California Cling Peach Advisory Board

2008 Annual Report

Project Title:	Assessment of Brown Rot Resistance in Advanced Experimental Selections of Peach
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Cooperating Personnel:	Carlos Crisosto, Eben Ogundiwin, David Slaughter
Location:	Departments of Plant Pathology and of Plant Sciences, University of California at Davis

Summary:
Over 330 peach and peach hybrid genotypes were evaluated in laboratory assays for resistance to brown rot disease caused by *Monilinia fructicola.* Mean lesion diameters and incidence (proportion of infected fruit) were determined in inoculated fruit for each genotype, and from these values disease severity values were calculated. Fruit color, an indicator of quality and maturity, also was estimated by color image analysis and light transmittance. This year's evaluation included material carried forward from previous years, including some of the advanced lines with heritage from peach x almond hybrids, cv. Bolinha and USDA lines. A number of the advanced selections have incorporated improved levels of brown rot resistance, and include Ultra-Early#1, Extra-Late#4, Extra-Late#5, Extra-Late#6, Extra-Late#7, and Compact#2. Our work this year also included a second year of evaluation of the progeny from two crosses between susceptible and previously determined resistant genotypes. This aspect of the project is part of a larger University of California ANR Core Issues grant (Crisosto and Ogundiwin, et al, principal investigators) to explore the feasibility of marker-assisted selection for brown rot resistance in cling peach breeding. For one of these populations, we have identified polymorphisms that identify 69 markers for linkage analysis. Locations of putative QTLs conferring resistance to brown rot were identified, and two candidate genes in the cutin and lignin biosynthesis pathways were mapped to regions on a *Prunus* reference map corresponding to locations of two putative QTLs detected for brown rot resistance. We also evaluated a collection of freestone peach and nectarine cultivars in collaboration with Dr. Carlos Crisosto, UC Kearney Agricultural Center, for brown rot resistance and other fruit maturity indices. Several of these displayed strong brown rot resistance over two years of testing.

Objective:
The primary objective of this research is to identify the most promising experimental selections that possess the desired characteristics of disease resistance and horticultural traits for subsequent multiplication and distribution in test orchards.

Overview of 2008 Research

Over 330 genotypes were evaluated for the period beginning 24 June to 15 September 2008. Fruit of similar maturity were selected based on visual inspection of size and color from among the experimental selections for comparison with fruit of similar maturity from commercial, susceptible or moderately resistant clingstone peach cultivars.

For most genotypes screened in the program this year, two inoculation methods were used – non-wounded and wounded. The non-wounded treatment consists of applying a droplet containing conidia (spores) of *Monilinia fructicola* directly on the intact peach surface. This provides an assessment of the epidermal and cuticular resistance of the fruit to direct penetration by the pathogen. The wounded treatment consists of making a shallow wound (1-2 mm deep) with a small syringe needle to breach the cuticle and epidermis (exocarp), and applying the inoculum to this wound. This provides an assessment of the flesh resistance. Most of our previous work has focused on the epidermal resistance, in part because of the heritage (i.e., from Bolinha) and heritability of this trait in the breeding program. However, some of the more recent material with heritage from other sources that has emerged in the program displays some flesh resistance as well.

Figure 1 shows the disease severity rankings for all genotypes evaluated during 2008 in the non-wounded treatment, in order from the most resistant (lowest disease severity) to the most susceptible (highest disease severity). These data are also presented in a more detailed fashion in **Table A** of the appendix. Of these, 103, or about 30%, had average lesion sizes less than or equal to 3 mm, which we consider to be highly resistant. In **Figure 2**, the disease severity values for wounded fruit are presented. Obviously, wounding compromises any epidermal/cuticular resistance and facilitates pathogen ingress and colonization. However, a few genotypes display a relatively good resistance even in the wounded format.

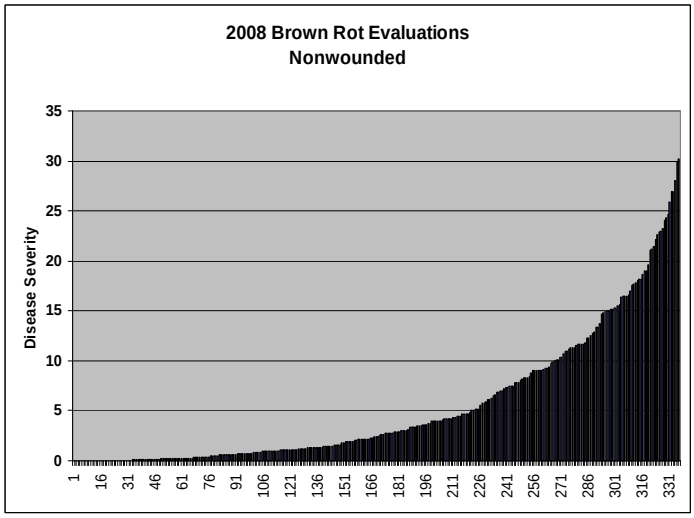

Fig. 1. Disease severity values of all genotypes evaluated in 2008, from the most resistant to most susceptible. Rankings for 336 individual genotypes are presented.

There is a moderately positive and statistically significant correlation among genotypes in lesion size between wounded and nonwounded treatments (Spearman's $\rho = 0.51$, $P < 0.0001$; **Fig. 3**). This indicates that in many of the lines evaluated that the epidermal/cuticular and flesh resistances are related, consistent with our seeing some selection for both forms of fruit resistance, which is certainly desirable.

Approximately 45% (151) of the genotypes evaluated this season were new lines or materials brought forward from the previous seasons, including commercial standards for comparison (e.g., Ross, Carson, Sherman). **Figure 4** illustrates the range in disease severity values for these lines. **Figure 5** shows the average disease severity values for the commercial standards, Bolinha, and some of the advanced selections developed in the program including peach X almond hybrids (F8 series, e.g. F8,5-

156 ExtraLate #5) and D62-193 (UltraEarly #1). The Bolinha value is uncharacteristically high this year, probably because these fruit were overly ripe at the time of analysis.

