Initial Characterization of a New Strain of *Cronartium ribicola* from the Cascade Mountains of Oregon

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ABSTRACT

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Inoculation of 20 families of *Pinus monticola* with two inoculum types of *Cronartium ribicola* demonstrated significant variation in virulence. Compared with wild-type inoculum from Oregon, a new strain (Champion Mine) increased the incubation period of the rust, increased rust intensity, reduced the proportion of seedlings shedding all infected needles within 1 yr of inoculation, reduced the proportion of seedlings showing retarded canker appearance, increased the proportion of seedlings with stunted leaders, reduced the period from inoculation to mortality, and reduced the proportion of trees healthy 2 yr after inoculation.

Additional key words: resistance mechanisms

Variation in virulence of rust isolates on host plants selected for resistance is well known (3). In the genus *Cronartium*, such variation is known for *C. quercuum* (Berk.) Miyabe: Shirai f. sp. *fusiforme* (14,16). Recently, variation in virulence of *C. ribicola* (J. C. Fisch.: Rabenh.) to a major gene type of resistance in sugar pine (*Pinus lambertiana* Dougl.) was reported (7). Variation in virulence of the pathogen on western white pine has not been reported. This is particularly significant because of potential effects on the breeding and deployment programs for rust-resistant white pine (5,9,11,12) in the western United States and Canada.

The existence of a race of *C. ribicola* capable of neutralizing particular resistance mechanisms in *P. monticola* has been suspected since 1973 by personnel at the Forest Service's resistant tree program at Dorena, OR. From 1960 to 1973, progeny from phenotypically resistant trees from many localities in Oregon and Washington were screened at the Dorena facility against a composite rust inoculum obtained from many locations in Oregon and Washington. Parent trees with a high frequency of resistant phenotypes among their progeny were identified. One source with a high frequency of resistant trees was a site near Champion Mine in the Cascade Range of central Oregon. In 1970, a marked increase of cankers on the previously canker-free parental trees was noticed. Progeny from these trees performed well after artificial inoculation in annual tests until 1973. Champion Mine inoculum has since been included with inoculum from other areas in most progeny tests. In general, spot frequency has increased, the shedding of infected needles as a mechanism of resistance has decreased, and appearance of symptoms (including mortality) appears to have accelerated. In addition, more infected seedlings are stunted than in earlier tests.

An experiment was designed to test the hypothesis that a new, virulent strain of *C. ribicola* was present at the Champion Mine site and, if so, to contrast it with the wild-type (WT) inoculum mixture with respect to definition and function of resistance mechanisms in western white pine populations.

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MATERIALS AND METHODS

Five sources of inoculum (*Ribes* leaves bearing telia) were used in the rust treatments of the test. Telia were collected at four locations in Oregon: Champion Mine (CM) (elevation 1,375 m, 45 km southeast of Cottage Grove), Still Creek (SC) (elevation 1,220 m at Mt. Hood, 195 km north of Champion Mine), Lower Grass Creek (GC) (elevation 375 m, 3 km east of Champion Mine in same drainage), and Dorena (D) (elevation 128 m, 30 km west of Champion Mine). *Ribes bracteosum* Dougl.: Hook., designated B, and *R. sanguineum* Pursh, designated S, were the telial hosts. At Dorena, telia on B were derived from aeciospores collected at Champion Mine, designated D/CM-B. The five sources of inoculum are described by the combined designations of geographic locality and telial host: CM-B, CM-S, SC-B, GC-B, and D/CM-B. *Ribes* leaves were collected 2 days before pine inoculation in September 1977.

In March and April 1977, bare-rooted pine seedlings beginning their second growing season were organized into 20 lots of 120 seedlings each. Lots 1 through 10 were from open-pollinated seed lots from 10 untested but phenotypically resistant western white pine trees growing in western Oregon. Lots 11 through 16 were from full-sib crosses between previously tested phenotypically resistant trees (genotypes unknown) located at Champion Mine. Lots 17 through 19 were from open-pollinated seed lots of phenotypically resistant trees growing in northern Idaho. Lot 20 was a full-sib cross between northern Idaho parents previously tested (genotypes unknown) with inoculum from northern Idaho.

