Variation among Persian Walnuts in Response to Inoculation with *Xanthomonas campestris* pv. *juglandis*

Keith E. Woeste and Gale H. McGranahan
Department of Pomology, University of California, Davis, CA 95616

M.N. Schroth
Department of Plant Pathology, University of California, Berkeley, CA 94720

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Abstract. Walnut blight of English walnut (*Juglans regia* L.), incited by *Xanthomonas campestris* pv. *juglandis* (Pierce) Dowson, causes significant crop loss in California. To assess levels of resistance in walnut germplasm, leaves and nuts of mature walnut genotypes were inoculated with *X. campestris* pv. *juglandis*. Significant differences were found among cultivars in size and frequency of lesions on leaves and in frequency of abscission of diseased leaves. Cultivars also varied in frequency of abscission of nuts following infection and in marketability of infected nuts. Afthough there was considerable variation in disease levels over 2 years, leaves of PI 159568 consistently received significantly higher disease ratings than leaves of 'Chandler' or 'Adams'. Nuts of 'Adams', 'Payne', PI 18256, and 'Sinensis 5' abscised less frequently following inoculation than nuts of other cultivars. In addition, the quality of infected nuts that did not abscise was consistently better for PI 18256 and 'Sinensis 5'. The rank of cultivars for levels of disease in inoculated leaves was not significantly correlated with the rank of cultivars for frequency of infestation of dormant buds associated with infected foliage. The apparent resistance of walnut germplasm may be affected by the abscission or necrosis of infected tissues.

The major nut and foliage disease of Persian walnut in California is walnut blight incited by the bacterium *Xanthomonas campestris* pv. *juglandis* (Univ. of California Statewide Integrated Pest Management Project, 1982). The disease also limits walnut production in other regions of the world (Chariot and Germain, 1988; Ramos, 1985; Vavasour, 1984). All succulent tissues may be attacked (Miller and Bollen, 1946). Symptoms on leaves begin as small, water-soaked spots that can expand to form angular necrotic lesions 2 to 4 mm across and typically extend along veins as the disease progresses (Ark, 1944). Infected male florets become necrotic and deform the expanding catkin. Infected distillate flowers often abscise, but later infections on the hull of developing fruit expand rapidly, creating dark sunken lesions. If infection occurs before shell-hardening, the kernels are usually destroyed; infection after shell-hardening results in dark kernels and poor-quality nuts (Miller and Bollen, 1946).

The primary inoculum is bacteria that have overwintered in dormant buds (Mulrean and Schroth, 1982). The inoculum is spread by the action of wind and rain, and infection requires the presence of free water (Miller and Bollen, 1946). Thus, blight is most severe in climates with rains throughout the growing season, causing yield losses up to 80% (Chariot and Germain, 1988). In California, cultivars that leaf out and bloom after the spring rains have subsided often escape blight. For this reason, late leafing has been a high-priority trait for selection in the breeding program at the Univ. of California (Forde, 1975).

Soon after walnut blight was first described (Pierce, 1901), there were many reports of resistance to the disease (Batchelor, 1921; Ramsey, 1908; Smith, 1907, 1910, 1921, 1922; Smith et al., 1912; Ware, 1903, 1904; Wickson, 1910). Although these accounts were later discredited (Miller and Bollen, 1946; Rudolph, 1933), reports of resistant cultivars continue to appear (Adhikari et al., 1988; Gibson, 1967; Miller, 1963; Shreve, 1989; Tamponi and Donati, 1990). Purported resistance has usually been based on natural infection, and it has not been clear whether the absence of blight was due to disease escape or other forms of resistance.