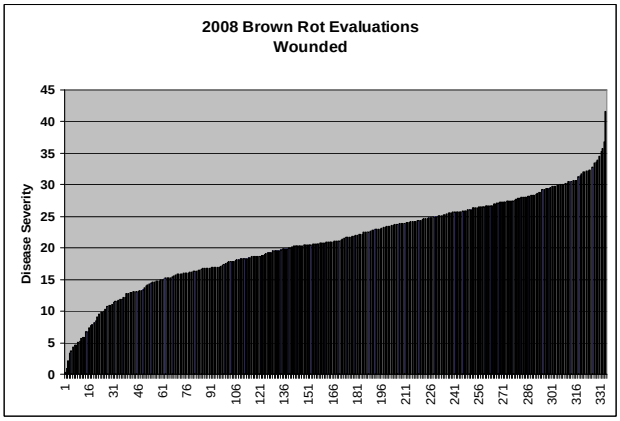

Fig. 2. Disease severity values of all genotypes evaluated in 2008, from the most resistant to most susceptible. Rankings for 336 individual genotypes are presented. Wound inoculation method.

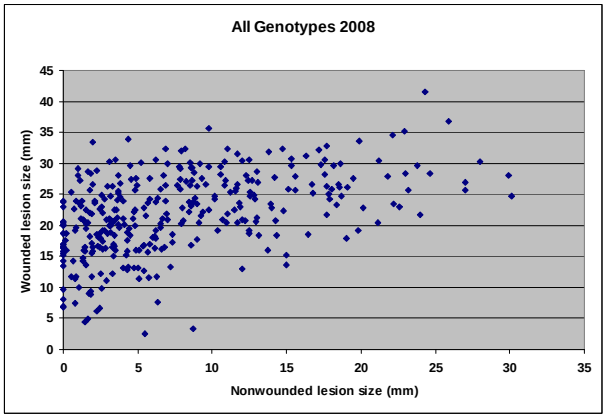

Fig. 3. Nonwounded lesion size vs. wounded lesion size plotted for each genotype. Spearman's correlation analysis, ρ = 0.51, *P* < 0.0001

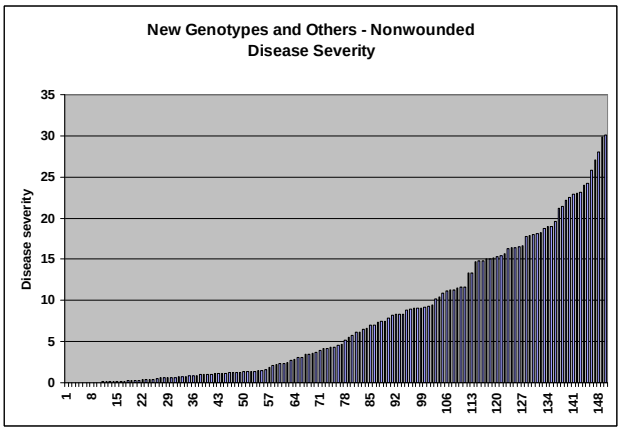

Fig. 4. Range of disease severity values for new experimental selections or materials brought forward from the previous seasons, including commercial standards, evaluated in 2008.

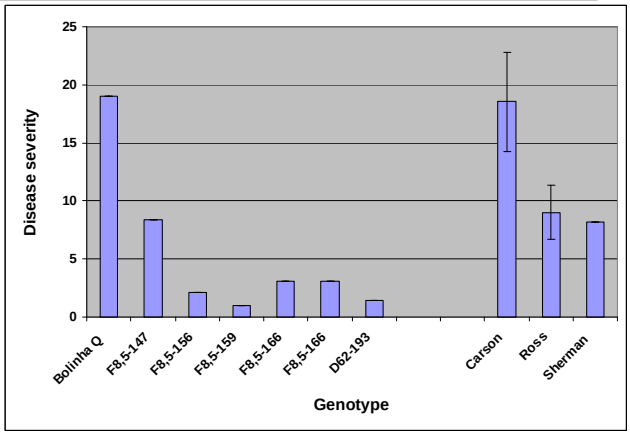

Fig. 5. Disease severity values of commercial cultivars used as comparative standards, Bolinha, and advanced selections (F8 series; D62-193) with strong brown rot resistance with heritage from different sources (2008).

Analysis of the PopBR populations

As part of a program to develop predictive tools for brown rot and sour rot resistance in peach and nectarines, in 2008 we evaluated 187 progeny lines from two mapping populations. The first population (Pop-BR1) was derived from the cross 'Dr. Davis' × 'F8,1-42', the latter having disease resistance heritage from almond. The second population (Pop-BR2) was developed from crossing the brown rot susceptible peach cultivar 'Loedel' to 'UCD96,4-55' a resistant experimental line derived from cv. 'Bolinha'. For most of these progeny lines we now have two consecutive years of brown rot data. The results for

these mapping populations for the nonwounded treatment are displayed in **Fig. 6** (composite of Pop-BR1 and Pop-BR2, 2007-2008 data combined). **Figure 7** shows frequency distributions for lesion size classes for the combined Pop-BR1 and Pop-BR2 populations compiled for both years. Brown rot disease reactions are arbitrarily divided among three classes based on disease severity: resistant (R), moderate reaction (M), and susceptible (S), and generally conform to the range of disease reactions illustrated in Fig. 5 for the comparative standards and advanced selections. The results also are consistent with quantitative (polygenic) inheritance of the fruit resistance phenotype. More detailed numerical data are presented in the appendix in **Table B**.

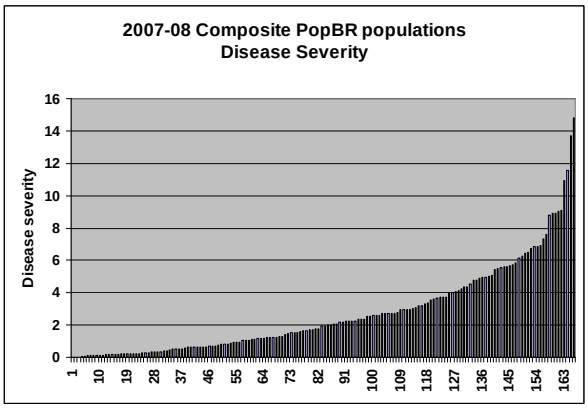

Fig. 6. Range of disease severity values for the two Pop-BR mapping populations, average of 2007 and 2008 data.

