

Mechanisms of Douglas-fir resistance to western spruce budworm defoliation: bud burst phenology, photosynthetic compensation and growth rate

ZHONG CHEN,^{1,2} THOMAS E. KOLB^{1,4} and KAREN M. CLANCY³

¹ School of Forestry, Northern Arizona University, Flagstaff, AZ 86011-5018, USA

² Present address: Dept. of Ecology and Evolutionary Biology, University of Michigan, Ann Arbor, MI 48109, USA

³ Rocky Mountain Research Station, 2500 S. Pine Knoll Drive, Flagstaff, AZ 86001, USA

⁴ Author to whom correspondence should be addressed

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Summary We compared growth rates among mature interior Douglas-fir (*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco) trees showing resistance or susceptibility to defoliation caused by western spruce budworm (*Choristoneura occidentalis* Freeman), and among clones and half-sib seedling progeny of these trees in a greenhouse. We also investigated bud burst phenology and photosynthetic responses of clones to budworm defoliation in greenhouse experiments. Resistant mature trees had a higher radial growth rate than susceptible trees, especially during periods of budworm defoliation. Clones from resistant trees grew larger crowns than clones from susceptible trees, whereas stem base diameter at the ground line and height did not differ. Half-sib seedling progeny from resistant trees had larger stem diameter, height, and total biomass than progeny from susceptible trees. Mean 5-year radial growth increment of mature trees was more strongly correlated with growth of seedlings than with growth of clones. Clones from resistant trees had later bud burst than clones from susceptible trees, and budworm defoliation of clones depended on the degree of synchrony between bud burst phenology and budworm larval feeding. Clones of resistant and susceptible mature trees showed similar responses of net photosynthetic rate to 2 years of budworm defoliation. We conclude that phenotypic differences in crown condition of Douglas-fir trees following western spruce budworm defoliation are influenced by tree genotype and that high growth rate and late bud burst phenology promote tree resistance to budworm defoliation.

Keywords: *Choristoneura occidentalis*, genetic variation, insect herbivory, photosynthesis, *Pseudotsuga menziesii*.

Introduction

Insect herbivores can change the growth and physiology of host trees by reducing foliage surface area and photosynthetic capacity, altering water relations, and influencing allocation

of photosynthate (Kozlowski 1969, Kulman 1971, Reich et al. 1993, Kolb et al. 1999, Vanderklein and Reich 1999). However, these effects often vary over different functional groups of insects and host tree species, and are influenced by numerous environmental factors (Parry 1982, Barbosa and Wagner 1989, Schoonhoven et al. 1998, Schowalter 2000). Moreover, effects of insect herbivory on tree performance can vary among trees of the same species, even within the same stand, suggesting intraspecific genetic variation in tree resistance to herbivory (Weis and Campbell 1992, Clancy et al. 1993, 1995, Quiring 1994). Such variation may be caused by differences among genotypes in bud burst phenology (Joly et al. 1989, Kolb and Teulon 1991, Hunter 1992, Quiring 1994, Lawrence et al. 1997), foliage chemical composition (Hanover 1975, Weis and Campbell 1992, Clancy et al. 1995), foliage toughness (McMillin and Wagner 1996), tree growth rate (Barbosa and Wagner 1989) and photosynthetic compensation to herbivory (Welter 1989, Trumble et al. 1993).

For example, variation in tree bud burst phenology can influence the distribution and abundance of insect herbivores on their hosts (Kolb and Teulon 1991, 1992, Hunter 1992, Quiring 1992, 1994, Fox et al. 1997). Variation in the chemical composition of plant tissues, including mineral nutrients, carbohydrates, and allelochemicals, can influence insect fitness by affecting development time and fecundity (Mattson et al. 1982, Mattson and Scriber 1987, Clancy et al. 1995). Likewise, variation in tissue toughness can influence feeding efficiency and digestion by insects and hence regulate their development (Wagner and Zhang 1993, Coley and Barone 1996, McMillin and Wagner 1996). Inherent variations in tree growth rate may influence the rate of tree recovery from herbivory (Barbosa and Wagner 1989). However, certain insect guilds, such as gall-makers and shoot borers, prefer more rapidly growing plants because of the availability of high-quality food sources (Price 1997).

Increased photosynthetic rate in response to defoliation, i.e., photosynthetic compensation, may also promote tree recovery

from insect herbivory by increasing carbon uptake of remaining leaves (Reich et al. 1993, Kolb et al. 1999, Vanderklein and Reich 1999). Photosynthetic compensation in response to defoliation, in combination with internal reallocation of nutrients and carbohydrates to regrowing tissues (Schoonhoven et al. 1998), can lead to compensatory growth in cases where defoliated plants have higher rates of biomass production than undefoliated plants (Belsky et al. 1993, Reich et al. 1993, Vanderklein and Reich 1999). However, intraspecific genetic variation in photosynthetic compensation has not been widely evaluated as a mechanism of tree resistance to insect herbivory.

Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) is a commercially important tree species in western North America (Silen 1978, Hermann and Lavender 1990, Hardin et al. 2001), and western spruce budworm (*Choristoneura occidentalis* Freeman) is one of its major defoliators (Brookes et al. 1987), especially of the interior variety (*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco). Western spruce budworm has a univoltine life cycle (one generation per year). Adults begin to emerge in early July. Mating typically occurs within 24 h of eclosion, after which females lay from two to eight clutches of eggs, with each egg mass averaging between 25 and 40 eggs (Brookes et al. 1987). The first instars disperse from the egg mass and spin hibernaculæ in sheltered locations on host trees after which they molt into second instars without feeding and overwinter in their hibernaculæ. Upon emergence in early May, most second instars passively disperse on silken threads and, landing on a suitable source, begin feeding (Brookes et al. 1987). Suitable food sources include current-year swollen buds and needles of true firs (*Abies*), Douglas-fir, and spruce (*Picea*) species. Larvae continue to feed, molting through a total of six instars, and later pupate on foliage. Pupation lasts about 10 days, after which adults emerge to repeat the life cycle (Brookes et al. 1987). Defoliation is caused by larvae feeding on swollen buds and needles, especially current-year needles (Frank and Jenkins 1986, Shepherd 1992, Dodds et al. 1996).

