Disease Detection and Losses

Effects of Comandra Blister Rust on Growth and Survival of Lodgepole Pine

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ABSTRACT

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Comandra blister rust, caused by the fungus *Cronartium comandrae*, reduced growth and survival of lodgepole pine trees (*Pinus contorta* subsp. *latifolia*) in the central Rocky Mountains. Stem analyses were used to determine annual increments of total cubic bolewood volume for 74 lodgepole pine trees 4–22 m tall, cankered by comandra blister rust. Mean annual increments after canker establishment were reduced 32% for trees with crown tops killed by comandra blister rust (spiked) and 9% for trees not yet girdled. Volume increments of spiked trees decreased each year after canker establishment until crown loss (top-kill) occurred; afterward, increments remained small and constant. Bolewood in spiked trees was sound but split and cracked above the canker. Reductions in radial increments for 140 spiked trees decreased with tree age when they were

top-killed and increased with severity of crown loss, measured as the proportion of stem length above canker center. Total and sapwood basal areas, crown ratio, and basal area increment were highly correlated among spiked trees. Sixty percent of 2,070 trees on either temporary or monitored plots survived top-kill; percent survival increased with canker height. On 15 remeasured plots, no spiked trees with cankers higher than 13 m had died during the past 5 yr; trees with cankers lower than 13 m died at rates inversely proportional to canker height. Functions describing expected reductions in radial increment and survival can be incorporated into stand-growth and yield models to provide better estimates of disease effects.

Additional keywords: stem rust, tree mortality.

Comandra blister rust, caused by the fungus *Cronartium comandrae* Peck, is a serious canker disease on many species of hard pines, including lodgepole pine (*Pinus contorta* Dougl. ex Loud. subsp. *latifolia* (Engelm. ex S. Wats.) Critchf.) in the central Rocky Mountains (22). This stem rust is especially common in portions of the Beaverhead, Shoshone, and Medicine Bow National Forests of Montana and Wyoming, and disease outbreaks in these forests are expected to continue (3,14,21). Damage to cankered trees includes mortality, growth loss, defect, and reduced cone production (11,36). Impact projections suggest that forest management of timber resources is seriously threatened in 40-yr-old stands with more than 20% of their trees cankered and in stands located within several kilometers of inoculum sources (15,17).

Comandra blister rust on lodgepole pine develops as a typical stem rust disease (24). Infection occurs through needles or young shoots, and disease usually begins on midcrown branches (16). Only half of the branch infections located 19 cm from the bole are likely to establish perennial stem cankers within 8 yr (16). After several years of expansion, stem cankers girdle their host, kill cambium, and retard transport of water, nutrients, and photosynthates (24). Girdling and attack by other agents lead to crown loss above the canker (top-kill) (Fig. 1) (20,34). Trees not killed immediately may remain alive for decades as "spiked" trees (Fig. 1). Original crown tops are occasionally and temporarily replaced by lower branches that assume terminal dominance, but expanding cankers eventually kill these branches as well. Growth rate and longevity of cankered trees are greatly reduced.

To quantify effects of comandra blister rust on growth and survival of lodgepole pine, we established several series of plots

in typical, infested stands and conducted stem analyses on selected trees from these stands. Our objectives were to develop simple functions for predicting increment loss and tree death with practical inventory variables and to determine if total and sapwood basal areas and crown ratio of spiked trees were correlated with basal area increment.

MATERIALS AND METHODS

Stem analyses. We adapted procedures and a computer program for stem analysis (19) to determine: 1) trends in annual volume-increment accretion, 2) reductions in periodic radial increment, and 3) correlations among total and sapwood basal areas, crown ratio, and basal area increment. We established 137 plots in infested stands in the Beaverhead (Montana), Shoshone (Wyoming), and Medicine Bow (Wyoming) National Forests (21) and cut 225 trees for stem analysis. Both plots and trees were selected to represent the range of stand and tree conditions for age, density, and disease severity found in the sampled forests. Using information from these trees, we assembled three data sets to compute: 1) annual volume increments, 2) periodic radial increments, and 3) periodic basal area increments, sapwood basal areas, and crown ratios.

The first data set was composed of 105 intensively sampled, live trees from 17 plots (total area 0.7 ha), including 31 trees without rust cankers (healthy), 51 trees with nongirdling stem cankers (cankered), and 23 trees with dead crowns above girdling cankers (spiked). We collected eight to 16 stem disks per tree at heights of 0.3 and 1.4 m, canker bottom, middle, and top, bottom of live crown, and at additional heights, so diameter differences between adjacent disks did not exceed 3 cm. Annual height-radius profiles were constructed with stem-analysis techniques described by Herman et al (19). We expanded Herman's method to include the use of a geometric mean of two to four

radii for eccentric disks and interpolation of height increments to be proportionate with radial increment (rather than constant). We added procedures for cross-checking ring ages among disks and for relating tree growth to stages of disease progression by dating relevant annual rings indicating when cankers entered stems (canker establishment) and when the crown above the canker died (top-kill). We also modified Herman's summary program to compute and display cumulative total cubic volume by year according to Smalian's formula (40). Mean annual volume incre-

ments were computed for one or more periods of indefinite length by fitting linear regression models to logarithmic transformations of volume and age. One period, 20–100 yr, was used for healthy trees. Two periods were used for cankered trees: 10 yr or more prior to canker establishment and after canker establishment. Three periods were used for spiked trees: 10 yr or more prior to canker establishment, between canker establishment and top-kill, and after top-kill. Expected volumes at time of sampling were determined by extrapolating volume increments from periods