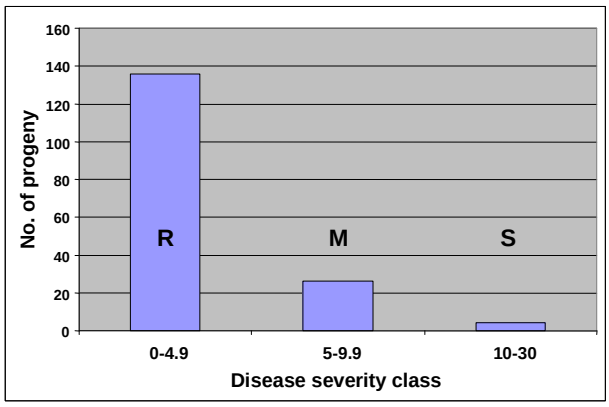

Fig. 7. Frequency plot of number of Pop-BR progeny in the three brown rot disease severity

Dr. Eben Ogundiwin has performed a preliminary analysis of DNA polymorphisms that associate with brown rot resistance in these progeny. A total of 230 simple sequence repeats (SSRs) and 37 candidate gene (CG) primer pairs were screened for polymorphism using the parents and progeny subsets of Pop-BR1 out of which 52 SSRs and two CGs were polymorphic. The polymorphic SSRs generated 59 SSR markers. In addition, eight resistance gene analog markers (RGAs) were generated. The total number of markers available for linkage analysis was 69 (59 SSRs, 8 RGAs and 2 CGs). A scaffold linkage map was constructed from these data consisting of 31 markers spread over 12 linkage groups of two to five markers each. These were organized into 7 linkage groups corresponding to the 'Texas' × 'Earlygold' *Prunus* reference map (T x E map) using common SSR markers (**Figure 8**). The locations of putative QTLs conferring resistance to BR also are indicated in **Fig. 8**.

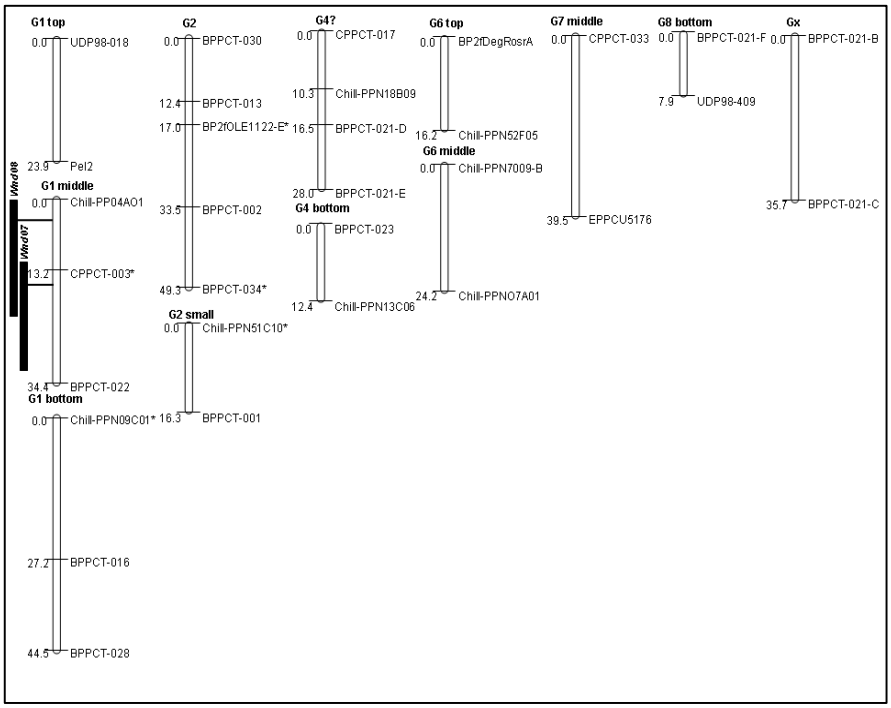

Fig. 8. Partial linkage map of Pop-BR1; linkage group numbers and orientation derived from SSR markers in common with the *Prunus* reference T×E map; Group Gx could not be assigned a known number; BR resistance QTL markers detected by non-parametric Kruskal-Wallis test asterisked; black bars represents putative BR resistance QTLs located by interval mapping analysis.

Putative QTLs detected by the non-parametric Kruskal-Wallis (KW) test and interval mapping are shown in **Tables 3 and 4**, respectively. The KW test detected three QTLs for nonwounded inoculation in 2007, two for wound inoculation in 2008 and one each for wound inoculation in 2007 and nonwounded inoculation in 2008. One QTL was stable for each inoculation method across the two years. Interval mapping analysis detected one QTL for wounded inoculation in 2007 on linkage group G1 controlling up to 52% of observed variation. One QTL was detected on the same linkage group for wounded inoculation in 2008 controlling about 24% of observed variation. The proximity of these two QTLs suggests that they may be controlled by the same gene. Marker saturation of this region will aid QTL position refinement. Two candidate genes in the cutin and lignin biosynthesis pathway mapped to regions on the T×E *Prunus* reference map corresponding to locations of two putative QTLs detected for brown rot resistance on Pop-BR1 (results not shown). Further work is needed to map these genes directly to Pop-BR1 and validate their relationship with the QTLs, as well as to conduct similar analyses of the Pop-BR2 population.