Pallets 102 × 122 cm were divided into 120 planting spots to provide spacing of 7.6 × 10.2 cm. Six seedlings from each of the 20 lots were placed randomly in the 120 planting spots in each of 10 pallets.

Seedlings in two adjacent pallets received inoculum from each source. For this, *Ribes* leaves were placed on 0.635-cm mesh hardware cloth screens over each pair of pallets. Screens protruded 30.5 cm beyond the pallet edges and were 1.20 m above the seedlings. Burlap side curtains were affixed. All seedlings were inoculated simultaneously in a chamber 13.9 m long × 10.1 m wide × 3.1 m high. Pairs of pallets were arranged one in each corner and one in the center of the chamber. Relative humidity was maintained at 99 ± 1% with a mist system and temperature was held at 19 ± 1 C.

Twenty microscope slides coated with rubber cement were placed randomly on each pallet to estimate inoculum density. Slides were removed at intervals, and spores were stained with glycerine-analine blue. Spores in 10 randomly selected microscope fields-of-view on

each slide were then counted. When inoculum density for a treatment exceeded 2,000 spores per square centimeter on the last slide removed, the entire screen support was removed from that pair of pallets and replaced with a clean sheet of plastic film. Seedlings were left in place until 12 hr after the last screen was removed. Pallets were then moved outside, where they remained under 50% shade cloth until the experiment ended 2 yr later.

Monthly inspections were conducted for 24 mo, beginning 3 mo after inoculation. Needles were inspected for spots, stems for yellow or orange discoloration, and bark for fusiform swelling, pycniospores, and aeciospores. Needle spot types (red or yellow) and frequency were recorded 8 and 12 mo after inoculation. Spots were counted by color (8) on two of the top secondary needle fascicles in the crown (10 needles) and on four primary needles, if present, per seedling. Number of rows of stomata and average length of needles on two needles from each of two fascicles were recorded. Needle spotting for each seedling was expressed as spots per square centimeter of susceptible surface (where susceptible surface = number of needles $\times$ $\bar{X}$ number of rows of stomata/needle $\times$ 0.01 cm $\times$ $\bar{X}$ needle length in centimeters) (12). Bark symptoms were described after 12 and 24 mo.

Incubation period (months to visible needle spots), rust intensity (spots per square centimeter of susceptible surface), proportion of spots that were red on secondary needles, proportion of seedlings shedding all infected needles after 1 yr, retarded appearance of cankers (number of trees on which cankers appeared in second year per total trees with cankers after 2 yr), proportion of seedlings with stunted leaders, mortality period (months from inoculation to death), and proportion of trees apparently free of infection after 2 yr were all analyzed by analysis of variance. A three-factor factorial in a completely random design with replicates fixed and inoculum and families random was used (1,17). Raw data were transformed as follows in order to stabilize variance as measured by skewness and kurtosis coefficients. Incubation period and red spot frequency (ln); rust intensity ($\ln(y+1)$); shedding of infected needles, canker appearance, proportion noninfected (1/arc sine $\sqrt{y}$); stunted tops (arc sine $\sqrt{y}$); and mortality period ($\sqrt{y}$).

RESULTS

Inoculum source CM-B released basidiospores most rapidly and produced the highest overall inoculum density. The average inoculum density and duration of inoculation varied among the five sources as follows: CM-B, 3,724 spores per square centimeter and 16 hr; SC-B, 2,027 and 33; GC-B, 2,370 and 42; CM-S, 1,753 and 42; and D/CM-B, 2,174 and 24. Variation in inoculum density within a treatment as indicated by spore catches on slides was high; the average coefficient of variation was 31%.

