California Cling Peach Advisory Board

2009 Annual Report

Project Title:	Assessment of Brown Rot Resistance in Advanced Experimental Selections of Peach
Project Leaders:	Richard Bostock & Tom Gradziel
Cooperating Personnel:	Carlos Crisosto, Riaz Ahmad
Location:	Departments of Plant Pathology and of Plant Sciences, University of California at Davis

Summary:

Over 292 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 provided a third and final year of evaluation of the progeny from two crosses between susceptible and previously determined resistant genotypes, designated Pop-BR1 and Pop-BR2. This aspect of the project has been part of a larger University of California ANR Core Issues grant (Crisosto, et al, principal investigators) to explore the feasibility of marker-assisted selection for brown rot resistance in cling peach breeding. In previous analyses, we identified polymorphisms that may identify genetic markers for linkage analysis. Our goal is to map QTLs and identify candidate genes associated with fruit resistance to brown rot. These analyses are ongoing and will be completed in 2010. In addition to the Pop-BR populations, we also evaluated 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 selected in previous years 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.

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 the cling peach breeding program and for subsequent multiplication and distribution in test orchards. A secondary objective is to identify genetic markers for brown rot resistance that can be used to facilitate the rapid selection of material for incorporation and to monitor for the presence of those markers as these materials progress through breeding program.

Overview of 2009 Research

Over 292 genotypes were evaluated for the period beginning 6 July to 21 September 2009. 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. About half of the genotypes (142) evaluated this year are new or are genotypes that have been carried forward from previous seasons for additional assessment.

A major part of our evaluations involved fruit from 150 progeny from the two populations that we constructed for segregation of molecular markers for brown rot resistance and other quality factors. The 2009 season was the **third year** of evaluation of these progeny, which were generated from two crosses between brown rot susceptible and previously determined 'resistant' genotypes from the breeding program ('Dr. Davis' × 'F8,1-42' and 'Loadel' × 'UCD96,4-55' aka "Pop-BR1" and Pop-BR2", respectively). Two inoculation formats were used to evaluate the PopBR populations – 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 then applying the inoculum in a 10 µl droplet to the wound with a pipette. 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 which has a strong epidermal resistance) 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 the new genotypes evaluated during 2009 in the non-wounded treatment, in order from the most resistant (lowest disease severity) to the most susceptible (highest disease severity). Approximately 49% (142) 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). These data are also presented in a more detailed fashion in **Table 1 of Appendix B**. Of these, 52, or about 37%, 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. Clearly, wounding compromises any epidermal/cuticular resistance and facilitates pathogen entry and colonization. However, a few genotypes display a relatively good resistance even in the wound inoculation format. **Figure 3** shows a highly resistant genotype and a susceptible genotype 72 h after inoculation in the nonwounded format.

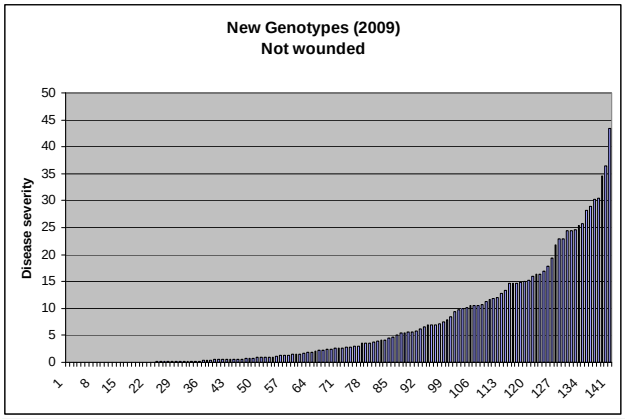

Fig. 1. Disease severity values of new genotypes evaluated in 2009, from the most resistant to most susceptible. Rankings for 142 individual genotypes are presented. Not wounded inoculation format.

