California Cling Peach Advisory Board

2010 Final Report

Project Title:	Assessment of Brown Rot Resistance in Advanced Experimental Selections of Peach
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Summary:

Approximately 182 cling peach 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 determinations a disease severity value for each genotype was calculated. Fruit color, an indicator of quality and maturity, also was estimated by color image analysis. We have carried forward and evaluated material from previous years, including some of the advanced lines with heritage from peach x almond hybrids, the brown rot resistant cv. Bolinha, and USDA lines. Lines with fruit that mature extra early or extra late have been targeted for selection, since these are often periods of high disease vulnerability. A number of promising lines have emerged from the program. These items are now in advanced regional grower trials as they also have good horticultural/production qualities, and are planted under the designation Ultra-Early#1 (D62-193) and the Extra-Lates #4, 5, 6 & 7 (the very late maturing lines with peach-almond heritage). In a student research project supported by the NSF during the summer, two bioreporter strains (epiphytic bacteria that have been engineered to express a reporter gene in the presence of an appropriate chemical stimulus) were used as sensors to visualize changes in the production of fructose sugar on the fruit surface. Through our qualitative testing, we observed that fructose is, in fact, present in a heterogeneous pattern on fruit surfaces, and that bioreporters, such as *fruB*, may be useful for obtaining quantitative data on chemical differences in surface chemistry that can influence infection and fruit susceptibility. These bioreporters also may be useful to study competitive interactions between microbes by providing a sensitive means to determine which nutrients are available, and when and where they become available.

Objective:

The primary objective of this research is to support the UC Davis cling peach breeding program by helping 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. 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 the breeding program.

Overview of 2010 Research

Evaluation of new genotypes and breeding selections. Over 182 genotypes were evaluated for the period beginning 12 July to 20 September 2010. Fruit of similar maturity were selected based on visual inspection of size and color from among the experimental lines. These were compared with fruit of similar maturity from commercial susceptible or moderately resistant clingstone peach cultivars. Two inoculation formats were used – non-wounded and wounded. The non-wounded treatment entails applying a droplet containing conidia (spores) of *Monilinia fructicola* directly on the intact peach surface with a pipette. This provides an assessment of the epidermal and cuticular resistance of the fruit to direct penetration by the pathogen. The wounded treatment involves 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 droplet to the 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 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 emerging in the program displays a degree of flesh resistance as well.

Figure 1 graphically represents the disease severity rankings of the genotypes evaluated during 2010 in the non-wounded treatment, in order from the most resistant (lowest disease severity) to the most susceptible (highest disease severity). The genotypes evaluated this season were new lines or materials brought forward from the previous seasons, but also included susceptible commercial standards for comparison (i.e., Ross, Carson, Sherman). These data are also presented in a more detailed fashion in **Table 1 of the Appendix**. Of these, 89 genotypes, or about 49%, 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 (note however that only wounded fruit values were obtained for 102 of the genotypes due to insufficient fruit and other unforeseen limitations to do both inoculation formats. Clearly, in the vast majority of genotypes wounding compromises any epidermal/cuticular resistance and facilitates pathogen entry and colonization. However, a few genotypes displayed 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.

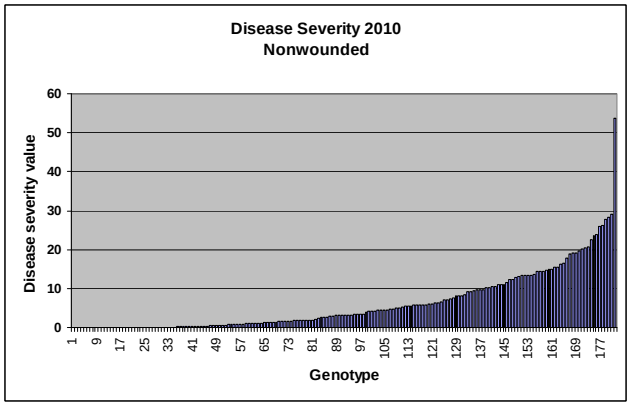

Fig. 1. Disease severity values of new genotypes evaluated in 2010, from the most resistant to most susceptible. Rankings for 182 individual genotypes are presented. Nonwounded inoculation format.

