

# Histological response of resistant and susceptible white spruce to simulated white pine weevil damage

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**Summary** The traumatic wound response of families of white spruce, *Picea glauca* (Moench) Voss, resistant or susceptible to the white pine weevil, *Pissodes strobi* (Peck), were compared after simulated weevil damage. Leaders from 331 trees were wounded just below the apical bud in the spring, coinciding with the natural time of weevil oviposition. A portable 1-mm diameter drill was used to drill 24 holes per leader. Leaders were removed in the fall and examined for evidence of traumatic resin canal formation. Drilled trees had a traumatic wound response 8 times greater than that of undrilled trees; however, undrilled trees also formed some resin canals in response to unknown causes. In the drilled trees, the traumatic wound response extended into the lower part of the leader, where it could possibly affect older larvae. Trees from resistant families responded with greater intensity than trees from susceptible families, by producing multiple rings of traumatic resin canals. Trees from resistant families also responded more rapidly than trees from susceptible families based on number of cells to the first ring of traumatic resin canals. Trees from some resistant families exhibited no traumatic resin canal formation, showing considerable within-family variation and suggesting that other resistance mechanisms might be important. In the year after drilling, there was a reduction in tree diameter growth and trees suffered a reduction in constitutive resin canals in the bark, which suggests some energetic cost of traumatic resin production. There was no indication that the extent of constitutive defenses, as measured by density of cortical resin canals before wounding, was related to the ability to produce traumatic resin canals. Screening trees based on their capacity to produce traumatic resin canals may be useful in selecting genotypes resistant to white pine weevil.

**Keywords:** host resistance, *Picea glauca*, *Pissodes strobi*, traumatic resin canals.

## Introduction

### *Role of the conifer resin canal system in resistance to pests*

Constitutive or preformed resin is considered to be the first line of defense in conifers against pathogens and stem-invading insects (Berryman 1972, Hanover 1975). The second line of

defense that follows wounding is the traumatic or induced response (Berryman 1972), which is characterized by the formation of a necrotic area in the immediate vicinity of the injury.

Resin canals that are present before injury are referred to as normal or constitutive resin canals and those produced in response to wounding are referred to as traumatic resin canals. Normal resin canals are elongated and usually occur singly, whereas traumatic resin canals are cyst-like and generally occur in tangential series (Thomson and Sifton 1926, Bannan 1933a, 1936, Esau 1965). Normal resin canals are not a constant feature in the xylem of *Picea* spp.; however, traumatic resin canals occur and extend both vertically and laterally (Bannan 1936).

Traumatic resin canals can be elicited by several stimuli, including mechanical trauma (Thomson and Sifton 1926, Bannan 1933b, Kuroda and Shimaji 1983, Chencilet 1987, Werner and Illman 1994), pathogens (Chencilet 1987, Lieutier and Berryman 1988, Werner and Illman 1994), plant hormones (Fahn et al. 1979), and exogenously applied elicitors derived from fungal cell walls such as chitosan and proteinase inhibitor inducing factor (PIIF) (Miller et al. 1986, Croteau et al. 1987, Lieutier and Berryman 1988).

Traumatic resinosis appears to be related to resistance of several conifers to bark beetles (Blanche et al. 1983, Raffa and Berryman 1983, Lorio and Hodges 1985). Resistance has been associated with the ability to respond rapidly (Tomiyama 1963, Berryman 1969, Berryman and Ashraf 1970) or strongly (Raffa and Berryman 1982a, Cook and Hain 1986, 1987), or both. The degree to which trees produce traumatic resin may be a species-specific characteristic and may depend on how frequently a species is attacked. Tree species subject to attack by many asynchronous generations of bark beetles in a year may be more likely to rely mainly on constitutive defenses, whereas those species attacked by few synchronous generations of beetles per year may rely heavily on induced resinosis. Preformed resin may be energetically less expensive than a response requiring repeated induction (Matson and Hain 1983). If trees of different genotypes relied more on one mechanism than the other, a negative correlation between the number of constitutive cortical resin canals and ability to produce traumatic resin canals would be predicted.