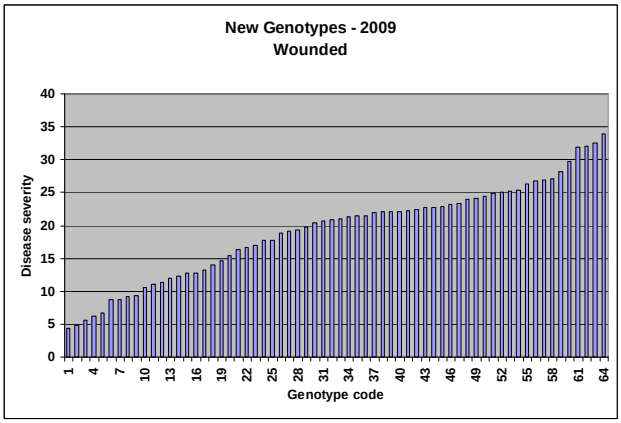

Fig. 2. Disease severity values of new genotypes evaluated in 2009, from the most resistant to most susceptible. Wound inoculation format.

Fig. 3. Representative reactions of highly resistant (left, 02,241) and susceptible (right, NSW5-26) genotypes. Photographs taken 72 hours after inoculation. Brown rot lesions (discolored fruit tissue) radiate out from the point of inoculation and are evident in the fruit pictured on the right. Nonwounded format.

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 2009 we evaluated 150 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 'Loadel' to 'UCD96,4-55' a resistant experimental line derived from cv. 'Bolinha'. The results for these mapping populations for the nonwounded treatment are displayed in **Fig. 4** (composite of Pop-BR1, 2007-2009 data) and **Fig. 5** (composite of Pop-BR2, 2007-2009 data). **Figures 6** and **7** show the results for Pop-BR1 and Pop-BR2, respectively, using the wound inoculation format.

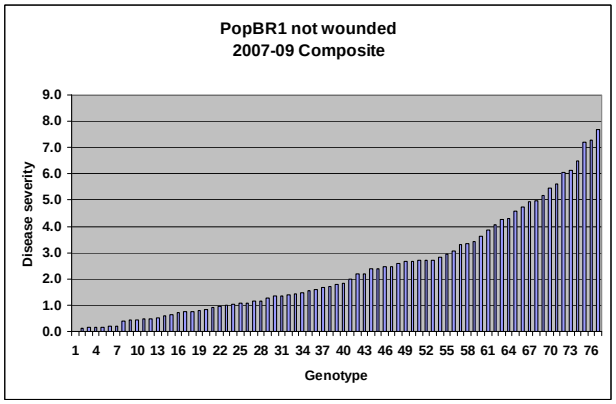

Fig. 4. Range of disease severity values for the Pop-BR1 mapping population, average of 2007, 2008 and 2009 data. Not wounded inoculation format.

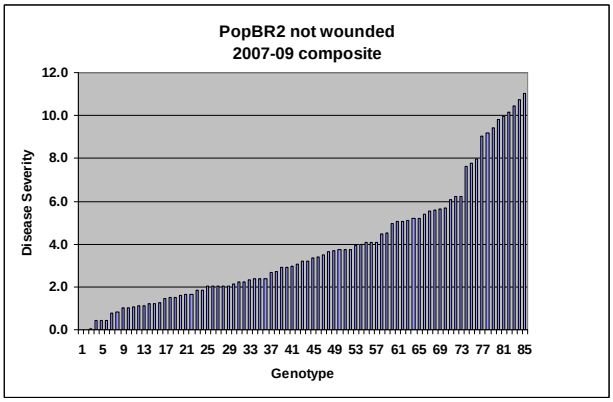

Fig. 5. Range of disease severity values for the Pop-BR2 mapping population, average of 2007, 2008 and 2009 data. Not wounded inoculation format.