Families were a significant source of variation in the analysis of variance of needle and stem symptoms. Inoculum type caused significant variation in all foliar and stem symptoms but not in incubation period (Tables 1 and 2). Interaction of replicate and inoculum occurred for rust intensity, red spot frequency, proportion stunted tops, and mortality period (Tables 1 and 2). There was no inoculum-by-family interaction.

Duncan's new multiple range test of differences among means of incubation periods, proportion of spots red, and rust intensity failed to differentiate inoculum from CM versus WT, but proportion of trees that shed infected needles, proportion with stunted tops, mortality period, and proportion uninfected after 2 yr clearly differentiated CM from WT (Table 3). On the basis of these results and the finding that family was a significant source of variation for all variables (Tables 1 and 2), we displayed average family means for each inoculum type and family selection history for all variables that differed significantly with inoculum source (Table 4).

Incubation period is a parameter of possible significance to resistance breeders (Table 4). Family averages ranged from 4.2 to 4.8 mo. The CM inoculum increased the incubation period slightly for both resistant and untested populations (Table 4).

Rust intensity (needle spots per square centimeter of susceptible surface) was roughly proportional to basidiospore production from the various sources. Values ranged from 0.23 spots per square centimeter for family 16 (resistant, control pollinated) inoculated with WT to 1.91 spots per square centimeter for family 18 (untested, open-pollinated) inoculated with spores from CM sources. Differences among inoculum sources are best seen by comparing average infection efficiency (needle spots per square centimeter of susceptible surface ÷ number of spores per square centimeter) (15) (Table 4). Infection efficiency differed both with inoculum source and

Table 1. Summary of analyses of variance for foliar symptoms caused by five sources of *Cronartium ribicola* inoculum on 20 families of *Pinus monticola*[a]

		Incubation period		Rust intensity		Shedding of infected needles		Red spot frequency	
Source	df	*MS*	*P* of >*F*	*MS*	*P* of >*F*	*MS*	*P* of >*F*	*MS*	*P* of >*F*
Replicates	1	0.0007	No test	12.49	No test	10.25	No test	0.46	No test
Inoculum type	4	0.0290	0.085	11.24	0.0001	12.77	0.0001	0.01	0.2500
Family	19	0.0410	0.0003	0.85	0.0001	7.27	0.0001	1.67	0.0001
Rep. × inoc.	4	0.0220	0.096	5.50	0.0001	0.87	0.5800	1.69	0.005
Rep. × fam.	19	0.0160	0.124	0.22	0.2600	0.72	0.0500	0.31	0.75
Inoc. × fam.	76	0.0140	0.166	0.16	0.2700	1.78	No test	0.72	0.70
Rep. × inoc. × fam.	76	0.0110	0.626	0.18	0.0800	1.21	Error	0.35	0.15
Residual	1,000	0.0120	Error	0.14	Error			0.30	Error

[a] Completely randomized design with replicates fixed and inoculum and families random.

Table 2. Summary of analysis of variance for stem symptoms caused by five sources of *Cronartium ribicola* on 20 families of *Pinus monticola*[a]

		Canker appearance		Stunted tops		Mortality period		Clean after 2 yr	
Source	df	*MS*	*P* of >*F*	*MS*	*P* of >*F*	*MS*	*P* of >*F*	*MS*	*P* of >*F*
Replicates	1	0.009	No test	0.291	No test	0.019	No test	5.10	No test
Inoculum type	4	0.062	0.008	0.658	0.0005	0.615	0.0001	12.24	0.0001
Family	19	0.031	0.028	0.162	0.005	0.030	0.0003	8.31	0.0001
Rep. × inoc.	4	0.007	0.830	0.176	0.005	0.553	0.0001	0.67	0.5500
Rep. × fam.	19	0.014	0.770	0.019	0.950	0.011	0.7300	1.11	0.2300
Inoc. × fam.	76	0.017	No test	0.058	No test	0.010	0.6500	1.63	No test
Rep. × inoc. × fam.	76	0.019	Error	0.037	Error	0.014	0.025	0.88	Error
Residual	1,000					0.010	Error		