Crown damage to Douglas-fir by western spruce budworm can vary considerably among trees of similar size within the same stand (Clancy et al. 1993, 1995), suggesting the existence of intraspecific genetic variation in resistance mechanisms. Despite numerous studies of the Douglas-fir–western spruce budworm system (Brubaker and Greene 1979, Alfaro et al. 1982, 1985, Thomson et al. 1982, Redak and Cates 1984, Brookes et al. 1987, Clancy et al. 1988, 1993, Ferguson 1988, Campbell 1993, Powell 1994, Kolb et al. 1999), there is little information on intraspecific genetic variation in resistance mechanisms of interior Douglas-fir.

Our overall objective was to evaluate growth rate, photosynthetic compensation, and bud burst phenology as resistance mechanisms of interior Douglas-fir to budworm defoliation. We used three experimental systems for this evaluation. First, we compared growth rates of mature trees showing phenotypic resistance with growth rates of trees showing susceptibility to budworm defoliation. Second, we cloned these

mature trees and measured growth and photosynthetic responses of the clones to budworm defoliation under controlled greenhouse conditions. Third, we compared growth rate and biomass allocation among half-sib seedling progeny from resistant and susceptible mature trees over 3 years in the greenhouse. We hypothesized that: (1) resistant mature trees and their clones and seedling progeny have higher growth rates compared with susceptible mature trees and their clones and seedling progeny (growth rate hypothesis); (2) bud burst phenology differs between clones of resistant and susceptible trees, and this difference influences the degree of synchrony between bud burst and budworm feeding, thereby regulating the amount of defoliation (bud burst phenology hypothesis); and (3) clones of resistant trees respond to budworm defoliation with a greater increase in net photosynthetic rate than clones of susceptible trees (photosynthetic compensation hypothesis).

Materials and methods

Mature tree growth rate

Mature trees were from the Pike National Forest near Deckers, CO (39.23° N, 105.23° W, elevation 2,573 m) and the Kaibab National Forest near Jacob Lake, AZ (36.71° N, 112.22° W, elevation 2,774 m). Both sites contain mixed coniferous species with a history of western spruce budworm defoliation (Clancy et al. 1993). After outbreaks of western spruce budworm in the mid-1980s, 12 pairs of mature trees, each comprising one phenotypically resistant tree and one susceptible tree, were identified based on the amount of crown damage. The resistant trees had a full and healthy crown, whereas the nearby (within 30 m) susceptible trees were severely defoliated. Both trees of each pair were of similar height and diameter at breast height (1.4 m, DBH), and comparable in site aspect and slope (Clancy et al. 1993). Measurements on mature trees included age, height, DBH, and radial growth increment over 5-year intervals from 1966 to 1990, which was determined by measuring yearly xylem production on increment cores sampled at breast height ($n = 1$ core per tree) with the aid of a binocular microscope equipped with an ocular micrometer (Clancy et al. 1993).

We used multivariate analysis of covariance (MANCOVA) to investigate effects of trait (resistant and susceptible), site (Deckers, CO and Jacob Lake, AZ), the interaction between trait and site, and tree age (covariate variable) on radial growth rate of each 5-year interval between 1966 and 1990. Following a significant ($P < 0.05$) Wilk's Lambda test in the MANCOVA, we performed univariate analyses of variance with the same model for each response variable. Mean separations were made by Tukey's test (Dean and Voss 1999) for all pairwise comparisons.

Growth rates of half-sib seedlings

Open-pollinated seeds were collected from 13 pairs of resistant and susceptible mature trees (i.e., 26 families) that were identified as described for mature trees. Seeds were collected from the Pike National Forest near Deckers, CO and the

Kaibab National Forest near Jacob Lake, AZ in 1991, and the San Isabel National Forest near Buena Vista, CO (38.75° N, 106.15° W, elevation 2,988 m) in 1994 (Clancy 2001). Five pairs of these trees corresponded with the mature tree used for radial growth analysis, whereas the other eight pairs were different because of uneven seed production among trees.

Collected seeds were sealed in plastic bags and stored in a freezer. In February 1998, 50 seeds per tree were germinated (overall germination rate of over 95%). The resulting half-sib seedlings were grown in a greenhouse (Rocky Mountain Research Station, Flagstaff, AZ) in plastic germination cells (5 × 6 × 20 cm) for the first year (1998). Seedlings were then transplanted to larger plastic pots (15 cm in diameter × 20 cm in height) filled with a mixture of screened peat moss and vermiculite for the second (1999) and third years (2000). Water and a balanced fertilizer were applied frequently to all seedlings. Air relative humidity and temperature in the computer-controlled greenhouse simulated seasonal variations in environmental conditions in natural forests and thus satisfied the chilling requirement of the seedlings. Maximum photosynthetically active radiation (PAR) in the greenhouse was about 1400 $\mu\text{mol m}^{-2} \text{s}^{-1}$ as measured with a Li-Cor quantum sensor (Li-Cor, Inc., Lincoln, NE) (about 64% of outdoor PAR) on several bright days throughout the summer.

We measured base diameter (1 cm above the soil surface), height, and number of branches (for 1998 and 1999 only) at the end of the 1998, 1999 and 2000 growing seasons. We also harvested 10 randomly selected seedlings per family each year for biomass determination. Each freshly harvested seedling was divided into shoot (stem + leaves) and root, oven dried at 75 °C for 48 h, and weighed. Growth and biomass data were analyzed by one-way analysis of variance (ANOVA). Linear regression was used to analyze the relationship between growth of half-sib seedlings and radial growth rate of their corresponding mature trees.

Clone growth and response to defoliation

We cloned each of the 24 mature trees by whip-grafting branches collected from the lower third of the crown onto 1-year-old seedling rootstocks in 1991 and 1992. Following grafting, all clones were planted in 15-l pots containing a mixture of screened peat moss and vermiculite, and watered and fertilized frequently until the start of the experiments in 1998. All clones were raised in a greenhouse, although they were over-wintered in an outdoor shade house from mid-August through January of 1992–1994.