Table 3: Putative QTLs for resistance to wounded and nonwounded BR inoculations detected by non-parametric Kruskal-Wallis test

Trait	Markers	LG	K*	*P*
Wound 07	BPPCT034	G2	10.14	0.05
Nonwound 07	CPPCT003	G1	6.85	0.01
	ChillPPN09C01	G1	7.10	0.01
	BP2fOLE1122-E	G2	7.57	0.01
Wound 08	CPPCT003	G1	9.46	0.005
	BPPCT034	G2	8.37	0.05
Nonwound 08	CPPCT003	G1	4.86	0.05

K* = the Kruskal-Wallis test statistic, LG = linkage group, *P* = significance level

Table 4: Putative QTLs for resistance to BR detected by interval mapping analysis of MapQTL®

Trait	Marker Interval	LG	Position (cM)	LOD	% explained
Wound 07	CPPCT003-BPPCT022	G1	28.18	3.74	52.0
Wound 08	ChillPPN04A01-CPPCT003	G1	2.0	3.39	24.0

Fruit color measurement

During the 2008 sampling, as in previous years, fruit maturity based on color was assessed for each genotype evaluated in the laboratory using digital color images and spectrometric analysis of transmitted light as measured with a hand-held spectrophotometer. In addition, in 2008 we again measured color with a nondestructive optical instrument, a light interactance sensor (Slaughter et al, 2006). We now have two seasons of measurements with the light interactance sensor. Color assessments relative to the disease data are ongoing in collaboration with Dr. David Slaughter, Dept of Biological and Agricultural Engineering, UC Davis. Initial analyses in 2007 indicated that hue angle, derived from the color measurements, and transmittance at 670 nm, related to chlorophyll content, are at best only weakly correlated with lesion size in a manner consistent with expectations over the entire set of genotypes analyzed. Our goal here is to see if these optical measures, in addition to having some utility as a measure of harvest maturity, have some predictive value in assessing brown rot susceptibility/resistance in these cultivars. In previous work, fruit firmness and lesion size were negatively correlated in all cases (nonwounded fruit inoculations; the firmer the fruit, the less susceptible). This is consistent with expectations. We will continue to tease apart the large datasets collected this season and last to look for associations as well as deviations from any perceived trends that may be significant relative to disease resistance or susceptibility of the genotypes.

Future plans

A goal of the program has been to identify the most promising early and late maturing genotypes, since these are often the most vulnerable to brown rot disease and present the most difficult challenge

for disease management. DC62-193 (UltraEarly#1) is a very early maturing genotype and is in its 9th year of evaluation. This genotype rated highly or moderately well for brown rot resistance in our laboratory evaluations, and is currently in regional field trials. This genotype has additional fruit quality attributes that make it attractive for continued testing and development.

Depending on the availability of funds, we will begin to assess the variation of *M. fructicola* and other *Monilinia* spp. in the California population. The importance of understanding the genetic variation of *M. fructicola* and other *Monilinia spp.* has a practical application for the breeding and selection of new cling peach genotypes and disease management. We would like to have a better handle on the range in variation in virulence of isolates common in California peach orchards by evaluating them on selected lines in side-by-side comparison with the highly aggressive isolate that we have used in our screening program (MUK-1). We propose to begin to collect several dozen isolates from various hosts in California, the US, and elsewhere to establish a comprehensive culture collection. We have colleagues who are willing to participate and provide isolates or DNA from those isolates for which we cannot obtain permits. Strains will be characterized using molecular markers and for virulence in disease assays. We also will coordinate with Drs. Jim Adaskaveg (UCR) and Themis Michailides (UCD-KAC), who have extensive collections of *Monilinia* already.

We will continue with the molecular marker analysis of resistance to both brown rot and to sour rot. The scaffold linkage maps will be constructed for both populations and QTL analysis of resistance will be conducted. Markers closely linked to the resistance QTLs will be identified for use in breeding programs. We will determine if a 3rd round of inoculation experiments on the progeny populations is necessary. This is very important for the reliability of QTL analysis because it will allow us to account for non-genetic variation due to experimental errors and environmental factors.

Materials and Methods

***Disease Assays*.** Disease assays were performed as described in previous reports. Briefly, freshly harvested fruit, selected at random from trees at the UC Davis Pomology Orchards or at the Kearney Agricultural Center, were stored at 4 C, usually 4 days to as much as 2 wks, in a few cases, until the day of the assay. Stored fruit were warmed to room temperature prior to inoculation. Fruit were surface sterilized for 30 sec by immersion in 10% bleach (0.6% NaOCl), rinsed, and dried.

Approximately 20 unblemished fruit of each genotype were placed in humidified plastic containers with fruit liners. The number varied depending upon the availability of fruit for each genotype. For some genotypes fruit also were punctured with a 22 gauge needle at the point to be inoculated to compare wounded and nonwounded lesion development. Each fruit was inoculated with a 10 µL droplet containing conidia of *Monilinia fructicola* at a concentration of 2.5×10^4 spores per mL from 7 to 10 day old cultures maintained on V-8 juice agar. Controls included fruit treated with a droplet of water. Lesion diameter (mm) was recorded 3 days after inoculation and incubation of the peaches in the humidified containers at room temperature (22±1°C). Disease severity for each genotype was calculated as the product of the average lesion diameter X proportion of symptomatic fruit (disease incidence). The data were collated and statistically analyzed using Microsoft Excel and JMP software version 7.0 (The SAS Institute, Cary, NC).

Fruit color determinations. Fruit color determinations as a measure of peach maturity were made using two methods. The first method is the standard method we have used in the past, which utilizes a hand-

held spectrophotometer (Minolta) that assays peel color as a measure of maturity. Transmittance values for the visible spectrum (400-700 nm) were collected for each fruit and recorded. A second instrument, developed for rapid determination of flesh color in clingstone peaches, was provided by Dr. David Slaughter of the Department of Agricultural and Biological Engineering (Slaughter et al., 2006). This instrument, designed by Drs. Slaughter and Crisostos, relies on a nondestructive optical method based upon near infrared and visible interactance spectroscopy. The instrument shines a hollow cone of light onto the fruit to make a transmission measurement through a portion of the fruit (the light goes through both the skin and the flesh). In clingstone peaches the device predicts the flesh hue angle without cutting the fruit with a high degree of accuracy (R^2 value of 0.8). The device provides the entire transmission spectrum of light through the peach, and software selects five wavebands to use in predicting the hue angle. We took readings on each of five peaches per genotype at two sites on each peach prior to inoculation. From these measures, the hue angle was also calculated. In addition, color photographs were taken with a digital camera for each genotype evaluated.

Acknowledgments

The advice of David Slaughter (UC Davis Dept. of Bio and Ag Engineering) and his sharing of the light interactance equipment, as well as the technical assistance of Timothy Weerts, Emilie Lee, Rachel Giffin, Tatiana Roubtsova, and Mary Ann Thorpe in this study are gratefully acknowledged. Research supported by grants from the California Cling Peach Growers Advisory Board and the UC ANR Core Issues program.