[a] Completely randomized design with replicates fixed and inoculum and families random.

family selection history (Table 4). Seedlings from families previously selected for resistance to WT inoculation produced fewer needle spots per spore than untested families regardless of inoculum source. CM inoculum produced more infections per spore than WT inoculum regardless of selection history. Proportion of needle spots red varied significantly with family, but not with inoculum source (Table 1).

Cankers appeared in both growing seasons. The ratio of stems with cankers appearing only in the second year to the total number of stems cankered varied significantly among families (Table 2).

Percentage of cankered stems with stunted tops varied significantly with family and inoculum type (Tables 2 and 3). Duncan's test clearly separated the inoculum sources into the CM versus WT groups. Inoculum sources from CM produced increased proportion of stunted tops in all but two of the 20 families. When challenged with WT inoculum, families from control-pollinated, phenotypically resistant parents displayed fewer stunted leaders than did families from open-pollinated parents. With CM inoculum, however, the opposite was true.

Retarded canker appearance and mortality period of cankered stems varied significantly with family and inoculum source (Tables 2 and 3), but the inoculum source difference was not strictly CM versus WT. The odd inoculum source was D/CM-B (Champion Mine aeciospores grown at Dorena). The family means were compared on the basis of the CM versus WT classification (Table 4). Cankers appeared sooner on seedlings exposed to CM inoculum, and cankered seedlings infected from CM sources died an average of 2 wk earlier. The resistant population inoculated with CM died more than 3 wk earlier than the comparable resistant population inoculated with WT (Table 4).

The ratios of infection-free seedlings 2 yr after inoculation showed differences attributable to both family and inoculum source (Tables 2 and 3). Duncan's multiple range test distinguished the CM sources from the WT sources. Inoculation with CM inoculum reduced the proportion of uninfected trees by a factor of four in the untested population and by a factor of six in the resistant population. All families, however, did not react similarly. Much family-to-family variation remained.

We noted in past studies (10) that shedding of infected needles within 1 yr of inoculation is strongly related to the disease-free condition after 2 yr. Four inoculated pine populations (resistant, control-pollinated pine-WT rust; resistant, control-pollinated pine-CM rust; untested, open-pollinated pine-WT rust; and untested, open-pollinated pine-CM rust) were compared (Table 5) to see the effect of the CM race on the yield of trees with each of four symptom combinations representing three resistance mechanisms. Bark reaction resistance types seldom occurred; they were included in the premature shedding of infected needles and fungicidal short shoot classes.

Selection for resistance against WT inoculum resulted in apparent increases of 3.5× in premature shedding of infected needles, 2.4× in fungicidal short shoots, and 1.5× in retarded canker appearance, respectively (Table 5). Inoculation of both resistant, control-pollinated and untested, open-pollinated pine populations with CM sources of the rust reduced the frequencies of premature shedding of infected needles and retarded appearance of cankers after infected needles were dropped by factors of 1.43–2.64 (Table 5). When infected needles were retained, greater effects of the CM source were

Table 3. Comparison of five sources of *Cronartium ribicola* for foliar and stem symptoms on 20 families of *Pinus monticola*

	Foliar				Stem			
					Proportion			
Inoculum source[t]	Incubation period (mo)	Rust intensity[u]	Needle shedding[v]	Red spot frequency[w]	Cankers delayed[x]	Tops stunted[y]	Uninfected	Mortality period (mo)
GC-B	4.5 n.s.	0.6 c[z]	0.22 a	0.66 n.s.	0.04 c	0.22 c	0.23 a	22.4 b
SC-B	4.5	0.5 c	0.16 a	0.72	0.12 a	0.20 c	0.17 a	22.9 a
CM-B	4.6	1.9 a	0.05 b	0.72	0.04 c	0.38 b	0.04 b	21.9 c
CM-S	4.6	0.5 c	0.08 b	0.65	0.04 c	0.37 b	0.03 b	21.9 c
D/CM-B	4.5	0.8 b	0.08 b	0.68	0.10 b	0.49 a	0.05 b	22.9 a