We used 12 ramets (individuals) of each clone in the study. All ramets had characteristics of their respective mature trees (e.g., thick blue-green needles, non-vertical or plagiotropic branch growth, and occasional strobilus production). Ramets averaged 2.1 ± 0.03 cm (mean \pm 1 SE) in base stem diameter (about 1 cm above the graft), 93.5 ± 1.6 cm in height, and 45.9 ± 0.7 cm in crown diameter (averaged over two directions) in fall 1997. Six ramets of each clone were randomly selected for the budworm defoliation treatment, and the other six ramets served as undefoliated controls. The experiment had a

completely randomized block design comprising six blocks, each containing a factorial combination of 24 clones (12 pairs of resistant and susceptible genotypes) × two defoliation treatments. In total, 288 grafted trees were included in the experiment. However, nine of the grafted trees died during the experiment (eight resistant trees: four defoliated and four undefoliated, and one susceptible tree in the defoliated treatment), resulting in four to six replications per treatment combination for each of the 12 pairs.

We used western spruce budworm larvae to defoliate half of the ramets of each clone over two consecutive years (1998 and 1999). The budworm larvae were from laboratory cultures of diapausing and non-diapausing western spruce budworms, maintained at Rocky Mountain Research Station's Entomology Laboratory in Flagstaff, AZ. Budworm larvae produced from the non-diapausing culture had growth rates and feeding behavior similar to those of a wild population (Leyva et al. 1995). Both cultures were maintained on a standard budworm artificial diet that is similar to the McMorran (1965) diet (see Clancy 1991a for a description of the variations and rearing conditions, and Clancy 1991b for the nutritional composition). The non-diapausing culture has been maintained since 1985 (Clancy 1991a). The diapausing culture was established from wild material collected in 1996–1998, so it has been in culture for fewer generations than the non-diapausing culture.

In April 1998, third and primarily fourth instar non-diapausing larvae were placed on each grafted tree (one larva per five terminal buds) assigned to the defoliation treatment when about 50% of the buds on the tree were in the fourth (columnar) bud burst developmental stage (Shepherd 1983). We had planned to use second instars in hibernaculæ from our diapausing culture to defoliate the grafted trees, but not enough larvae were available from this culture in 1998. The number of buds on each ramet was estimated by a regression equation that predicted the total number of terminal buds from stem base diameter and crown diameter ($r^2 = 0.72$, $P < 0.001$, $n = 31$). This method resulted in the placement of larvae on a grafted tree at the bud burst stage when bud and needle tissues were highly palatable. Both defoliated and undefoliated grafted trees were caged with nylon "No-See-um" netting bags (The Rain Shed Corp., Corvallis, OR) that allowed penetration of about 80% of full light in order to contain larvae and maintain all trees in a similar light environment. The bags were not removed until 95% of the larvae had pupated, which required approximately 40 days. The actual amount of defoliation was calculated for each grafted tree based on the number of damaged terminal buds divided by total number of terminal buds. In 1999, we defoliated the same ramets of each clone that were defoliated in 1998, but we used a slightly different method of larval placement. In May 1999, second instar diapausing larvae in hibernaculæ (over-wintering structures) were placed on all previously defoliated ramets on the same day (one larva per four terminal buds) when approximately 50% of all ramets were in the second (yellow) bud burst developmental stage (Shepherd 1983). The initial number of terminal buds on each grafted tree was counted before the defoliation

treatment. Pieces of gauze containing pre-counted second instar larvae in hibernacula (1–20 larvae per gauze) were pinned to the stem in the upper crown of each defoliated ramet. Thus, in 1999, we did not attempt to place larvae on each tree when its buds were most suitable for feeding as we did in 1998. The actual number of damaged terminal buds was counted immediately after budworm pupation. The cumulative amount of defoliation over both years was calculated as $(1 - (1 - \% \text{ defoliation } 98)(1 - \% \text{ defoliation } 99))100$. Stem base diameter, height, and crown diameters of clones were measured at the end of the 1998 and 2000 growing seasons.

Bud burst phenology and shoot development were monitored on randomly selected, labeled terminal buds (8–20 buds depending on tree size; 20 buds were assessed on most trees) from the upper two-thirds of the crown on each undefoliated grafted tree in the 1998 experiment. From March 19 to May 7, we recorded the developmental stage of each labeled bud/shoot weekly based on Shepherd's (1983) bud burst scale. In total, 2620 current-year buds and shoots were monitored (1284 on resistant clones, and 1336 on susceptible clones). Monitoring date was used as a repeated factor in analysis of variance (ANOVA) on bud burst stage.

We measured net photosynthetic rate (P_n) and stomatal conductance (g_w) on fully developed, undamaged foliage from the terminal portion of mid-crown lateral branches of all grafted trees at the end of July in both 1998 and 1999. Both first- (current) and second-year needles were measured in 1998 (one measurement per grafted tree for each needle age class), but only first-year needles were measured in 1999 (two measurements on different twigs on each tree). We performed all measurements over a 30-s period in a 0.25-l cuvette with a closed-circuit portable photosynthesis system (LI-6200, Li-Cor, Inc.) between 1000 and 1400 h under high light conditions (mean PAR 1141.3 ± 8.4 (1 SE) $\mu\text{mol m}^{-2} \text{s}^{-1}$) created by a combination of sunlight and artificial light from a metal halide lamp (MH-250W; Hubbell Lighting, Inc., Christiansburg, VA). Air temperature and relative humidity during these measurements averaged 28.2 ± 0.2 (1 SE) °C and $41.5 \pm 0.5\%$, respectively. Both P_n and g_w were expressed on a projected leaf area basis (one side). We measured nitrogen (N) concentration of all foliage used for the gas exchange measurements in 1998 and 1999, and also measured stable carbon isotope ratio ($\delta^{13}\text{C}$) on foliage sampled in 1999. These measurements were made with a continuous-flow mass spectrometer (Ceinstrument NC2100 and Delta Plus XL, Finnigan, MAT, San Jose, CA). Finally, base stem diameter, height, and mean crown diameter of all ramets were measured at the end of the 1998 and 2000 growing seasons. We used a mixed ANOVA statistical model (Table 1) to analyze the responses of growth, P_n , g_w , N concentration, and $\delta^{13}\text{C}$ of clones to budworm defoliation. The model was based on four assumptions: (1) the two clones from paired mature trees at a site were considered the basic sampling unit; (2) each pair included trees that differed in resistance trait (resistant versus susceptible); (3) each trait within a pair was further split to incorporate the treatment in the greenhouse (defoliated versus undefoliated); and (4) tree pairs were random factors and other factors were fixed. We fo-