The objective of this study was to determine if there is genetic variation in walnuts in response to controlled inoculation with *X. campestris* pv. *juglandis.*

Materials and Methods

Walnut germplasm. Genotypes used in the study represented the diversity of germplasm available in the Univ. of California, Davis, walnut collection. A range of origins, leafing dates, and reported susceptibility to blight (Table 1) was included. The leafing date falls when the average length of terminal buds is 2.5 cm (Forde, 1975). 'Payne', 'Sunland', and 'Serr' are considered very susceptible, whereas blight is not considered a problem on 'Hartley', 'Chandler', or 'Franquette' (Teviotdale et al., 1985). PI 159568 (Forde, 1975) and 'Howe' (Miller, 1963) are potentially resistant. Four mature trees of each genotype were used: two grafted on northern California black walnut [*J. hindsii* (Jeps.) Jeps.] and two on Paradox walnut (*J. hindsii* × *J. regia*) rootstock. Trees were irrigated and sprayed for pests in accordance with current horticultural practices (Ramos, 1985; Univ. of California Statewide Integrated Pest Management Project, 1982), except no effort was made to control blight during the years of the investigation.

Pathogen sources and inoculum production. Isolates of *X. campestris* pv. *juglandis* were recovered from dormant, healthy-appearing walnut buds using the methods of Mulrean and Schroth (1981). Additional *campestris* pv. *juglandis* strains numbered 110, 117, and 124 were obtained from M.N.S. The numbered

Table 1. Phenology and origin of walnut cultivars evaluated for response to inoculation with *Xanthomonas campestris* pv. *juglandis.*

Species and cultivar	Origin[y]	Leafing date[z]		Full bloom		Avg harvest date
		1988	1989	1988	1989	
Juglans regia						
PI 18256	China	5 Mar.	16 Mar.	26 Mar.	4 Apr.	18 Sept.
PI 159568	Afghanistan	5 Mar.	17 Mar.	27 Mar.	6 Apr.	18 Sept.
Sinensis 5	Japan	5 Mar.	17 Mar.	27 Mar.	7 Apr.	21 Sept.
Payne	California	7 Mar.	19 Mar.	27 Mar.	8 Apr.	15 Sept.
Sunland	California	9 Mar.	22 Mar.	5 Apr.	12 Apr.	6 Oct.
Serr	California	12 Mar.	20 Mar.	1 Apr.	9 Apr.	15 Sept.
Hartley	California	25 Mar.	4 Apr.	18 Apr.	20 Apr.	8 Oct.
0-13-1048	Afghanistan	26 Mar.	---	---	---	23 Sept.
Adams	Oregon	27 Mar.	4 Apr.	2 Apr.	7 Apr.	10 Oct.
Howe	Oregon	27 Mar.	4 Apr.	---	---	9 Oct.
Chandler	California	27 Mar.	6 Apr.	14 Apr.	19 Apr.	3 Oct.
Meylan	France	1 Apr.	---	---	---	26 Sept.
Franquette	France	7 Apr.	13 Apr.	26 Apr.	1 May	10 Oct.
Juglans hindsii						
Rawlins	California	2 Apr.	28 Mar.	---	---	---

[z]Leafing date falls when the average length of terminal buds is 2.5 cm.
[y]Origin indicates where cultivar was originally selected or collected.

and wild-type strains were mixed by culturing them together in 100 ml nutrient broth (Difco, Detroit, Mich.) + 2.5% dextrose. Cultures were gently agitated with a rotary shaker for 36 h, diluted with glycerol [30% (v/v)], and stored as l-ml aliquots at – 81C. These mixed strains were used for all inoculations to reduce the likelihood of observing a strain-specific response. Inoculum was quantified by standard dilution-plating and turbidimetric assays using light at 420 nm.

Leaf inoculations. Cultivars were inoculated when the terminal leaflet of the first emerged leaves unfolded, about 1 week after the leafing date (Table 1). Three shoots on each of 20 branches randomly distributed over the lower half of the canopies of the four trees were inoculated. Shoots were gently sprayed to run off between 0700 and 1000 HR with a suspension of *X. campestris* pv. *juglandis* adjusted to 2×10^9 colony-forming units (cfu)/ml of deionized water and enclosed (three per bag) for 24 h in bags, 60 × 90 cm, made of white spunbonded olefin (DuPont, Wilmington, Del.). To maintain high humidity around the shoots, the interior of the bag was premoistened with deionized water and a wet sponge was placed inside. Controls (45 shoots distributed on 15 branches of four trees of each genotype) were treated similarly, except they were sprayed with water instead of inoculum. This study was repeated the following year on different branches of the same trees.

