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New Genes for the Battle against Sorghum Ergot

[1,2]Damian J. Herde, [3]David R. Jordan, [3]Robert G. Henzell, [4]Scott D. Foster, [2]Malcolm J. Ryley and [1]Victor J. Galea.

[1] School of Agriculture and Horticulture, University of Queensland Gatton Campus, QLD 4343; [2] Queensland Department of Primary Industries, 203 Tor St, Toowoomba QLD 4350; [3]Queensland Department of Primary Industries, Hermitage Research Station, Warwick QLD 4370; [4]Queensland Department of Primary Industries, Centre for Tropical Agriculture, Mareeba QLD 4880.

Abstract

Sorghum ergot (*Claviceps africana*) remains a serious threat to the Australian sorghum industry since it's discovery in 1996. This is due to ongoing toxicity issues, narrowing of the planting window, and increased cost of hybrid seed production. Successful pollination ("escape" resistance) is the only means of avoiding disease in susceptible lines. Ergot resistance is reported to show quantitative inheritance with considerable genotype-environment interaction. This paper reports on studies carried out to establish the inheritance of ergot resistance and related traits in F1, F2 and BC1F1 populations derived from crosses between the resistant germplasm line IS8525 and two susceptible inbred lines (31945-2-2 and 60535-2-2). Significant levels of dominance were observed for resistance. This result, combined with frequency distribution data from the F2 and BC (data not presented), indicates the possibility of major gene action, an unexpected but beneficial discovery. Pollen traits were also studied, with significant dominance found towards decreased pollen viability. Despite this, ergot resistance was undiminished, leading to the conclusion that resistance in IS8525 is a non-pollen based mechanism, which remains to be identified.

Introduction

C. africana is a non-systemic floral pathogen, which infects unfertilised florets, replacing the developing ovary with a fungal mass (sphacelium). Sticky honeydew oozes from infected spikelets, carrying spores for further infections.

IS8525 has been tested in a number of countries (Australia, South Africa and the USA), and shown very high levels of ergot resistance across a range of environments (Dahlberg *et al.*, 2001). Experimental work was undertaken to determine the ease of breeding ergot resistant sorghum lines based on IS8525.

Pollen traits need consideration, as poor pollen viability or quantity will lead to poor fertilisation and increased ergot infection (McLaren, 1997). However, ergot resistance that depends on pollen cannot be incorporated into a male sterile line for use in hybrid seed production. Ergot studies have included close scrutiny of pollen traits to determine whether this is the source of resistance in IS8525.

Materials and Methods

Trial design and set-up:

A generation means design was followed to produce two populations (Kearsy and Pooni, 1996). The first population was based on IS8525 (ergot resistant), and 31945-2-2 (ergot susceptible), while the second was based on IS8525 and 60535-2-2 (moderately ergot susceptible). Each population consisted of two homogeneous parents, the F1 cross between these parents, the F2 generation produced by self pollination of the F1, and two backcross generations (BC1 and BC2) developed by crossing the F1 to each parent. Control plots were included.

Panicles were artificially inoculated with a conidial suspension ($1x10^6$ conidia/ml) at 50% anthesis, and non-flowering spikelets removed. Pollen quantity was assessed visually (score from 0 to 10, 0 = no pollen, 10 = abundant pollen); pollen viability (%) was scored by staining pollen grains with 0.25% iodine solution and assessing microscopically; and ergot severity was visually assessed three weeks after inoculation (% infected spikelets/panicle).

This experiment was conducted at Hermitage Research Station, Warwick Qld. There were three planting dates, with two replications per planting date.

Analysis

A mixed model using smoothing splines (Verbyla, 1995) was fitted to each of the three outcome variables (ergot percentage data was arcsine square root transformed prior to analysis). These models accounted for possible flowering date effects as well as any possible correlations induced by repeatedly measuring the same plots (Verbyla *et al.*, 1999). Sorghum Line by time interactions were also considered in the mixed model. Genetic mean parameters were estimated using the line main fixed effects calculated at the average time.

The convention of using the susceptible line as the P1 parent (Kearsy and Pooni, 1996) has been used. Caution should be taken when interpreting the results as this will not always be the parent with the higher phenotype value.

Results

As results for the two populations show similar trends, only results for the first population (IS8525 and 31945-2-2) are displayed.

Table 1 displays the outcome of the Kearsy and Pooni (1996) analysis. As the susceptible line is always the P1 parent (by definition) then a value of [d] which has the

same sign as the corresponding [a] shows dominance towards the susceptible line. Conversely an opposite sign shows dominance by the non-susceptible line.

Table 1. Estimated values for genetic mean parameters and respective p values (for tests against zero) for the population of IS8525 and 31945-2-2.

Mean Parameter	Transformed % Ergot	% Pollen Viability	Pollen Quantity (0-10)
Parent Mean, m	23.73 ($p<0.001$)	71.38 ($p<0.001$)	1.61 ($p<0.001$)
Additive genetic component of means, [a]	13.35 ($p<0.001$) [A]	-4.08 ($p=0.003$) [B]	-0.48 ($p=0.006$) [B]
Dominance genetic component of means, [d]	-11.55 ($p=0.002$) [B]	-25.94 ($p<0.001$) [A]	1.84 ($p=0.002$) [B]
Dominance Ratio [d]/[a] [C]	-0.87	6.37	-3.79
Fit of Model (p value) [D]	0.12	0.86	0.34

[A] Value towards 31945-2-2.

[B] Value towards IS8525.

[C] Value of 1 or –1 shows complete dominance.

[D] P value >0.05 indicates the model fits assumptions of additive and dominance gene effects.

The first main conclusion from Table 1 is that dominance is significant for ergot resistance and pollen quantity, despite the significant dominance for decreased pollen viability. Another important conclusion is that the observed traits are explained sufficiently by additive and dominance gene effects, pointing towards no maternal effects or interactions between genes (epistasis).

Discussion

Additional analysis (data not presented) also reinforce the conclusion that ergot resistance in IS8525 is a major gene effect rather than being quantitative in nature.

The strong dominance for ergot resistance will be very useful in the production of sorghum hybrid seed. There need only be one parent containing resistance genes to produce resistant hybrids.

Future work will provide predictions of ergot resistance heritability from IS8525, and determine the mechanisms(s) responsible.

Acknowledgements

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