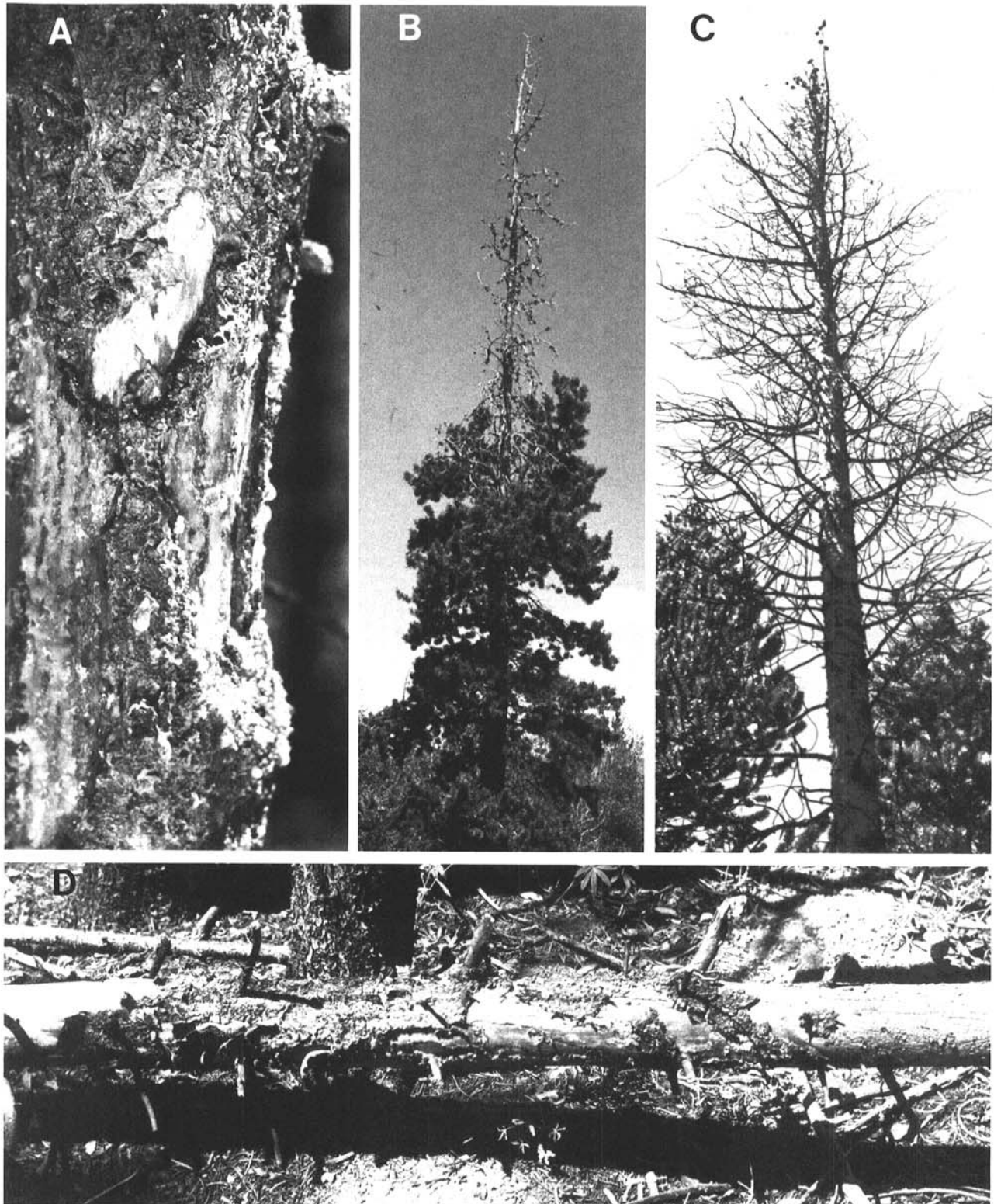**Fig. 1.** Lodgepole pine trees girdled by comandra blister rust (*Cronartium comandrae*). **A,** 7-cm-diameter tree with a girdling stem canker (much of the bark has been removed by squirrels except for several small bridges of intact bark); **B,** spiked tree; **C,** recently killed tree; and **D,** fallen tree, long dead but still retaining rough, pitchy bark in a concentric pattern diagnostic for stem rust cankers.

prior to canker establishment (1). The validity of these extrapolations was assessed by comparisons with increment trends of healthy trees. Volume loss was computed as a percent, the percent reduction in total cubic bolewood volume relative to expected volume. Significance of differences in average tree size, age, increment, and volume loss were evaluated with general linearized models (SAS, release 6.03, SAS Institute Inc., Cary, NC); univariate differences (by analysis of variance and Tukey's HSD test) were considered significant at $P < 0.05$.