Nut inoculations. Similar techniques were used to inoculate nuts. In 1988, nuts were inoculated when their outside diameter measured 35 to 40 mm, ≈5 to 6 weeks after bloom; in 1989 nuts were inoculated at 7 mm and 10 to 15 mm, ≈1 to 2 weeks and 3 to 4 weeks after bloom, respectively. Typically two to four nuts per branch on 20 to 40 branches distributed over four trees per genotype were inoculated. Control nuts, typically three nuts per branch on 15 to 20 branches per cultivar, were treated at the same stage of maturity as inoculated nuts, but with deionized water only.

Disease assessment. Leaves were rated for disease 21 and 35 days after inoculation using a O to 5 disease severity scale based on number, size, and distribution of lesions and the number of leaflets abscised. Leaves rated 5 if they were completely necrotic or abscised. Two leaves of each shoot were rated: the

most diseased leaf and the leaf that was most nearly opposite and distal to that leaf. Thus, six leaves (three shoots, two leaves per shoot) were rated on each branch.

Mesh bags were tied around fruits that remained attached to trees in mid-August. As each genotype reached its average harvest date (Table 1), the bags and nuts were collected. Nuts were hulled if necessary, air-dried in the bags for 1 week, and stored at 4C for 3 to 6 weeks until they were evaluated for kernel color according to the Walnut Color Chart (Dried Fruit Assn. of California, n.d.). Kernels darker than light amber were considered unmarketable.

A small subset of leaves (three per cultivar) and nuts (five per cultivar) were tested to confirm the presence of the disease. Bacteria were reisolated and identified using the methods of Mulrean and Schroth (1982), except leaf tissue was triturated instead of buds. In addition, overwintering buds from branches inoculated in 1989 (n = 17 to 20 per genotype) and control branches (n = 9 to 15) were collected to determine the relationship between severity of leaf symptoms and subsequent overwintering populations of *X. campestris* pv. *juglandis.* Treated branches, typically bearing 15 to 40 buds, were harvested in random order over 5 weeks, placed in plastic bags, and stored at 4C for 1 to 3 days. Samples of four healthy-appearing buds were taken from each branch; when possible, two catkin and two vegetative/distillate buds were chosen. If four healthy-appearing buds could not be found on a branch, then buds with necrotic areas were used. Both necrotic (Miller and Bollen, 1946) and healthy-appearing dormant buds (Mulrean and Schroth, 1982) are able to support *X. campestris* pv. *juglandis* over winter. All four buds from each branch were triturated together in 10 ml of 0.01 M phosphate buffer using a mortar and pestle. After trituration, 1.0 ml of the suspension was removed and serially diluted in sterile buffer. Aliquots of 0.1 ml of the serially diluted suspension were spread onto a semiselective medium, incubated at room temperature for ≈5 days, and quantitated using the methods of Mulrean and Schroth (1981).

Data analysis. Differences in lesion development were evaluated using a completely nested design (branch(tree(cultivar))). General Linear Models procedure of SAS (SAS Institute, Carey,

N.C.) was used to perform analysis of variance (ANOVA) with Tukey's test for mean separations. The Kruskal-Wallis test and Spearman's coefficient of rank correlation were used to evaluate and correlate ranked treatment results. The Ryan–Einot–Gabriel–Welsch multiple F test of SAS was used for rank mean separation. Wilcoxon's rank sums with a chi-square test was used to compare cultivars for the frequency of a given rank. A chi-square test was used to determine if cultivars differed in frequency of bud infestation.

Results

Leaf inoculations. Genotype was a highly significant $(P \leq 0.001)$ source of variation in disease severity in both years and at both rating times, contributing between 57% (1988, 35 days postinoculation) and 74% (1989, 35 days) of the total variation. In the first year, tree within genotype was also significant at 21 days $(P \leq 0.01)$ and 35 days $(P \leq 0.05)$. Leaves of PI 159568 consistently were given significantly higher disease ratings than other cultivars tested (Fig. 1). 'Adams' and 'Chandler' ranked lower than 'Franquette', 'Howe', 'Payne', PI 159568, 'Serr', and 'Sunland' 35 days after inoculation in 1988 and 1989, although their means were not significantly different than 'Sunland', 'Payne', and 'Serr' in 1988.