### *Resistance by conifers to the white pine weevil*

The white pine weevil, *Pissodes strobi* (Peck), is the most damaging pest of regenerating spruces in the Pacific Northwest (Silver 1968, Mitchell et al. 1990). The main hosts in this region are Sitka spruce, *Picea sitchensis* (Bong.) Carr, white spruce, *Picea glauca* (Moench) Voss, Engelmann spruce, *Picea engelmannii* Parry, and hybrids of the latter two species (Humble et al. 1994). (The complex of white spruce, Engelmann spruce, and their hybrids in British Columbia is referred to as interior spruce (Kiss and Yanchuk 1991).) Weevil eggs are laid just below the apical bud of leaders in the spring, and when larvae hatch, they mine downward through the phloem tissue, girdling and killing the leader (Silver 1968). Repeated leader destruction reduces height growth and causes stem deformities that significantly reduce tree value (Alfaro 1982). Direct control techniques for the weevil are either ineffective, impractical, or too costly to be used on a large scale (Stiell and Berry 1985, McMullen et al. 1987, Alfaro and Omule 1990, deGroot and Helson 1993, Rankin and Lewis 1994).

Given the inadequacy of direct control methods, genetic resistance may be the most promising tool for managing *P. strobi* (Alfaro et al. 1995). Host resistance has been identified in both Sitka spruce (Alfaro and Ying 1990, Ying 1991) and interior spruce (Kiss and Yanchuk 1991, Kiss et al. 1994), and a number of potential resistance mechanisms have been identified (see review by Tomlin and Borden 1997). They include feeding or oviposition deterrence, repellency, inhibition of reproductive maturation, high density of cortical resin canals, bark thickness, and resin chemistry. These traits are all preformed characteristics of the tree and are not induced.

There is evidence that *P. strobi* is affected by the traumatic wound response of its host. Resin was observed as the major cause of brood mortality during early development in eastern white pine, *Pinus strobus* L., Sitka spruce and Norway spruce (*Picea abies* (L.) Karst) (Sullivan 1960, Overhulser and Gara 1981a, Dixon and Houseweart 1982, Therrien 1994). Traumatic resin canals form in interior spruce in response to injury by the white pine weevil. Resin from these canals appeared to flow into oviposition cavities, presumably killing eggs and larvae (Alfaro 1995). Trees that were successfully attacked had only 62% of the response level observed in trees with failed broods (Alfaro et al. 1996a). Traumatic resin canals have also been observed in Sitka spruce (Jou 1971, Tomlin 1996), with the response extending into the lateral branches (Alfaro and Tomlin, unpublished results).

We examined the traumatic wound response to simulated weevil attack in families of white spruce of known resistance and susceptibility to the white pine weevil. Our long-term goal is to incorporate the trait of traumatic resinosis into a multi-component resistance index (Brooks and Borden 1992, Alfaro et al. 1996b, Tomlin and Borden 1997) for use in screening parent trees and their progeny for a resistance breeding program. The specific hypotheses tested were: (1) mechanical wounding produces a consistent traumatic resin canal response that could be used as a resistance screening tool; (2) the extent and timing of the traumatic wound response in the xylem and bark is related to resistance status of the family; (3) the trau-

matic response is systemic; (4) traumatic resin canals are also induced in the bark of the leader; and (5) the capacity for induced resinosis varies with level of constitutive defense.

## Materials and methods

### *Study site*

In summer 1995, we conducted a simulated weevil wounding experiment in an interior spruce progeny test plantation established in 1984 near Clearwater, B.C. (51°42' N, 120°03' W) on seedlings from 142 open-pollinated parent trees that had been selected for superior growth characteristics from various regions across the B.C. interior. Each of eight replicates had 142 rows containing four seedlings from the same parent. Families were randomly allocated within a replicate. The first white pine weevil attacks in the plantation occurred in 1986. In 1994, a retrospective survey of the number of weevil attacks and tree growth in each year from 1986 to 1994 was done to determine the resistance ranking of the families (Alfaro et al. 1996c).

