Resistance to *Fusarium oxysporum* f. sp. *fragariae* and Predicted Breeding Values in Strawberry

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ABSTRACT. Fusarium wilt of strawberry, incited by *Fusarium oxysporum* f. sp. *fragariae* (*Fof*), is a major disease of the cultivated strawberry (*Fragaria ×ananassa*) worldwide. An increase in disease outbreaks of the pathogen in Western Australia and Queensland plus the search for alternative disease management strategies place emphasis on the development of resistant cultivars. In response, a partial incomplete diallel cross involving four parents was performed for use in glasshouse resistance screenings. The resulting progeny were evaluated for their susceptibility to *Fof*. Best-performing progeny and suitability of progenies as parents were determined using data from disease severity ratings and analyzed using a linear mixed model incorporating a pedigree to produce best linear unbiased predictions of breeding values. Variation in disease response, ranging from highly susceptible to resistant, indicates a quantitative effect. The estimate of the narrow-sense heritability was 0.49 ± 0.04 (SE), suggesting the population should be responsive to phenotypic recurrent selection. Several progeny genotypes have predicted breeding values higher than any of the parents. Knowledge of *Fof* resistance derived from this study can help select best parents for future crosses for the development of new strawberry cultivars with *Fof* resistance.

Fusarium vascular wilt disease is incited by pathogens of *Fusarium oxysporum* and comprises major soilborne fungal pathogens of many important crops. The *F. oxysporum* complex comprises both pathogenic and non-pathogenic strains, many being indistinguishable using phenotypic characters. Pathogenic strains are grouped into *formae speciales* depending on host specificity. *F. oxysporum* f. sp. *fragariae* is specific (pathogenic) to strawberry and considered a worldwide major disease of the cultivated strawberry. *Fof* was first identified and described in 1962, when it spread rapidly in the strawberry production areas of Brisbane, Queensland, Australia (Winks and Williams, 1965). Subsequently, *Fof* has been reported in many countries including Japan (Mori et al., 2005; Takahashi et al., 2003), Mexico (Dávalos-González et al., 2006), Korea (Nagarajan et al., 2006), China (Zhao et al., 2009), Spain (Arroyo et al., 2009), and the United States (Koike, 2009).

Fusarium oxysporum species reproduce asexually, producing microconidia, macroconidia, and chlamydospores. Chlamydospores can remain viable in the soil for many years (Smith and Snyder, 1975) making fusarium wilt diseases difficult to control. Pathogenic strains enter the host through the roots moving into the vascular system where it colonizes in the xylem vessels and impedes water movement, causing the plant to wilt and die (Lagopodi et al., 2002; Xiao-min et al., 2011). Newly infected plants may die within a few weeks if weather conditions are hot and wet (Hancock, 1999; Winks and

Williams, 1965). Large areas of strawberry production can rapidly succumb to this disease.

Commercial strawberry growers have typically relied on disease-free runners and the pre-plant soil fumigant methyl bromide as their major management strategies for soilborne pathogens, including *Fof*. Outbreaks of fusarium wilt were relatively uncommon under a regime of methyl bromide fumigation so that neither the disease nor resistance breeding was considered important. However, after the phase-out of methyl bromide in 2005 under the “Montreal Protocol on Substances That Deplete the Ozone Layer,” outbreaks of the disease have caused up to 50% plant mortality in some fields in the Perth district of Western Australia (Golzar et al., 2007) and up to 10% mortality in the southeastern regions of Queensland. The incidence of severe outbreaks is likely to increase in subtropical Queensland if susceptible cultivars become popular for marketing reasons. Currently, there is no effective treatment for *Fof*-infected plants, and cultivars with resistance to fusarium wilt are required to limit disease outbreaks (Dávalos-González et al., 2006; Herrington et al., 2007).

