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Impact of *Cre1*, *Cre8* and *Cre3* genes on cereal cyst nematode resistance in wheat

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Abstract The cereal cyst nematode (CCN; *Heterodera avenae*), a root disease of cereal crops, is a major economic constraint in many wheat (*Triticum aestivum*)-growing areas of the world. The objective of this study was to assess the impact of the *Cre1*, *Cre8* and *Cre3* genes on CCN resistance. A population of 92 doubled-haploid (DH) lines derived from a cross between wheat cvs. Frame and Silverstar as well as 1,851 wheat breeding lines were screened for CCN resistance at the Primary Industries Research Victoria (PIRVic). A second population of 9,470 wheat breeding lines was screened at the South Australian Research and Development Institute (SARDI). *Cre3* had the largest impact on reducing the number of female cysts, followed by *Cre1* and *Cre8*. There was no significant difference in number of cysts between DH lines with or without the *Cre8* marker, suggesting that the marker is not perfectly linked to *Cre8*. The estimated heritabilities were 0.32 in the DH population, 0.48 in the PIRVic data set and 0.32 in the SARDI data set, which confirm that this is a trait of low heritability. The repeatability of CCN resistance improved with an increase in the number of plants assessed per line—up to ten. However, 85–88% of the improvement was achieved with the assessments of the first five plants.

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Introduction

The cereal cyst nematode (CCN; *Heterodera avenae*), a root disease of cereal crops, is a major economic constraint in many wheat (*Triticum aestivum L.*)-growing areas of the world. In south-eastern Australia, CCN accounts for 8% of the annual yield loss (Eastwood et al. 1991). Consequently, breeding for resistance to CCN has been a major objective of wheat breeding programmes in this area since the 1980s (Rathjen et al. 1998).

To incorporate resistance to CCN (capacity to prevent reproduction of the nematode) into commercial cultivars, breeding programmes have traditionally relied on a biological assay. However, due to poor reliability and the high cost of the bioassay (Rathjen et al. 1998; Eagles et al. 2001), marker-assisted selection (MAS) has been advocated as an alternative and efficient selection tool (Tanksley et al. 1989; Eagles et al. 2001).

Several genes in both hexaploid wheat and its relatives have been identified as sources of resistance to CCN. A gene designated *Cre1* on chromosome 2B was identified in the wheat landrace AUS10894 (Slootmaker et al. 1974) and subsequently widely used across Australia and Europe to incorporate resistance into new cultivars. A second gene, *Cre8*, identified in the Festiguay cultivar (Paull et al. 1998), has also been used as a source of CCN resistance in south-eastern Australian wheat breeding programmes. A third dominant gene, *Cre3*, located on chromosome 2D, was identified in *Aegilops tauschii* (Eastwood et al. 1991) and was recently introduced in the wheat breeding programmes of south-eastern Australia.

The ability to predict the outcome of a proposed breeding programme using these genes to breed resistant cultivars requires a knowledge of the effect of each

individual gene and their combined effects as well as their contributions to the total genetic variance. Anecdotal evidence suggests that while the introduction of two or more CCN resistant genes into a single cultivar may enhance the degree of resistance and reduce the probability of resistance breakdown, the expression of resistance is affected by the genotypic background. However, there is no information available on this topic in the literature other than the work of Ogbonnaya et al. (2001a), which dealt with the impact of *Cre1* and *Cre8* on the inhibition of female nematode reproduction.

The aim of the study reported here was to assess the relative importance of the *Cre1*, *Cre8* and *Cre3* genes in CCN resistance. Genetic components for the variability of resistance to CCN and the number of plants required to be assessed to identify resistant lines in applied wheat breeding programmes were also estimated.

Materials and methods

Genetic materials

The genetic material used in the first part of this study was a population of 92 doubled-haploid (DH) lines derived from a cross between wheat (*Triticum aestivum*) cultivars Frame and Silverstar. These lines were assessed in Primary Industries Research Victoria (PIRV). Frame and Silverstar are known to carry the *Cre8* and *Cre1* resistance genes, respectively (Ogbonnaya et al. 2001a).

In the second part of the investigation, data were obtained as part of applied breeding programmes on a wide range of genetic materials, including introductions, released cultivars and unreleased breeding lines, with the overwhelming majority of the data from breeding lines. In the following sections we refer to all of the genetic material as lines. For these analyses data were obtained on 9,470 lines from 840 different pedigrees assessed in the South Australian Research and Development Institute (SARDI) over a 5-year period (1997–2000) and on 1,851 lines from 310 different pedigrees in PIRV assessed over a 4-year period (1998–2001).