Reference

D. C. Slaughter, C. H. Crisosto, J. K. Hasey, J. F. Thompson. 2006. Comparison of instrumental and manual inspection of clingstone peaches. Appl. Eng. Agric. 22: 883-889.

Appendix

Table A below contains a listing in order of most resistant to most susceptible to brown rot of the peach genotypes that were evaluated during 2008 in the nonwounded format for the new selections. Mean lesion diameters and standard deviations (SD), disease incidence (proportion of fruit infected), and disease severity (lesion diameter x incidence) for each genotype are presented. Harvest dates are indicated. Peaches were evaluated for resistance soon after harvest, according to the following schedule: group A, June 24; B, July 1; C, July 7; D, July 14; E, July 21; F, July 28; G, August 8; H, August 11; I, August 18; J, August 25; K, September 2; L, September 8; M, September 15.

Table B below contains a listing in order of most resistant to most susceptible to brown rot of the Pop-BR progeny populations, compiled over the 2007 and 2008 seasons. Only the progeny lines for which we have two years of data are presented. Key to analysis date codes same as in Table A.

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
Table A					
04,2-238	2-Jul	0.00	0.00	0.00	0.00
03,1-162	10-Jul	0.00	0.00	0.00	0.00
03,1-286	10-Jul	0.00	0.00	0.00	0.00
02,9-292	11-Jul	0.00	0.00	0.00	0.00
02,7-78	9-Jul	0.00	0.00	0.00	0.00
02,2/279	9-Jul	0.00	0.00	0.00	0.00
02,2-45	31-Jul	0.20	0.89	0.05	0.01
02,2-275	9-Jul	0.80	3.58	0.05	0.04
01,9-3	20-Aug	1.05	4.59	0.05	0.06
01,9-107	29-Aug	1.19	5.46	0.05	0.06
05, 18-221	23-Jun	0.50	1.41	0.13	0.06
03,1-287	10-Jul	0.83	2.89	0.08	0.07
04,2-131	22-Jul	0.88	2.47	0.13	0.11
02,8-76	23-Jul	0.86	2.27	0.14	0.12
02,11-184	10-Jul	1.00	2.83	0.13	0.13
02,7-103	29-Jul	1.33	4.00	0.11	0.15
04,2-218	24-Jul	1.14	3.02	0.14	0.16
02,11-139	10-Jul	1.00	2.16	0.20	0.20
02,8-48	22-Jul	1.86	4.91	0.14	0.27
04,1-159	22-Jul	1.40	3.13	0.20	0.28
02,2-96	21-Jul	2.00	5.29	0.14	0.29
02,9-286	10-Jul	3.10	9.80	0.10	0.31
05, 17-151	23-Jun	1.76	5.03	0.18	0.31
05,17-114	18-Aug	1.80	4.05	0.20	0.36
02,7-101	8-Aug	2.60	6.92	0.15	0.39
02, 6-89	23-Jun	2.22	4.41	0.22	0.49
02,2-829	10-Jul	2.25	4.50	0.25	0.56
02,9-162	10-Jul	1.83	2.86	0.33	0.61
01,9-188	8-Sep	2.47	4.03	0.26	0.65
03,1-293	10-Jul	2.63	4.87	0.25	0.66
01,9-156	12-Aug	2.45	4.37	0.27	0.67
02,7-79	10-Jul	3.13	7.55	0.22	0.68
02,9-291	10-Jul	3.70	9.78	0.20	0.74
01,9-185	27-Aug	3.00	6.54	0.25	0.75
01,9-109	8-Aug	3.52	6.98	0.24	0.84
03,1-289	10-Jul	2.80	6.03	0.30	0.84
04,5-166	10-Jul	3.00	6.03	0.29	0.86
F8,5-17	3-Sep	3.20	6.81	0.30	0.96
02,2-90	21-Jul	3.64	7.93	0.27	0.99
F8,5-159	3-Sep	4.00	7.43	0.25	1.00
05,17-39	18-Aug	3.40	6.52	0.30	1.02
04, 213	27-Jun	3.70	7.24	0.30	1.11
04,2-195	2-Jul	2.80	4.76	0.40	1.12
04,1-152	22-Jul	4.50	9.00	0.25	1.13

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
02,11-146	25-Jul	4.00	6.86	0.29	1.14
01,9-20	29-Aug	3.75	6.66	0.33	1.25
04, 2-216	27-Jun	4.50	8.89	0.28	1.25
05,17-139	18-Aug	3.45	4.74	0.36	1.25
01,9-42	8-Aug	3.20	4.94	0.40	1.28
02,2-68	7-Jul	4.30	7.12	0.30	1.29
01,9-47	27-Aug	2.60	3.97	0.50	1.30
Ross/PSA,6-11	6-Aug	4.40	6.28	0.30	1.32
02,7-105	23-Jul	5.09	8.84	0.27	1.39
D62-193/PG8-6+7	10-Jul	4.80	7.77	0.30	1.44
02,6-143	23-Jun	4.56	9.84	0.33	1.52
05,17-69	18-Aug	3.71	6.24	0.43	1.59
04,3-221	10-Jul	6.29	9.39	0.29	1.80
F8,5-156	3-Sep	4.25	5.63	0.50	2.13
02,7-73	14-Jul	4.38	7.54	0.50	2.19
02,9-289	10-Jul	6.89	10.62	0.33	2.30
02,2-287	10-Jul	5.80	10.08	0.40	2.32
02,2-303	9-Jul	7.33	11.64	0.33	2.44
Carson/ PG 1-20+21	10-Jul	6.11	8.96	0.44	2.72
04,4-32	28-Jul	6.90	11.26	0.40	2.76
04,2-208	2-Jul	7.00	9.75	0.43	3.00
F8,5-166	3-Sep	6.11	7.55	0.50	3.06
05,17-138	19-Aug	6.75	8.06	0.50	3.38
05,14-196	23-Jul	6.00	8.35	0.57	3.43
04,4-30	16-Jul	8.80	12.77	0.40	3.52
02,11-182	14-Jul	8.57	11.47	0.43	3.67
02,2-92		7.88	9.88	0.50	3.94
04,5-159	10-Jul	6.22	6.48	0.67	4.15
02,8-74	7-Jul	6.33	10.97	0.67	4.22
05,14-67	23-Jul	7.82	10.80	0.55	4.27
04,2-193	2-Jul	8.63	12.06	0.50	4.32
02,7-110	29-Jul	9.00	10.39	0.50	4.50
02,11-144	10-Jul	7.00	9.03	0.67	4.67
05,17-227	18-Aug	8.60	7.09	0.60	5.16
05,13-217	23-Jul	11.00	13.34	0.50	5.50
05,12-138	2-Jul	10.14	11.70	0.57	5.79
04,2-196	10-Jul	9.75	10.50	0.63	6.09
05,15-229	23-Jul	7.33	9.61	0.83	6.11
04,4-84	16-Jul	13.00	15.03	0.50	6.50
01,9-55	28-Jul	12.00	13.54	0.56	6.67
96,8-192/NSW 5-12+13	13-Aug	9.33	8.60	0.75	7.00
02,8-49	25-Jul	11.70	12.13	0.60	7.02
04,7-211	16-Jul	9.75	6.95	0.75	7.31
Ross/FSA, 6-11	6-Aug	11.22	9.76	0.67	7.48
04,2-267	10-Jul	9.00	6.45	0.83	7.50
04,1-125	22-Jul	10.38	8.96	0.75	7.79