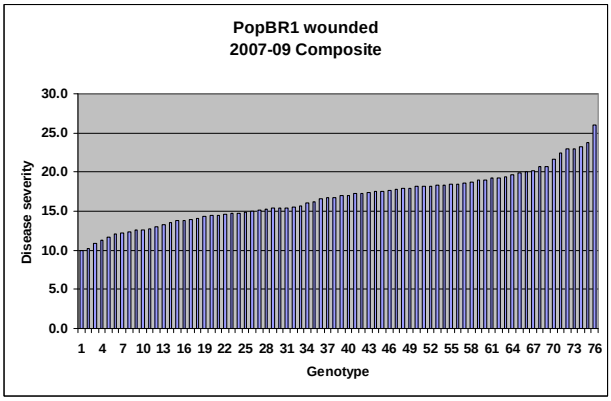

Fig. 6. Range of disease severity values for the Pop-BR1 mapping population, average of 2007, 2008 and 2009 data. Wounded inoculation format.

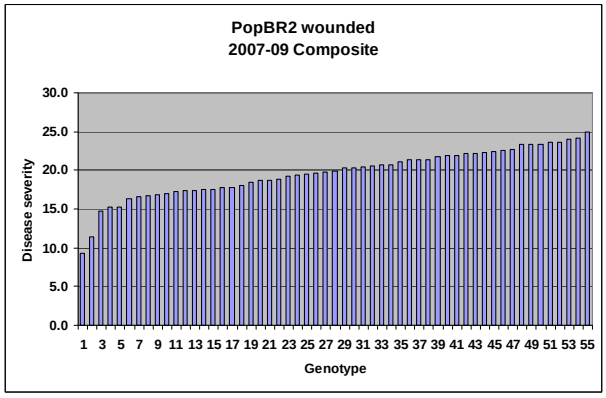

Fig. 7. Range of disease severity values for the Pop-BR2 mapping population, average of 2007, 2008 and 2009 data. Wounded inoculation format.

The results also are consistent with quantitative (polygenic) inheritance of the fruit resistance phenotype. More detailed numerical data are presented in **Table 2 of Appendix B**.

Pop-BR genotypes were identified as showing consistently either high resistance or high susceptibility over the three years of sampling. These results are summarized in **Appendix A** to this report. Dr. Riaz Ahmad is continuing the analysis of DNA polymorphisms begun by Dr. Eben Ogundiwin, with a focus on the genotypes that associate with brown rot resistance in these progeny. This work is in progress and we should have a clear assessment of the strength of the molecular data in late 2010. We will provide a summary of the results of this work in the 2010 mid-year and annual reports.

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 10th year of evaluation. This genotype rated highly or moderately well for brown rot resistance in our laboratory evaluations, and has been planted in a variety of locations for regional field trials. This genotype has additional fruit quality attributes that make it attractive for continued testing and development. These trees will be coming into fruit-bearing maturity during the next year. There are other genotypes particularly those with peach x almond heritage that show promise, and we will continue to move those through the program. The immediate goal for the next year will be to continue with the molecular marker analysis of resistance to brown rot. We will know this year whether our populations that appear to segregate for brown rot resistance reveal genetic markers that associate with the resistance phenotype. 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. The three years of evaluation of these populations conducted from 2007-2009 is critical to the reliability of the QTL analyses because these data will help us account for non-genetic variation from inherent experimental variation 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, 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.

Unblemished fruit of each genotype were placed in humidified plastic containers with fruit liners. 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. Approximately 20-40 fruit per genotype were prepared, with the number varying depending upon the availability of fruit for that genotype and whether both inoculation formats were to be used. Each fruit was inoculated with a 10 μL droplet containing conidia of *Monilinia fructicola* at a concentration of 2.5 x 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 a standard method we have used in the past, which utilizes a hand-held spectrophotometer (Minolta) that assays peel color as a measure of maturity. In addition, color photographs were taken with a digital camera for each genotype evaluated.

Acknowledgments

The technical assistance of Eva Gutierrez, Alex De Beaumont-Felt, Emilie Trinh, Tatiana Roubtsova, Mary Ann Thorpe, and field assistants to the Gradziel program are gratefully acknowledged. Research supported by grants from the California Cling Peach Growers Advisory Board and the UC ANR Core Issues program.

References

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Ebenezer Ogundiwin, Richard Bostock, Tom Gradziel, Themis Michailides, Mohammad Yaghmour, Dan Parfitt, and Carlos Crisosto (2008). Towards molecular genetic analysis of resistance to brown rot and sour rot in *Prunus persica*. Plant & Animal Genome Conference XVI, San Diego, 12-16 January 2008.

