus contorta*) and ponderosa pine (*Pinus ponderosa*), are susceptible to this rust. Presently, western gall rust-caused damage, aside from localized stands, is mainly a management concern in plantations and seed orchards (Martinsson 1980). Some practices associated with intensive forest management, like replanting, are likely to result in an increase in this disease. It is imperative, therefore, to secure knowledge about the biological dynamics of these rust:pine systems before the natural environment is badly disturbed. Knowledge of the kinds and levels of host responses relative to locations of the provenances, and how the rust varies with provenance and pine host, would aid the forest manager in the transfer of seed and in the selection of better timber types.

Variation among provenances, together with some information on host responses, has been reported for both lodgepole and ponderosa pine (Martinsson 1980; Thomas and Hart 1983). In addition, host responses on various pines infected by western gall rust have been reported (Hutchinson 1935; York 1938; Nelson 1972; Hiratsuka and Maruyama 1983). This paper reports on variation among populations of ponderosa pine from central Idaho in response to artificial inoculation by western gall rust.

MATERIALS AND METHODS

Seeds from 39 populations from central Idaho (fig. 1, table 1) were sown in 10-in³ containers in May of 1981. The seedlings were grown and overwintered in a shade-house at the Forestry Sciences Laboratory, Moscow, ID. During the summer, the seedlings were watered and lightly fertilized with the objective of producing seedlings that are not stressed but without accelerating their growth. During the winter, the seedlings were set on the ground within a screened cage to protect them from large accumulations of ice and snow. Bases were surrounded with sawdust so the roots would not freeze.

¹Principal plant geneticist located at Intermountain Station's Forestry Sciences Laboratory, Moscow, ID.

Figure 1.—Location of populations of ponderosa pine included in this test.

The following spring, the seedlings were put back on benches in the shadehouse. In May of 1982, when the needles were just beginning to break out of the fascicle sheaths, the seedlings were moved into a mist chamber in preparation for inoculation. Mist nozzles operated so that a fog was constantly visible throughout the chamber. Twenty-four hours later, the stems of the seedlings were wet down and allowed to set for 1 hour to remove excess moisture. At this point, the seedlings were ready to inoculate.

Experimental design:

- 39 provenances
- 3 replications
- 7 seedlings/replication

Sixty-three seedlings randomly selected from all provenances served as uninoculated comparison trees.

Fresh inoculum was collected from at least 50 galls from a young lodgepole pine stand located in the Moose Creek drainage (lat. 46°51' N., long. 116°23' W., elevation 2,900 ft) about 3 miles west of Bovill, ID. Inoculation was accomplished by blowing spores over the seedlings using an air-sprayer adapted from an

Erlenmeyer flask. Each replication was inoculated separately with a mixture of 355 mg of aeciospores and 2 g of talc. This gave a spore-to-seedling ratio of about 30,000 to 1. The seedlings were maintained in the mist chamber for 72 hours, again with a constant, visible fog. The mist nozzles were then turned off and the seedlings allowed to slowly dry. When dry, the seedlings were moved back to the shadehouse. Frequent inspections for signs of rust infection were made throughout the summer. Data concerning presence of galls and bark reactions were taken each spring and fall.

In September, the seedlings were moved to Priest River Experimental Forest and planted at a spacing of 6 by 6 inches. The seven seedlings from each population per replication were planted in row plots. The comparison trees (three rows of seven trees per replication) were evenly spaced within the replication.

Analysis of variance and the associations of geographic patterns with infection were completed using SAS programs (SAS 1982). Provenance means (percent nongalled) were transformed to $\arcsin \sqrt{\text{percent}}$ (Steel and Torrie 1960). Values of 0 percent or 100 percent were assigned values of $\frac{1}{4}n$ and $100 - \frac{1}{4}n$, respectively (Steel and Torrie 1960).

