Heritability of *Leucostoma persoonii* Canker Resistance among Diverse Peach Genotypes

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Abstract. Heritability and the genetic and environmental variance components of resistance to the canker-causing pathogen *Leucostoma persoonii* were estimated in a population of diverse peach (*Prunus persica* L.)-genotypes. Disease resistance was measured as the length of necrotic tissue, i.e., canker length, following artificial inoculation in the field. Genetic and environmental variations were partitioned as variance components of the linear statistical model. Heritability was estimated by regressing average performance of seedlings on performance of their maternal parent. The genetic variance was highly significant, and the heritability for canker necrotic length was relatively high (0.72), suggesting that it should be possible to select *L. persoonii*-resistant individuals within the population.

Perennial canker, incited by *Leucostoma persoonii* Fr. (Nits.) Hohn or *L. cincta* (Pers. ex Fr.), seriously limits peach longevity and productivity in cold areas of peach production (Cline, 1982; Gairola and Powell, 1970; Hildebrand, 1947; Jones and Luepschen, 1971; Layne, 1976, 1984; Luepschen, 1981; Wensley, 1964). Since cultural practices and chemical treatments do not adequately control the disease, the ultimate approach must be through host-plant resistance. Studies of peach susceptibility to *Leucostoma* infection, based on either natural infection or artificial inoculation, have been conducted, and almost all commercial peach cultivars failed to show resistance (Dhanvantari, 1978; Dhanvantari and Dirk, 1983; Gairola and Powell, 1970; Hildebrand, 1947; Layne,

1984; Luepschen, 1981; Palmiter and Hickey, 1970). Possibly, this lack of resistance to *Leucostoma* can be attributed to the narrow genetic base of peach cultivars in North America (Layne, 1976, 1984). Where some level of resistance was found, the reaction tended to be inconsistent among experiments. Following inoculation of a diverse peach population with *L. persoonii*, we reported that some peach genotypes may have a higher tolerance to *L. persoonii* canker than has been found previously in U.S. germplasm (Chang et al., 1989).

Studies of various tree crops, including sweet cherry, peach, nectarine, walnut, and plum, have shown considerable additive genetic variance for commercially important traits (Hansche, 1986a, 1986b; Hansche and Boyton, 1986; Hansche et al., 1966; Hansche et al., 1972a; Hansche et al., 1972b; Kester et al., 1977). Estimates of heritability and the relative contributions of genetic and environmental variability are useful to breeders as guides in improving tree crops and in maximizing breeding efficiency.

This study was undertaken to evaluate genetic and environmental variation and to estimate the heritability of *L. persoonii* resistance in a diverse peach population that previously had been shown to segregate for

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Table 1. Parents of the 15 half-sib peach families used to calculate the heritability estimates of *L. persoonii* resistance from parent-offspring regression (POR) and the least squares model (LS).

Parent	Pedigree	Model	
		POR	LS
Babygold 8	PI35201 x Ambergem		x
Canadian Harmony	Redskin x Sunhaven	x	x
Elberta	Chinese Cling (open-pollinated)	x	
Harken	Redskin x Sunhaven	x	x
Loring	Frank x Halehaven	x	x
Red Hale	Unknown		x
B8-11-147	(K82 x Sunrise) x [(Red Ceylon x NJ191) x Okinawa]	x	x
B8-20-171	(5110417 x Ta Tao 3 ^a) x C2R31T45		x
B8-21-20	Orange Cling x RR65-1		x
C2-28-89	Kasna Dupnisha open-pollinated	x	x
C4-11-97	Peach x almond	x	x
NJ257	Honeydew Hale x Jefferson		x
NJN69	(NJN55 x NJC68) x Marzochella	x	x
NJ672017002	(PI35321 x Cherryred) x <i>Prunus kansuensis</i>		x
RR37-15	NJ174 x <i>Prunus kansuensis</i>		x

^aTa Tao 3 = P.I. 101665.

Table 2. The analysis of variance of the linear statistical model used to calculate the heritability of canker resistance among 14 peach half-sib families.

Sources of variation	df	Mean squares
Among peach families (F)	13	179.03***
Progeny within family (P _{ij})	68	38.48 [†]
Year (Y)	2	6.28
Year x peach family (FY) _a	26	32.96
Year x progeny within family (Y x P _{ij})	136	25.02**
Sampled branch within progeny within family (S _{ijk})	82	8.54
Y x S _{ijk} (E _{ijkl})	163	13.55

***, **, [†] Significant at P = 0.005, 0.01, and 0.05, respectively.

Table 3. Mean values of canker necrotic length for open-pollinated progeny generated from eight diverse peach parents.

Parent	Parent canker necrotic length ^a (cm)	Offspring canker necrotic length ^b (cm)
NJN69	13.5	11.1
Elberta	13.1	12.8
Harken	11.0	9.4
Loring	10.3	9.6
Canadian Harmony	9.7	8.3
C2-28-89	9.3	9.4
B8-11-147	8.3	7.4
C4-11-97	5.1	5.7

^aMean of 10 clones over 2 years. The performance of female parent was estimated by the mean of the necrotic length ratings from 10 replicated clones following inoculations in Fall 1986 and 1987.

^bMean of 20 progeny per clone over 2 years.