Lee M-H, Chiu C-M**,** Roubtsova T, Chou C-M, and Bostock RM. (2010) Overexpression of a redox-regulated cutinase gene *MfCUT1* increases virulence of the brown rot pathogen *Monilinia fructicola* on *Prunus spp.* Molecular Plant Microbe Interactions 23:176-186

Appendix A – summary of Pop-BR populations

Appendix B

Table 1 below contains a listing in order of most resistant to most susceptible to brown rot of the peach genotypes that were evaluated during 2009 in the nonwounded format for the new and carry forward 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, July 6; B, July 13; C, July 20; D, July 27; E, Aug 3; F, Aug 10; G, Aug 17; H, Aug 24; I, Aug 31; J, Sept 7; K, Sept 14; L, Sept 21.

Table 2 below contains a listing in order of most resistant to most susceptible to brown rot of the Pop-BR progeny populations, compiled over three seasons (2007-2009). Only the progeny lines for which we have three years of data are presented, and only the nonwounded inoculation results are presented. The Pop-BR1 lines have "..01,.." and the Pop-BR2 lines have "..02,…" in their genotype designations. In the table, the ordering from resistant to susceptible starts with Pop-BR1 and then begins anew with the Pop-BR2 lines.

Appendix A
Assessment of PopBR progeny – 3 seasons composite (2007-2009)

PopBR1 – genotypes with consistent, relatively **good resistance** (listed in order of rank) over 3 seasons

Not wounded:

Tr010 01,9-38
Tr127 01,9-234
Tr009 01,9-35
Tr002 01,9-27
Tr093 01,9-173
Tr094 01,9-176
Tr039 01,9-88
Tr081 01,9-150

Wounded:

Tr018 01,9-53
Tr093 01,9-173
Tr122 01,9-225

PopBR1 genotypes w/ potentially good unwounded and wound resistance:
Tr018 01,9-53

PopBR1 genotypes **most susceptible**

Not wounded:

Tr078 01,9-144
Tr126 01,9-233
Tr123 01,9-230
Tr131 01,9-270

Wounded:

Tr123 01,9-230
Tr047 01,9-99

PopBR2 - genotypes with consistent, relatively **good resistance** (listed in order of rank) over 3 seasons

Not wounded:

Tr073 02,7-68
Tr009 02,2-237
Tr034 02,2-280
Tr031 02,2-274
Tr010 02,2-238
Tr058 02,7-39
Tr001 02,2-44
Tr002 02,2-45

Wounded:

Tr066 02,7-54
Tr058 02,7-39
Tr028 02,2-270
Tr049 02,7-24

PopBR2 genotypes w/ potentially good unwounded and wound resistance:

Tr049 02,7-24
Tr028 02,2-270

PopBR2 genotypes **most susceptible**

Not wounded:

Tr054 02,7-30
Tr046 02,7-18
Tr101 02,7-113
Tr043 02,2-302
Tr070 02,7-61

Wounded:

Tr014 02,2-244
Tr077 02,7-25
Tr047 02,7-21
Tr070 02,7-61

Appendix B - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
W 15-43	3-Jul	0.0	0.0	0.0	0.00
02,2-241	9-Jul	0.0	0.0	0.0	0.00
02,2-289	9-Jul	0.0	0.0	0.0	0.00
W15-16	19-Jul	0.0	0.0	0.0	0.00
W15-31	19-Jul	0.0	0.0	0.0	0.00
W14-75	19-Jul	0.0	0.0	0.0	0.00
W15-57	19-Jul	0.0	0.0	0.0	0.00
05,10-100	26-Jul	0.0	0.0	0.0	0.00
05,18-168	2-Aug	0.0	0.0	0.0	0.00
01,9-42	6-Aug	0.0	0.0	0.0	0.00
01,9-109	13-Aug	0.0	0.0	0.0	0.00
01,9-195	20-Aug	0.0	0.0	0.0	0.00
W15-47	19-Jul	0.3	1.1	0.1	0.01
01,9-194	23-Jul	0.4	1.6	0.1	0.02
01,9-158	9-Jul	0.4	1.7	0.1	0.02
NSW1-31	26-Jul	0.4	1.8	0.1	0.02
01,9-156	13-Aug	0.5	2.1	0.1	0.02
02,2-268	9-Jul	0.6	2.7	0.1	0.03
01, 9-57	20-Aug	0.7	3.1	0.1	0.04
W15-50	12-Jul	1.0	4.5	0.1	0.05
NSW3-25	26-Jul	0.6	1.8	0.1	0.06
01, 9-77	27-Aug	1.4	6.3	0.1	0.07
W14-23	19-Jul	0.7	2.2	0.1	0.08
W14-54	19-Jul	1.0	3.3	0.1	0.10
01,9-96	27-Aug	1.0	2.9	0.1	0.10
05,18-221	3-Jul	1.4	4.2	0.1	0.14
02,2-260	6-Aug	1.4	4.2	0.1	0.14
05,16-223	19-Jul	1.6	5.0	0.1	0.16
01, 9-31	20-Aug	1.6	4.8	0.1	0.16
W 15-1	3-Jul	1.2	2.8	0.2	0.17
NSW4-2	26-Jul	1.8	5.9	0.1	0.18
02,2-279	9-Jul	1.3	3.4	0.2	0.19
02,2-245	6-Aug	1.8	5.2	0.1	0.20
05,19-206	19-Jul	1.1	2.4	0.2	0.22
02,7-110	6-Aug	1.7	4.3	0.2	0.26
02,7-101	6-Aug	1.5	2.7	0.3	0.38
01,9-107	3-Sep	1.6	2.7	0.3	0.4
02,7-64	30-Jul	3.0	7.9	0.2	0.45
01,9-162	13-Aug	2.7	6.0	0.2	0.53
NSW2-10	26-Jul	2.0	3.2	0.3	0.59
02,7-29	16-Jul	2.4	5.1	0.3	0.59
02,2-275	9-Jul	2.4	5.2	0.3	0.60
W 15-22	3-Jul	3.1	6.7	0.2	0.61
01,9-215	30-Jul	3.1	7.2	0.2	0.62
01, 9-46	20-Aug	2.5	5.2	0.3	0.63
01, 9-67	27-Aug	2.2	3.9	0.3	0.64
NSW2-32	26-Jul	2.4	4.9	0.3	0.66
06,3-183	12-Jul	2.9	6.0	0.3	0.71
NSW4-20	26-Jul	3.1	5.5	0.3	0.82