Leaf lesions appeared on all cultivars ≈ 14 days after inoculation, and infections became increasingly severe. The severely infected leaves of PI 159568 began to abscise by 21 days after inoculation, and all of its infected leaves abscised by day 40 (observation only). There were significant differences among cultivars in the frequency of severe infections as measured by the proportion of leaves rated 5 on the 0 to 5 disease severity scale 35 days after inoculation (Table 2). 'Chandler' consistently had significantly fewer leaves rated 5 than 'Payne' or PI

159568. 'Chandler', 'Sinensis 5', PI 18256, 'Howe', and 'Adams' had fewer than the average number of leaves rated 5 in 1988 and 1989. Disease severity ratings were higher in 1989 than 1988 (Fig. 1), but the rank of cultivars remained fairly consistent at 35 days postinoculation (Spearman rank correlation coefficient = 0.73, $P \leq 0.01$). The rank correlation coefficient between the two rating times was 0.75 $(P \leq 0.003)$ and 0.83 $(P \leq 0.003)$ in 1988 and 1989, respectively. Levels of natural infection on control branches did not vary significantly among cultivars $(P \leq 0.5)$, except for PI 18256 in 1989, which had significantly more infected branches. All control branches had a mean branch disease rating of less than one (data not presented).

Dormant bud samples. Cultivars had significantly $(P < 0.01)$ different ratios of infested to noninfested dormant-bud samples when samples were from branches that bore inoculated leaves the previous growing season (Table 3). Cultivars were not significantly different when samples were from control branches. 'Chandler' and PI 159568 had the lowest ratios of infested to noninfested bud samples from inoculated branches, accounting for 50% of the chi-square value for difference among cultivars. 'Adams' and 'Payne' had the highest ratios, accounting for 23.5% and 12.4% of the total chi-square value, respectively. The rank of cultivars for the ratio of infested to noninfested dormant bud samples was not significantly correlated with the rank of cultivars for leaf lesion rating at any rating time.

Nut inoculations. Walnut cultivars were variable in the number of infected nuts retained until harvest and the marketability of infected nuts at harvest (Table 4). PI 18256, 'Payne', and 'Sinensis 5' retained an average of 79% of their infected nuts in 1988 and 67% in 1989. 'Sunland' and PI 159568 retained an average of 9% and 0% in the same years. 'Serr' retained 70%
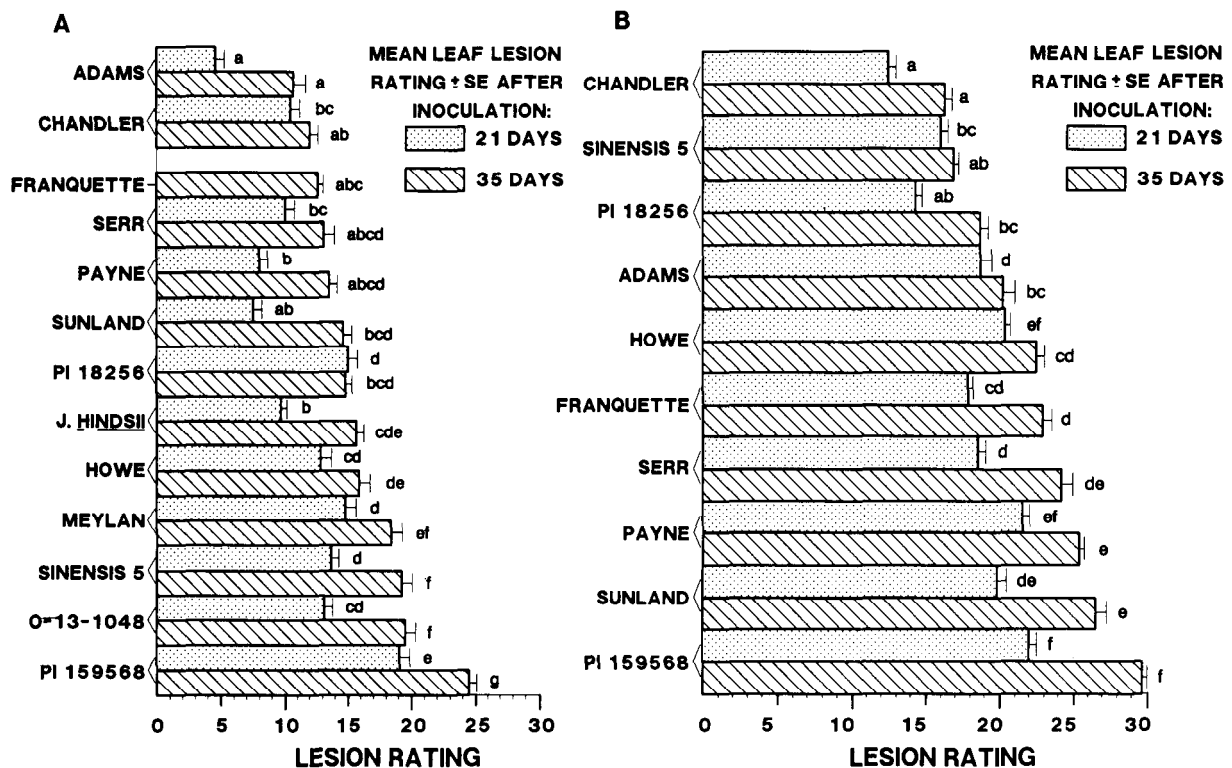