As a result of the lack of effective fumigants and also the large plant losses occurring from fusarium wilt in Australia, the Queensland strawberry breeding program identified a need to include fusarium wilt resistance as part of the primary selection criteria of its multi-trait breeding strategy. Varietal field screenings for susceptibility to fusarium wilt began in 2002 at Maroochy Research Facility, Nambour, Queensland. Screening of cultivars in *Fof*-infested field plots identified large variation in cultivar response. Assessed on a 0 to 10 symptom severity scale (Hutton and Gomez, 2006), the cultivars Maroochy Jewel and Kabarla were described as susceptible (greater than 5) and ‘Festival’ and ‘Sugarbaby’ as resistant (1 or less) (Hutton and Gomez, 2006). Further glasshouse experiments, testing nine cultivars for symptoms of *Fof*, confirmed ‘Kabarla’ and ‘Camarosa’ as susceptible to fusarium wilt and ‘Sugarbaby’ and ‘Festival’ as resistant (M.L. Paynter, unpublished data). Additionally, a study conducted recently in Western Australia

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also described 'Festival' as resistant to fusarium wilt and 'Camarosa' as susceptible (Fang et al., 2012). 'Camarosa' (susceptible) is the major cultivar grown in Western Australia, and its high susceptibility may partially explain the greater losses to fusarium wilt experienced in Western Australia.

The genetic base in the cultivated strawberry is relatively narrow (Sjulin and Dale, 1987); however, high levels of heterozygosity in the strawberry genome and the hybrid nature of *F. ×ananassa* (Hancock, 1999; Maas, 1998) make breeding for disease resistance a viable option. Despite much knowledge on fusarium wilt diseases in other crops, little is known of the genetics of resistance in strawberry, where most inheritance studies on strawberry populations have been on fruit traits (Murti et al., 2012; Shaw and Sacks, 1995; Verma et al., 2003). From a study in Japan of *Fof* resistance in strawberry, Mori et al. (2005) reported bimodal segregation of disease resistance to fusarium wilt in F₁ hybrid seedlings of strawberry and concluded that major genes were involved, but there was also a multigenic component, because among susceptible cultivars, the disease severity index varied continuously. Continuous variation in resistance to *Fof* among strawberry cultivars has also been observed by Hutton and Gomez (2006). Information about the heritability of the resistance in strawberry and estimation of the breeding value of individual plants would be beneficial in identifying highly resistant genotypes and using them in breeding programs.

Best linear unbiased predictions [BLUPs (Henderson, 1984)] of breeding values have been used in many breeding programs to increase the frequency of desired phenotypes in progeny (Davik and Honne, 2005; Hardner et al., 2012; Kennedy, 1981). Use of specialized crossing designs (e.g., full or partial diallel) and statistical models can generate individual breeding values, and so determine their suitability as parents, from an observed sample of progeny. If pedigree data are included in the model, the information on the relatedness of the genotypes allows better estimates of total genetic effects and predicted breeding values because the effective number of observations available increases (White and Hodge, 1989). Fitting models with pedigree information to estimate genetic effects have been used for apple [*Malus ×domestica* (Durel et al., 1998)], peach [*Prunus persica* (de Souza et al., 1998)], and strawberry (Davik and Honne, 2005). Strawberry is an ideal crop for selection using predicted breeding values because the short generation interval allows for progeny measurements to be performed and available for analysis within one season.

In this report, we used progeny resulting from a partial incomplete diallel crossing to test for resistance to fusarium wilt to obtain estimates of individual predicted breeding values and genetic parameters relevant to fusarium wilt resistance in our strawberry population. This knowledge can be used to enable better predictions about progeny response to selection for the resistance trait and assist in the breeding of strawberry cultivars with increased resistance to fusarium wilt.