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
Sherman 86-28A	1-Jul	10.20	6.81	0.80	8.16
04,4-23	28-Jul	12.50	9.95	0.67	8.33
Ross/FSA 6-11	6-Aug	12.53	10.39	0.67	8.35
F8,5-147	3-Sep	10.94	7.64	0.76	8.37
98,9-7/NSW 5-18+19	26-Aug	13.82	12.54	0.64	8.79
03,1-196	29-Jul	12.57	10.06	0.71	8.98
03,1-143	10-Jul	12.00	10.30	0.75	9.00
03,1-150	10-Jul	10.83	10.15	0.83	9.03
Carson PG1-20+21	16-Jul	10.57	9.38	0.86	9.06
02,2-260	14-Aug	12.00	9.47	0.76	9.18
Carson/PG-1-20+21	10-Jul	11.60	10.29	0.80	9.28
05,17-140	18-Aug	11.80	9.52	0.80	9.44
03,1-141	10-Jul	17.71	18.10	0.57	10.12
03,1-303	10-Jul	11.88	10.91	0.88	10.40
03,1-184	10-Jul	17.18	13.56	0.64	10.93
05,12-259	10-Jul	14.33	10.72	0.78	11.15
02,6-267	12-Aug	12.50	5.38	0.90	11.25
04,2-194	2-Jul	15.00	10.98	0.75	11.25
04, 2-253	27-Jun	17.33	15.36	0.67	11.55
05,13-230	6-Aug	16.29	14.16	0.71	11.64
04, 2-183	27-Jun	14.00	8.92	0.83	11.67
04, 212	27-Jun	15.57	10.67	0.86	13.35
03,1-169	2-Jul	15.57	12.01	0.86	13.35
02,11-201	28-Jul	19.50	13.10	0.75	14.63
04,3-175	16-Jul	14.75	6.24	1.00	14.75
9,20-C/PG1-29+27a	10-Jul	19.11	12.46	0.78	14.86
04,3-120	5-Aug	15.00	6.68	1.00	15.00
05,17-147	7-Aug	18.36	11.23	0.82	15.02
05,17-134	19-Aug	17.71	11.86	0.86	15.18
02,9-283	10-Jul	15.33	9.12	1.00	15.33
04,4-89	16-Jul	19.89	12.86	0.78	15.47
05,17/155	26-Aug	17.56	12.40	0.89	15.61
02,10-291	25-Jul	18.63	10.17	0.88	16.30
99,4-123/NSW 5-20+21	13-Aug	17.92	8.04	0.92	16.43
04,6-242	5-Aug	16.43	7.30	1.00	16.43
05,11-261	19-Aug	18.56	7.81	0.89	16.50
98,4-177/NSW 5-16+17	13-Aug	18.50	8.29	0.90	16.65
03,1-277	25-Jul	17.71	6.85	1.00	17.71
05,14-213	6-Aug	17.83	5.85	1.00	17.83
03,1-198	7-Jul	18.00	14.78	1.00	18.00
02,9-285	10-Jul	18.17	9.20	1.00	18.17
92,10-136/PG5-25+25a	30-Jul	19.83	8.10	0.92	18.18
02,11-215	12-Aug	18.67	5.22	1.00	18.67
Bolinha Q/PG 8-26+27	13-Aug	20.11	5.10	0.94	18.99
05,11-249	31-Jul	19.00	5.10	1.00	19.00
05,16-177	14-Aug	21.80	10.52	0.90	19.62

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
02,6-252	30-Jul	21.13	5.14	1.00	21.13
02,10-297	25-Jul	23.80	9.09	0.90	21.42
05,13-136	6-Aug	22.18	2.68	1.00	22.18
02,6-258	30-Jul	22.57	4.31	1.00	22.57
Carson	30-Jul	22.89	6.51	1.00	22.89
04,4-29	28-Jul	23.00	5.97	1.00	23.00
03,1-237	10-Jul	23.17	7.49	1.00	23.17
02,10-294	6-Aug	24.00	2.65	1.00	24.00
03,2-203	10-Jul	24.29	5.31	1.00	24.29
Carson/PG1-20+21	30-Jul	25.86	5.61	1.00	25.86
04,2-190	2-Jul	27.00	2.24	1.00	27.00
02,7-65	25-Jul	28.00	11.11	1.00	28.00
Carson	30-Jul	29.91	4.59	1.00	29.91
Carson/PG1-20+21	16-Jul	30.14	8.34	1.00	30.14