Table 1.—Location and host responses of seedlings of ponderosa pine inoculated with western gall rust

Stand	Eleva- tion	Latitude	Longitude	Non-galled				Galls present			
				No symp- toms	Red spots and streaks	Necrotic tissue	Total	Rate ¹ of appear- ance	Number ²	Dead ³ rust	Total infected
	Feet	°N.	°W.	Percent				Percent			
1	6,000	44–57	114–20	10	5	5	19	34	2.4	24	51
2	4,400	45–30	113–55	0	14	19	33	72	2.5	43	67
3	6,150	45–30	114–10	5	0	5	10	40	3.7	47	90
4	5,720	45–27	114–13	5	20	0	25	32	3.2	53	75
5	5,400	45–27	114–13	0	5	10	15	84	3.3	35	85
6	4,680	45–25	114–11	0	14	10	24	62	3.7	56	76
7	4,700	43–51	115–53	5	6	11	11	68	3.2	75	89
8	5,200	44–19	116–14	0	5	24	29	67	2.9	53	71
9	6,000	44–47	115–52	5	19	5	29	68	3.2	40	71
10	4,100	44–40	116–52	0	29	14	43	91	3.1	33	57
11	6,000	43–48	115–43	0	5	24	29	73	2.6	27	71
12	4,400	45–03	116–23	5	2	7	14	55	3.2	17	86
13	3,700	44–54	116–29	0	0	5	5	61	3.3	22	95
14	4,200	45–03	116–16	0	10	5	15	44	2.8	59	85
15	4,400	44–59	116–13	0	0	5	5	44	4.1	50	95
16	5,500	43–44	115–27	5	13	0	18	63	3.2	52	82
17	4,000	44–18	115–52	5	5	20	30	73	2.7	43	70
18	3,100	44–04	115–56	0	0	5	5	43	3.2	42	95
19	4,300	44–53	115–42	5	15	25	45	64	2.5	45	55
20	4,800	45–07	115–45	0	15	7	22	77	3.2	43	78
21	5,800	43–37	115–17	0	0	5	5	80	3.7	50	95
22	4,300	43–37	115–43	0	14	19	33	39	3.5	57	67
23	4,100	43–50	115–43	0	5	0	5	36	3.1	50	95
24	4,400	43–50	115–22	10	18	10	38	62	2.6	46	62
25	5,100	44–11	115–13	5	5	0	10	37	3.7	47	90
26	4,100	44–07	115–37	0	9	5	14	90	2.6	50	86
27	3,400	44–13	115–50	0	14	0	14	37	3.1	56	86
28	4,600	44–55	116–40	0	5	19	24	41	2.5	31	76
29	5,800	44–42	116–40	0	19	14	33	76	3.7	64	67
30	4,700	44–48	116–17	5	5	0	10	65	3.0	26	90
31	6,200	45–15	115–37	0	9	5	14	56	4.3	33	86
32	4,700	43–37	115–14	0	5	20	25	72	2.6	53	75
33	4,600	43–37	115–13	5	11	5	21	36	3.1	73	79
34	4,600	43–35	115–14	0	5	5	10	44	3.6	67	90
35	5,000	43–36	115–04	0	0	14	14	33	3.8	11	86
36	5,300	44–48	116–17	0	9	5	14	61	3.3	22	86
37	5,600	45–19	114–20	5	4	10	19	50	3.7	41	81
38	5,600	45–38	114–00	5	27	11	43	62	3.5	38	57
39	5,900	47–27	113–58	0	9	24	33	62	3.2	43	67
Average				2	9	10	21	58	3.2	44	79

¹Ratio of galls appearing at 4 months to 28 months after inoculation.

²Galls per infected seedlings.

³Infected seedlings that died.

RESULTS

Several host reactions were observed.

1. Red spots, streaks, and blotches on stems within a month of inoculation.
2. Small dead patches on the stems within 2 months of inoculation (fig. 2).
3. Small swellings and bumps indicating incipient galls within 4 months of inoculation (fig. 3).

Figure 2.—Small necrotic areas (arrows) on the stems of ponderosa pines 2 months after inoculation with western gall rust.

Figure 3.—Incipient galls of western gall rust on seedlings of ponderosa pine 4 months after inoculation.

4. Large, dead patches on the stem that increased in size from 4 to 28 months after inoculation (fig. 4). Some of these reactions were so severe that they caused malformation of the seedling (fig. 5).

5. Collapse and death of galls.

6. Variation in size of galls (fig. 6).

Out of 819 trees inoculated, only 17 had no symptoms (table 1). None of the 98 control seedlings had symptoms.

Figure 4.—Large necrotic areas on the stems of ponderosa pines 4 to 12 months after inoculation.

Figure 5.—Malformed seedling caused by many bark reactions.

Figure 6.—Variation in size of galls of western gall rust on ponderosa pines.

The percentage of seedlings without galls (that is, with evidence of resistance) was 21 percent. This ranged from 5 to 45 percent among populations (table 1). These means were significantly different at the 2 percent level (table 2).