L. persoonii resistance. Genetic and environmental variations were partitioned as variance components of the linear statistical model. Heritability was estimated from the linear regression of offspring performance on the average performance of its female parent.

Linear statistical model. Open-pollinated seedlings from diverse backgrounds were planted in Spring 1984 in a completely randomized design at the Horticultural Research Center, East Lansing, Mich. (Chang et al., 1989). Fourteen open-pollinated peach families with at least six progeny were randomly selected to estimate the heritability of *L. persoonii* resistance (Table 1).

Two 2-year-old branches, ≈17 mm in di-

ameter, on each seedling were inoculated with 20 µl of a suspension of 10⁷ *L. persoonii* conidia per milliliter derived from isolates collected from cankers on peach at Clarksville and Hartford, Mich. The wound-freezing inoculation technique developed by Scorza and Pusey (1984) was followed. Trees were inoculated in early Oct. 1985, 1986, and 1987, then evaluated the following springs when trees initiated growth. Inoculated branches were measured for length of canker necrosis distal to the point of inoculation.

Phenotypic variability of all observations of canker necrotic length on this collection of peach progeny over all 3 years is described by the following model: $Y_{ijkl} = u + F_i + P_{ij} + S_{ijk} + Y_l + (FY)_{il} + (PY)_{ijl}$

+ E_{ijkl} , where Y_{ijkl} represents the kth sample branch within the jth progeny within the ith peach family in the lth year. The effects in this model stand for the overall population mean (u), a random effect due to each family (F), the effect of progeny within family (P_{ij}), the effect of sampling branches within progeny within peach family (S_{ijk}), the year effect (Y), the interaction of family and year [(FY)_{il}], and the error due to the year x S_{ijk} (E_{ijkl}).

Parent-offspring regression. Eight randomly selected female parents from the original population (Chang et al., 1989) were grafted onto Tennessee Natural peach rootstock and planted in 1985 in blocks adjacent to the orchard of diverse peach progeny (Table 1). Ten grafted trees per female parent plus 20 open-pollinated progeny per female parent were selected for artificial inoculation. Two randomly selected healthy branches of each grafted tree and each progeny were inoculated on the same day in 1986 and 1987 as described above. Female parent performance was rated as the mean canker length of 10 replicated individuals over 2 years. Progeny performance from the eight families was calculated as mean canker length over the two years.

Results were based on unadjusted data for the year effect because no significant variation due to year effect was evident in previous studies (Chang et al., 1989). Heritability was based on the regression of the mean performance of their maternal parent.

Linear statistical model. The experimental error can be divided into the error due to year x sampling branches within progeny within family (E_{ijkl}) and that due to sampling branches within progeny within family (S_{ijk}) (Table 2). The variation due to sampling error was not significant. The attempt to choose branches of equal size may have effectively controlled this potential source of variation. However, the second error term was quite large. This variation could be attributed to branch size, age, and directional orientation that may have interacted differently between years.

Variation attributed to years and the interaction between year and peach family were not significant and, therefore, would not dramatically decrease the efficiency of selection of more disease-resistant individuals from this population. However, the year x progeny within family variation was relatively large. Therefore, statistically removing the yearly environmental effect would increase the selection efficiency and the rate of genetic gain.

The variation due to the progeny within family was significant. Doubtless, this variation for peach families was due to a combined effect of genetic segregation within the families and environmental variability. Therefore, it is not appropriate to estimate how much variability is due to the environmental effect within families without clonal propagation and replicated evaluation of the individual seedlings. The mean squares due to variation among peach families were highly significant, indicating a large genetic component for canker length in this population.

Parent-offspring regression. The parent-offspring regression is commonly used to estimate the heritability of quantitative traits in various crop species. Progeny mean values are usually regressed on values from one or both parents, depending on pollination control. Since peach is presumably highly self-pollinated (Hesse, 1976), the performance of the female parent has been used as an estimate of the mid-parent performance (Hansche, 1968a, 1968b). The linear regression coefficient gives the estimate of heritability as the ratio of additive genetic variance : phenotypic variance.

Generally, the mean canker necrotic length rating was lower in the seedling population than in the female parent performance over the two years of observations (Table 3). The apparent increase in resistance to *L. persoonii* in the progeny may have been due to self-pollination increasing the additive effect or, alternatively, to an effect of age or grafted vs. own-rooted trees. The heritability estimate and standard deviation, based on parent-offspring regression of the canker necrotic length ratings, were 0.76 ± 0.11 . According to Falconer (1981), use of the parent performance will cause the heritability estimate to be biased downward by $\approx 5\%$. Therefore, the heritability of *L. persoonii* resistance in this diverse population can be calculated as $0.76 \times 0.95 = 0.72$. This relatively high heritability estimate is consistent with the highly significant genetic component (variation among peach families) from the linear statistical model.

Several factors influence the breeder's selection of a breeding scheme to improve genetic stocks. These include: 1) quantity and types of genetic variance, 2) environmental effects, 3) the interaction between environment and genotype, and 4) linkage effects. Genetic and environmental effects and interactions between genotype and environment were estimated in this population. The estimate of heritability for *L. persoonii* resistance, measured as canker length, was relatively high. Therefore, it should be possible to select *L. persoonii*-resistant individuals in this population and maintain them in the breeding program without the masking effect of year X year variation.

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