For a set of 140 live, spiked trees on 120 plots, we measured total stem length and collected disks from 1.4 m above ground, top of live crown, and middle of canker. Ten-year radial increments were determined for the 1.4-m-above-ground disks: a reference period prior to canker establishment and a response period after top-kill. Proportional reductions in radial increments were modeled by nonlinear regression (SAS, release 6.03) with independent variables for tree age when the crown died and severity of crown loss measured as the proportion of stem length above canker middle.

Many of the spiked trees used to predict reductions in radial increment also were used to evaluate correlations among total and sapwood basal areas, crown ratio, and basal area increment since top-kill. From the stem-analysis data described above for 133 trees, we read four average radial lengths per tree at the 1.4 m height for three measures (pith to cambium, to heartwood-sapwood interface, and to the ring produced in the year of top-kill). Using these radii, we computed total and sapwood basal areas and basal area increment since top-kill. Although crown-length data were incomplete for 34 trees, we were able to determine live-crown ratios for 99 trees with measurements of total tree height and live-crown length. Pearson's correlation coefficients were then computed for total and sapwood basal areas, crown ratio, and basal area increment since top-kill.

Plot observations. We established 217 temporary and 15 monitored plots across the range of stand conditions for age, density, and disease severity in the Beaverhead, Shoshone, and Medicine Bow National Forests. Using data from these plots, we determined rates of rust-associated mortality that occur in cankered trees immediately after top-kill and in spiked trees during subsequent decades.

On 217 temporary plots (total area 9.2 ha), we measured canker heights for all girdled lodgepole pine trees, including those still alive (spiked) and those dead but still standing (rust-killed). Mean canker heights of spiked and rust-killed trees were compared with the Student t test (at $P < 0.05$). For 1-m canker-height classes, we computed percentages of girdled trees that were spiked and developed a sample-weighted linear model relating percent survival to canker height.

On 15 plots (total area 0.9 ha), we measured canker heights, monitored crown color and canker extent, and observed mortality in rust-infected trees. Infected trees were classified in 1982 as

cankered if they bore nongirdling stem cankers or as spiked if they had dead crowns above girdling cankers. The same trees were reclassified in 1987 as green (green foliage throughout crown), yellow (yellow foliage above cankers), spiked (sparse, red foliage above cankers and green foliage below cankers), or dead (all foliage red or absent).

Trees that were cankered in 1982 and either spiked or dead in 1987 were grouped into three damage classes by canker height (<5, $5-10$, or >10 m). Observed survival rates were determined by damage classes; expected survival rates were computed with the relationship between survival and canker height developed from trees on temporary plots.

Trees that were spiked in 1982 were grouped into 2-m canker-height classes. Proportions of trees by height class that had died during a 5-yr observation period were computed and related to canker height with sample-weighted linear regression.

RESULTS

Volume increment. The 105 trees sampled for volume increment (Table 1) ranged from 4 to 47 cm in diameter, 4 to 22 m tall, 22 to 168 yr old, 0.003 to 1.3 m^3 volume of bolewood, and a mean annual increment of 0.1 to 11.5 dm^3/yr. The 74 cankered and spiked trees had been infected in the bole for 2–36 yr; cankers were 0.4–17 m from the ground, and annual volume increments after canker establishment ranged from 0.23 to 15 dm^3/yr. In aggregate, sample trees included ranges in size, age, and increment representative of lodgepole pine trees found in infested stands of central Rocky Mountain forests.

Healthy, cankered, and spiked lodgepole pine trees were similar in mean diameter, height, age, volume, and mean annual increment, but cankered and spiked trees displayed notable differences in volume increment measured as volume loss (Table 1). Loss for healthy trees ranged from -7 to $+6\%$ (trees with recent accelerations in volume increments show negative loss). Although some cankered trees increased their increment after becoming cankered (minimum -7%), cankered trees had an average of 8.7% less volume than they were expected to produce (maximum 33%). For spiked trees, loss not including defect averaged 31.8% (range 2–87%).

Although cankers on spiked trees were higher on average than were cankers on nongirdled trees and required fewer years to girdle host stems, differences in annual volume increment in the period between canker establishment and top-kill were not significant (Table 2). Mean annual volume increment for spiked trees decreased after top-kill from 4.7 to 1.6 dm^3/yr.