cused on the effects of trait, budworm defoliation treatment, and their interaction (Table 1) because these sources of variation are most relevant to our hypotheses. If the F -tests of these factors were significant in the mixed model ANOVA, all pair-wise comparisons were made with Tukey's test to separate the means of these effects at $P = 0.05$. All statistical analyses were performed with SAS JMP software (SAS Institute, Inc., Cary, NC). The relationship between growth of clones and that of their corresponding mature trees was evaluated by linear regression analysis.

Results

Mature tree growth rate

Overall, 5-year radial growth rate differed significantly between phenotypically resistant and susceptible mature trees (MANCOVA, $P = 0.004$). Site, interaction between site and trait, and tree age (as a covariate variable) did not affect radial growth rates (MANCOVA, $P \geq 0.177$). Thus, mature trees at the Deckers, CO and Jacob Lake, AZ sites had similar growth rates, and differences in radial growth rate between resistant and susceptible trees were similar at each site. Resistant trees had a greater growth rate than susceptible trees at every measurement interval from 1966 to 1990 (Table 2). However, these differences were significant only for the interval from 1986 to 1990 ($P < 0.001$) (Table 2) when the western spruce budworm population outbreak occurred.

The difference in radial growth rate between resistant and susceptible mature trees was greatest between 1986 and 1990 (about 120%), intermediate during 1966–1970 (29%) and 1981–1985 (25%), and lowest during 1971–1975 (8%) and 1976–1980 (2%). Radial growth rate was about 28% higher in resistant trees than in susceptible trees ($P = 0.088$) averaged over all 1966–1990 measurements (Table 2).

Growth rates of half-sib seedlings

In the absence of western spruce budworm defoliation, height and base diameter of half-sib seedlings from resistant trees were significantly greater compared with seedlings from susceptible trees in all 3 years of growth in the greenhouse ($P < 0.001$) (Table 3). Seedlings from susceptible trees had significantly more lateral branches than seedlings from resistant trees during the first year (1998) ($P < 0.001$), but not in the second year (1999) (Table 3). Total biomass was similar for seedlings from resistant and susceptible trees for the first year ($P = 0.469$) (Table 3). However, seedlings from resistant trees had significantly ($P < 0.001$) greater total biomass than seedlings from susceptible trees in the second and third years. Shoot/root biomass ratio was similar for seedlings from resistant and susceptible trees in all years ($P \geq 0.128$) (Table 3).

Clone bud phenology and defoliation

Clones of susceptible trees had earlier bud burst than clones of resistant trees ($P < 0.001$) (Figure 1). Resistant clones required about 90–110 more degree-days to reach the same bud burst stage as susceptible clones.

Table 1. Statistical model for analysis of variance of growth rate and physiological characteristics of Douglas-fir clones in the 1998 and 1999 experiments. The major sources of variation that are directly related to our hypotheses appear in bold face.

Sources	Effect	df	F-Test denominator
Block	Fixed	5	Block × Pair (Site)
Site	Fixed	1	Pair (Site)
Trait	Fixed	1	Trait × Pair (Site)
Treatment	Fixed	1	Treatment × Pair (Site)
Block × Site	Fixed	5	Block × Pair (Site)
Block × Trait	Fixed	5	Residual
Block × Treatment	Fixed	5	Residual
Site × Treatment	Fixed	1	Treatment × Pair (Site)
Site × Trait	Fixed	1	Trait × Pair (Site)
Trait × Treatment	Fixed	1	Trait × Treat × Pair (Site)
Trait × Treatment × Site	Fixed	1	Trait × Treatment × Pair (Site)
Pair (Site)	Random	10	Residual
Treatment × Pair (Site)	Random	10	Trait × Treatment × Pair (Site)
Trait × Pair (Site)	Random	10	Trait × Treatment × Pair (Site)
Block × Pair (Site)	Random	50	Residual
Trait × Treatment × Pair (Site)	Random	10	Residual
Residual		161	
Total		278 ¹	

¹ Originally 288 grafted trees were included, but nine died during the experiments.

Table 2. Mean 5-year radial growth increment (± 1 SE) over 25 years (1966–1990) for Douglas-fir trees showing phenotypic resistance ($n = 12$ trees) or susceptibility ($n = 12$ trees) to western spruce budworm defoliation.

Trait	Five-year radial growth increment (mm)					Mean 5-year radial growth over 25 years (mm)
	1966–70	1971–75	1976–80	1981–85	1986–90	
Resistant	8.21 \pm 0.90	6.92 \pm 0.63	5.88 \pm 0.61	5.13 \pm 0.69	7.26 \pm 0.64	6.68 \pm 0.58
Susceptible	6.35 \pm 0.91	6.41 \pm 0.63	5.78 \pm 0.62	4.10 \pm 0.70	3.29 \pm 0.65	5.19 \pm 0.58
P-Value	0.170	0.578	0.918	0.313	< 0.001	0.088

In the 1998 experiment, when larvae were placed on each grafted tree when buds were highly suitable for feeding, between 9 and 84% of the terminal buds were damaged and defoliation averaged 37%. Resistant clones were defoliated more (41.7 \pm 1.70%) (mean \pm 1 SE) than susceptible clones (34.0 \pm 1.65%) ($P = 0.002$) (Figure 2). In the 1999 experiment, when larvae were placed on all trees on the same date regardless of bud burst stage, defoliation ranged between 5 and 85%, and averaged 30%. Resistant clones were defoliated less (26.3 \pm 2.17%) than susceptible clones (33.0 \pm 2.10%) in 1999 ($P = 0.032$) (Figure 2). Susceptible clones were defoliated to about the same extent in both years, but resistant clones were significantly less defoliated in 1999 than in 1998 (i.e., trait \times year interaction; $P = 0.001$) (Figure 2). Cumulative defoliation over 2 years was similar for resistant (56.4 \pm 1.89%) and susceptible clones (56.1 \pm 1.83%) ($P = 0.90$) (Figure 2).