The PIRV and SARDI data were used to classify the lines into different genotypic groups(*Cre1*, *Cre8*, *Cre3*, *Cre1*/*Cre8*, *Cre1*/*Cre3*, *Cre8*/*Cre3*, and those without resistant genes) based on pedigree and the source of the resistance genes. The lines were designated to the different genotypic groups using both breeding and marker information. These data were also classified according to parent lines, based on the pedigree of the donors of the respective resistance genes. Lines that could not be unambiguously classified were excluded from the analyses.

Identification of resistance genes

Alleles for resistance at *Cre1* and *Cre8* loci in the DH population were identified using the diagnostic DNA methods described by Ogbonnaya et al. (2001b). The genomic and cDNA clones used as probes for the analysis of *Cre1*—*G*4, *G*12 and *CD*2.2—were kindly supplied by Dr. Evans Lagudah (CSIRO Plant Industry, Canberra-ACT, Australia). The diagnostic DNA marker, *Xcdo*347, described by Paull et al. (1998) was used for identifying lines with *Cre8*. In addition, lines carrying alleles for tolerance to boron at the *Bo1* and *Bo3* loci and glutenin alleles at different loci were identified by the procedure described by Eagles et al. (2001).

Screening for CCN resistance

Doubled-haploid population and PIRV data

The inoculum used was a CCN-infested, sandy-loam soil collected from a field plot in Rainbow, Victoria, with an initial population of approximately 20 eggs per gram. The soil was collected after harvest and before a rainfall, and stored air-dry in a bin until used. Before being used as inoculum, the nematodes in the soil were hatched by moistening the soil, mixing it thoroughly so that it was damp but friable and then storing the moistened soil in 45-l plastic bins for 6 weeks at 15°C. The soil was then placed in pots (285–300 g per pot), and seeds (pre-germinated on filter paper at 24°C) were planted in the potted soil (one seed per pot). The pots were placed in a growth cabinet maintained at 15°C under artificial light and a 12/12-h (light/dark) photoperiod in a completely randomised layout for 6 weeks before being transferred to a greenhouse (temperature range: 15–25°C) for a further 6 weeks. At least five plants per genotype were used in the CCN bioassay, which consisted of the soil being carefully washed from the roots and the resistance subsequently being assessed by counting the number of white female cysts on the roots of each plant using a stereomicroscope at a 6.4× magnification. If the number of white female cysts was above 50, it was classified as 50.

SARDI data

The CCN inoculum was a mixture of inoculum concentrate containing brown cysts (300 eggs per cyst) mixed with air-dried Tailem Bend Sandy Loam (TBSL) soil to a density of 25 eggs per gram. CCN-laden soil was obtained from a heavily infested field at Pinery, South Australia. Gadac p2 black tubes (length: approx. 10 cm; diameter: 5 cm at the top, tapering to 4 cm at the bottom) were filled with inoculum prior to seeding. These tubes were placed in wire crates in a five row by ten column arrangement. Seeding took place during June, and the crates were placed on terraced plant-beds to grow under natural conditions supplemented by irrigation. Resistance was assessed during October and early November by counting the number of white females on the outside of the rootball using a

stereomicroscope at a 6.4× magnification. If the number of female cysts was more than 21, it was classified as 21.

Data analysis

A square root transformation was applied to the data to remove mean-variance dependence. Following transformation the number of cysts was analysed using the linear mixed model method. Results were back-transformed into the original scale and presented both in square root and original scales. The initial model for the DH population included *Cre1*, *Cre8*, *Bo1*, *Bo2*, *Glu-A1*, *Glu-B1* and *GluD1* as fixed factors and line identification as a random factor. Since the effects of *Bo1*, *Bo2* and the glutenin genes were not significant, they were dropped from the final model.

Three types of statistical models were used. Year, line and genotypic classification were included in Model 1. In Model 2, pedigree was fitted as another random factor, while in Model 3, origin of resistance was fitted as an extra random factor. In all of the models genotypic classification was fitted as fixed, with year and line as random factors. All data were analysed on a per-plant basis, and the intraclass correlation was defined as the ratio of variance components due to line to total variance, calculated from the model which included year and line as random effects. ASREML (Gilmour et al. 1999) was used for all the analyses.