Materials and Methods

Genetic material

Four parents were hand-pollinated in 2009 using a partial incomplete diallel cross design to generate 245 progeny from 14 full-sib families, numbering from six to 27 progeny per family (Table 1). The parents chosen for crossing were derived from ancestors containing several connecting relatives (pedigree

Table 1. Crossing scheme used in screening for resistance to *Fusarium oxysporum* f. sp. *fragariae*.^z

Family	Parent cultivars used in cross		Progeny (no./family)
	Female	Male	
2772	Festival	Festival	15
2773	Festival	Maroochy Jewel	26
2774	Festival	Kabarla	22
2775	Festival	Sugarbaby	27
2776	Maroochy Jewel	Maroochy Jewel	19
2777	Maroochy Jewel	Festival	10
2778	Maroochy Jewel	Kabarla	14
2779	Maroochy Jewel	Sugarbaby	23
2780	Kabarla	Kabarla	22
2781	Kabarla	Festival	19
2782	Kabarla	Maroochy Jewel	14
2783	Kabarla	Sugarbaby	22
2786	Sugarbaby	Sugarbaby	6
2787	Sugarbaby	Maroochy Jewel	6

^zIndicated are family number, parents used, and number of strawberry progeny per family.

linkages) and were considered representative of a diverse range of *Fof* susceptibility. In Feb. 2011, up to five freshly emerged runners from each progeny were potted into 100 × 140-mm plastic pots (one plant per pot) containing steam-sterilized potting mix composed of double-washed river sand and coir (by volume:1:1) with a pre-mixed fertilizer of (in g·L⁻¹) 5.1 nitrogen, 7.2 phosphorus, 4.6 potassium, 60.4 calcium, 0.08 copper, 0.06 iron, 0.32 magnesium, and 0.15 zinc.

The cultivar Kabarla, reported to be susceptible in the southeastern regions of Queensland (Hutton and Gomez, 2006), was used to validate the inoculation procedure. Certified disease-free 'Kabarla' runners were grown under the same conditions as described for strawberry progenies.

Fungal isolates and inoculum preparation

Two isolates of *Fof*, originally collected in Queensland, from the crowns of infected strawberry plants showing typical severe symptoms of fusarium wilt disease (N13581 harvested from 'Kabarla' in 2002 and N15309 harvested from 'Camarosa' in 2005), were used as inoculum in screening trials. These were obtained from the Maroochy Research Facility culture collection at Nambour. Both isolates belonged to the same vegetative compatibility group and had been identified as *Fof* based on spore and colony morphology, cultural characteristics, and their high virulence confirmed by pathogenicity tests performed at the Maroochy Research Facility (M.L. Paynter, unpublished data). Cultivar responses of these two highly pathogenic isolates of *Fof* were confirmed by previous trials (M.L. Paynter, unpublished data) and results were similar to Fang et al. (2012) and Hutton and Gomez (2006).

For preparation of inocula, single spore accessions of N13581 and N15309 were plated onto one-quarter strength potato dextrose agar amended with 50 mg·L⁻¹ streptomycin sulphate and incubated at 24 °C for 2 weeks. The spores were collected from culture plates after addition of sterile deionized water and rubbing the agar surface with a glass spreader. The spore suspension was then filtered through four layers of cheesecloth. The conidial concentration (mainly microconidia and macroconidia with some chlamydospores) was determined using a hemocytometer and adjusted with sterile water to 1 × 10⁶

conidia/L. Before use, both N13581 and N15309 conidial suspensions were combined equally.

The inoculation procedure combined a root dip technique (Pastor-Corrales and Abawi (1987) with the addition of sterilized ryegrass seed (*Lolium perenne* cv. Tetila) to assist the pathogens survival and proliferation (Smith et al., 2008). To prepare sterilized ryegrass seed, the ryegrass seed was rinsed under running tap water, drained, and soaked overnight in distilled water, after which the water was strained off and the seed rinsed several times with tap water until water was clear. The seed was then autoclaved at 121 °C for 20 min on 3 consecutive days.