### *Experimental methods*

Ten trees from each of the 10 most resistant and the 10 most susceptible families were selected to be wounded; at least one tree from each of the same families served as an unwounded control, for a total of 331 trees. On April 18, 1995, before weevil activity was reinitiated, leaders were protected from weevil damage by means of crinoline bags. The bags were attached to the stem just below the apical bud and above the first whorl of branches using wire twisted around foam collars. This allowed new leaders to grow but prevented weevils from feeding or ovipositing on the old leader. On May 30, 1995, coinciding with the natural oviposition period of the weevils, 24 holes, 1 mm in diameter, were drilled into leaders with a portable electric drill. The holes, 0.5 cm apart and penetrating the bark only, were made along vertical lines (eight holes per line), on three sectors around the leader circumference, starting 5 cm below the apical bud. To exclude the weevils, the mesh bags were replaced on the leaders following drilling.

Leaders were removed on September 12, 1995 after the 1995 growing season. Ten-cm portions of the leader from the top (drilled section) and bottom (undrilled section) were fixed in FAA (formalin-acetic acid-alcohol) (Johansen 1940). Similar portions were removed from the top and bottom of unwounded controls. All samples were sectioned to 30 µm with a sliding microtome, and stained with 1% aqueous safranin.

Xylem of the current-year growth (formed in the year of wounding) was examined in cross section at 6× magnification and scored for degree of traumatic wound response based on a modification of the scoring system described by Alfaro et al. (1996a), where 0 represents no traumatic resin canals, 1 represents one ring of traumatic resin canals, 2 represents one ring plus a few scattered canals, and 3 represents two or more complete, or incomplete traumatic rings. The relative rapidity of the traumatic response was determined by measuring the distance and by counting the number of cells, from the outer edge of the 1994 growth ring to the first ring of traumatic resin canals with an ocular micrometer. The widths of the 1995

growth rings were compared between drilled trees and controls to determine if wounding had any effect on growth.

To determine if wounding caused any change in the constitutive resin canals in the bark, the numbers of inner and outer resin canals were counted in each cross section. Inner resin canals are the ring of large, uniformly distributed ducts closest to the cambium, and outer ducts refer to all other ducts, which are generally smaller and located close to the outer bark (Tomlin and Borden 1994). In addition, the radius of the cross section from the pith to outer bark was measured so that the density of resin canals (no. per cm of circumference) could be calculated.

To determine if the ability to produce traumatic resin canals in 1995 was related to the number of resin canals present in the bark formed in 1994, the bark resin canals were counted in sections made from an upper lateral branch removed from the base of each leader on April 18, 1995. These data provide the number of constitutive resin canals present before wounding and have been described by Alfaro et al. (1996c). The relationship between number of resin canals in the leader bark versus the number in the bark of lateral branches was determined using the regression equation,  $y = 0.536 + 0.442x$  (Alfaro et al. 1996c). This equation was developed based on data obtained from leader and lateral samples collected from an additional replicate at the same site. The relationship was used to convert resin canal densities observed in the branches to those that would have been in the leaders had they not been wounded by drilling.

#### Statistical analysis

The intensity of traumatic resin canal formation was compared between resistant and susceptible families, and drilled and undrilled trees by a Kruskal-Wallis test performed with the SAS software package Proc NPAR1WAY (SAS Institute 1988,

Cary, NC), except where analysis of covariance was required. The frequency distributions of scores between resistant and susceptible families and drilled and undrilled trees were compared by a  $\chi^2$  test (Zar 1984). The wound response was correlated with the initial diameter of the leader (before drilling) by Spearman's Rank Correlation (Proc Corr, SAS software package). All other variables were compared between trees from resistant and susceptible families, drilled and undrilled trees, and upper and lower parts of the leader by analysis of variance and analysis of covariance where appropriate (Proc GLM, SAS Software package). Spearman's Rank Correlation was used to determine if the number of constitutive resin canals was correlated with traumatic wound response score. In all cases,  $\alpha = 0.05$ .