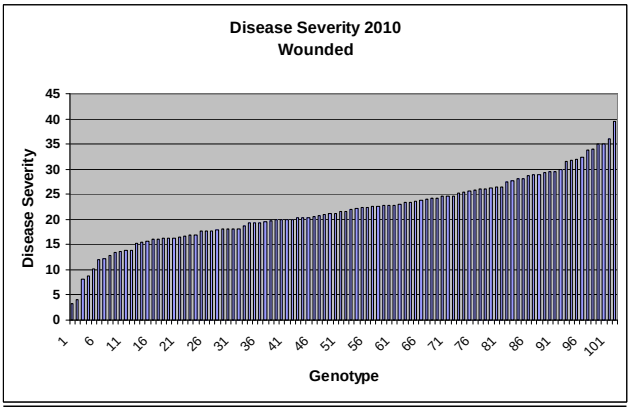

Fig. 2. Disease severity values of new genotypes evaluated in 2010, from the most resistant to most susceptible. Rankings for 102 individual genotypes are presented. Wound inoculation format.

Fig. 3. Representative reactions of highly resistant (left, Dixon/PG1-18+19) and susceptible (right, 96, 3-153/NS04-4+5) genotypes from 2010 analyses. 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. Noninoculated controls (CK) are indicated. Nonwounded format.

*Status of analysis of the PopBR populations for genetic markers***.** As part of a program to develop predictive tools for brown rot and sour rot resistance in peach and nectarines, we previously had evaluated progeny lines from two mapping populations (Ogundiwin et al., 2008). 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 disease assay results from these evaluations are consistent with quantitative (polygenic) inheritance of the fruit resistance phenotype. In 2009, a series of Pop-BR genotypes were identified after 3 consecutive seasons of evaluation as showing consistently either high resistance or high susceptibility. Because of interruption in staffing of this aspect of the project, we were forced to temporarily stop the analysis of DNA polymorphisms associated with brown rot resistance in these progeny during 2010. We anticipate that we will resume these analyses soon and will have a clear assessment of the strength of the molecular data in late 2011. We will provide a summary of the results of this work at that time.

Chemical prospecting of the fruit surface with bioreporters. Stone fruits, such as peaches and nectarines, become increasingly susceptible to pathogens as they mature and ripen. Associated with this increased susceptibility are structural changes in the fruit surface, which includes thinning of the cuticle, as well as changes in fruit surface chemistry, such as production of sugars and a decline of certain phenolic compounds. Also, there are significant differences among different varieties in their susceptibility to postharvest diseases. For example, a white flesh cultivar, Bright Pearl, develops smaller lesions and is considered more resistant to sour rot (*Geotrichum candidum*) than the yellow flesh cultivar, May Grand. Gil et al. (2002) compared fruit peel and flesh chemistry in yellow and white flesh

cultivars and found significantly higher levels of phenolic compounds in some cultivars, such as Snow King and Bright Pearl. These cultivars were tested for their susceptibility to sour rot and found to be more resistant than Spring Lady, which had significantly lower amounts of phenols. In addition, titratable acidity was higher in yellow flesh than in white flesh peach cultivars. We have observed similar trends in fruit peel phenols among processing peach genotypes that differ in their resistance to the brown rot pathogen (Lee and Bostock, 2007). Prusky (1996) and Prusky and Lichter (2007) have reviewed pathogen quiescence in post-harvest diseases and discuss how fruit factors such as high acidity and phenols in unripe fruits can contribute to disease resistance.

Pathogens such as *M. fructicola* respond to these changes by expressing genes and proteins that are important for the pathogen to successfully infect the fruit (Lee et al., 2010). We are interested in ripening-associated changes in fruit surface chemistry that may contribute to this increased susceptibility. Of particular interest are changes in sugars, pH and redox chemicals (antioxidants and pro-oxidants), as these are known factors that can influence the expression of pathogenicity factors by fungi such as plant cell wall degrading enzymes. There is a strong desire within the industry and among consumers to reduce the use of chemical fungicides, as well as concern about the development of fungicide-resistance in pathogen populations (Adaskaveg et al., 2005; Ma and Michailides, 2005). Due to this perception, alternative methods such as biocontrol are needed. Competitive exclusion is one of the suggested mechanisms for biocontrol, whereby the biocontrol agent competes with the pathogen for nutrients and space (Janisiewicz and Korsten, 2002; Janisiewicz and Buyer, 2010). However, providing evidence for this and other postulated mechanisms for biocontrol has been difficult due to the absence of experimental methods with sufficient resolution to study the microbial ecology and chemical dynamics of the fruit surface.