Inoculation process

In Nov. (late spring) 2011, plants were removed from their pots and ≈3 cm cut from the bottom of the root ball (soil and roots). The 3-cm section was put back into the pot and 20 mL of sterilized ryegrass seed was spread on top. The plants were inoculated in a randomized order by immersing the plant root ball in the inoculum (to 1 cm past the top of crown) for 10 min and then placed back (on top of ryegrass) into their pot. The plants were then firmly situated in their pots using potting medium (as described previously). Approximately 1 cm of sterile gravel (3 to 5 mm in diameter) was added around the plant on top of the soil mix to prevent splash. To validate the inoculation procedure, commercial runners of ‘Kabarla’ were used as positive and negative controls. Six ‘Kabarla’ were inoculated as described previously, whereas another six plants were immersed in sterile water only. Additionally, one to two plants from each family were also inoculated using sterile water only. Plants were randomly allocated, spaced at ≈25 cm apart, onto five heated benches at 30 °C in a glasshouse. Non-inoculated control plants were placed on a bench separated from inoculated plants by ≈1 m to avoid contamination from splash and insects. Plants were watered with tap water daily for up to 5 d and approximately three times per week thereafter and fertilized at 2-week intervals. The trial design was an incomplete block design. There were between two to five replicate plants (ramets) per progeny with a mean number of four.

Disease severity assessment

VISUAL ASSESSMENT. Disease development was monitored weekly on the individual plants and visual severity ratings taken from 4 weeks post-inoculation. Severity of foliar symptoms was assessed on a 0 to 10 disease severity score (Hutton and Gomez, 2006) where: 0 = plant healthy with erect growth and full vigor; 1 = plant healthy, with a smaller canopy and moderate vigor; 3 = plant with a slight wilt, with the lower leaves affected; 5 = plant with a moderate wilt, with the mature leaves collapsing but young leaves still healthy; 7 = plant with a severe wilt, with most of the plant collapsed and mature leaves desiccated; 9 = plant with a very severe wilt, with the entire plant collapsed and most of plant desiccated; and 10 = dead plant. A mean disease severity score for each progeny was calculated across replicates.

CLASSIFICATION OF RESISTANCE. The degree of resistance (x) to *Fof* was determined from the mean disease severity score by the following scale: $x \leq 2$ = resistant, $2 < x \leq 4$ = moderately resistant, $4 < x \leq 6$ = moderately susceptible, $6 < x \leq 8$ = susceptible, $x > 8$ = very susceptible.

SAMPLING. Random sampling from symptomatic (six to nine) and healthy (one to two) plants of each family was

performed at 14 weeks post-inoculation. The crowns were washed clean and surface-sterilized using sodium hypochlorite and rinsed three times in sterile water. Crowns were cut in cross-sections and discolored pieces of crown plated onto potato dextrose agar and incubated at 24 °C. After 1 week, plates were inspected and analyzed microscopically for the presence of *Fof*.

Statistical analysis

Analysis of the disease response from severity ratings taken at 8, 10, and 14 weeks post-inoculation was performed using a linear mixed model incorporating a pedigree (interline relationships) on individual plant records (Hardner et al., 2012; Henderson, 1975; Oakey et al., 2006). The pedigree information included ancestors traced back up to four generations. The model included terms for the random additive genetic effects for each of the genotypes (including parents), random family effects, residual non-additive genetic variance, replicate clone effects, and table (location) effects. Control data were not included in analysis as a result of their placement on a separate table to restrict contamination. The analyses were performed using the ASReml-R package (Butler et al., 2009), which provides residual maximum likelihood (REML) estimates of the variance components and BLUPs of the random effects in the mixed model.

To investigate the genetic effects influencing resistance to fusarium wilt, the disease severity scores for the screenings were analyzed using the following linear mixed model: $y = X\tau + Z_g u_g + Z_o u_o + e$, where y is the vector of observed disease ratings, τ is a vector of fixed effects (e.g., overall mean) with design matrix X , u_g is a vector of random total genetic effects with design matrix Z_g , u_o is a vector of other non-genetic random effects (e.g., replicate and table effects) with design matrix Z_o , and e is the vector of random residual effects.

All random effects in the model are assumed to be normally distributed with mean zero and the three random effect vectors (u_g , u_o , e) are assumed pairwise-independent. The variance models for the random non-genetic and residual effects are given by: $Var(u_o) = \sigma_o^2 I$ and $Var(e) = \sigma_e^2 I$.