[t] GC and SC are wild type (WT); CM is a new strain from Champion Mine, OR; B and S designate basidiospores from *Ribes bracteosum* and *R. sanguineum*, respectively; D indicates inoculum produced at Dorena, OR.
[u] Spots per square centimeter of susceptible surface 9 mo after inoculation.
[v] Seedlings without needle spots after 1 yr as proportion of total seedlings initially spotted.
[w] Red spots as proportion of total spots.
[x] Cankers that appeared in second year/total cankers.
[y] Proportion of cankered seedlings that showed stunted tops.
[z] Treatment means followed by the same letter are not significantly different according to Duncan's multiple range test ($P = 0.05$).

Table 4. Summary of *Pinus monticola* family means for seven resistant, control-pollinated and 13 untested, open-pollinated families for seven responses to inoculation with two Oregon wild-type (WT) and three Champion Mine (CM) sources of *Cronartium ribicola*

		Pine-rust combination			
		Resistant, control-pollinated		Untested, open-pollinated	
Response	Units or ratio	CM	WT	CM	WT
Incubation period	Months	4.6	4.5	4.6	4.5
Infection efficiency	Spots/spore	0.00034	0.00018	0.00047	0.00027
Shedding of infected needles	Seedlings lacking spots/ seedlings in family	0.10	0.45	0.05	0.11
Retarded appearance of canker	Seedlings cankered in second year/total seedlings cankered after 2 yr	0.06	0.07	0.04	0.06
Stunted leader	Seedlings with stunted leaders/ seedlings with cankers in second year	0.46	0.20	0.39	0.24
Interval from inoculation to mortality	Months	22.30	23.10	22.10	22.60
Infection-free second year	Seedlings not infected/ seedlings in family	0.08	0.48	0.01	0.04

Table 5. Summary of means for seven families of resistant, control-pollinated and 13 families of untested, open-pollinated *Pinus monticola* for occurrence of three kinds of resistance mechanisms after inoculation with two Oregon wild-type (WT) and three Champion Mine (CM) sources of *Cronartium ribicola*

Symptom combinations			Proportion in pine-rust combination[b]							
			Resistant, control-pollinated				Untested, open-pollinated			
			CM		WT		CM		WT	
Infected needles after 1 yr	Cankers	Resistance mechanism[a]	N	X/N[c]	N	X/N	N	X/N	N	X/N
Absent	None	PSN	32	0.38	76	0.80	25	0.16	35	0.23
Absent	Appeared second year	RCA	20	0.25	15	0.47	21	0.14	27	0.37
Present	None	FSS	220	0.0004	92	0.24	443	0.002	277	0.01
Present	Appeared second year	RCA	211	0.005	70	0.05	442	0.004	274	0.03

[a] PSN = premature shedding of infected needles defined as infected needles present at 9 mo, absent at 12 mo, and no canker at 24 mo; RCA = retarded canker appearance; FSS = fungicidal short-shoot, defined as infected needles present at 9 and 12 mo and no canker at 24 mo.
[b] Number of seedlings in each pine-rust combination: resistant, control-pollinated-CM = 252, WT = 168; untested, open-pollinated-WT = 312; CM = 468.
[c] X = number of resistant seedlings; N = number in symptom combination. N in PSN + FSS rows sum to total seedlings; N in RCA rows sum to total cankered.

evident. Inoculum from CM reduced the frequency of the fungicidal short-shoot mechanism in the open-pollinated population by a factor of 10, and in the resistant population by a factor of 600. Frequencies of retarded canker appearance were reduced by factors of 7.5 and 10 in open-pollinated and resistant populations, respectively (Table 5).