Graphs of total volume by tree age for cankered and spiked trees revealed consistent patterns of increment reduction compared to healthy trees (example graphs for three typical trees are illustrated in Fig. 2). Plots for the 31 healthy trees showed generally small changes in volume increment over 20- to 30-yr

TABLE 1. Effects of cankering and top-kill caused by comandra blister rust (*Cronartium comandrae*) on volume increment of lodgepole pine trees

Damage class[1]	No. of trees	DIB (cm)[u]	Height (m)[v]	Age (yr)[w]	Volume (m^3)[x]	MAI (dm^3/yr)[y]	Volume loss[z]
Healthy	31	17	14	76	0.22	2.6	−0.2a
		(1.1)	(0.9)	(6.4)	(0.04)	(0.3)	(0.5)
Cankered	51	16	13	68	0.22	2.7	8.7b
		(1.1)	(0.7)	(5.0)	(0.03)	(0.3)	(1.3)
Spiked	23	21	14	85	0.29	3.2	31.8c
		(1.6)	(0.7)	(3.8)	(0.57)	(0.5)	(4.4)

[1] Healthy trees were not infected; cankered trees had nongirdling stem cankers; spiked trees had the upper portion of their crown killed by girdling cankers. There were no significant differences in mean values (except for volume loss) by damage class as determined by analysis of variance ($P < 0.05$).
[u] Mean (SE) diameter inside bark at 1.4 m when tree was cut.
[v] Mean (SE) total stem length including dead top.
[w] Mean (SE) total tree age.
[x] Mean (SE) total cubic volume of bolewood.
[y] Mean annual increment (SE) (total cubic volume/total tree age).
[z] Mean (SE) reduction of volume at time of sampling as a percentage of expected volume if tree had maintained previous annual volume increment. Different letters designate significant mean differences at $P < 0.05$ (Tukey's HSD test).

TABLE 2. Comparisons of canker height and periodic volume increment for lodgepole pine trees infected by comandra blister rust (*Cronartium comandrae*)

Damage class[v]	No. of trees	Canker height (m)[w]	Period length (yr)[x]	Periodic annual increment (dm³/yr)[y]
Cankered	51	5.4a[z] (0.7)	12a (1.0)	4.3a (0.4)
Spiked	23	8.1b (0.7)	9b (1.0)	4.7a (0.7)

[v] Cankered trees had nongirdling stem cankers; spiked trees had the upper portion of the crown killed by girdling cankers.

[w] Mean (SE) height to center of canker.

[x] Mean (SE) period from time when canker became established in the stem until the tree was cut (cankered trees) or until the upper portion of the crown was killed (spiked trees).

[y] Mean (SE) annual rate of volume accretion during the period after canker establishment and before top-kill.

[z] Different letters designate significant differences at $P < 0.05$ (analysis of variance).
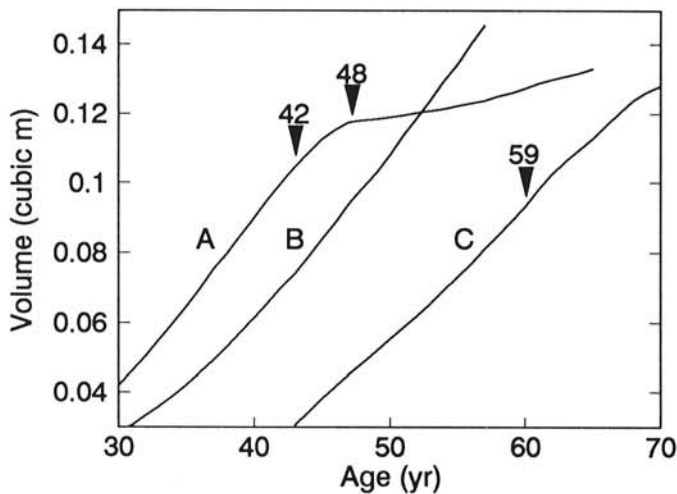
periods. A decline in growth of older healthy trees was evident over periods longer than 30 yr. An increase in annual increment was exhibited in all sampled trees of one stand entered 5 yr prior to the study for a shelterwood-seed cut. Most of the 51 cankered trees, however, exhibited a noticeable decrease in annual increment after boles were cankered. The 23 spiked trees not only displayed reductions in increment after canker establishment but reduced growth rates were sustained after top-kill occurred (height-radius profile for a typical tree is illustrated in Fig. 3). Interpretation of stem profiles of spiked trees suggested that reductions in volume increment resulted from complete cessation of xylem production above cankers and from large uniformly distributed reductions in increment below cankers.

Radial increment. Reductions in radial increment were related to tree age and severity of crown loss. The 140 sampled trees were 23–298 yr old and were 6–19 m tall when they were girdled and top-killed. From 8 to 71% of the stem length was killed, and radial increment after top-kill was reduced by 1–92%. For any tree with a proportion, X_1, of stem killed at age X_2 yr, the nonlinear function $Y = 0.29 + 0.57(1 + e^{(-5.77X_1)}) - 0.0014X_2$ best described expected reduction in radial increment, Y ($R^2 = 0.38$, MSE = 0.03). In general agreement with our observations,