Fig. 1. Leaf lesion ratings of Persian walnut cultivars after inoculation with *Xanthomonas campestris* pv. *juglandis.* Six leaves per branch were rated using a O to 5 disease severity scale and these values were summed. The mean of 20 rated branches is shown for (A) 1988 and (B) 1989. Means within years and days after inoculation (21 or 35) were separated by Tukey's test $(P \leq 0.05)$.

Table 2. Wilcoxon scores[z] for the proportion of leaves of Persian walnut cultivars inoculated with *Xanthomonas campestris* pv. *juglandis* that became severely infected.

	No.	Sum of rank scores ± SD	
Cultivar	trees	1988	1989
Chandler	3	48.0 ± 13.4	15.0 ± 18.8
Sinensis 5	4	80.0 ± 15.2	20.0 ± 21.4
PI 18256	4	64.0 ± 15.2	38.0 ± 21.4
Howe	4	64.0 ± 15.2	68.0 ± 21.4
Adams	4	64.0 ± 15.2	72.0 ± 21.4
Franquette ·	4	64.0 ± 15.2	83.5 ± 21.4
Serr	4	64.0 ± 15.2	106.0 ± 21.4
Sunland	4	64.0 ± 15.2	127.0 ± 21.4
Payne	4	127.0 ± 15.2	100.0 ± 21.4
PI 159568 ·	4	140.0 ± 15.2	150.0 ± 21.4

[z]Wilcoxon scores (rank sums) for the proportion of the six rated leaves on each of 20 branches per cultivar that rated 5 on a 0 to 5 disease severity scale, 35 days after inoculation. For 'Chandler', the expected sum of the rank scores under the H_o was 60 in 1988 and 80 in 1989; for all other cultivars these values were 80 in 1988 and 80 in 1989. In 1988, x^2 = 29.3; probability of a larger value = 0.0006. In 1989, x^2 = 32.2; probability of a larger value = 0.0002.

Table 3. Influence of inoculation with *Xanthomonas campestris* pv. *juglandis* on frequency of infestation of samples'[z] of dormant buds of Persian walnut cultivars.