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity

Table B. Pop-BR populations

02,7-95	9-Jul	0.00	0.00	0.00	0.00
02,2-238	9-Jul	0.00	0.00	0.00	0.00
01,9-158	9-Jul	0.00	0.00	0.00	0.00
02,2-237	9-Jul	0.00	0.00	0.00	0.00
02,7-98	23-Jul	0.00	0.00	0.00	0.00
02,7-25	31-Jul	0.00	0.00	0.00	0.00
01,9-171	24-Jul	0.00	0.00	0.00	0.00
01,9-172	14-Aug	0.00	0.00	0.00	0.00
01,9-31	20-Aug	0.00	0.00	0.00	0.00
01,9-32	25-Jul	0.00	0.00	0.00	0.00
02,7-68	20-Aug	0.00	0.00	0.00	0.00
01,9-119	29-Aug	0.00	0.00	0.00	0.00
01,9-38	29-Aug	0.00	0.00	0.00	0.00
01,9-28	27-Aug	0.00	0.00	0.00	0.00
01,9-219	27-Aug	0.00	0.00	0.00	0.00
01,9-146	29-Aug	0.00	0.00	0.00	0.00
01,9-200	29-Aug	0.00	0.00	0.00	0.00
01,9-198	29-Aug	0.00	0.00	0.00	0.00
01,9-206	3-Sep	0.00	0.00	0.00	0.00
01,9-58	21-Aug	0.15	0.67	0.00	0.00
01,9-153	29-Aug	0.21	1.02	0.04	0.01
02,2-241	9-Jul	1.08	5.31	0.04	0.05
01,9-136	29-Aug	0.53	1.61	0.11	0.06
02,7-93	7-Jul	0.75	2.60	0.08	0.06
01,9-105	27-Aug	0.79	2.07	0.11	0.08
01,9-180	27-Aug	0.78	2.33	0.11	0.09
01,9-167	29-Aug	0.67	1.63	0.17	0.11
01,9-57	21-Aug	1.43	4.37	0.09	0.12
01,9-168	29-Aug	1.22	3.67	0.11	0.14
02,7-45	9-Jul	1.62	5.57	0.10	0.15
02,7-100	23-Jul	1.25	3.54	0.13	0.16
01,9-199	14-Aug	1.31	3.70	0.13	0.16
01,9-234	27-Aug	1.67	5.00	0.11	0.19
02,2-263	9-Jul	1.43	4.01	0.13	0.19
01,9-56	20-Aug	1.53	3.60	0.13	0.20
02,2-46	31-Jul	2.10	6.47	0.10	0.21
01,9-222	9-Jul	1.33	3.23	0.17	0.22
01,9-178	27-Aug	2.30	7.27	0.10	0.23
01,9-232	27-Aug	1.50	1.93	0.17	0.25
01,9-95	25-Aug	1.50	3.67	0.17	0.25
01,9-113	26-Aug	2.00	3.51	0.13	0.25
01,9-128	4-Sep	1.70	4.23	0.15	0.26
01,9-186	3-Sep	1.63	4.07	0.16	0.26
02,2-49	30-Jul	1.78	5.33	0.16	0.28
01,9-114	29-Aug	1.55	3.45	0.18	0.28
01,9-34	20-Aug	1.37	2.77	0.21	0.29
02,7-86	29-Jul	2.14	5.67	0.14	0.31

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
01,9-138	8-Sep	1.44	2.64	0.22	0.32
02,7-80	24-Jul	1.92	5.26	0.17	0.32
01,9-169	29-Aug	2.43	6.43	0.14	0.35
01,9-85	21-Aug	2.45	5.21	0.15	0.37
02,7-85	20-Aug	1.92	3.63	0.25	0.48
01,9-67	25-Aug	2.40	3.91	0.20	0.48
01,9-35	8-Aug	2.57	4.90	0.19	0.49
02,2-240	30-Jul	1.91	5.35	0.26	0.50
01,9-142	14-Aug	1.90	3.58	0.29	0.54
02,2-288	9-Jul	3.65	8.19	0.15	0.55
01,9-80	4-Sep	1.83	2.86	0.33	0.61
01,9-46	20-Aug	3.08	6.61	0.21	0.64
02,2-236	29-Aug	2.63	5.24	0.25	0.66
02,2-280	30-Jul	3.30	7.20	0.20	0.66
02,7-39	20-Aug	2.00	3.16	0.33	0.67
01,9-71	11-Aug	3.35	7.60	0.20	0.67
01,9-211	29-Aug	2.83	5.42	0.25	0.71
01,9-202	29-Aug	3.44	7.37	0.22	0.76
01,9-40	18-Aug	2.63	4.41	0.29	0.77
01,9-145	27-Aug	2.00	2.83	0.40	0.80
02,7-55	22-Jul	2.67	5.20	0.33	0.89
02,7-47	9-Jul	3.67	7.91	0.25	0.92
01,9-48	8-Aug	3.85	7.13	0.25	0.96
02,7-27	22-Jul	3.43	6.80	0.29	0.98
01,9-190	3-Sep	2.50	3.31	0.40	1.00
01,9-97	5-Sep	2.88	4.40	0.35	1.02
01,9-189	30-Jul	3.57	7.16	0.29	1.02
01,9-129	26-Aug	3.11	4.23	0.33	1.03
01,9-135	27-Aug	3.50	5.68	0.30	1.05
01,9-103	29-Jul	2.91	4.85	0.36	1.06
02,7-46	29-Jul	3.20	5.07	0.33	1.07
01,9-230	27-Aug	3.33	5.20	0.33	1.11
01,9-195	29-Aug	2.25	2.63	0.50	1.13
01,9-115	26-Aug	3.42	5.79	0.33	1.14
01,9-102	2-Sep	4.09	6.67	0.30	1.24
01,9-27	8-Aug	4.91	9.15	0.26	1.28
01,9-140	27-Aug	4.00	6.23	0.33	1.33
01,9-176	14-Aug	3.14	3.21	0.43	1.35
01,9-173	14-Aug	4.31	6.56	0.31	1.35
01,9-144	2-Sep	3.35	4.70	0.41	1.38
01,9-165	12-Aug	4.29	5.26	0.33	1.43
01,9-203	27-Aug	5.75	11.50	0.25	1.44
02,2-257	29-Aug	3.33	4.21	0.44	1.48
01,9-39	18-Aug	5.00	9.17	0.30	1.50
02,7-82	7-Jul	3.80	8.07	0.40	1.52
01,9-65	8-Aug	5.20	9.23	0.30	1.56
01,9-112	8-Aug	4.72	6.74	0.39	1.84
01,9-233	27-Aug	2.50	1.73	0.75	1.88
01,9-72	21-Jul	6.91	12.54	0.27	1.88