Fig. 2. Annual volume increments of three lodgepole pine trees **A**, cankered by comandra blister rust (*Cronartium comandrae*) at age 42 and top-killed at age 48; **B**, healthy; **C**, cankered at age 59 and girdled at age 72.
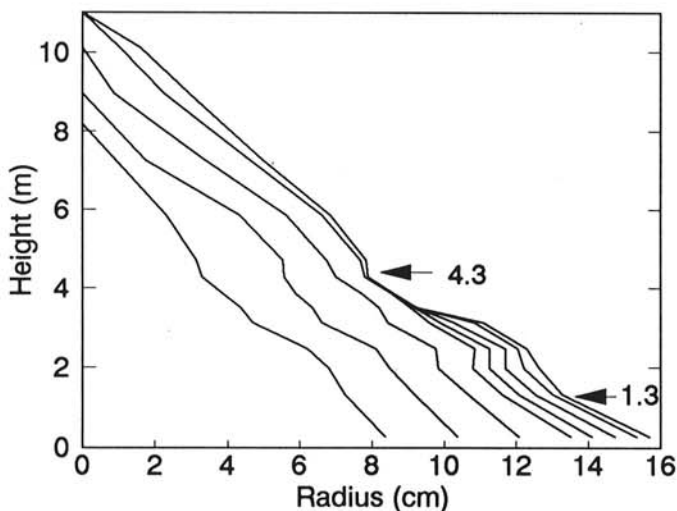

Fig. 3. Stem profiles from ages 20 to 55 (5-yr increments) for a lodgepole pine tree cankered by comandra blister rust (*Cronartium comandrae*). A stem canker at 4.3 m developed on the bole at age 30; top-kill occurred at age 35. At age 55, the live crown began at 1.3 m (total crown length was 2 m).
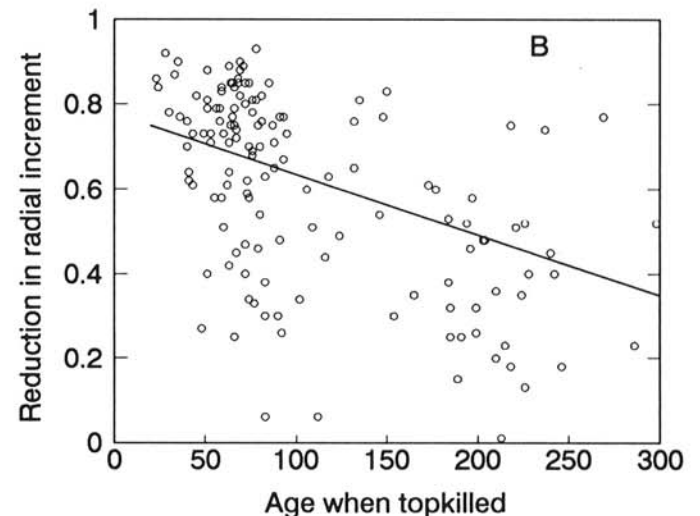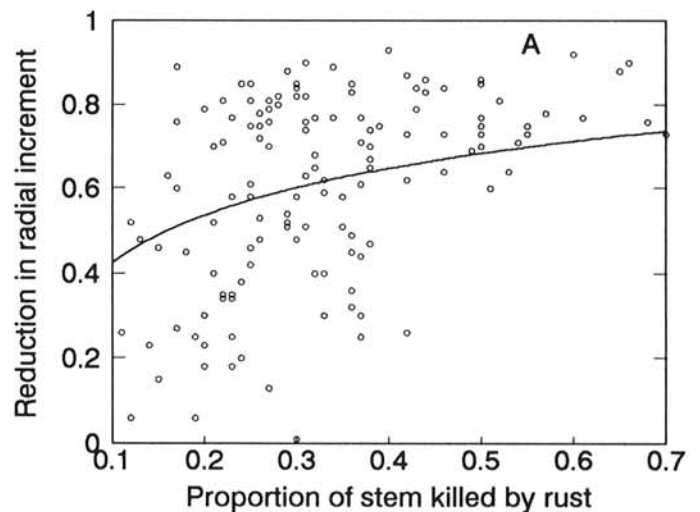

Fig. 4. Reduction in radial increment of lodgepole pine trees cankered by comandra blister rust (*Cronartium comandrae*); radial growth loss is the proportional reduction in radial increment after top-kill relative to radial increment before tree was cankered. **A**, observed (circles) and predicted (solid line) radial growth loss (Y) by proportion of total stem length above canker middle (X_1) (assuming top-kill occurred when tree was 100 yr old). **B**, observed (circles) and predicted (solid line) radial growth loss by age of tree at time of top-kill (X_2) (assuming upper 33% of stem was killed by a girdling canker). $Y = 0.29 + 0.57(1 + e^{(-5.77 \times X1)}) - 0.0014X_2$. ($R_2 = 0.38$, MSE = 0.03).

this model projected less increment reduction in older trees and greater reduction in trees with a higher proportion of stem killed (Fig 4.).

Decay and defect. We found no decay but some defect associated with comandra blister rust cankers. Resin usually flowed over and down from cankers, but it penetrated into the wood only near the middle of cankers. Bark above cankers was rapidly shed after top-kill, and boles soon cracked and split but remained sound. Branches <3 cm in diameter were retained on spiked trees for several decades.

Sapwood basal area. Among spiked trees, total and sapwood basal areas and crown ratio were highly correlated ($P < 0.001$) with each other and with basal area increment after top-kill (Table 3). Partial correlations after adjusting for crown ratio and for total basal area were also high ($P < 0.001$) between sapwood basal area and basal area increments.