## Results and discussion

### Traumatic resin canal induction in drilled trees

A typical response of interior spruce leaders to drilling is shown in Figure 1. Although the position of the cambium at the time of wounding was not determined, a reaction zone, presumably enriched with resin and phenolics (Shain 1967), formed in the xylem prior to the band of traumatic resin ducts (Figure 1). We hypothesize that the inner edge of this region, labeled 'C' in Figure 1, indicates the cambium location at the time of wounding (Yoshimura et al. 1981, Kuroda and Shimaji 1983). Drilled trees had a mean traumatic wound response score 8 times higher than that of undrilled trees (1.75 versus 0.22;  $P < 0.0001$ , Kruskal-Wallis test) (Table 1). The distribution of scores also differed significantly between drilled and undrilled trees (Figure 2). Drilled trees had a median score of two and had observations in all score categories. Undrilled trees had either no response or a score of one. The low level of traumatic wound response observed in the untreated trees

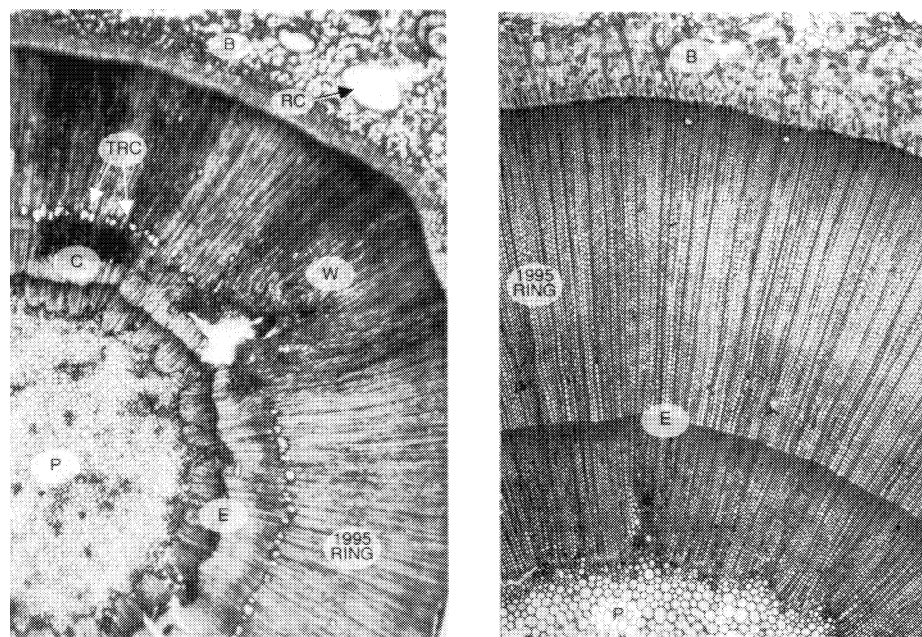


Figure 1. Photomicrograph of cross section of an interior spruce leader showing typical traumatic wound response to drilling (left) compared to an undrilled control leader (right). E = Edge of 1994 ring, C = possible location of cambium at time of wounding, P = pith, B = bark, RC = constitutive resin canal, W = region of xylem below the wounding site in the bark, TRC = ring of traumatic resin canals. Based on a 0–3 scoring system, the drilled leader would have a score of 2.

Table 1. Comparison of measurements (mean  $\pm$  SE) between the upper portions of drilled and undrilled white spruce leaders, averaged across resistant and susceptible trees.