Of the nongalled seedlings, 10 percent (2 percent overall) had no symptoms; 42 percent (9 percent overall) exhibited red spots, streaks, or blotches on the stem; and 48 percent (10 percent overall) had dead patches of various sizes on the stem, or had developed a gall (table 1).

The percentages of nongalled seedlings by population were plotted against elevation, latitude, and longitude. The R^2 values were 0.02, 0.03, and 0.01, respectively. The percentages of nongalled appear to be random (table 1) for these factors.

Table 2.—Analysis of variance of transformed means of seedlings without galls from 39 populations of ponderosa pine

	Degrees of freedom	Mean square	F	Probability > F
Block	2	0.121	2.95	0.057
Population	38	.072	1.76	.017
Block × population	76	.041		

Fifty-eight percent of the galls that were present at 28 months first appeared at 4 months (table 1). This varied among the populations from 32 to 91 percent. Probability of a higher F value for differences among the population means was 0.15. And the R^2 values for elevation, latitude, and longitude were essentially zero.

The percentage of infected trees in the population that had died by 28 months after inoculation averaged 44 percent, ranging from 11 to 73 percent (table 1). Probability of a higher F value was 0.23, and there were no associations with elevation, latitude, or longitude.

DISCUSSION

It is rare to find natural stands of ponderosa pine severely damaged by western gall rust in the Northern Rocky Mountains. And yet, in this test, 79 percent of the seedlings were infected, and nearly half of those were dead within 28 months after inoculation. The cause was probably the high spore density applied under optimal conditions—a situation not likely to occur very often in nature. Rust infection most frequently occurs in wave years (Leaphart 1955; Peterson 1959). Years that are foggy with intermittent showers during the high spore production time would seem to be ideal.

High resistance does not seem to be necessary in maintaining natural balance with this host-pest system. The probability of high spore density, coupled with optimal environmental conditions, may be quite low. In working out an artificial inoculation procedure, I found that a visible fog was not enough. I had to make sure the stems of the seedlings were wet. This method provided just the right conditions. These conditions may not occur in nature very often.

Even though the total level of resistance was only 21 percent, the rich assortment of host responses suggests that there are several defense mechanisms and, consequently, many genes involved.

Signs of Defense Mechanisms

1. No symptoms: Infection was so high that it is hard to believe that these seedlings had escaped infection. Resistance could be due to prevention of spore germination, formation of an effective barrier by the cuticle or epidermal cell walls, or destruction of the penetrating germ tube, which results in a very small host sign not readily visible.

2. Red spots, streaks, or blotches with no visible sign of necrotic stem patches or gall symptoms: This reaction was not severe enough to cause a visible necrotic patch on the stem, so presumably the fungus was either prevented from entering because the chemicals causing

red coloration in that seedling possibly were toxic, or the fungus was destroyed soon after penetration, but the defense reaction was so severe or quick that no necrosis was observed.

3. Necrotic patches on the stem: These ranged from very small patches that would soon be overgrown or sloughed off, to very large patches, at times so severe that they caused seedlings to be malformed.

Hutchinson (1935) reported similar reactions in *Pinus sylvestris* infected with western gall rust. He found that the first type was a fairly quick response by the host that killed the fungus, but the more severe reactions were often typified as a seesaw reaction by host and fungus: the host produced a wound periderm, then the fungus broke through, then the host would produce another periderm, and so on, until either the host or the fungus succumbed, or they both lived in some kind of a physiological balance.

4. Gall death: This may just be an extension of the severe stem reactions. But in these cases, there were no early signs of a resistant host reaction.

5. Gall size: The galls in some seedlings were so small that very little damage would be expected.

6. Rate of gall appearance: Although this trait was nonsignificant in this provenance test, I expect that a family test may show that this is an important defense mechanism. Slow appearance and slow growth of the fungus would not likely result in causing much damage to the host.

7. Tolerance: This is the ability of infected seedlings to remain alive and, especially, to grow normally or close to normal. Again, this trait was nonsignificant in this provenance test.

Population differentiation appeared to vary randomly over the landscape. But because the degree of resistance is usually thought of as being a result of disease severity (Leppik 1970), why would geographically close stands have such large differences in resistance? One cause could be the varying and random probability of infection during regeneration; that is, conditions for inoculation appear quite demanding, so the probability of optimal conditions occurring when trees are most

vulnerable to infection and damage (when young) are small. A test of individual families with varying levels of spore density using rust collections from several sources now seems extremely valuable.

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