Survival of top-killed trees. Nearly 60% of top-killed lodgepole pine trees found on 217 temporary plots were alive. Mean canker

TABLE 3. Correlations among crown ratio and basal area variables for lodgepole pine trees spiked by comandra blister rust (*Cronartium comandrae*)

Variables	Total basal area (cm²)[t]	Sapwood basal area (cm²)[u]	Basal area increment (cm²/yr)[v]
Crown ratio[w,x]	0.21	0.40	0.29
Total basal area[x]	...	0.79	0.53
Sapwood basal arena[x]	...	...	0.63
Total basal area[y]	...	0.77	0.54
Sapwood basal area[y]	...	...	0.61
Crown ratio[z]	...	0.39	0.21
Sapwood basal area[z]	...	...	0.41

[t] Mean total basal area at 1.4 m when trees were cut was 31.3 cm² ($n = 133$, SE = 1.3).

[u] Mean sapwood basal area was 17.2 cm² ($n = 133$, SE = 0.8).

[v] Mean basal area increment since trees were top-killed was 0.16 cm²/yr ($n = 133$, SE = 0.009).

[w] Mean live-crown ratio of spiked trees when they were cut was 0.19 ($n = 99$, SE = 0.01).

[x] Correlation coefficient.

[y] Partial correlation coefficient, adjusting for crown ratio.

[z] Partial correlation coefficient, adjusting for total basal area.
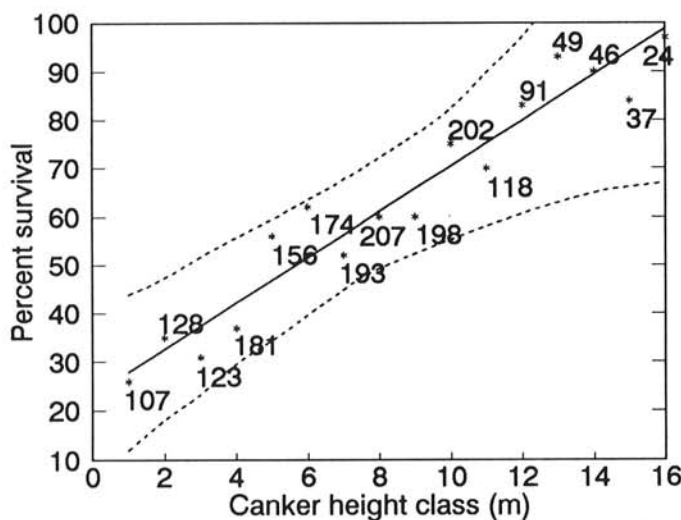

Fig. 5. Percentage of lodgepole pine trees surviving top-kill by girdling cankers caused by comandra blister rust (*Cronartium comandrae*): percentage of all spiked and rust-killed trees in a 1-m canker-height class that were live at time of examination (asterisks). Numbers indicate total sample in each class, predicted survival (solid line) by sample-weighted linear regression of percent survival (*Y*) on canker height in meters (*X*), and 95% prediction band (dotted lines). $Y = 23.3 + 4.7 \times X$. ($R^2 = 0.90$, MSE = 4,884.)

height for 1,228 spiked trees (9.0 m, SE = 0.1) was significantly greater ($P < 0.001$) than the mean canker height for 842 rust-killed trees (6.1 m, SE = 0.1). No rust-killed trees were found with cankers higher than 17 m. For trees with canker heights less than 17 m, the linear regression $Y = 23.3 + 4.7X$ related percent survival, *Y*, with canker height (meters), *X* ($R^2 = 0.90$, MSE = 4,884) (Fig. 5).

Five-year disease progression. The 15 monitored plots established in 1982 included 94 cankered trees and 180 spiked trees. Only 26 cankered trees were top-killed 5 yr later, and only 15 spiked trees had died (Table 4). Among cankered trees that were later top-killed, survival rates for canker-height classes of 5–10 and 10–20 m were nearly equal to rates projected by the model developed from trees on temporary plots (Table 5). But, only 8% of trees with cankers 0–5 m above ground survived top-kill; the proposed model suggested that 31% would survive.

For trees that were already spiked in 1982, continued survival depended on canker height. No spiked trees with cankers higher than 13 m died. For spiked trees with cankers below 13 m, the 5-yr mortality rate, *Y*, decreased in proportion to canker height (meters), *X*, according to the relationship $Y = 0.21 - 0.02X$ ($R^2 = 0.95$, MSE = 0.0065) (Fig. 6).