Results

DH population

No significant differences were observed between plants with or without the *Cre8* marker (Table 1), indicating that the *Cre8* gene has little effect on the number of cysts. Conversely, the *Cre1* gene significantly ($P < 0.001$) reduced the number of cysts. There was no significant interaction between the *Cre1* and *Cre8* genes. The intraclass correlation for the number of cysts was estimated to be 0.32 ± 0.05. Repeatability of number of cysts increased from 0.32 to 0.82 with assessment of ten plants (Fig. 1). Although repeatability was improved up to ten plants, the rate of improvement was very small after seven plants, indicating that any gain achieved with assessing more than seven plants is negligible. *Bo1*, *Bo2* and the glutenin protein profiles (*Glu-A1a*; *Glu-B1b*, *Glu-B1c*, *Glu-B1I*; *GluD1a*, *GluD1d*; *GluA3c*; *GluB3b*, *GluB3h*; *GluD3c* and *GluD3b*) did not have a significant effect on the mean of the resistance genotypes, and the results of these analyses are not presented here. These genes are all located on homologous chromosomes 1 (Gupta and Shepherd 1990), suggesting that genes on these chromosomes do not influence resistance to CCN.

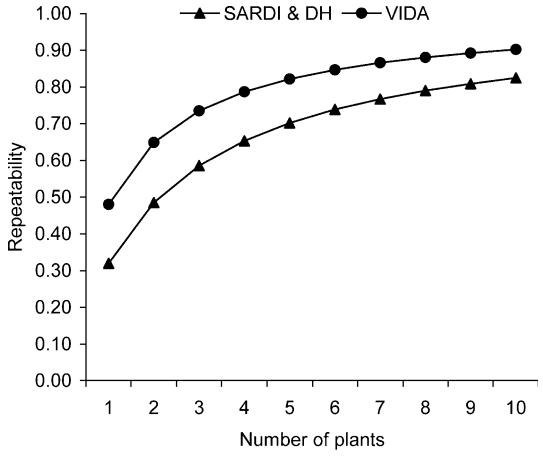

Fig. 1 Repeatability of number of cysts in relation to number of plants assessed

PIRV data

There were significance differences among the genotypes for the number of cysts (Table 2). Plants without a resistance gene had 93%, 41% and 12% more cysts on their roots than their counterparts with the *Cre3*, *Cre1* and *Cre8* gene, respectively. While the mean number of cysts for plants with the *Cre8* gene was not significantly different from that of plants without a resistance gene, the means for all other genotypes were significantly ($P < 0.01$) different from each other. A similar pattern of genotype effects was observed in Models 1, 2, and 3, with one exception. In Model 3, the inclusion of the lines that provided the resistant gene increased the difference between plants with the *Cre8* gene and those with no gene to 35%, which was then a significant difference ($P < 0.01$). While plants with two genes (*Cre1*/*Cre3* and *Cre1*/*Cre8*) were significantly ($P < 0.05$) different from

Table 1 Number of observation (*n*) and least squares means ± standard errors for the number of CCN cysts in square root and original scales on roots of DH lines derived from a cross between cvs. Silverstar and Frame

Cre8	*Cre1*					
	Absence			Presence		
	n	Square root	Original scale	*n*	Square root	Original scale
Absence	125	4.30 ± 0.23	18.52 ± 1.97	64	3.35 ± 0.27	11.24 ± 1.84
Presence	147	4.60 ± 0.21	21.14 ± 1.96	104	3.06 ± 0.24	9.35 ± 1.48

Average standard error of difference = 0.32

Table 2 Number of observations (*n*) and least squares means ± standard errors for the number of CCN cysts in square root and original scales for six genotypes included in the PIRV data[a]

Genotype	*n*	Model 1		Model 2		Model 3	
		Square root	Original	Square root	Original	Square root	Original
Cre3	192	2.78 ± 0.27e	7.75 ± 1.48	2.54 ± 0.39d	6.48 ± 2.00	2.64 ± 0.34d	7.00 ± 1.81
Possibly *Cre1*/*Cre3*	56	3.34 ± 0.42d,e	11.17 ± 2.90	3.20 ± 0.52c,d	10.27 ± 3.34	3.49 ± 0.50c,d	12.20 ± 3.52
Cre1	6,451	3.80 ± 0.16c,d	14.47 ± 1.18	3.91 ± 0.15b,c	15.29 ± 1.21	3.85 ± 0.19b,c	14.87 ± 1.48
Possibly *Cre1*/*Cre8*	85	4.16 ± 0.3b,c	17.31 ± 2.90	4.31 ± 0.55b	18.59 ± 4.75	4.49 ± 0.57b	20.14 ± 5.13
Cre8	963	4.78 ± 0.18a,b	22.93 ± 1.71	4.61 ± 0.22a,b	21.30 ± 2.04	4.04 ± 0.32b,c	16.35 ± 2.60
No gene	274	5.36 ± 0.33a	28.77 ± 3.57	5.45 ± 0.34a	29.75 ± 3.75	5.47 ± 0.33a	29.93 ± 3.67
S.E.d[b]		0.35		0.50		0.48	

[a]Estimates followed by the same letter differ by less than twice the standard error of the difference
[b]S.E.d, Average standard error of difference

each other, they were not different from plants with either gene.