Although previous studies in our lab have examined some of these changes by analysis of fruit tissue extracts, bioreporters have the potential to visualize chemical changes on the fruit surface with unprecedented spatial clarity. Bioreporters are epiphytic bacteria that have been engineered to express a reporter gene in the presence of an appropriate chemical stimulus (Mercier and Lindow, 2000; Miller et al., 2001). During the summer of 2010 we conducted a proof-of-concept study to see whether bioreporters responsive to changes in surface sugars can be used to sense and visualize changes on the surface of peach and nectarine fruit following different treatments. Bioreporter strains were used as sensors to visualize changes in the production of fructose sugar on the fruit surface. The epiphytic bioreporter *EH299R* (*Pantoea agglomerans*) with *gfp* (encoding green fluorescence protein) under control of the *E. coli fruB* promoter (Leveau and Lindow, 2001) was used to evaluate fruit surface chemistry. The bioreporter *EH299R* with gfp under the control of the promoter *nptII* was used as a fructose nonresponsive, GFP-positive control. These preliminary experiments demonstrated that the *EH299R* strains correctly respond to the present or absence of fructose, and that fructose is, in fact, present in a heterogeneous pattern on the surfaces of peach and nectarine fruit. In addition, *fruB* may be useful for obtaining quantitative data on chemical differences in surface chemistry that can influence disease development and for studying competitive interactions between microbes by providing a sensitive means to determine which nutrients are available, and when and where they become available.

Future plans

An ongoing 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. With a number of these now in regional trials and coming into fruit-bearing maturity, a rigorous assessment of their performance in the field will now be possible. An immediate goal for the next year will be to complete the molecular marker analysis of resistance to brown rot, and to further develop the bioreporter system for studying fruit surface chemistry.

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 analyzed using Microsoft Excel.

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.

Bioreporters and fruit surface chemistry - The epiphytic bacterial bioreporter *EH*299R (*Pantoea agglomerans*) expressing the green fluorescent protein (*gfp*) under control of the fructose-responsive *fruB* promoter (Leveau and Lindow, 2001) was used to evaluate fructose availability in peach and nectarine fruit in the laboratory. *EH*299R with *gfp* consitutively expressed under the control of the promoter *nptII* was used as a fructose-nonresponsive control. Fruits were harvested at various stages of maturity and wounded or left unwounded and then inoculated by spraying the fruits with a bacterial suspension (5 x 10^6 cfu/ml). Fruit were placed in humidified plastic crispers and incubated at 22°C. To monitor the bioreporter populations, bacteria were collected at various time points by washing the fruits with K-PO_4 buffer and examining the collected bacteria under a fluorescence microscope.

Acknowledgments

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Appendix

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 2010 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 12; B, July 19; C, July 26; D, Aug 2; E, Aug 9; F, Aug 16; G, Aug 23; H, Aug 30; I, Sept 6; J, Sept 13; K, Sept 20.