DISCUSSION

Severity of damage caused by stem rusts depends on canker height and increases as the disease progresses (29,43). For comandra blister rust, initial damage is limited to minor loss of infected branches. But as cankers expand into stems, more cambium is killed, and after girdling, either the upper crown or entire crown is killed. Unlike other stem rusts (18), decay is rarely associated with comandra blister rust cankers, but like other stem diseases, sound defect can be important (11,31). On trees that survive top-kill, cankers continue to expand down stems and kill more crown at an average rate of 4.4 cm/yr (16). Because of top-kill, attack by other agents (20,34), and competition from other trees (18,37), cankered trees eventually die. The impacts of comandra blister rust on timber management (32) depend on stage of disease pro-

TABLE 4. Crown conditions for 274 lodgepole pine trees either cankered or spiked by comandra blister rust (*Cronartium comandrae*) 5 yr after an initial observation

1982 damage class[v]	1987 crown condition (number of trees)				
	Green[w]	Yellow[x]	Spiked[y]	Dead[z]	Total
Cankered	48	20	11	15	94
Spiked	...	...	165	15	180

[v] In 1982, cankered trees had nongirdling stem cankers and either green or yellow upper crowns, and spiked trees were alive but had girdling stem cankers and upper crowns that were already top-killed.

[w] A green crown indicated the canker had not yet girdled the stem.

[x] A yellow or fading crown indicated the canker had girdled the stem, but it was unknown whether the whole tree would die or only the crown above the canker.

[y] The crown of a spiked tree was dead above the canker and green below.

[z] Crowns with no green foliage were classified as dead.

TABLE 5. Observed and predicted survival of lodgepole pine trees recently top-killed by cankers of comandra blister rust (*Cronartium comandrae*)

Canker height class (m)	No. of trees	Mean canker height (m)	Percent surviving	
			Observed[y]	Predicted[z]
0–5	13	2	8	31
5–10	5	8	60	59
10–20	6	13	88	84

[y] Percentage of trees cankered in 1982 that were alive in 1987, spiked trees.

[z] Percentage of trees predicted by canker height to survive top-kill (percent survival = 23.3 + 4.7 × canker height in meters).

gression, height of initially infected branch, and involvement of other damage agents.

Effects on growth. Quantitative comparisons among healthy, cankered, and spiked trees suggest how disease progression affects volume accretion. Although healthy and rust-diseased trees were located in the same stands, were measured over common periods, and were similar in size and age, diseased trees showed striking reductions in volume increment, whereas healthy trees maintained nearly constant growth (Table 1). Even though cankers on spiked trees were higher and observation periods were shorter than on nongirdled trees, mean annual increments before top-kill did not differ (Table 2). Mean annual increments of spiked trees were reduced 34% after top-kill. We suggest that canker development causes a slight reduction in volume increment before the bole is girdled and severe, sustained reductions in increment after top-kill. In spite of recent growth losses, these diseased trees had lifetime annual increments similar to healthy trees, implying that earlier these trees must have been among the larger and faster growing trees in their respective stands (12,29).

Volume-age graphs and stem profiles illustrate on an individual tree basis how disease progression relates to volume increment (Fig. 2–3). Localized reductions in annual xylem layers (10) are apparent in stem profiles and clearly are associated with canker development. Small but increasing reductions in volume increment appear on volume-age graphs after canker establishment. Top-kill is evident in stem profiles and marks distinct changes in growth rate and tree form. Previously declining rates of volume increment stabilize at reduced levels and xylem is laid thinly but uniformly below the girdling canker.

Modeling effects of comandra blister rust on volume increment can be accomplished by disregarding losses before top-kill and breaking the problem into components for taper and height and radial increments. Proper taper and height increments for spiked trees can be obtained by truncation at canker height (30). This truncated height may be either fixed at the height of the canker center or lowered as a function of the rate of canker expansion (16). Radial increment for spiked trees can be modeled as a proportional reduction from the increment expected for trees of similar size and competitive status (usually measured as tree diameter and stand basal area). We suggest that the proportional reduction in radial increment can be projected from tree age, stem length, and canker height.

The proportional reduction in radial increment of spiked trees is fundamentally related to change in crown size (13,25). Unfortunately, it is not feasible to model growth directly from crown

Fig. 6. Five-year mortality rate of lodgepole pine trees previously top-killed by comandra blister rust: proportion of trees in a 2-m canker-height class that died within 5 yr of initial observation (asterisks). Numbers indicate total sample in class, predicted mortality (solid line) by linear regression of mortality rate (weighted by sample size) on canker height in meters (X), and 95% prediction band (dotted lines). $Y = 0.21 - 0.02 \times X$. ($R^2 = 0.95$, MSE $= 0.0065$.)

size, because data on crown size (length, width, and density) are usually not available or are subject to considerable estimation error and inherent variation (5,13). A useful variable that can substitute for severity of crown loss is the proportion of stem length above the girdling canker. This proportion is easily determined from simple inventory variables (stem length and canker height) and along with tree age, is highly correlated to proportional reduction in radial increment (Fig. 4).

Consistent with other studies (25,28), we found that the upper crown contributed more to radial increment than did the lower crown. Based on our observations (Fig. 4), if a tree was top-killed at age 100 and the upper 20% of the stem was killed, we would expect a 54% reduction in radial increment. If stem loss was 40 or 60%, then increment reductions would be 67 or 70%, respectively. The marginal change of increment reduction decreases with additional stem loss.

Because of the wide range of ages included in our data (23–298 yr), tree age at the time of top-kill could be identified as a significant term for predicting increment reduction (Fig. 4). Although increment reduction is not very sensitive to tree age, the effect of this variable becomes important when comparing trees with large age differences. For equal amounts of stem loss in trees of similar size, top-kill proportionally reduces radial increment by much less in 150-yr-old trees (with long, narrow crowns and small increments) than in 50-yr-old trees (with short, broad crowns and large increments).