The intraclass correlation was estimated to be 0.48 ± 0.02 from Model 1. The repeatability of CCN resistance increased from 48% to 90% with the assessment of ten plants per line (Fig. 1). However, 88% of the improvement was achieved by the assessment of the first five plants, and improvement after the seventh plant was negligible.

SARDI data

There were significant differences among the genotypes for number of cysts (Table 3). Plants without any resistant gene had 64%, 57%, and 49% more cysts on their roots than their counterparts with the *Cre3*, *Cre1* and *Cre8* gene, respectively. The only significant ($P < 0.05$) difference between plants with a single gene was between *Cre3* and *Cre8*. While plants with *Cre1*/*Cre3* and *Cre1*/*Cre8* were not significantly different from each other, they were significantly ($P < 0.01$) different from *Cre8*/*Cre3* plants and all of the plants with a single gene. A similar pattern of genotypic effects was observed in all models, and the introduction of the source of resistance into the model had no impact on this pattern, in contrast to what occurred with the PIRV data.

The intraclass correlation was estimated to be 0.32 ± 0.02 from Model 1. The repeatability of CCN resistance increased from 0.32% to 82% with the assessment of ten plants per line (Fig. 1). However, 85% of this improvement was achieved by the assessment of the first five plants, with negligible improvement after plant eight.

Discussion

Cre3 had the largest impact on reducing the number of female cysts followed by *Cre1* and *Cre8*. The outcome of both analyses is therefore consistent with the earlier observations of Ogbonnaya et al. (2001a) who also ranked *Cre1* higher than *Cre8* in terms of inhibiting female nematode reproduction. However, contrary to results based on the PIRV data, the differences between *Cre3* and *Cre1*, and *Cre1* and *Cre8* were not statistically significant in the SARDI data. This may be due to a wider genetic background in the lines evaluated at SARDI than in those at PIRV or to temperature differences between the SARDI and PIRV assays. Modifications in the expression of CCN resistance genes in different genetic backgrounds have previously been observed in wheat, although they have not been documented. There is also circumstantial evidence that high

Table 3 Number of observations (*n*) and least squares means ± standard errors for the number of CCN cysts in square root and original scales for seven genotypes in the SARDI data[a]

Genotype	*n*	Model 1		Model 2		Model 3	
		Square root	Original	Square root	Original	Square root	Original
Cre3	1,416	1.79 ± 0.17b	3.19 ± 0.64	1.71 ± 0.21b,c	2.92 ± 0.73	1.82 ± 0.22b,c	3.32 ± 0.82
Possibly *Cre1*/*Cre3*	1,329	1.46 ± 0.17c	2.13 ± 0.50	1.39 ± 0.22d,e	1.94 ± 0.60	1.49 ± 0.22c,d	2.24 ± 0.65
Cre1	45,766	1.86 ± 0.17b	3.48 ± 0.63	1.88 ± 0.19b	3.53 ± 0.70	1.87 ± 0.19b	3.50 ± 0.70
Possibly *Cre1*/*Cre8*	5,809	1.45 ± 0.17c	2.12 ± 0.52	1.45 ± 0.20c,d	2.09 ± 0.57	1.42 ± 0.20d,e	2.02 ± 0.56
Possibly *Cre8*/*Cre3*	203	1.16 ± 0.23d	1.34 ± 0.54	1.11 ± 0.31e	1.23 ± 0.69	1.08 ± 0.30e	1.17 ± 0.66
Cre8	7,115	1.97 ± 0.17b	3.88 ± 0.67	2.00 ± 0.19b	3.99 ± 0.77	2.02 ± 0.20b	4.10 ± 0.79
No gene	2,950	2.93 ± 0.18a	8.58 ± 1.04	2.75 ± 0.23a	7.55 ± 1.26	2.79 ± 0.23a	7.78 ± 1.28
S.E.d[b]		0.09		0.16		0.17	

[a]Estimates followed by the same letter differ by less than twice the standard error of the difference
[b]S.E.d, Average standard error of difference

temperatures may weaken the resistance in wheat (Cook and McLeod 1980). Consequently, one likely explanation for the differences between the PIRV and SARDI data is that plants in the former assay, which were kept under glasshouse conditions, experienced higher temperatures than those in the latter assay, which were in the field. Person and Doussinault (1979) suggested that the resistance of a barley genotype to French Pathotype 2 might be overcome at 23–25°C.