Appendix B - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
NSW2-14	26-Jul	2.9	4.9	0.3	0.86
W 15-10	3-Jul	3.6	7.0	0.2	0.86
Carson/PG 1-20+21	8-Jul	2.7	4.5	0.4	0.95
NSW2-19	26-Jul	2.7	4.3	0.4	0.95
05,1-138	26-Jul	3.0	4.8	0.3	1.00
NSW4-7	26-Jul	2.8	4.2	0.4	1.10
Carson SP/PG 1-20+21	19-Jul	3.6	5.4	0.4	1.24
W15-1E	19-Jul	1.6	3.9	0.8	1.36
PP Ross PG 1-2+3	11-Aug	3.3	4.4	0.4	1.37
NSW1-26	26-Jul	3.5	4.6	0.4	1.46
Carson/PG 1-20+21	20-Jul	3.4	4.1	0.5	1.51
NSW5-6	26-Jul	4.2	6.9	0.4	1.55
05,4-237	26-Jul	4.0	5.7	0.4	1.65
W4-60	12-Jul	4.5	6.9	0.4	1.80
02,7-105	30-Jul	7.5	14.2	0.3	1.88
02,2-268	16-Jul	5.9	8.9	0.4	2.05
W 13-27	3-Jul	5.7	9.5	0.4	2.28
NSW1-36	26-Jul	3.9	3.6	0.6	2.31
02,7-35	27-Aug	5.3	6.7	0.5	2.38
02,7-75	13-Aug	5.0	6.0	0.5	2.50
Sherman/PG 1-26+26a	1-Jul	5.4	6.6	0.5	2.55
01, 9-28	20-Aug	7.6	13.5	0.4	2.66
NSW4-41	19-Jul	6.0	8.0	0.4	2.67
W14-17	19-Jul	6.3	8.4	0.5	2.84
D,62-193/PG 8-6+7	1-Jul	6.5	8.2	0.5	2.91
05,26-233	12-Jul	6.6	10.0	0.5	3.10
W 11-19	3-Jul	7.2	11.9	0.4	3.10
05,17-151	3-Jul	7.1	8.2	0.5	3.53
01,9-86	27-Aug	8.9	10.1	0.4	3.54
01,9-185	13-Aug	8.1	10.3	0.5	3.65
02,7-60	6-Aug	6.3	6.3	0.6	3.70
NSW2-17	19-Jul	7.3	8.6	0.6	4.02
05,20-116	12-Jul	7.4	8.8	0.6	4.17
05,17-136	19-Jul	6.5	6.2	0.7	4.19
W 14-42	3-Jul	8.1	9.1	0.6	4.54
05,27-130	12-Jul	8.5	10.1	0.6	4.65
NSW2-36	19-Jul	8.6	8.5	0.6	5.16
05,19-253	12-Jul	7.8	6.9	0.7	5.36
01, 9-40	20-Aug	11.1	13.5	0.5	5.53
02,7-28	16-Jul	8.9	8.4	0.6	5.55
W12-75	12-Jul	12.0	11.6	0.5	5.64
02,7-92	16-Jul	9.4	11.4	0.6	5.74
01, 9-68	27-Aug	11.1	12.2	0.6	6.14
W 12-50	3-Jul	10.9	11.0	0.6	6.54
01, 9-79	27-Aug	11.6	11.4	0.6	6.98
W 11-23	3-Jul	8.7	7.5	0.8	7.04
01, 9-58	20-Aug	12.8	12.3	0.6	7.04
W15-55	12-Jul	9.0	5.8	0.8	7.16
PP Ross PG 1-2+3	9-Aug	10.1	7.9	0.8	7.55

Appendix B - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
02,7-103	6-Aug	10.7	7.1	0.8	7.99
W 9-27	3-Jul	14.2	15.5	0.6	8.52
NSW1-40	19-Jul	11.2	7.9	0.8	9.35
NSW3-38	19-Jul	14.0	10.9	0.7	9.80
01,9-47	27-Aug	6.2	8.2	0.5	9.90
07,19-15	2-Aug	17.0	14.9	0.6	10.20
NSW5-7	19-Jul	14.0	9.9	0.8	10.50
NSW5-1	19-Jul	13.2	8.1	0.8	10.52
07,20-117	2-Aug	15.8	12.4	0.7	10.55
01,9-208	27-Aug	14.4	9.3	0.8	10.78
01,7-96	27-Aug	17.4	10.7	0.7	11.34
W15-28	12-Jul	12.9	8.5	0.9	11.57
NSW3-30	2-Aug	17.8	13.9	0.7	11.85
01, 9-34	20-Aug	15.1	10.5	0.8	12.04
NSW5-26	19-Jul	18.2	11.7	0.7	12.72
01,7-85	27-Aug	19.1	8.2	0.7	13.35
PP Carson PG	6-Aug	16.3	8.4	0.9	14.63
01,9-30	27-Aug	8.4	7.0	0.8	14.66
01, 9-93	20-Aug	16.4	9.2	0.9	14.72
01, 9-180	20-Aug	18.6	10.5	0.8	14.90
NSW4-37	2-Aug	18.7	11.5	0.8	14.94
NSW3-2	2-Aug	19.2	12.1	0.8	15.17
01, 9-127	20-Aug	20.1	13.2	0.8	16.04
01,9-188	27-Aug	19.2	9.8	0.9	16.28
01,9-232	27-Aug	20.5	13.6	0.8	16.40
PP Ross PG 1-2+3	5-Aug	17.9	3.2	0.9	16.83
01, 9-63	20-Aug	19.9	10.3	0.9	17.87
PP Ross PG 1-2+3	11-Aug	21.5	9.7	0.9	19.38
PP Ross PG 1-2+3	11-Aug	21.9	3.7	1.0	21.85
W 13-16	3-Jul	22.9	19.8	1.0	22.86
01, 9-169	27-Aug	25.6	11.8	0.9	23.00
01,7-80	27-Aug	27.1	7.8	0.9	24.35
02, 7-51	20-Aug	27.2	12.2	0.9	24.48
02, 7-15	20-Aug	35.1	7.3	0.7	24.55
02, 2-257	27-Aug	27.4	10.5	0.9	25.41
01, 9-178	27-Aug	27.2	12.2	1.0	25.84
01, 9-266	27-Aug	30.0	9.8	0.9	28.13
Carson/PG 1-20+21	2-Aug	30.5	9.9	0.9	28.92
01, 9-32	20-Aug	30.3	7.1	1.0	30.25
01, 9-186	27-Aug	32.1	9.6	1.0	30.45
PP Ross PG 1-2+3	5-Aug	34.7	5.6	1.0	34.65
01, 9-220	27-Aug	36.5	8.7	1.0	36.50
01, 9-216	20-Aug	46.2	85.6	0.9	43.41