Sapwood basal area. Numerous studies have identified strong correlations between leaf area index (LAI) and sapwood basal area (6,26,39), and several authors have used this relationship to describe tree vigor and growth (7,38,42). The observed high correlations are explained by physical and physiological factors—mechanical support, water balance, photosynthesis, respiration, and hormonal control (23,27,35). Although LAI provides useful information (41), it is difficult to measure directly; sapwood basal areas, however, can be determined easily from increment cores.

Few studies have investigated responses of sapwood basal area to defoliation, pruning, or top-kill. Britton (4) did not find significant 1-yr differences in sapwood area between control and defoliated lodgepole pine populations. Effects on sapwood basal area of pruning lower branches of balsam fir (*Abies balsamae* (L.) Mill.) varied with severity (28). Balsam fir trees pruned to 60% crown ratios showed no reductions; for trees pruned to 40%, sapwood reductions matched reductions in basal area increments; and for trees pruned to 20%, sapwood areas were reduced by decreases in basal area increments and by increases in heartwood areas.

Methods used in this study were not suitable to identify either how quickly a lodgepole pine-sapwood basal area responds to crown loss or what changes occur in ratios of sapwood/heartwood or sapwood/basal area increment. We were able to confirm, however, that total and sapwood basal areas, crown ratio, and basal area increment are highly correlated in spiked lodgepole pine trees (Table 3). Because sapwood basal areas are highly correlated with basal area increments after adjusting for crown ratios or total basal areas, sapwood basal area is a good candidate variable for describing effective crown size and for modeling basal area increment.

Effects on survival. Likelihoods of top-killed lodgepole pine trees dying from comandra blister rust depend on the extent of crown loss, but quantifying this relationship is difficult with available data. Although methods for estimating mortality rates with monitored plots are straightforward (8), we observed only 30 rust-killed trees during one short period on several plots (Table 4). Estimating mortality rates with temporary plots is less direct but uses many observations from plots across wide ranges of stand ages, densities, and levels of disease severity.

The proportion of trees girdled by comandra blister rust that were alive on temporary plots varied as a function of canker height (Fig. 5). Except for the rust-killed trees that were no longer standing and therefore not sampled, this proportion should reflect cumulative mortality rates of top-killed trees. If losses of rust-killed trees from cutting or toppling were equal across canker-
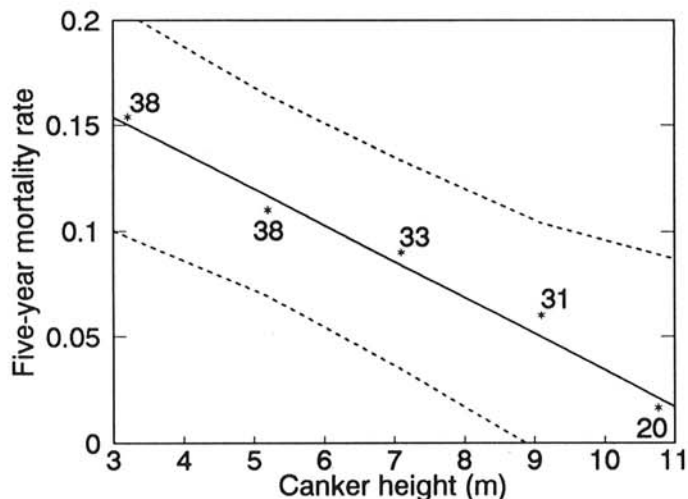
height classes, then differences in proportions of live trees should indicate effects of canker height on survival of top-killed trees. Because stumps and logs were rarely found on these temporary plots, most spiked and rust-killed trees would have been tallied, except for those <5 m tall, which would quickly fall and decay. Loss of these small trees would lead us to overpredict survival of trees with low cankers and explain the poor match between observed and predicted survival of trees with cankers <5 m above the ground (Table 5). Because small trees have low cankers and high-mortality rates (Fig. 6), the discrepancy disappears after several projection cycles. In contrast, the good match between observed and predicted survival (Table 5) of spiked trees >5 m tall supports the proposed survival model (Fig. 5). Based on observed mortality rates (Fig. 6), spiked trees with cankers <13 m high are soon overtopped and killed by neighboring dominant trees (18,37).

Management application. Functions described here for predicting reduction in radial increments, survival of top-killed trees, and 5-yr mortality rates of spiked trees could be incorporated into risk-rating systems (32), stand-growth models (9), and other forest-planning tools (17). Along with predictions of infection rates (3) and disease distribution (21), these decision tools provide information immediately useful for timber management. Additional research would be needed to relate the effects of crown loss to other resource values. Techniques have been developed to relate various forms of partial crown loss to visual quality (2). Although numerous species feed on aeciospores and rust-infected cambium (33), little information is available on secondary effects of rust outbreaks on wildlife habitat, (forage, perching, and cavity sites), ground cover, and downed, woody material.

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