Variable	Drilled leader	Undrilled leader	<i>P</i> Value, drilled versus undrilled <sup>1</sup>	<i>P</i> Value, effect of covariate (1994 radius)
Wound response score	1.75 $\pm$ 0.13	0.22 $\pm$ 0.13	0.0001	0.1813
Distance from 1994 annual ring to 1st traumatic resin canal ring (mm)	2.46 $\pm$ 0.04	3.02 $\pm$ 0.27	0.0001	0.0001
No. of cells to 1st traumatic ring	31.36 $\pm$ 0.93	46.07 $\pm$ 8.98	0.0001	0.0004
Radius of 1994 ring (mm)	1.84 $\pm$ 0.03	2.01 $\pm$ 0.05	0.0060	–
Width of 1995 ring (mm)	4.06 $\pm$ 0.08	4.42 $\pm$ 0.06	0.0036	0.0001

<sup>1</sup> The *P* value is for ANOVA after effect of radius of 1994 growth ring was removed as a covariate, except in the case of the 1994 radius.

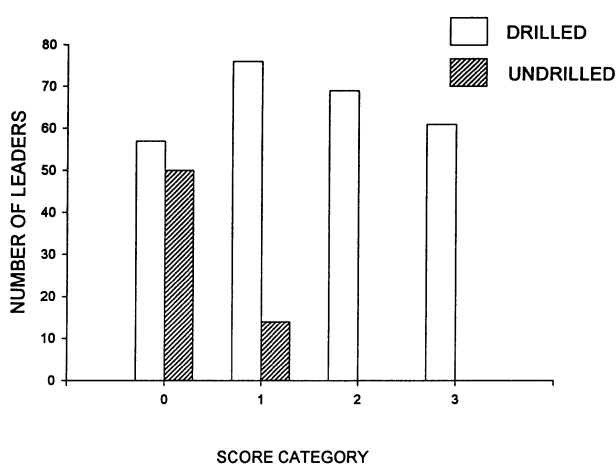


Figure 2. Frequency distribution of traumatic wound response scores in the upper xylem of 264 drilled and 64 undrilled leaders of interior spruce, including both resistant and susceptible families. Distributions were significantly different,  $\chi^2$  test,  $P < 0.001$ .

could have been caused by frost, mechanical stress or feeding by other insects. Fifty-seven of the drilled trees had a score of zero, including 22 from resistant families and 35 from susceptible families.

When a response occurred in undrilled trees, both the number of cells and the distance from the 1994 annual ring to the first traumatic resin canal ring were greater than on drilled trees (Table 1), suggesting that the severity of wounding by drilling increased the speed of the wound response, or that the damage to undrilled trees occurred earlier in the season. Rapidity and extent of traumatic resinosis are known to be affected by the type of stimulus (Lieutier and Berryman 1988).

The radius of the 1994 growth ring was smaller in drilled trees than in undrilled trees, which could have been caused by chance or researcher bias in tree selection. Width of the 1994 growth ring was thus included as a covariate in the comparison of all variables between drilled and undrilled trees, and trees from resistant and susceptible families. There was significantly less radial growth in 1995 (the same year as drilling) in drilled trees than in undrilled trees (Table 1) after removal of

the effects of the differential 1994 radial growth, suggesting that 1995 radial growth was reduced by the drilling treatment.

#### *Extent and timing of response compared between resistant and susceptible families*

On average, the intensity of the traumatic wound response was 1.3 $\times$  higher in resistant families than in susceptible families (Table 2). Both resistant and susceptible families produced responses in all score categories; however, there was a significant difference in the response profiles between the two populations (Figure 3). More susceptible families had a score of 0 (no response) or 1 (1 traumatic ring) than resistant families, whereas significantly more resistant families than susceptible families had a score of 3 (2 rings or more). This means that resistant families had a higher frequency of trees with the ability to produce multiple responses to a single event than susceptible families. This confirms the hypothesis of Alfaro et al. (1996a) who proposed that resistant trees are able to produce a stronger traumatic response for the same stimulus level than susceptible trees.