Appendix B - Table 2. Pop-BR populations

Genotype	Disease Severity	SE (n=3)
Pop-BR1		
Tr010 01,9-38	0.00	0.00
Tr127 01,9-234	0.12	0.06
Tr058 01,9-114	0.16	0.08
Tr061 01,9-119	0.16	0.16
Tr009 01,9-35	0.16	0.16
Tr052 01,9-105	0.21	0.13
Tr074 01,9-138	0.21	0.11
Tr119 01,9-219	0.41	0.41
Tr002 01,9-27	0.43	0.43
Tr093 01,9-173	0.45	0.45
Tr094 01,9-176	0.46	0.44
Tr087 01,9-165	0.49	0.47
Tr025 01,9-65	0.53	0.52
Tr020 01,9-56	0.62	0.30
Tr039 01,9-88	0.65	0.65
Tr071 01,9-132	0.73	0.72
Tr036 01,9-85	0.76	0.42
Tr045 01,9-97	0.77	0.39
Tr028 01,9-72	0.81	0.56
Tr067 01,9-128	0.83	0.29
Tr115 01,9-211	0.93	0.32
Tr081 01,9-150	0.96	0.96
Tr048 01,9-101	1.01	0.92
Tr132 01,9-271	1.02	0.99
Tr049 01,9-102	1.08	0.15
Tr038 01,9-87	1.09	0.58
Tr033 01,9-82	1.16	0.84
Tr043 01,9-95	1.16	1.04
Tr118 01,9-216	1.28	0.71
Tr004 01,9-29	1.35	1.22
Tr108 01,9-200	1.37	1.12
Tr018 01,9-53	1.39	1.39
Tr109 01,9-202	1.42	0.51
Tr121 01,9-222	1.49	1.10
Tr082 01,9-153	1.56	1.18
Tr023 01,9-61	1.57	1.55
Tr001 01,9-24	1.69	1.69
Tr057 01,9-113	1.72	1.60
Tr084 01,9-157	1.81	0.93
Tr065 01,9-126	1.83	0.78
Tr051 01,9-104	2.00	1.98
Tr113 01,9-206	2.20	2.07
Tr080 01,9-146	2.21	1.44
Tr047 01,9-99	2.38	1.43
Tr069 01,9-129	2.40	1.91
Tr040 01,9-89	2.47	2.41
Tr062 01,9-122	2.49	2.39
Tr073 01,9-136	2.59	1.84