Clone growth and response to defoliation

Before the budworm defoliation experiment (1997), resistant and susceptible clones had similar base diameters and heights

($P > 0.22$). However, resistant clones had greater crown diameter (48.3 \pm 0.95 cm) than susceptible clones (43.8 \pm 0.93 cm) ($P < 0.001$). After the 1998 defoliation, base diameter and height did not differ between resistant and susceptible clones ($P = 0.542$), but crown diameter was still greater in resistant clones (62.2 \pm 1.13 cm) than in susceptible clones (56.7 \pm 1.08 cm) ($P < 0.001$). The 1998 budworm defoliation did not influence mean base diameter, height, or crown diameter ($P > 0.367$) (Figures 3A, 3C, and 3E) or their relative growth rates between 1997 and 1998 ($P \geq 0.108$). Although the trait \times defoliation treatment interaction was not significant ($P \geq 0.629$) for 1998 base stem diameter and height (Figures 3A and 3C), this interaction was significant for crown diameter ($P = 0.033$). Defoliation in 1998 decreased crown diameter in resistant clones and caused a small increase in crown diameter in susceptible clones (Figure 3E).

After two consecutive years of defoliation (2000), resistant clones still had a greater mean crown diameter (70.2 \pm 2.71 cm) than susceptible clones (66.2 \pm 2.73 cm) ($P = 0.005$) (Figure 3F), whereas base stem diameter and height did not

Table 3. Height, base stem diameter, number of branches, total biomass, and shoot/root dry mass ratio (± 1 SE) over three growing seasons (1998–2000) of half-sib seedlings from Douglas-fir trees showing phenotypic resistance ($n = 13$ trees) or susceptibility ($n = 13$ trees) to western spruce budworm defoliation.

Year/trait	Height (cm)	Diameter (mm)	No. branches	Total biomass (g)	Shoot/root ratio
<i>1998</i> ¹					
Resistant	25.82 \pm 0.34	3.26 \pm 0.04	4.43 \pm 0.14	4.95 \pm 0.21	1.84 \pm 0.28
Susceptible	22.95 \pm 0.33	2.96 \pm 0.04	5.78 \pm 0.15	4.73 \pm 0.21	1.92 \pm 0.28
<i>P</i> -Value	< 0.001	< 0.001	< 0.001	0.469	0.831
<i>1999</i> ²					
Resistant	44.00 \pm 1.09	7.26 \pm 0.15	15.15 \pm 0.57	29.94 \pm 0.80	1.56 \pm 0.04
Susceptible	38.24 \pm 1.09	6.57 \pm 0.15	16.42 \pm 0.57	20.83 \pm 0.80	1.52 \pm 0.04
<i>P</i> -Value	< 0.001	< 0.001	0.113	< 0.001	0.383
<i>2000</i> ³					
Resistant	66.10 \pm 1.40	13.27 \pm 0.24	–	58.71 \pm 1.81	1.84 \pm 0.04
Susceptible	58.57 \pm 1.40	11.83 \pm 0.24	–	49.82 \pm 1.81	1.75 \pm 0.04
<i>P</i> -Value	< 0.001	< 0.001	–	< 0.001	0.128

¹ Height, diameter and number of branches were based on the measurement of 1,014 seedlings; 34–40 seedlings per family ($n = 26$). Among them, 509 seedlings were from resistant families ($n = 13$), and 505 from susceptible families ($n = 13$). Total biomass and shoot/root ratio dry mass values were based on a subsample of 10 seedlings per family.

² Height, diameter and number of branches were based on the measurement of 520 seedlings; 20 seedlings per family ($n = 26$). Total biomass and shoot/root dry mass ratio values were based on a subsample of 10 seedlings per family.

³ The number of branches was not counted in 2000. All other measurements were based on a subsample of 10 seedlings per family ($n = 26$).

differ ($P > 0.499$) (Figures 3B and 3D). However, 2 years of defoliation reduced mean base stem diameter ($P = 0.058$) and its relative growth rate between 1997 and 2000 ($P = 0.010$; data not shown). In 2000, defoliated clones had similar mean height to undefoliated clones ($P = 0.972$, Figure 3D), but defoliation reduced relative growth rate of height (1997–2000) by 17% ($P = 0.013$; data not shown). In contrast, 2 years of defoliation did not influence mean crown diameter (Figure 3F) or its relative growth rate over 3 years ($P \geq 0.178$). The trait \times defoliation treatment interaction did not have a significant influence on base diameter, height, and crown diameter ($P \geq 0.129$)

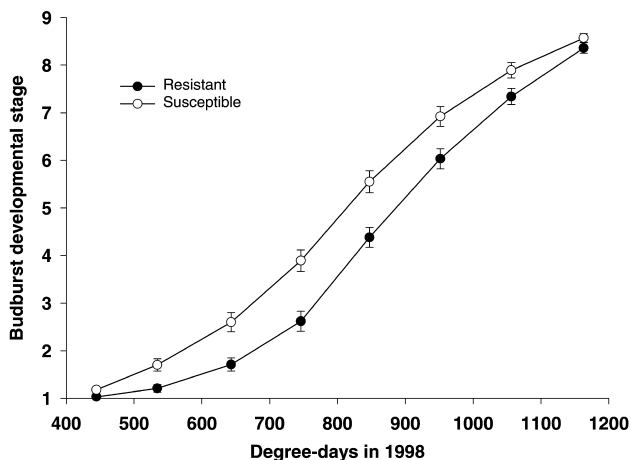


Figure 1. Mean bud burst phenological stage (± 1 SE) of clones from interior Douglas-fir trees showing phenotypic resistance (●) or susceptibility (○) to western spruce budworm defoliation versus degree-days in 1998.

after two consecutive years of defoliation (2000) (Figures 3B, 3D and 3F). Overall, cumulative defoliation over both years was negatively correlated with relative growth rate (1997–2000) of base stem diameter ($r = -0.304$, $P = 0.006$), and height ($r = -0.236$, $P = 0.036$), but it was unrelated to relative growth rate of crown diameter ($r = -0.091$, $P = 0.428$, $n = 138$).