The vector of total genetic effects can be partitioned into three components, namely additive, non-additive, and family genetic effects. That is: $u_g = u_a + u_{na} + Z_f u_f$, where u_a represents the vector of additive genetic effects with distribution $u_a \sim N(0, \sigma_a^2 A)$ where A is the known additive relationship matrix based on the pedigree information, u_{na} represents the non-additive or residual genetic effects with distribution $u_{na} \sim N(0, \sigma_{na}^2 I)$, and u_f represents the family genetic effects with distribution $u_f \sim N(0, \sigma_f^2 I)$.

Using the REML estimates of the variance components in the linear mixed model, the narrow-sense heritability (proportion of additive genetic variance over the total variance) was estimated by: $h^2 = \sigma_a^2 / (\sigma_a^2 + \sigma_{na}^2 + \sigma_f^2 + \sigma_e^2)$, where σ_a^2 is the additive genetic variance of the individual genotypes, σ_{na}^2 is the non-additive genetic variance, σ_f^2 is the genetic variance between families, and σ_e^2 is the variance of the random residuals. Breeding values were predicted for each of the 245 progeny and four parents obtained by the BLUPs. Broad-sense heritability was estimated as: $H^2 = (\sigma_a^2 + \sigma_{na}^2 + \sigma_f^2) / (\sigma_a^2 + \sigma_{na}^2 + \sigma_f^2 + \sigma_e^2)$.

Results

PROGENY RESPONSE. Four weeks post-inoculation, external foliar symptoms of fusarium wilt were apparent. These included wilting, stunting of leaves, and lesions on petioles. Typically, susceptible plants were showing symptoms by Week 6. Plant deaths of up to 13% were observed by Week 8 and 56% by Week 14. Disease severity ranged from plants showing no symptoms of wilting to the complete collapse and death of plants. Non-inoculated control plants showed no symptoms. The response to *Fof* in the population was quantitative with continuous variation in susceptibility ranging from mildly resistant to very susceptible. Families that scored a low mean visual rating (i.e., showed good resistance) included: 2772, 2775, and 2786. Those with high scores (i.e., highly susceptible) included: 2776, 2778, and 2782. The remaining families showed variation in disease expression to *Fof* from moderately resistant to susceptible as shown in Figure 1.

The crown isolations carried out on symptomatic plants produced significant fungal colonies that were identified by morphological characteristics as *F. oxysporum*. The severity of discoloration in the vascular tissues of the crown was consistent with that of foliar disease severity and typically plants showing no symptoms had clean, disease-free crowns, whereas those showing symptoms exhibited vascular discoloration and rots.

The BLUP estimates of breeding values at Week 14 post-inoculation for the four parental genotypes ranged from 2 to 9.6 as shown in Figure 2. Higher breeding value estimates signify individuals that will pass on greater susceptibility to the next generation. Low BLUP values indicate cultivars that will pass

on good resistance. ‘Maroochy Jewel’ (9.61) and ‘Kabarla’ (7.85) were the most susceptible parental genotypes, whereas ‘Festival’ (2.04) and ‘Sugarbaby’ (2.42) were the most resistant.

The family mean breeding values predicted for disease severity for each of the 14 families covered a broad range from 1.52 to 9.43 (average SE of difference equal to 0.55) as shown in Figure 3. Several progeny from the families 2775, 2786, and 2772 had high predicted levels of resistance (i.e., low disease severity score), whereas progeny from the families 2780, 2778, 2782, and 2776 had the highest predicted levels of susceptibility (i.e., high disease severity score).

The range of BLUP breeding values across the 245 individual genotypes was from 0.62 to 10.15 (average SE of difference equal to 2.00). Among the most resistant 10% of progeny, the five best genotypes for resistance included: 2772-14, 2772-15, 2772-05, 2786-01 and 2775-30 as shown in Figure 4. The best predicted breeding value for the individual progeny for the resistance trait belonged to 2772-14, which had a breeding value of 0.62 as shown in Figure 4.