The finding that 11% of trees from resistant families failed to respond to wounding and that 17% of trees from susceptible families responded with a score of 3 shows considerable within-family variation in response. This variation was expected because it was the open-pollinated families, and not the individual trees that were originally rated as resistant or susceptible (Alfaro et al. 1996c). Another possible source of variation in response may be the differential effects of the wounding treatment on leaders of different diameters. It is possible that the wounding treatment had a greater impact on small diameter leaders than on large diameter leaders (greater wound density). If so, a negative correlation between traumatic wound response and leader diameter would be expected. In support of this hypothesis, we found a weak, but significant relationship between the width of the 1994 growth ring (grown before wounding) and the wound response score for all resistant and susceptible families combined (Spearman's Rank Correlation coefficient  $-0.15$ ,  $P < 0.0063$ ). Trees from resistant families had greater radial growth than trees from susceptible families in 1994, but not in 1995 (Table 2), suggesting that the greater traumatic wound response of resistant trees to drilling

Table 2. Comparison of measurements (mean  $\pm$  SE) between resistant and susceptible trees exposed to drilling treatment. Data include upper section of the leader only, and undrilled trees are excluded.

Variable	Resistant	Susceptible	<i>P</i> Value, resistant versus susceptible <sup>1</sup>	<i>P</i> Value, effect of covariate (1994 radius)
Wound response score	1.74 $\pm$ 0.09	1.34 $\pm$ 0.09	0.0001	0.0284
Distance from 1994 annual ring to 1st traumatic resin canal ring (mm)	2.36 $\pm$ 0.04	2.49 $\pm$ 0.04	0.0191	0.0001
No. of cells from 1994 annual ring to 1st traumatic resin canal ring	30.00 $\pm$ 1.47	35.01 $\pm$ 1.49	0.0001	0.0001
Radius of 1994 annual ring (mm)	1.94 $\pm$ 0.03	1.74 $\pm$ 0.03	0.0020	–
Width of 1995 annual ring (mm)	3.71 $\pm$ 0.12	3.65 $\pm$ 0.12	0.1608	0.0001

<sup>1</sup> The *P* value is for ANOVA after effect of radius of 1994 growth ring was removed as a covariate, except in the case of the 1994 radius.

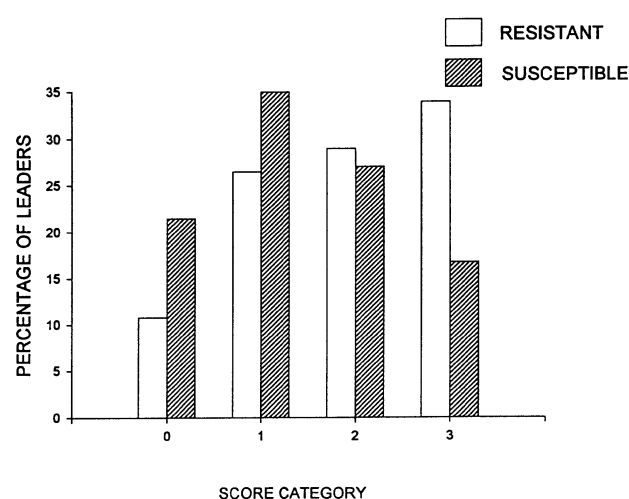


Figure 3. Frequency distribution of traumatic wound response scores in the upper xylem of 187 resistant and 141 susceptible leaders of interior spruce subjected to drilling treatment. Distributions significantly different,  $\chi^2$  test,  $P < 0.004$ .

resulted in reduced diameter growth, i.e., growth was sacrificed in favor of defense.

In addition to the intensity, the rapidity of the traumatic wound response may also be important (Tomiya 1963, Berryman 1969, Berryman and Ashraf 1970, Lieutier and Berryman 1988). In trees from resistant families there were 14.3% fewer cells in the xylem before the first traumatic resin canal ring than in trees from susceptible families (Table 2), suggesting that the response occurred faster in resistant families. Although it is not known when spring cambial growth began in each tree, a difference in phenology between resistant and susceptible families may explain this difference in response time. Time of bud burst, which is correlated with onset of cambial activity, is under strong genetic control (Cannell et al. 1985). In Sitka spruce, the initiation of bud burst can span a 2–3-week period (Cannell et al. 1985), which is about half to two-thirds of the time taken to produce traumatic resin canals in some conifers (Fahn et al. 1979, Kuroda and Shimaji 1983).