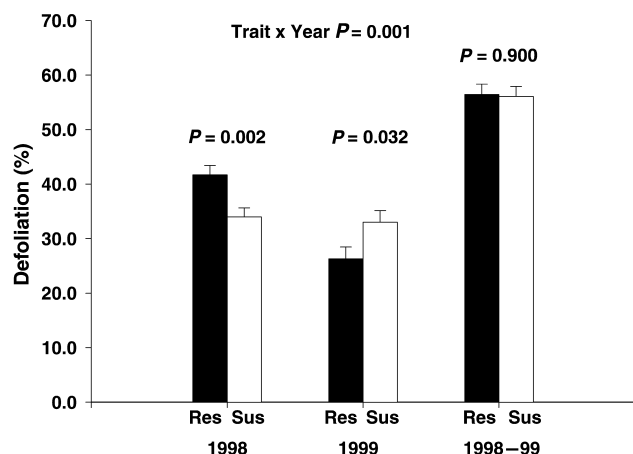


Figure 2. Mean defoliation (± 1 SE; % of all terminal buds damaged by budworm larvae) in the 1998 and 1999 experiments, and cumulative defoliation over both years (1998–1999) of clones of interior Douglas-fir trees showing phenotypic resistance (Res, ■) or susceptibility (Sus, □) to western spruce budworm defoliation. The *P*-value for the trait \times year (1998–1999) interaction is shown, along with *P*-values for trait (resistant versus susceptible) in 1998, 1999 and 2000.

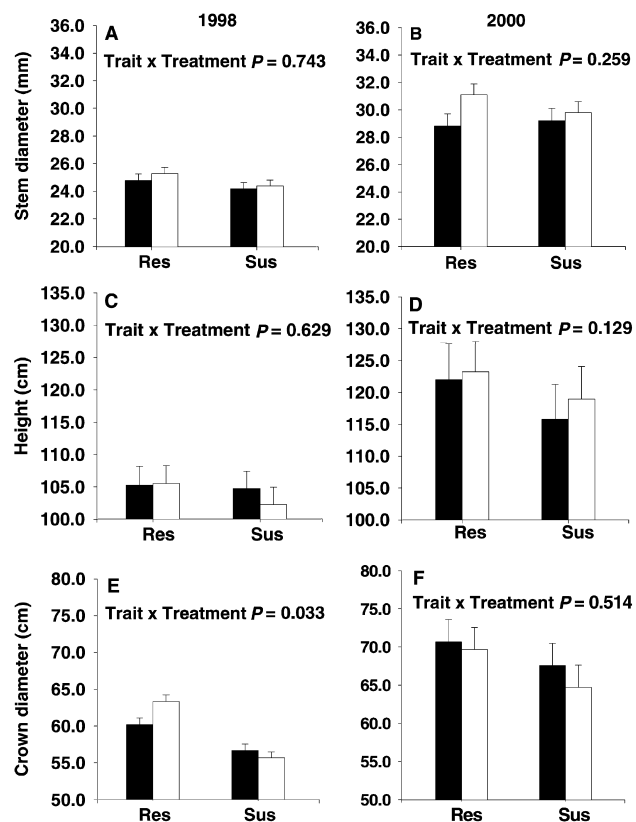


Figure 3. Mean (+ 1 SE) stem base diameter (A, B), height (C, D) and crown diameter (E, F) for defoliated (■) and undefoliated (□) clones of interior Douglas-fir trees showing phenotypic resistance (Res) or susceptibility (Sus) to western spruce budworm defoliation. Data are for 1 year of defoliation in 1998 (A, C, E) and after 2 years of consecutive defoliation in 2000 (B, D, F). The P -value for the trait \times defoliation treatment interaction is shown for each variable.

Net photosynthetic rate (P_n) of both first- and second-year needles did not differ significantly between resistant and susceptible clones or between defoliated and undefoliated clones after the first year of defoliation (1998) ($P \geq 0.297$) (Figures 4A and 4B). However, the trait \times defoliation treatment interaction was significant for P_n of first-year needles in 1998 ($P = 0.049$). Defoliation increased P_n of first-year needles of resistant clones, but decreased P_n of susceptible clones (Figure 4A). This pattern also occurred in second-year needles in 1998 (Figure 4B) ($P = 0.148$). In 1998, P_n was 20–25% greater in first-year needles than in second-year needles. The P_n of first-year needles was negatively correlated with the amount of defoliation in 1998 ($r = -0.169$, $P = 0.053$), whereas P_n of second-year needles was not correlated with defoliation in 1998 ($r = -0.117$, $P = 0.176$, $n = 138$).