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
01,9-88	11-Aug	4.27	3.26	0.45	1.94
02,2-294	9-Jul	5.83	9.17	0.33	1.94
01,9-87	11-Aug	4.75	5.72	0.42	1.98
02,2-274	5-Aug	6.22	10.39	0.33	2.07
02,2-270	30-Jul	6.24	9.87	0.33	2.08
02,7-51	20-Aug	5.08	7.30	0.42	2.12
01,9-225	12-Aug	4.78	5.47	0.44	2.12
01,9-79	25-Aug	5.40	5.13	0.40	2.16
01,9-132	12-Aug	4.33	3.74	0.50	2.17
01,9-125	26-Aug	4.85	5.74	0.45	2.18
01,9-157	2-Sep	5.71	7.82	0.41	2.35
01,9-216	27-Aug	4.90	6.61	0.50	2.45
02,2-44	8-Aug	4.95	6.78	0.50	2.48
02,7-90	1-Aug	5.83	8.42	0.44	2.59
01,9-93	25-Aug	5.40	5.32	0.50	2.70
02,7-24	30-Jul	5.50	9.05	0.50	2.75
01,9-82	21-Aug	6.20	7.58	0.45	2.79
01,9-44	9-Jul	6.33	7.88	0.44	2.81
01,9-101	2-Sep	5.42	5.33	0.53	2.85
01,9-150	12-Aug	6.88	9.86	0.42	2.87
01,9-126	8-Aug	6.56	9.55	0.44	2.87
01,9-204	27-Aug	4.80	5.22	0.60	2.88
01,9-271	27-Aug	6.00	7.66	0.50	3.00
02,7-15	14-Aug	5.71	5.64	0.53	3.01
02,2-287	10-Jul	5.58	7.64	0.56	3.10
02,2-235	8-Aug	8.18	11.30	0.41	3.35
02,2-273	7-Aug	7.95	10.54	0.42	3.35
02,7-35	26-Aug	5.38	5.01	0.63	3.36
02,7-54	29-Jul	8.70	13.46	0.40	3.48
02,7-34	4-Aug	8.65	11.94	0.41	3.56
02,2-249	29-Aug	6.58	7.23	0.54	3.56
02,2-282	7-Aug	8.05	10.73	0.45	3.62
01,9-29	11-Aug	6.53	7.19	0.58	3.78
02,7-88	28-Jul	7.83	10.93	0.50	3.92
02,2-47	4-Aug	7.88	10.81	0.50	3.94
02,7-113	20-Aug	6.65	6.32	0.60	3.99
02,2-283	7-Aug	6.57	6.79	0.61	4.00
02,2-301	5-Aug	8.43	9.78	0.48	4.01
01,9-53	18-Aug	6.83	6.74	0.61	4.17
01,9-81	11-Aug	8.00	9.96	0.53	4.21
02,7-106	20-Aug	7.93	9.80	0.53	4.23
01,9-55	30-Jul	13.17	20.40	0.33	4.39
02,2-45	4-Aug	8.53	10.23	0.53	4.49
01,9-268	26-Aug	9.00	9.38	0.50	4.50
02,7-52	29-Jul	10.86	13.86	0.43	4.65
01,9-61	4-Sep	7.20	6.32	0.65	4.68
02,7-72	10-Jul	10.56	12.91	0.44	4.69
02,2-239	7-Aug	8.26	10.00	0.58	4.78
01,9-99	25-Aug	9.18	10.11	0.55	5.01

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Severity
01,9-24	11-Aug	8.45	8.02	0.60	5.07
02,7-21	30-Jul	7.67	7.28	0.67	5.11
01,9-127	26-Aug	8.60	8.29	0.60	5.16
02,2-258	12-Aug	9.55	9.62	0.60	5.73
01,9-104	24-Jul	8.50	7.38	0.70	5.95
02,2-248	10-Jul	11.04	11.48	0.57	6.24
02,2-247	2-Sep	9.36	8.23	0.73	6.81
02,2-255	5-Aug	11.55	11.69	0.59	6.83
01,9-122	26-Aug	10.90	10.62	0.67	7.27
01,9-89	25-Aug	9.10	7.61	0.80	7.28
01,9-108	26-Aug	8.71	5.53	0.86	7.47
02,7-25	27-Jun	8.88	8.66	0.88	7.77
02,2-244	12-Aug	12.71	11.15	0.62	7.87
02,7-96	29-Aug	11.50	10.05	0.70	8.05
01,9-77	25-Aug	9.79	5.45	0.84	8.24
02,2-261	12-Aug	10.68	7.10	0.84	8.99
02,2-290	12-Aug	12.26	8.98	0.74	9.03
01,9-270	26-Aug	9.25	3.45	1.00	9.25
01,9-231	27-Aug	12.20	7.85	0.80	9.76
01,9-84	24-Aug	12.40	8.34	0.80	9.92
02,2-271	12-Aug	12.95	8.75	0.77	10.01
02,2-304	31-Jul	13.09	10.98	0.77	10.12
02,7-38	26-Aug	13.00	12.39	0.80	10.40
02,7-18	30-Jul	14.25	10.21	0.75	10.69
02,7-70	26-Aug	12.50	5.26	0.88	10.94
02,7-61	7-Aug	14.20	8.64	0.80	11.36
01,9-63	25-Aug	11.67	12.50	1.00	11.67
02,7-23	22-Jul	11.71	5.79	1.00	11.71
01,9-205	8-Aug	14.80	10.66	0.80	11.84
02,2-259	12-Aug	13.96	7.19	0.88	12.22
02,7-76	8-Aug	15.30	11.96	0.80	12.24
02,2-234	7-Aug	16.70	13.94	0.75	12.53
01,9-141	12-Aug	12.80	3.03	1.00	12.80
01,9-94	26-Aug	12.90	4.79	1.00	12.90
01,9-117	26-Aug	13.75	6.34	1.00	13.75
02,7-30	26-Aug	15.11	7.29	1.00	15.11
02,2-302	7-Aug	16.79	10.79	0.91	15.26
01,9-193	8-Aug	21.20	13.70	0.80	16.96
01,9-266	26-Aug	17.57	10.56	1.00	17.57
01,9-130	26-Aug	22.09	8.67	0.95	21.09
02,2-266	12-Aug	24.65	6.46	1.00	24.65
02,7-78	23-Jul	27.00	10.15	1.00	27.00