Appendix - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
D, 6-15W/PG1-18+19	27-Jul	0.0	0.0	0.00	0.0
Dixon/PG1-18+19	27-Jul	0.0	0.0	0.00	0.0
06, 1-89	28-Jul	0.0	0.0	0.00	0.0
01, 9-104	6-Aug	0.0	0.0	0.00	0.0
01, 9-103	6-Aug	0.0	0.0	0.00	0.0
01, 9-42	10-Aug	0.0	0.0	0.00	0.0
01, 9-208	2-Aug	0.0	0.0	0.00	0.0
01, 9-34	2-Aug	0.0	0.0	0.00	0.0
01, 9-142	2-Aug	0.0	0.0	0.00	0.0
01,9-71	26-Aug	7.9	9.1	0.00	0.0
01,9-67	26-Aug	0.0	0.0	0.00	0.0
01,9-32	19-Aug	0.0	0.0	0.00	0.0
01,9-46	26-Aug	0.0	0.0	0.00	0.0
01,9-58	19-Aug	0.0	0.0	0.00	0.0
01,9-82	26-Aug	0.0	0.0	0.00	0.0
01, 9-99	30-Aug	0.0	0.0	0.00	0.0
01, 9-101	30-Aug	0.0	0.0	0.00	0.0
01, 9-115	30-Aug	0.0	0.0	0.00	0.0
01, 9-114	30-Aug	0.0	0.0	0.06	0.0
01, 9-95	30-Aug	0.0	0.0	0.00	0.0
01, 9-169	30-Aug	0.0	0.0	0.00	0.0
01, 9-188	30-Aug	0.0	0.0	0.00	0.0
01, 9-186	30-Aug	0.0	0.0	0.00	0.0
98, 9-7 (NSW 5-18+19)	1-Sep	0.0	0.0	0.00	0.0
01, 9-157	30-Aug	0.0	0.0	0.00	0.0
01, 9-89	2-Aug	0.3	1.3	0.05	0.0
01, 9-35	2-Aug	0.3	1.2	0.06	0.0
NJC 83/ PG 5-15	5-Aug	0.4	1.5	0.08	0.0
01, 9-140	30-Aug	0.3	1.0	0.10	0.0
01,9-30	26-Aug	0.7	2.1	0.05	0.0
07, 6-227	6-Aug	0.7	2.0	0.06	0.0
01, 9-39	2-Aug	0.6	1.7	0.13	0.1
01, 9-24	2-Aug	0.9	2.9	0.10	0.1
01, 9-63	2-Aug	0.9	2.8	0.12	0.1
08, 19-97 (+pic)	28-Jul	1.1	3.3	0.11	0.1
01, 9-129	30-Aug	1.7	5.0	0.11	0.2
01, 9-125	30-Aug	1.4	3.8	0.14	0.2
01, 9-107	30-Aug	1.0	2.1	0.22	0.2
98, 4-177/NSW 15-16+17	18-Aug	1.3	2.8	0.19	0.2
PPD 62-193 PG 8-6+7	8-Jul	2.0	4.5	0.13	0.3
01, 9-198	30-Aug	1.3	2.7	0.20	0.3
08, 20-44	6-Aug	1.1	2.2	0.25	0.3
01, 9-40	2-Aug	1.5	3.4	0.19	0.3
01, 9-180	2-Aug	2.5	7.1	0.13	0.3
92, 14-6/NSW 5-647	27-Jul	2.0	4.8	0.18	0.4
01, 9-81	10-Aug	2.2	5.2	0.17	0.4
01, 9-135	30-Aug	2.0	4.2	0.20	0.4
05, 26-251	28-Jul	2.3	5.5	0.19	0.4

Appendix - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
01, 9-234	2-Aug	2.9	7.1	0.18	0.5
01,9-117	19-Aug	2.3	4.5	0.25	0.6
01, 9-97	30-Aug	5.7	9.8	0.11	0.6
01, 9-144	8-Sep	3.2	8.0	0.20	0.6
99,12-155/NSW5-647	27-Jul	2.8	5.5	0.25	0.7
01, 9-112	10-Aug	2.4	3.4	0.33	0.8
08, 13-132	6-Aug	2.6	4.2	0.33	0.9
05-16-242	11-Jul	3.9	8.1	0.22	0.9
01, 9-190	30-Aug	3.9	10.1	0.22	0.9
01, 9-150	2-Aug	2.9	5.2	0.30	0.9
01, 9-65	2-Aug	3.1	5.4	0.30	0.9
08, 31-161	16-Aug	3.8	8.6	0.25	0.9
05, 17-113	17-Aug	4.2	8.3	0.25	1.0
90,9-116/NSW2-34-38	27-Jul	3.2	7.2	0.33	1.1
01, 9-130	2-Aug	3.9	6.7	0.29	1.1
01, 9-204	31-Aug	2.9	4.5	0.39	1.1
01. 9-178	2-Aug	4.4	8.0	0.29	1.3
05,31-150	28-Jul	4.6	10.3	0.29	1.3
08, 12-165 (+pic)	28-Jul	4.5	8.1	0.29	1.3
08,1-49	11-Jul	4.3	7.6	0.31	1.4
01, 9-48	10-Aug	4.3	7.4	0.33	1.4
05, 24-25	14-Aug	3.0	4.2	0.50	1.5
01, 9-122	30-Aug	3.9	5.7	0.40	1.6
97, 2-152/ NSW 4-647	27-Jul	4.6	7.5	0.35	1.6
05-26-233	11-Jul	5.8	12.7	0.29	1.6
05, 17-152	14-Aug	1.7	2.9	1.00	1.7
01, 9-173	2-Aug	5.2	9.0	0.33	1.7
01, 9-203	30-Aug	5.2	8.5	0.33	1.7
01, 9-156	2-Aug	7.0	16.8	0.25	1.8
05-27-51	11-Jul	4.1	7.4	0.44	1.8
01, 9-77	8-Sep	3.7	4.2	0.50	1.8
99, 4-123/NSW 5-20+21	18-Aug	4.6	6.4	0.40	1.8
05-16-244	11-Jul	5.7	9.1	0.33	1.9
F8, 5-156/NSW 1-4	15-Sep	4.6	4.6	0.44	2.0
08, 33-69	29-Jul	5.8	9.5	0.42	2.4
01,9-165	19-Aug	5.8	8.4	0.44	2.5
05, 17-56	14-Aug	3.6	5.1	0.71	2.5
96, 8-192/ NSW 5-12+13	1-Sep	5.6	7.8	0.47	2.7
08, 33-237	16-Jul	7.3	11.4	0.39	2.8
06, 2-92	7-Aug	4.5	4.7	0.67	3.0
01, 9-72	6-Aug	4.6	6.3	0.67	3.0
05-16-218	11-Jul	7.0	7.5	0.44	3.1
07-13-191	11-Jul	8.4	12.7	0.38	3.1
F8, 5-147/NSW 1-1-3	15-Sep	5.4	5.1	0.60	3.2
05-24-24	11-Jul	7.9	11.9	0.41	3.2
F8, 5-166/NSW 1-7-9	15-Sep	6.5	8.7	0.50	3.3
05, 24-149	28-Jul	6.7	11.6	0.50	3.3
05, 18-82	14-Aug	5.9	5.7	0.56	3.3
05, 16-220	16-Jul	4.7	8.5	0.72	3.4