In 1998, stomatal conductance (g_w) of first- and second-year needles did not differ significantly between resistant and susceptible clones or between defoliation treatments ($P \geq 0.082$) (Figures 4C and 4D). The trait \times defoliation treatment interaction was not significant for g_w in 1998 ($P \geq 0.184$). Stomatal conductance was positively correlated with P_n in 1998 ($P < 0.001$, $n = 279$), and this correlation was stronger in first-year

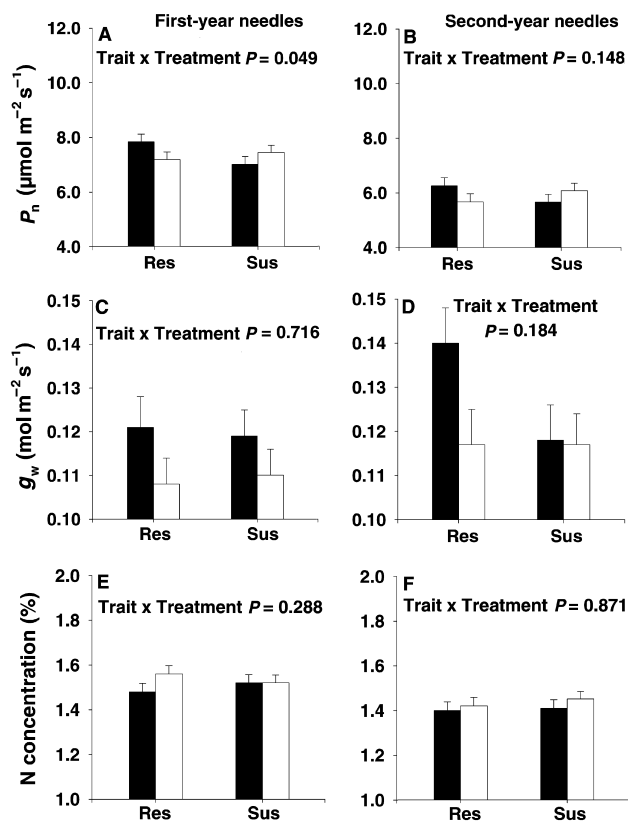


Figure 4. Mean (+ 1 SE) 1998 net photosynthetic rate (P_n) (A, B), stomatal conductance (g_w) (C, D), and nitrogen (N) concentration (E, F) for first- (A, C, E) and second-year (B, D, F) needles for defoliated (■) and undefoliated (□) clones of interior Douglas-fir trees showing phenotypic resistance (Res) or susceptibility (Sus) to western spruce budworm defoliation. The P -value for the trait \times defoliation treatment interaction is shown for each variable.

needles ($r = 0.671$) than in second-year needles ($r = 0.574$). In 1998, defoliation-induced stimulation of P_n in resistant clones (Figures 4A and 4B) was associated with increased g_w for both needle age classes (Figures 4C and 4D), but not with changes in foliar N concentration (Figures 4E and 4F). Trait, defoliation treatment, and their interaction did not influence foliar N concentration in first- or second-year needles in 1998 ($P \geq 0.288$) (Figures 4E and 4F). Foliar N concentration was not related to P_n in first-year needles ($r = 0.039$, $P = 0.651$), but was positively related to P_n in second-year needles ($r = 0.249$, $P = 0.004$, $n = 279$).

In 1999 after 2 years of defoliation, P_n of first-year needles did not differ significantly between resistant and susceptible clones or between defoliated and undefoliated clones ($P \geq 0.587$) (Figure 5A). The trait \times defoliation treatment interaction was not significant for P_n in 1999 ($P = 0.883$). In first-year needles, P_n was similar in 1998 and 1999 and was positively correlated for individual trees ($r = 0.167$, $P = 0.054$, $n = 279$). Linear regressions pooled over all clones showed that P_n and g_w of first-year needles in 1999 were positively correlated with cumulative defoliation over both years (P_n : $r = 0.218$, $P = 0.012$, $n = 138$; g_w : $r = 0.171$, $P = 0.048$, $n = 138$) (Figure 5C).

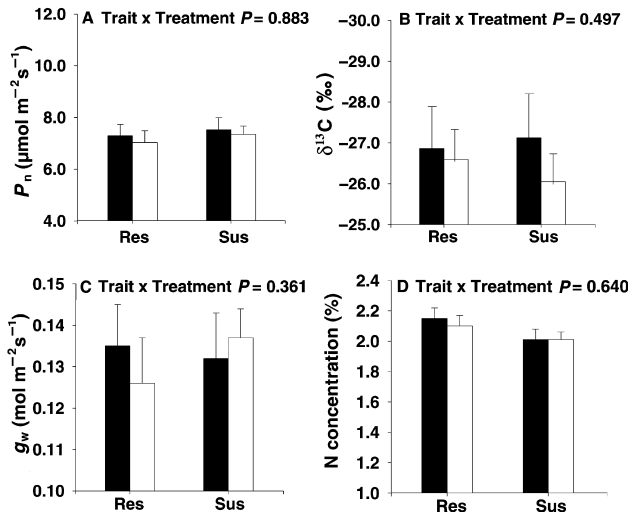


Figure 5. Mean (± 1 SE) 1999 net photosynthetic rate (P_n) (A), $\delta^{13}\text{C}$ (B), stomatal conductance (g_w) (C), and N concentration (D) of first-year needles of defoliated (■) and undefoliated (□) clones of interior Douglas-fir trees showing phenotypic resistance (Res) or susceptibility (Sus) to western spruce budworm defoliation. The P -value for the trait \times defoliation treatment interaction is shown for each variable.

Mean N concentration in first-year needles was significantly higher in 1999 ($2.02 \pm 0.03\%$) than in 1998 ($1.52 \pm 0.02\%$) ($P < 0.001$), but trait, defoliation treatment, and their interaction were not significant ($P \geq 0.074$) (Figure 5D). Although there were no significant treatment effects on $\delta^{13}\text{C}$ of first-year needles in 1999 (Figure 5B), $\delta^{13}\text{C}$ was significantly correlated with P_n ($r = -0.195$, $P = 0.007$) and g_w ($r = -0.361$, $P = 0.016$, $n = 236$).

Relationships among growth rates of mature trees, seedlings, and clones

Mean 5-year radial growth rate of mature trees was not significantly correlated with mean stem base diameter, height, or crown diameter of their greenhouse-grown clones ($r \leq 0.311$, $P \geq 0.653$, $n = 24$). There was a positive but weak correlation between mean 5-year radial growth rate of mature trees and stem base diameter ($r = 0.363$, $P = 0.096$) and height ($r = 0.342$, $P = 0.120$) of their greenhouse-grown seedling progeny ($n = 26$).

