Unravelling the Genetics of Fusarium Wilt Resistance in Cotton

L. AUGUSTO BECERRA LOPEZ-LAVALLE, BRONWYN MATHESON, WALTER TATE, HELEN McFADDEN, GREG REBETZKE & CURT L. BRUBAKER

CSIRO Plant Industry, GPO Box 1600, Canberra ACT 2601, Australia

Introduction

Despite its widespread occurrence, the genetics of fusarium wilt resistance in cotton has still not been clearly elucidated. This is a result, in part, of the complex phenotyping involved, but also, in some cotton growing regions, the confounding effects of root knot nematode interactions with the severity of fusarium wilt symptoms. More importantly, the genetic relationships among fusarium wilt pathogens around the world are complex, suggesting that a single genetic model may not be applicable universally. To elucidate the genetics of fusarium wilt resistance against the Australian fusarium wilt pathogens (*Fusarium oxysporum* f.sp. *vasinfectum* VCGs 00011 & 00012), two genetic families have been used. The purpose of this paper is to present evidence that *G. Sturtianum* and *G. barbadense* harbour genes of interest for the genetic improvement of cultivated cotton.

Material and Methods

Plant material and experimental design

Plants were selected from two genetic populations. The first population, 46 BC_1 MACAL families carrying 0 to 4 C-genome chromosome additions were challenged with *For*. Accession CPI-138969 was included in every experiment as a susceptible control. The second population, 266 F_2 from a cross *G. hirsutum* x *G. barbadense* were used to generate a 20 cM framework cotton map. Of the 266 F2 lines, 162 F_3 families were challenged with *For* in a series of two glasshouse trials. Each trial comprised 3,600 plants arranged in a row x column plot design including 900 Siokra 1-4 (*For* susceptible industry standard), 900 Sicot 189 (*For* resistant industry standard), 900 Sicot F-1 (best *For* resistant cultivar), and 900 *G. barbadense* (*For* resistant) as *For* disease controls. In both genetic populations the extent of fusarium wilt resistance was measured by the vascular browning index (VBI). Only the F3 families were also access for foliar and general appearance (FGA).

Logistic regression on BC_1 families

The association between FWR and the presence of C-genome chromosomes in the *G. hirsutum* background was evaluated using binomial logistic regression. The binomial logistic regression was used to model the relationship between resistant and susceptible fusarium wilt symptoms ["0"=resistant (VBI 0&1), "1"=susceptible (VBI 4&5)] and a set of explanatory variables (*e.g.*, the *G. sturtianum* chromosomes or part thereof present in each individual).

Estimation of heritability and genetic correlation

Variance components were used to estimate heritability on an entry-mean basis and defined in the narrow-sense (h2). Confidence intervals were obtained for heritability estimates. Genetic covariance estimates were obtained and used with genetic variance estimates to obtain genetic correlations among selected traits. Standard errors for these estimates were calculated.

QTL mapping

Linkage or recombination frequencies between markers were calculated using MAPMAKER/ EXP Version 3.0 QTLs linked to markers for *For* resistance were determined using the mixed-model based composite interval mapping method of QTL Network software.

Figure 1: Frequency distribution of quantitative resistance to Fov in the F3 progeny derived from the cross GB (8810) x Siokra 1-4. Phenotypic values are shown on the x=axis. The position of the mean resistance value of the parents and the industry-standard for Fow resistence are indicated by blue, red and orange arrows, respectively

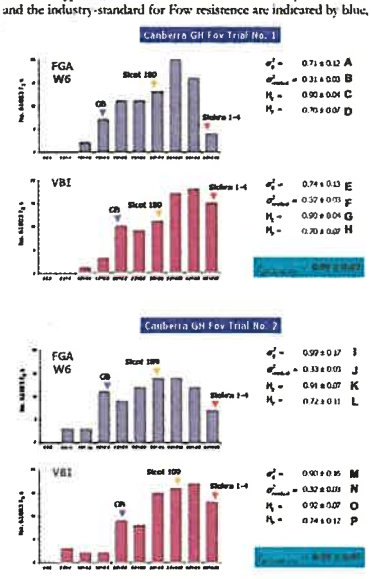
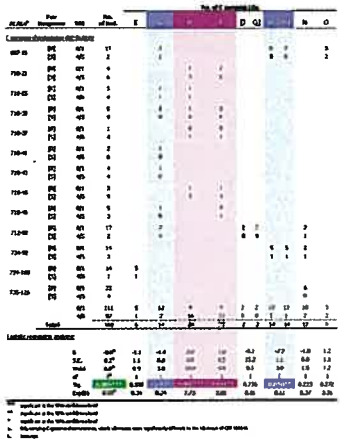


Table 1: C-genome alien addition lines, assigned *For* disease response, distribution of *G. sturtianum* chromosomes per *For* disease response category, and results of logistic regression analyses showing *G. sturtianum* chromosomes significantly associated with *For* wilt symptoms.