Appendix - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
97,7-75/NSS4-20421	27-Jul	5.0	6.9	0.71	3.5
08, 8-75 (+pic)	28-Jul	7.8	10.1	0.50	3.9
08, 20-12(+pic)	22-Jul	6.6	7.9	0.63	4.1
05, 16-223 (+pic)	22-Jul	7.6	8.6	0.56	4.2
99, 12-155/NSW5-6+7	2-Aug	5.9	6.4	0.71	4.2
Stanislaus (PG 1-28+28a)	20-Jul	6.1	5.2	0.71	4.3
06, 2-210	16-Sep	13.0	9.9	0.33	4.3
07, 6-218 (+pic)	21-Jul	6.6	8.0	0.67	4.4
96,9-292/NSW4-41-14-2	27-Jul	8.1	8.2	0.56	4.5
F8, 5-171/NSW 1-10	15-Sep	8.0	7.2	0.60	4.8
05, 16-196	14-Aug	6.7	6.0	0.72	4.8
96, 1-171/NSW4-2+3	5-Aug	7.3	6.9	0.67	4.9
07, 26-5	16-Jul	9.2	12.3	0.54	5.0
01, 9-189	10-Aug	12.3	12.5	0.43	5.3
07, 15-175 (+pic)	28-Jul	9.7	10.3	0.56	5.4
05, 18-205	22-Jul	10.0	9.4	0.56	5.6
91, 12-54 NSW 2-11-18	10-Aug	6.4	5.4	0.88	5.6
01,9-205	19-Aug	9.9	14.5	0.57	5.6
08, 35-206	16-Aug	9.2	9.9	0.63	5.8
08, 32-109 (+pic)	22-Jul	8.1	7.4	0.72	5.9
Carson/ PG1-20+21	20-Jul	7.7	7.4	0.76	5.9
01,9-176	19-Aug	8.0	7.1	0.74	5.9
9-161 NSW 2-19-23	10-Aug	7.6	7.5	0.78	5.9
05-27-130	11-Jul	9.9	10.5	0.62	6.1
01, 9-61	8-Sep	8.3	6.2	0.75	6.2
Diamonte/NSW 3-41+42	20-Jul	8.3	10.5	0.77	6.4
PP Sherman 86-28A PG 1-26+26a	8-Jul	11.1	10.3	0.59	6.5
05, 17-73	14-Aug	8.5	6.2	0.82	7.0
08, 35-68	16-Aug	9.4	6.9	0.76	7.2
04, 2-178	19-Aug	10.0	8.2	0.75	7.5
07, 2-173	16-Jul	11.6	12.4	0.67	7.7
90, 10-162 (NSW 3-37+38)	20-Jul	14.0	5.4	0.57	8.0
05,19-206(+pic)	22-Jul	10.7	8.8	0.75	8.0
01, 9-55	10-Aug	9.4	7.3	0.86	8.0
08, 4-177	16-Jul	9.0	12.7	0.94	8.5
08, 34-88	16-Aug	11.4	5.7	0.80	9.1
Carson/PG 1-20+21	20-Jul	12.2	9.1	0.76	9.3
05-18-221	11-Jul	12.1	9.9	0.78	9.4
06, 2-99	16-Jul	13.7	13.0	0.70	9.6
05, 18-205(+pic)	22-Jul	12.4	9.6	0.78	9.6
05, 17-101	14-Aug	11.0	7.1	0.88	9.7
Carson/PG 1-20+21	13-Jul	11.9	12.4	0.86	10.2
06, 2-107	13-Jul	14.6	12.2	0.71	10.3
05, 17-73	14-Aug	11.1	7.2	0.94	10.4
08, 1-125	16-Jul	14.0	12.6	0.75	10.5
Carson PG 1-20+21	28-Jul	10.9	8.2	1.00	10.9
05, 17-92	14-Aug	11.0	5.5	1.00	11.0
Carson/PG 1-20+21	2-Aug	13.8	10.9	0.80	11.0