Hulme (1995) observed that buds flushed earlier in resistant genotypes of Sitka spruce than in susceptible genotypes. Gravid weevils caged on resistant clones before the normal oviposition period were able to kill the leaders, whereas those caged later could not. Ability to damage the leader was inversely correlated with external signs of resin flow. These results suggest that the cambium of resistant trees is activated earlier than the cambium of susceptible trees, possibly allowing for a more rapid wound response. Active cambial growth is necessary for production of traumatic resin canals (Fahn et al. 1979, Blanche et al. 1992). If weevils were to attack a tree before cambial growth starts, presumably the tree would be unable to produce a traumatic resin response and the leader would be killed.

It is possible that, as for bark beetles (Rudinsky 1962, Whitney 1982, Webber and Gibbs 1989), attack by the white pine weevil may be associated with a symbiotic microorganism. If so, the physical effect of drilling might have been enhanced if we had inoculated the wounds with a specific microorganism or chemical elicitor (Raffa and Berryman 1982b, Lieutier and Berryman 1988), making it possible to reveal a larger difference in intensity of response between resistant and susceptible families.

#### *Comparison of traumatic wound response in the upper and lower parts of the leader*

The wound response score of  $0.61 \pm 0.70$  in the lower (undrilled) section of drilled leaders (Table 3) was 2.8 $\times$  higher than the score of  $0.22 \pm 0.13$  in the undrilled leaders (Table 1), indicating that the traumatic resin canal response extended well beyond the wounding site. There were significant differences between the upper (drilled) and lower parts of the leader with respect to intensity of traumatic wound response, and distance and number of cells to the first ring of traumatic resin canals (Table 3). These results are similar to those of Thomson and Sifton (1926) who observed resin canals some distance from the wound site in white spruce, although the greatest density was closest to the injury. The latter two measurements show that the response may take longer to be expressed in the lower part of the leader than in the upper part of the leader. Extension of the wound response into the lower part of the

Table 3. Comparison of measurements (mean  $\pm$  SE) between the upper (drilled) and lower (undrilled) portion of the leader. Measurements are averaged across resistant and susceptible trees, and undrilled trees are excluded.

Variable	Upper section of leader (drilled)	Lower section of leader (undrilled)	P Value, upper versus lower <sup>1</sup>
Wound response score	1.42 $\pm$ 0.06	0.61 $\pm$ 0.07	0.0001
Distance (mm) 1994 annual ring to first TRC <sup>2</sup> ring	2.43 $\pm$ 0.04	3.34 $\pm$ 0.07	0.0001
No. of cells to 1st traumatic ring	32.29 $\pm$ 1.06	40.41 $\pm$ 3.22	0.0028
Radius of 1994 ring (mm)	1.84 $\pm$ 0.03	2.53 $\pm$ 0.04	0.0001
Radius of 1995 ring (mm)	4.02 $\pm$ 0.07	5.06 $\pm$ 0.09	0.0001

<sup>1</sup> The *P* value is for wound response by Kruskal-Wallis test. The *P* value for all other paired data is by ANOVA.

<sup>2</sup> TRC = Traumatic resin canal.

leader may result in death of older larvae, should they escape the effects of traumatic resinosis in the upper part of the leader.