Discussion

We obtained evidence to support our first hypothesis that resistant mature trees, their clones, and half-sib seedling progeny have higher growth rates than their susceptible counterparts (growth rate hypothesis). Growth rates were higher in resistant mature trees than in susceptible trees in the forest. This growth difference was largest between 1986 and 1990 when the stands were defoliated by budworm, suggesting that the greater growth rate of resistant mature trees compared with susceptible mature trees was largely a consequence of a differ-

ence in defoliation intensity rather than genetic variation in growth rate. However, in the absence of budworm defoliation in a uniform greenhouse environment, clones and half-sib seedling progeny of resistant trees also had higher growth rates than those of susceptible trees. Thus, we conclude that resistant trees were genetically predisposed to grow faster than susceptible trees. Inherently higher growth rate may promote tree resistance or recovery from budworm defoliation by rapid regrowth.

Radial growth rate of resistant mature trees increased 42% between the 1981–85 and 1986–90 periods, whereas radial growth rate of susceptible mature trees decreased 20% over the same period. These results suggest less budworm defoliation during 1986–90 on resistant trees than on susceptible trees in the forest. The bud burst phenology of susceptible trees may have favored budworm feeding. That is, defoliation may have been greater on trees whose bud burst occurred when larvae were starting to feed because larvae feed preferentially on swollen buds and young needles (Shepherd 1992, Dodds et al. 1996). Consistent with this idea, bud burst was earlier for susceptible mature trees than for resistant mature trees at both the Arizona and Colorado study sites (Clancy et al. 1993), and this difference also occurred in the greenhouse-grown clones. Thus, there is evidence that the difference in bud burst phenology reported by Clancy et al. (1993) for these mature trees growing in the same stand resulted from genotypic differences. This is consistent with the finding of high heritability for bud burst phenology in Douglas-fir (Li and Adams 1993).

The budworm defoliation experiments on the clones provided additional support for a role of genetic variation in bud burst phenology as an influence on budworm defoliation (bud burst phenology hypothesis). In the 1998 experiment, when clones of resistant trees were defoliated more than clones of susceptible trees, the influence of bud burst phenology on the amount of defoliation was minimized because we introduced larvae to each tree at the same bud burst stage. In contrast, genetic differences in bud burst phenology influenced defoliation in the 1999 experiment when we introduced larvae to all trees on the date when 50% of all buds in the pooled population were expanding. Under this scenario, clones of resistant trees were defoliated less than clones of susceptible trees because more of their buds were tightly closed and thus not available for larval feeding. Thus, our second hypothesis, that bud burst phenology differs genetically between resistant and susceptible trees and that this difference influences defoliation, was supported. Our findings are consistent with other studies highlighting the importance of intraspecific variation in bud burst phenology as a mechanism of tree resistance to insects that feed on swollen buds and young leaves in both broad-leaved and coniferous trees (Kolb and Teulon 1991, Hunter 1992, Kolb et al. 1992, Lawrence et al. 1997, Teulon et al. 1998, Ostaff and Quiring 2000). Moreover, because foliar nutrients (Clancy et al. 1995) and allelochemicals (e.g., monoterpenes) (Wagner et al. 1989, Gambiel and Cates 1995, Zou and Cates 1995) often change during tissue maturation, which

is related to bud burst phenology, they also likely influenced budworm defoliation in our study.

Our third hypothesis that clones of resistant trees respond to budworm defoliation by a greater increase in P_n than clones of susceptible trees (photosynthetic compensation hypothesis) was not supported. Although we found evidence of a stronger compensatory photosynthetic response to defoliation in resistant clones than in susceptible clones after the first defoliation in 1998, this difference was probably caused by greater defoliation of resistant clones, rather than a genetic difference in compensatory capacity. For example, the degree to which P_n was stimulated by budworm feeding on 3-year-old Douglas-fir seedlings was positively related to the amount of defoliation (Kolb et al. 1999). Increased P_n in 1998 in defoliated resistant clones likely resulted from increased g_w caused by a more favorable balance between leaf and root area (Kruger and Reich 1993, Reich et al. 1993), and not an increase in leaf N concentration as can occur following heavy defoliation (e.g., Kolb et al. 1999). The similar responses of P_n , g_w , N concentration, and $\delta^{13}C$ of the clones to defoliation over 2 years, when cumulative defoliation was similar for resistant and susceptible clones, also suggest a similar capacity for compensatory photosynthesis in resistant and susceptible trees. Because late bud burst phenology apparently enabled resistant trees to avoid heavy budworm defoliation, there is probably little selection for mechanisms leading to compensatory photosynthesis in resistant genotypes.

We acknowledge that our mature-tree clones growing in the greenhouse do not behave exactly as mature trees of the same genotype growing in the forest. Resource availability, water relations, and growing environment obviously differ between small, container-grown trees given ample water and nutrients in the greenhouse and mature forest trees that are subject to the rigors of nature. Nevertheless, the clones allowed us to study the relationship between bud burst phenology, budworm defoliation, and leaf gas exchange using mature tissues that could be easily manipulated in greenhouse experiments and provided a useful model system for phenological studies. However, they were less useful for genetic studies of growth rate, because the correlation with radial growth of mature trees was stronger for half-sib seedling progeny than for clones in the greenhouse.

In summary, we obtained evidence that two mechanisms, growth rate and bud burst phenology, are important in explaining variation in crown condition of mature Douglas-fir trees following western spruce budworm outbreaks. In contrast, we found little support for the idea that resistant trees have a greater inherent capacity for photosynthetic compensation after budworm defoliation than susceptible trees. Further, the pattern of differences in growth rate and bud burst phenology between resistant and susceptible trees was similar between mature trees in the forest and clones or half-sib seedling progeny in the greenhouse, indicating genetic control of these traits, which might be useful in Douglas-fir genetic improvement programs that include resistance to western spruce budworm.

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