Narrow-sense heritability was estimated at 0.49 ± 0.04 (SE) for the severity of disease, indicating that the observed phenotypic variation was moderately influenced by genetic factors. Broad-sense heritability was estimated at 0.50 ± 0.04 . Variance components estimated from the linear mixed model at 14 weeks post-inoculation included the additive, non-additive, family, replicate, residual, and table (glasshouse location) effects (Table 2). There is a high correlation of breeding values among the assessment dates 8, 10, and 14 weeks after inoculation as shown in Figure 5.

Fig. 1. Disease ratings for severity of disease symptoms in strawberry caused by *Fusarium oxysporum* f. sp. *fragariae* for each family taken at Weeks 4, 7, 8, 9, 10, 11, 12, 13, and 14 post-inoculation (0 = healthy plant and 10 = dead plant) showing variation among families over time. Each line represents the mean disease severity over time for each family. Dotted lines represent ± 2 SD from the mean.

The realized response to selection for selecting the best p^{th} percent ($p\%$) of lines as parents can be calculated as the mean of the BLUPs of breeding values of the top $p\%$ of ranked cultivars (Cullis et al., 2006). Therefore, the realized response to selection for *Fof* disease rating by selecting the best (most resistant; i.e., the lowest disease ratings) 10% of progeny as parents (and assuming they are randomly mated) is given by 0.98. The overall population mean genetic effect for *Fof* rating before selection is 5.74. Hence, the predicted genetic gain in the first generation of selection is a decrease in disease rating of 4.76 or 83% of the original mean.

Discussion

This study aimed to deliver two fundamental objectives relevant to the breeding of fusarium wilt resistance in strawberry. The first was to evaluate strawberry progeny for their susceptibility to *Fof* and identify suitable parents for transferring the resistance trait. The second was to obtain estimates of genetic parameters (breeding values, variances,

Fig. 2. Best linear unbiased prediction (BLUP) estimates of average breeding value for severity of disease symptoms in strawberry caused by *Fusarium oxysporum* f. sp. *fragariae* for parents taken at Week 14 post-inoculation. Parent BLUP average SE of differences = 0.78.

Fig. 3. Best linear unbiased prediction (BLUP) estimates of average breeding value for severity of disease symptoms in strawberry caused by *Fusarium oxysporum* f. sp. *fragariae* for each family taken at Week 14 post-inoculation. Family BLUP average SE of differences = 0.55.

Fig. 4. Predicted breeding values (best linear unbiased predictions) of the most resistant (lowest symptom expression) 10% of strawberry progeny to *Fusarium oxysporum* f. sp. *fragariae* at 14 weeks.

Table 2. Table of residual maximum likelihood estimates of variance components (additive, non-additive, family, replicate, residual, and table effects) and SEs of strawberry progeny estimated from the linear mixed model fitted to *Fusarium oxysporum* f. sp. *fragariae* disease severity rating at 14 weeks post-inoculation.

Variance component			SE
Additive genetic variance	σ_a^2	10.09	1.45
Non-additive genetic variance	σ_{na}^2	0.00 ^z	—
Family	σ_f^2	0.08	0.24
Replicate (clone)		0.40	0.32
Bench (location in glasshouse)		0.02	0.07
Residual variance	σ_e^2	10.33	0.55

^zVariance component estimate constrained to be fixed at a very small positive value.

and heritability) relevant to our population. Our study documents cultivar response to *Fof*, then estimates the breeding values for the parents and progeny and heritability for the breeding population. The procedure used in this study predicted the potential as a parent of individual genotypes (through additive genetic effects) as well as the overall or total genetic effect of each genotype (by combining both additive and non-additive genetic effects) and provided estimates of both narrow- and broad-sense heritability.