DISCUSSION

In order of appearance, the effects of the CM source compared with WT were longer spot incubation period, increased rust intensity, reduced proportion of seedlings shedding all infected needles within 1 yr of inoculation, less frequent retarded canker appearance, increased proportion of seedlings with stunted leaders in the first growing season after inoculation, reduced mortality period, and reduced proportion of trees free of disease 2 yr after inoculation.

Blister rust passes through five major phases to reach the canker stage: penetration of needles, growth of a pseudosclerotium in the needle, growth of mycelial strands (hyphal runners) down the needle, penetration of the short shoot by the runners, and growth in the bark leading to discoloration, fusiform swelling, and bark cracking or scaling (2). Both interruptive and growth-retarding resistance mechanisms function within these developmental phases. The interruptive mechanism (premature shedding of infected needles) apparently involves dropping infected needles after the pseudosclerotium forms but before the hyphal runners penetrate the short shoot. McDonald and Hoff (10) observed that in resistant families, about 54% of trees that dropped all of their infected needles within 1 yr failed to produce cankers. In this study, 80% of seedlings from phenotypically resistant, control-pollinated parents shed their infected needles prematurely and failed to produce cankers (Table 5). Among progeny of open-pollinated trees, by contrast, 33% from susceptible parents dropped infected needles early in the previous study. After exposure to WT inoculum in this study, 23% of the progeny of open-pollinated "resistant phenotypes" shed their infected needles and did not display cankers (Table 5). Inoculum from CM reduced the needle-shedding trait regardless of previous selection history (Table 5).

Another kind of interruptive resistance is the fungicidal short shoot (4). In a previous progeny test, 17% of seedlings in resistant families versus 3% in susceptible families displayed this mechanism (10). Corresponding data in the present study were 24 and 1% (Table 5). Champion Mine inoculum negated this mechanism, though not completely.

Analysis of variance showed that inoculum source caused significant variation in six of eight symptoms studied (Tables 1 and 2). Duncan's multiple range test showed that the means could be placed into CM versus WT groups for three traits: stunted infected leader, proportion of seedlings free of infection, and proportion that shed infected needles early.

Rust intensity could influence the ranking of such characters as spot appearance, canker appearance, proportion of seedlings that dropped all infected needles, and mortality period. When the five inoculum sources were combined into two principal sources (CM and WT) and the families and selection histories compared (Table 5), selection for resistance to WT inoculum decreased rust intensity by a factor of about 1.5. CM inoculum increased rust intensity about 2× in both selected and nonselected populations, but selection for resistance to WT inoculum decreased rust intensity by a factor of 1.5 after inoculation with both inoculum types. Thus, selection for resistance to WT reduced the intensity of rust caused by each inoculum source.

As expected, retarded appearance of cankers and mortality period were correlated. The pine-rust combination in which retarded appearance was most common also gave the longest mortality period and vice versa.

One of the most interesting traits studied was stunted leaders. The CM source was clearly differentiated from WT on most families by the frequency of stunted, infected leaders. Stunting is apparently caused by growth of the rust into the bud before host growth begins. The evidence for this is that stunting often occurs in conjunction with formation of numerous spots on needles produced by the stunted leader when the needles have not been exposed to basidiospores. Also, the new stunted growth often supports many pycnia (McDonald et al, *unpublished*). In this case, the CM rust sources penetrated the dormant bud about twice as often as did WT sources.

Decreased mortality period, more rapid appearance of cankers, and increased proportion of stunted infected leaders all fit with the idea that the CM sources were growing at a faster rate in the stem and branch tissues than the WT sources were. Only the increased needle incubation period is an unexpected result.