	No. bud samples infested/ no. taken	
Cultivar	Inoculated[y]	Control
Chandler	1/19	1/15
PI 159568	3/18	1/9
Serr	5/17	3/13
Sinensis 5	6/17	1/10
PI 18256	7/18	1/13
Sunland	7/17	2/13
Howe	9/20	5/13
Franquette	10/17	5/13
Payne	11/18	3/12
Adams	13/19	2/15

[z]Four buds per sample.
[y]Samples were from branches that bore inoculated or control leaves during the 1989 growing season.

in 1988 but only 15% in 1989. Over the two years of the study, an average of 48% of the infected nuts of PI 18256 and 'Sinensis 5' were marketable. 'Serr' and 'Payne' averaged 43% marketable nuts in 1988 but 1.5% in 1989. Almost all the infected nuts of 'Sunland' were unmarketable at harvest. "

Discussion

Significant differences in disease ratings and frequency of severe leaf infection were detected among cultivars of Persian walnut in response to inoculation with X. *campestris* pv. *juglandis*. There also were differences in the susceptibility of nuts with respect to amount of abscission and marketability following infection.

Only PI 18256 and 'Sinensis 5' had a consistently large percentage of marketable nuts at harvest following inoculation. These cultivars are derived from germplasm from the Far East, probably China; by contrast, most of the advanced breeding lines in California are of French provenance.

PI 159568 seemed highly susceptible; its leaves consistently received significantly higher disease severity ratings than other genotypes, and all its infected leaves and nuts abscised. This result contrasts with that of Forde (1975), who reported that PI 159568 appears blight resistant. However, PI 159568 also had a low ratio of infested to noninfested buds when dormant buds were sampled from branches that bore inoculated leaves the previous growing season (Table 3). These apparently contradictory results might be resolved if abscission and/or necrosis of severely infected tissues interrupts the disease cycle. Leaf abscission did not appear to be related to the restriction of bacteria from the vasculature of PI 159568, since shoots of PI 159568 were often killed by blight infections that began in the leaves.

The relative importance of different disease responses in relation to economically significant levels of resistance is uncertain. By abscising infected tissues, trees can partition resources to healthy leaves and nuts. Abscission of diseased tissues may also interrupt the disease cycle. Conversely, since abscised nuts cannot be sold, cultivars such as PI 18256 and 'Sinensis 5' that retain diseased but marketable nuts might prove more profitable

These results suggest there is genetic variability among Persian walnuts in response to infection by X. *campestris* pv. *juglandis*. Methods for evaluating walnut germplasm for resistance to blight should consider the entire disease cycle, since the severity of blight during some parts of the growing season may not be significantly correlated with subsequent overwintering populations of the pathogen.

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Table 4. Abscission of nuts of Persian walnut cultivars inoculated with *Xanthomonas campestris* pv. *juglandis* and the marketability of nuts at harvest.

Year	Treatment[z]	Adams	Franquette	Hartley	Payne	PI 18256	PI 159568	Serr	Sinensis 5	Sunland
				No. nuts harvested/no. inoculated						
1988	I	---	---	---	45/57	48/60	0/60	42/60	41/53	11/60
1989	Ia	42/100	9/100	1/94	58/103	83/134	0/50	0/100	68/122	0/100
1989	Ib	76/96	46/100	8/60	76/109	53/60	0/35	30/100	103/130	0/100
1988	C	---	---	---	33/33	26/31	26/30	28/30	37/39	27/30
1989	Ca	40/50	55/60	35/51	86/100	59/60	35/50	53/59	78/100	47/50
1989	Cb	52/52	60/60	33/50	55/60	51/51	21/25	42/51	54/61	53/71
				Marketable nuts (%)[y]						
1988	I	---	---	---	45.6	53.3	0.0	40.0	58.5	10.0
1989	Ia	17.0	1.0	0.0	0.0	28.0	0.0	0.0	46.7	0.0
1989	Ib	46.0	0.0	0.0	0.0	53.0	0.0	6.0	58.4	0.0
1988	C	---	---	---	87.8	83.8	70.0	83.3	89.7	76.0
1989	Ca	68.0	72.0	66.6	61.0	70.0	64.0	84.7	75.0	74.0
1989	Cb	96.0	93.0	66.0	91.6	75.0	80.0	86.3	78.7	61.9

[z]I = inoculation; C = control; a and b indicate size of nut at inoculation, ≈7 and 15 mm o.d., respectively. Nuts were ≈35 mm o.d. at inoculation in 1988.
[y]Expressed as percentage of all nuts inoculated.

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