Typically, genetic variance among progenies is divided into both additive and non-additive components, the additive component being important because it determines how well a progeny will perform as a parent and for evaluating the potential for genetic gain. In this study, the total genetic effect of the progenies is influenced mostly by additive variance (Table 2). Because we are interested in identifying the best potential parents for future crosses, we have focused on the additive genetic variance component (or breeding value) of progenies rather than the total genotypic genetic effect. By incorporating the pedigree information in the linear mixed model, the relationships between the genotypes are taken into account and this increases the accuracy of the genotypic effects (Oakey et al., 2006; Piepho et al., 2007).

Previous studies on the inheritance of fusarium wilt resistance in strawberry have suggested *Fof* resistance is inherited as both a qualitative and quantitative trait (Mori et al., 2005). In our population we found varying degrees of susceptibility to *Fof* ranging from mildly resistant to very susceptible among strawberry cultivars and progeny with resistance best described as under multigenic control. The lowest occurrence of fusarium wilt was observed in the families 2772 and 2775 (i.e., progeny from crosses involving 'Festival' or 'Sugarbaby'), whereas the highest level of fusarium wilt symptoms occurred in 2776 and 2778 (i.e., progeny from crosses involving 'Maroochy Jewel' or 'Kabarla'). This study confirms previous findings of fusarium wilt resistance in 'Festival' and 'Sugarbaby' (Fang et al., 2012; Hutton and Gomez, 2006). 'Festival' is one of the major cultivars grown in Queensland and, from a field trial conducted in Nambour, is considered resistant to fusarium wilt (Hutton and Gomez, 2006). Fang et al. (2012) also found 'Festival' plants to be resistant to fusarium wilt in Western Australia. Although 'Festival' shows effective levels of resistance, *F. oxysporum* has been isolated from several severely wilted 'Festival' plants in Nambour and further work on the pathogenicity and variability of *F. oxysporum* strains that affect strawberry is required.

Fig. 5. Relationships at 8, 10, and 14 weeks of breeding values (best linear unbiased predictions) of symptom expression after inoculation of strawberry progeny to *Fusarium oxysporum* f. sp. *fragariae*. Above diagonal shows pairwise plot; below diagonal gives corresponding correlation coefficient.

The narrow-sense heritability for the resistance trait was estimated at 0.49 ± 0.04 . This implies the observed phenotypic variation is influenced by genetic factors and indicates that the trait can be improved by phenotypic selection.

BLUPs are considered important for determining progeny worth or a progeny's genetic value for use in breeding programs. The suitability of progeny as parents can be decided from their individual predicted breeding values (Fig. 4). In our population, the individuals in the families 2772, 2775, and 2786 have the highest predicted breeding values for resistance and would make the best parents. The most likely best future parents in generating resistant progeny include: 2772-14, 2772-15, 2772-05, 2786-01, and 2775-30. There were many progeny that were predicted to have a better breeding value than any of the parents. These included several progeny from the family 2775. For example, progenies 2775-30, 2775-22, and 2775-13 have breeding values of 0.82, 0.88, and 0.95, respectively, whereas their parent 'Festival' and 'Sugarbaby' have breeding values of 2.04 and 2.42, respectively.

Several of the progeny most suitable as future parents are the result of self-pollination (e.g., 2772-14, 2772-15, and 2772-05 from 'Festival' and 2786-01 from 'Sugarbaby'). This suggests different loci may be involved in the cultivars Festival and Sugarbaby and that the size of the progeny was not large enough to fully capture the range of recombination between the loci involved in these two parents. It is reasonable to expect that advantageous transgressive segregants will arise with recombination of additive genes among these parents. In support of this, the moderate heritability (0.49) implies substantial additive gene action. Self-pollination to recover transgressive segregants, by concentrating alleles, may hold promise for improving the breeding values of parental lines. In this case, improvements in breeding lines could be made using smaller populations.

The identification and incorporation of host plant resistance into susceptible plants is a highly desirable objective for many breeding programs (Maas and Galletta, 1989). The high

additive variance in our population, together with the genotypes with low (desirable) predicted breeding values and high heritability in our evaluation system, will allow us to continually select for the very best performers and provide the basis for our resistance breeding program and lead to reduced losses to *Fof* in Australia.

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