The CM source appears to be a real entity that carries implications for both management and research of white pine blister rust. For example, the differential infection efficiencies of these inoculum sources could be used to study infection mechanics, as discussed by Patton and Spear (13). Because most highly resistant trees utilize needle-shedding and short-shoot resistance (6), the geographic extent of the CM strain, its influence on resistant white pine selected from exposure to northern Idaho inoculum, its influence on sugar pine selections, and its interaction with species and geographic origins of *Ribes* should be determined soon.

LITERATURE CITED

1. Barr, A. J., Goodnight, J. H., Sall, J. P., Blair, W. H., and Chilko, D. M. 1979. SAS User's Guide. SAS Institute Inc., Raleigh, NC. 494 pp.
2. Bingham, R. T., Squillace, A. E., and Wright, J. W. 1960. Breeding blister rust resistant western white pine. II. First results of progeny tests including preliminary estimates of heritability and rate of improvement. Silvae Genet. 9:33-64.
3. Day, P. R. 1974. Genetics of Host-Parasite Interaction. W. H. Freeman & Co., San Francisco. 238 pp.
4. Hoff, R. J., and McDonald, G. I. 1971. Resistance to *Cronartium ribicola* in *Pinus monticola*: Short shoot fungicidal reaction. Can.

J. Bot. 49:1235-1239.
5. Hoff, R. J., and McDonald, G. I. 1980. Improving rust-resistant strains of inland western white pine. U.S. For. Serv. Res. Pap. INT-245. 13 pp.
6. Hoff, R. J., McDonald, G. I., and Bingham, R. T. 1973. Resistance to *Cronartium ribicola* in *Pinus monticola*: Structure and gain of resistance in the second generation. U.S. For. Serv. Res. Note INT-178. 8 pp.
7. Kinlock, B. B., Jr., and Comstock, M. 1980. Cotyledon test for major gene resistance to white pine blister rust in sugar pine. Can. J. Bot. 58:1912-1914.
8. McDonald, G. I. 1978. Segregation of "red" and "yellow" needle lesion types among mono-aeciospore lines of *Cronartium ribicola*. Can. J. Genet. Cytol. 20:313-324.
9. McDonald, G. I. 1979. Resistance of western white pine to blister rust: A foundation for integrated control. U.S. For. Serv. Res. Note INT-252. 5 pp.
10. McDonald, G. I., and Hoff, R. J. 1970. Resistance to *Cronartium ribicola* in *Pinus monticola*: Early shedding of infected needles. U.S. For. Serv. Res. Note INT-124. 8 pp.
11. McDonald, G. I., and Hoff, R. J. 1981. Engineering rust resistant white pines. Pages 404-411 in: Resistance to Disease and Pests in Forest Trees. H. M. Heybroek, R. Stephan, and K. Von Weissenberg, eds. Pudoc, Wageningen, Netherlands.
12. McDonald, G. I., Hoff, R. J., and Wykoff, W. R. 1981. Computer simulation of white pine blister rust epidemics: Model formulation. U.S. Dep. Agric. For. Serv. Res. Pap. INT-258. 136 pp.
13. Patton, R. F., and Spear, R. N. 1980. Stomatal influences on white pine blister rust infection. Phytopathol. Mediterr. 39:1-7.
14. Powers, H. R., Jr., Matthews, F. R., and Dwinell, L. D. 1977. Evaluation of pathogenic variability of *Cronartium fusiforme* on loblolly pine in the southern USA. Phytopathology 67:1403-1407.
15. Schein, R. D. 1964. Design performance and use of a quantitative inoculator. Phytopathology 54:509-513.
16. Snow, G. A., Dinus, R. J., and Kais, A. G. 1975. Variation in pathogenicity of diverse sources of *Cronartium fusiforme* on selected slash pine families. Phytopathology 65:170-175.
17. Steel, R. G. D., and Torrie, J. H. 1960. Principles and Procedures of Statistics. McGraw-Hill, New York. 481 pp.