Appendix B - Table 2. Pop-BR populations

Genotype	Disease Severity	SE (n=3)
Tr054 01,9-108	2.66	2.41
Tr059 01,9-115	2.66	2.05
Tr122 01,9-225	2.70	1.75
Tr072 01,9-135	2.70	2.01
Tr102 01,9-190	2.70	2.19
Tr014 01,9-44	2.85	1.65
Tr079 01,9-145	2.96	2.44
Tr050 01,9-103	3.05	2.52
Tr034 01,9-84	3.31	3.31
Tr101 01,9-189	3.36	1.54
Tr124 01,9-231	3.42	3.17
Tr031 01,9-80	3.64	3.30
Tr078 01,9-144	3.88	2.12
Tr130 01,9-268	4.08	2.22
Tr077 01,9-142	4.28	3.62
Tr042 01,9-94	4.31	4.30
Tr075 01,9-140	4.59	3.94
Tr060 01,9-117	4.75	4.50
Tr126 01,9-233	4.95	3.79
Tr088 01,9-167	4.97	4.82
Tr064 01,9-125	5.17	4.11
Tr123 01,9-230	5.45	4.18
Tr112 01,9-205	5.61	3.23
Tr105 01,9-198	6.07	5.06
Tr103 01,9-193	6.12	5.42
Tr131 01,9-270	6.51	3.08
Tr089 01,9-168	7.19	5.53
Tr070 01,9-130	7.30	6.90
Tr106 01,9-199	7.68	7.39
Pop-BR2		
Tr073 02,7-68	0.43	0.40
Tr009 02,2-237	0.80	0.80
Tr034 02,2-280	0.81	0.52
Tr031 02,2-274	1.00	0.60
Tr010 02,2-238	1.03	1.02
Tr058 02,7-39	1.13	0.80
Tr001 02,2-44	1.13	0.72
Tr003 02,2-46	1.48	1.36
Tr002 02,2-45	1.51	1.49
Tr060 02,7-46	1.51	0.60
Tr055 02,7-34	1.63	1.02
Tr025 02,2-263	1.82	1.69
Tr036 02,2-283	1.83	1.17
Tr067 02,7-55	2.03	1.25
Tr012 02,2-240	2.03	1.79
Tr005 02,2-49	2.04	1.17
Tr011 02,2-239	2.12	1.41
Tr075 02,7-72	2.22	1.24
Tr041 02,2-294	2.36	1.29

Appendix B - Table 2. Pop-BR populations

Genotype	Disease Severity	SE (n=3)
Tr082 02,7-82	2.37	1.67
Tr035 02,2-282	2.40	1.19
Tr049 02,7-24	2.69	1.52
Tr021 02,2-258	2.74	1.65
Tr014 02,2-244	2.90	2.50
Tr037 02,2-287	3.07	1.52
Tr040 02,2-290	3.18	2.93
Tr013 02,2-241	3.20	2.51
Tr093 02,7-100	3.33	1.80
Tr038 02,2-288	3.51	3.03
Tr024 02,2-261	3.68	2.72
Tr004 02,2-47	3.72	2.08
Tr029 02,2-271	3.74	3.15
Tr059 02,7-45	3.91	3.57
Tr077 02,7-25	3.97	2.00
Tr008 02,2-236	4.08	2.11
Tr085 02,7-88	4.09	1.02
Tr078 02,7-76	4.49	3.88
Tr007 02,2-235	4.52	2.98
Tr066 02,7-54	4.93	2.81
Tr018 02,2-249	5.05	1.87
Tr086 02,7-90	5.05	1.95
Tr006 02,2-234	5.09	3.80
Tr084 02,7-86	5.21	2.51
Tr016 02,2-247	5.22	0.84
Tr057 02,7-38	5.37	2.86
Tr047 02,7-21	6.09	3.60
Tr023 02,2-259	6.21	3.04
Tr074 02,7-70	7.95	3.78
Tr097 02,7-106	9.05	6.05
Tr026 02,2-266	9.19	7.77
Tr054 02,7-30	9.43	3.60
Tr046 02,7-18	9.84	3.69
Tr079 02,7-78	9.97	8.54
Tr101 02,7-113	10.16	6.44
Tr043 02,2-302	10.73	2.28
Tr070 02,7-61	11.04	4.41