Results and Discussion

BC_1 MACALs

Approximately, 168 BC_1s individuals were used to determine if the enhanced *For* resistance observed in the 13 BC_1 MACALs could be attributed to eight specific LGs Sturt-E, -G, -H, -I, -J&Q, -K&P, -N or –O. A binary logistic regression based on model *For* disease resistance and susceptibility using C-genome-specific chromosome inheritance as predictor variables was used. This model correctly classified 72% of the 168 lines by giving the probabilities of contribution of the Sturt-LGs to resistant and susceptible fusarium wilt symptoms as shown in Table 1. Linkage groups Sturt-G and Sturt-KP were significantly associated with *For* resistance, while LGs Sturt-H and Sturt-I were associated with *For* disease susceptibility. The fact that multiple C-genome chromosome segments (Sturt-LGs) were associated with fusarium wilt resistant and susceptible symptoms in the BC_1 MACALs support the conclusion that fusarium wilt resistance in cotton is multigenically controlled. It is clear that it will not be possible to transfer these genes to cultivated cottons by traditional breeding means (data not presented), and that we need to adopt more technically complex methods. The next step in this process is to undertake a quantitative trait loci (QTL) analysis using an experimental breeding population derived from a cross between susceptible and resistant parents. This will identify specific genomic regions in the cotton genome were import genes for fusarium wilt resistance are located and mark their location with molecular markers.

Fov resistance in cultivated cotton

A total of 8,100 F_2 individuals have been tested for "foliar & general appearance" (FGA) and "vascular browning index" (VBI) in the glasshouse. The distribution of *Fov* disease symptoms for trial#1 and trial#2 are shown in Figure1. The resistance levels of the parentals (GB: resistant & Siokra 1-4: susceptible) and the industry standard Sicot 189 are also indicated on Figure 1. On these distributions, the resistant parent (GB) was positioned at the left hand end of the distribution, whereas the susceptible parent (Siokra1-4) was positioned at the right hand end of the distribution indicating a good phenotypic separation of the two parental cultivars. High genetic correlations were observed between FGA and VBI in trial#1 ($r=0.99 \pm 0.03$) and trial#2 ($r=0.99 \pm 0.07$). Broad-sense heritability was estimated for FGA and VBI. Heritabilities were high for both traits in both trials, indicating a small genotype x environment interactions ($H>0.90$). Genotypic variance components were large and significantly different from zero ($P<0.05$) for FGA (Fig. 1A and 1I) and VBI (Fig. 1E and 1M). In contrast, the genotype x environment interaction variance were typically smaller than the genotypic variance for FGA (Fig. 1B and 1J) and VBI (Fig. 1G and 1N). The large broad-sense heritabilities for FGA and VBI and the high genetic correlations between environments indicates that a large component of the genotype x environment interaction for FGA and VBI reflected changes in the magnitude of the genetic variance in each environment with only small changes in genotype ranking. Hence, genotypes effects on Fusarium wilt resistance were large and robust owing to weal interactions with environment

Identification of quantitative trait loci (QTLs)

A preliminary analysis to identify QTLs linked with Fusarium wilt resistance in cultivated cotton was undertaken. Overall, the correlation of phenotypic and genotypic variances in 61003 F_3 and F_2 allowed the identification of 2 QTLs associated with Fusarium wilt resistance. A QTL with large effect in FGA (35%) was detected on LG.15 (Chr.1 or Chr.15). A second QTL, which explain an additional 22% of the phenotypic variation in FGA, was identified on LG.D08. These two loci together explain 57% of the FGA phenotypic variation among lines. For VBI, a QTL with a large effect (29%) was also found in the same location on chromosome LG.15. A second QTL, which explain an additional 19% of the phenotypic variation in VBI, was identified on LG.D08. These two loci together explain 48% of the VBI phenotypic variation among lines.

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