Appendix - Table 1

Genotype	Harvest Date	Mean lesion diameter (mm)	SD	Incidence (lesion>3mm)	Disease Severity
D62, 193	20-Jul	13.0	7.7	0.89	11.5
05,16-213	14-Aug	12.3	4.2	1.00	12.3
05, 16-218	16-Jul	15.7	11.7	0.79	12.4
05, 18-221	13-Jul	16.6	12.6	0.78	12.9
05, 17-23	14-Aug	14.0	7.7	0.94	13.2
01, 9-138	8-Sep	22.1	14.4	0.60	13.3
05, 17-65	14-Aug	14.2	8.3	0.94	13.3
F8, 5-159/NSW 1-6	15-Sep	18.7	12.2	0.71	13.3
01, 9-84	31-Aug	16.7	11.8	0.80	13.4
01, 9-80	31-Aug	7.1	3.8	0.55	13.7
05, 18-217	22-Jul	15.2	8.4	0.94	14.3
05, 11-132	17-Aug	15.9	8.3	0.90	14.3
Sherman 86-28A/PG 1-26+26a	8-Jul	14.4	7.3	1.00	14.4
96, 3-153/NS04-4+5	27-Jul	17.5	10.8	0.83	14.6
19, 4-40 (NSW 3-37+38)	20-Jul	14.9	14.9	1.00	14.9
07, 4-172	17-Aug	16.4	16.1	0.92	15.0
05, 16-246	16-Jul	17.7	12.2	0.88	15.5
E 6-27 (NSW 3-26+27)	8-Jul	15.5	6.5	1.00	15.5
05, 20-116	14-Aug	19.5	9.2	0.83	16.3
08, 15-85	16-Aug	16.5	0.7	1.00	16.5
E, 5-43(NSW 2-1+PG 3-8)	7/20 & 7/13	17.8	8.8	1.00	17.8
01, 9-85	31-Aug	21.1	11.4	0.90	19.0
01, 9-200	31-Aug	19.0	8.9	1.00	19.0
Carson P6 1-20	10-Aug	19.3	5.4	1.00	19.3
89, 15-309 (PG10-25 at 26)	8-Jul	19.7	8.5	1.00	19.7
01, 9-47	8-Sep	20.1	3.9	1.00	20.1
NJC 86/ PG 5-16	13-Jul	22.5	12.0	0.91	20.4
Ross/ PG 1-2+3	18-Aug	20.7	2.1	1.00	20.7
08, 32-11	16-Jul	22.5	8.6	1.00	22.5
Ross/PG 1-2+3	18-Aug	17.5	13.4	1.00	23.5
08, 3-252	16-Jul	23.9	8.6	1.00	23.9
08, 4-91	16-Jul	25.9	9.7	1.00	25.9
01, 9-206	8-Sep	26.1	9.0	1.00	26.1
01, 9-233	31-Aug	27.9	10.6	1.00	27.9
Ross / PG 1- 2+3	18-Aug	28.3	4.1	1.00	28.3
01, 9-234	31-Aug	29.0	3.6	1.00	29.0
01,9-185	19-Aug	26.8	5.4	2.00	53.6