In the PIRV data, plants with both genes (*Cre1*/*Cre3*, *Cre1*/*Cre8*) were significantly ($P < 0.05$) different from each other, and their performances were close to the average performance of the contributing single genes, suggesting that the presence of both genes was not able to enhance the degree of resistance. On the other hand, in the SARDI data, plants with both genes performed significantly ($P < 0.01$) better then plants with either gene alone. Only the performance of the *Cre3*/*Cre8* plants was significantly ($P < 0.01$) better than plants with both genes, an outcome consistent with the epistatic effect of genes, assuming that these genes occur at independent loci (Ogbonnaya et al. 2001a). This may also be the product of a greater genetic diversity in the SARDI data, with the 840 different pedigrees providing, in addition to *Cre1*, *Cre8* and *Cre3*, many more genes with small effects acting on resistance to CCN.

Outcomes from the analyses of both data sets confirmed that all three genes and their joint actions significantly reduced the number of cysts. The statistically nonsignificant differences between plants with the *Cre8* gene and plants without any resistance gene in Models 1 and 2 of the PIRV data were due to the influence of the Bowie cultivar, which provided more than 80% of the plants since following the introduction of origin of the resistance gene in Model 3 the difference between plants with *Cre8* and plants without any resistance gene became significant ($P < 0.01$). An analysis of a subset of PIRV data with the *Cre8* genotype also showed a significantly ($P < 0.05$) higher number of cysts in plants with Bowie background than in plants without the Bowie background (4.65 vs. 4.20). The perception among plant breeders that the effect of the *Cre8* gene is always weaker in the Bowie background is thereby confirmed here.

Lack of support for the effectiveness of the *Cre8* gene in the DH population suggests that either the *Cre8* marker is not tightly linked or that the *Cre8* gene is not able to express itself in the Silverstar background. Fisher et al. (1981) reported that Festiguay, the source of *Cre8* in Frame, expresses both resistance and tolerance to CCN and that these traits are linked in this germplasm. These researchers defined tolerance as the ability to maintain yield despite high CCN infection levels. According to Williams et al. (personal communication) either one gene or more than one gene, all closely linked, are present at the *Cre8* locus affecting both resistance and tolerance to CCN; as well there are extra modifier genes needed for the full expression of resistance and tolerance. The results of analyses of both the PIRV and SARDI data suggest that the *Cre8* marker is loosely linked to the *Cre8* gene, since plants carrying the gene did have a reduced numbers of female cysts compared to plants without any gene in both data sets. However, given the large DH population size, the impact of genetic background (Bowie) seems more plausible, an outcome which supports the finding of Williams et al. (personal communication).

The difference in numbers of cysts between the presence and absence of the *Cre1* marker in the DH population was 28%, while the difference between lines with and without the *Cre1* gene in the PIRV and SARDI data was 41% and 63%, respectively. This difference might be a reflection of the fact that the impact of a specific gene can be diluted or enhanced by the pleiotropic action of other genes. Part of the failure of MAS compared to phenotypic selection (Van Berloo and Stam 1999) may be due to the fact that the selective value of a gene is a product of both the gene and its founding genetic background.

The higher estimated heritability for CCN resistance in the PIRV data (0.48) compared to the SARDI data (0.32) was expected since most of the PIRV lines were more advanced in terms of genetic structure than the SARDI lines. In the present study, all of the genotypes were assumed to be known without error. Nevertheless there is always the probability that some of the genotypes, as a result of segregation, were misclassified as carriers of resistant genes, and this would lead to an underestimation of the magnitude of the effects of the resistance genes. However it is difficult to determine the probability of misclassification in a diverse and complicated pedigree as our data sets.

In plant breeding, gene effects are generally estimated from experiments specifically designed for that purpose. The cross between Frame and Silverstar is an example of such an experiment. However, large plant breeding data sets provide an alternative. Eagles et al. (2002) showed that data from the wheat breeding programmes in southern Australia can be effectively used to estimate the effects of glutenin alleles on key quality traits. We have extended this to show that such data sets can be used to estimate the effects of genes for resistance to CCN. In this analysis we were also able to identify specific background effects, such as Bowie with the *Cre8* gene. For traits which are expensive to assess, such as resistance to CCN and for glutenin traits, the analysis of plant breeding data provides a powerful and practical alternative to designed experiments.

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