#### Effect of wounding on constitutive resin canals in the bark

Drilling caused a significant reduction in the density of inner and outer constitutive resin canals in the 1995 bark of drilled trees compared to undrilled trees (Figure 4). Because resin ducts are formed from the apical meristem, no new resin canals are normally added in the 2-year-old bark. Therefore, this reduction is possibly the result of reabsorption of resin canals or a process of deactivation and cellular ingrowth. In several cross sections, it was observed that resin canals in the two-

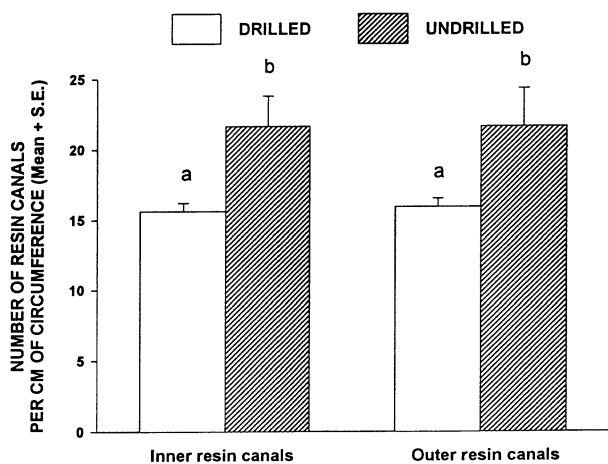


Figure 4. Density of inner and outer constitutive resin canals in the upper bark that grew after the drilling treatment (1995). Paired bars with different letters are significantly different, ANOVA, *P* < 0.05.

year-old bark appeared occluded by cellular growth, similar to that reported by Overhulser and Gara (1981*b*). This finding is consistent with the hypothesis that production of resin is metabolically expensive to a tree and results in the loss of carbon that could otherwise be allocated to growth and reproduction (Matson and Hain 1983, Lorio 1986). Traumatic resin is more costly than constitutive resin in terms of carbon consumption because it contains higher concentrations of monoterpenes (Matson and Hain 1983). Loss of constitutive resin canals after wounding could make a tree more susceptible to further attack in the years after the leader is killed. There was no evidence that traumatic resin canals were produced in the bark.

#### Correlation of constitutive and induced resin canals

There was no correlation between the density of constitutive resin canals in the bark prior to wounding (estimated by regression analysis) and the intensity of traumatic resinosis based on either the inner, outer, or total number of resin canals ( $r^2 = 0.02$ ,  $P < 0.07$ ;  $r^2 = 0.01$ ,  $P < 0.20$ ;  $r^2 = 0.01$ ,  $P < 0.11$ , respectively), suggesting that the constitutive resin system and the ability of the tree to produce traumatic resin canals are independent. These traits could thus be considered as separate resistance mechanisms that are under different genetic controls. Alternatively, they may reflect a trade-off in terms of energy use by the tree.

#### Conclusions

Simulated weevil damage by drilling the tree leader induced a traumatic wound response of higher intensity, and greater rapidity in resistant families than in susceptible families of white spruce. However, studies examining the effects of different wounding stimuli, the rate of response of different families to wounding, and the possible induction of radial resin canals must be performed before the utility of using traumatic resinosis as a resistance screening tool can be confirmed. The resin chemistry of the traumatic wound response might also differ between resistant and susceptible families. Preliminary analyses of the monoterpene and resin acid composition of traumatic resin shows that there is a difference between drilled and undrilled trees (E. Antonijević, Simon Fraser Univ., Burnaby, Canada, unpublished results).

Some trees from resistant families appeared incapable of producing a traumatic resin response. These may either be susceptible (families are open-pollinated) or may rely on other resistance mechanisms. Because trees from resistant families have more dense cortical resin canals than trees from susceptible families, weevils or other stem-invading insects may be less likely to wound these trees than trees with few cortical resin canals. Thus some resistant trees may not need to produce traumatic resin.

Production of traumatic resin canals appears to be a generalized defense system in conifers (Thomson and Sifton 1926, Bannan 1933*b*, Fahn et al. 1979, Kuroda and Shimaji 1983, Miller et al. 1986, Chencilet 1987, Croteau et al. 1987, Lieutier and Berryman 1988, Werner and Illman 1994) that is important in defending spruces against weevil colonization. If the weevil

breaches the constitutive resin canal defense system in the bark (also a nonspecific defense system), the inducible traumatic wound response may determine the success of the weevil brood, particularly if resistant trees can respond rapidly and repeatedly.

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