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Y. Z. Tao · A. Hardy · J. Drenth · R. G. Henzell · B. A. Franzmann · D. R. Jordan · D. G. Butler · C. L. McIntyre

Identifications of two different mechanisms for sorghum midge resistance through QTL mapping

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Abstract Sorghum midge is the one of the most damaging insect pests of grain sorghum production worldwide. At least three different mechanisms are involved in midge resistance. The genetic bases of these mechanisms, however, are poorly understood. In this study, for the first time, quantitative trait loci associated with two of the mechanisms of midge resistance, antixenosis and antibiosis, were identified in an RI (recombinant inbred) population from the cross of sorghum lines ICSV745 × 90562. Two genetic regions located on separate linkage groups were found to be associated with antixenosis and explained 12% and 15%, respectively, of the total variation in egg numbers/spikelet laid in a cage experiment. One region was significantly associated with antibiosis and explained 34.5% of the variation of the difference of egg and pupal counts in the RI population. The identification of genes for different mechanisms of midge resistance will be particularly useful for exploring new sources of midge resistance and for gene pyramiding of different mechanisms for increased security in sorghum breeding through marker-assisted selection.

Keywords Sorghum · Midge resistance · QTL mapping · Marker-assisted selection

Introduction

The development of cultivars resistant to insect pests is a very important task in plant improvement. Although there have been many notable successes in conventional breeding for improved plant resistance to insects, the breeding process is often slow and laborious, and sufficient levels of resistance have not been achieved for some pests because of the fact that plant resistance to insects is most often a quantitatively inherited trait. Strong effects of both the environment (light, wind, temperature, plant and insect nutritional status, etc.) and the genetic variability within insect pest populations on the assessment of bioassays have resulted in a high degree of genotype-by-environment error (Smith 1989; Smith et al. 1994).

Molecular markers and quantitative trait locus (QTL) mapping offer entomologists and plant breeders a more efficient approach for working with quantitative traits, whether or not specific genes or gene products are known (Tanksley 1993). In addition, since molecular markers can be employed to analyse any quantifiable phenotypic trait, biochemical and/or physical mechanisms and direct measures of insect resistance can be simultaneously mapped. Comparisons of the resulting QTLs identified for each trait would then increase our understanding of genetic and physiochemical mechanisms of plant defence. The advantage of this strategy has been well illustrated by the studies focused on developing new potato and maize varieties with resistance to the Colorado potato beetle and the corn earworm, respectively (Byrne et al. 1996, 1998; Yencho et al. 1996, 2000).

Sorghum midge *Stenodiplosis sorghicola* (Coquillett) is the most damaging pest of grain sorghum worldwide (Young and Teetes 1977). At flowering, female midges oviposit into spikelets, and the larvae feed on the ovary during the following 2 weeks, resulting in the failure of kernel development. Prior to the introduction of resistant varieties, the pest cost Australian sorghum producers an estimated $8–10 million per year in chemical control and loss of grain yield (Henzell et al. 1994). Early breeding for host-plant resistance to the sorghum midge and associated entomological research brought reports of worthwhile resistance in sorghum (Johnson et al. 1973; Wiseman et al. 1973; Rossetto et al. 1975).

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Y. Z. Tao (✉) · R. G. Henzell · D. R. Jordan
QDPI Hermitage Research Station, Warwick, QLD 4370, Australia
e-mail: taoy@dpi.qld.gov.au

A. Hardy · B. A. Franzmann · D. G. Butler
QDPI Toowoomba, QLD 4350, Australia

J. Drenth · C. L. McIntyre
CSIRO Plant Industry, 120 Meiers Road, Indooroopilly, QLD 4068, Australia

Three different mechanisms of insect resistance have been observed to be involved in insect-host plant interactions: antixenosis, antibiosis and tolerance (Painter 1951; Kogan and Ortman 1978). Ovipositional antixenosis to sorghum midge, or a reduction in the number of eggs laid in spikelets, is the most common resistance mechanism presented in international breeding lines (Henzell et al. 1994). Antibiosis to larvae feeding within the spikelet is a second less common mechanism of midge resistance observed in sorghum, and results in the death of larvae before they cause the seed to abort (Sharma 1985). Tolerance to larval feeding has only been reported recently in midge resistant studies, where midge develop through to pupation and emerge, leaving a slightly undersized kernel (Hardy and Franzmann, unpublished). While a number of mechanisms of resistance have been identified, the genetic control and components of such resistance are very poorly understood.

Molecular marker development for important traits in Australian sorghum breeding programs has been conducted in parallel to the construction of restriction fragment length polymorphism (RFLP) maps developed by several other groups all over the world (McIntyre et al. 1997). A genetic map has been established using a recombinant inbred (RI) population and has been aligned with other major published sorghum maps (Tao et al. 1998a, 2000). QTLs for some agronomic traits such as rust resistance and stay green have also been identified (Tao et al. 1998b, 1999, 2000). In this paper we report on the identification of QTLs associated with midge resistance in sorghum.

Materials and methods

Genetic stocks

One hundred and twenty random recombinant inbred lines (RILs) were developed from the cross of lines ICSV745 × 90562. Line ICSV 745 was developed in ICRISAT (International Crop Research Institute for the Semi-Arid Tropics) and is believed to carry gene(s) of antibiosis to sorghum midge (Sharma et al. 1993), while another parental line 90562 was developed by the Queensland Department of Primary Industries and is known to carry gene(s) for antixenosis (Henzell, unpublished). Line 90562 has the following midge resistance source lines in its pedigree: TAM2566 (Johnson et al. 1973) and AF28 (Rossetto et al. 1975). Line ICSV745 derives its antibiosis from DJ6514 (Shyamsunder et al. 1975). The cross was made in 1996 and the F_5 RILs were obtained after four generations of selfing in the following 2 years.

Construction of genetic map

A total of 1,140 RFLP probes and 120 simple sequence repeat (SSR) primers were screened for polymorphism for constructing a genetic map of the RI population mentioned above. The type, source and code of the markers mapped, the RFLP and SSR techniques and the procedure for linkage mapping were as previously reported (Tao et al. 1998a, 2000). An additional 400 sugarcane expressed sequence tag (EST) clones were also screened as RFLP probes in this study. The mapped loci were coded as EST, FC, JH, GE, MT, RG, SG and STR, respectively, on the map presented.

Assessment of midge resistance

The methods and procedures for assessing midge resistance were as described by Franzmann (1996) with some modifications. Trials were conducted on all lines to determine the level of antixenosis (reduced egg lay) and antibiosis (larval mortality) presented in each RI line. The two parents of the F_5 RI lines – line 90562, a highly antixenotic line without antibiosis, and ICSV745, a line with little antixenosis and high levels of antibiosis – were also included.

Plants were grown in pots in the glasshouse and selected for evaluation in the summers of 1998–2000. On each trial day, flowering RI panicles and at least one panicle of ICSV745 and 90562 were trimmed back to contain 120–150 flowering spikelets and then caged by wire cages enclosed with white nylon gauze bags. Forty mated female sorghum midges were introduced into each cage between 9 a.m. and 11 a.m. and left to lay in each enclosed panicle. Two samples (each of 50–80 sessile spikelets) were then taken from each panicle: an egg sample (1 day after trial commencement) and a pupal sample (taken upon the emergence of the first adult from the remaining spikelets on each panicle). The percentage of spikelets infested with eggs and pupae were obtained by microscope dissections of each sample.

Data analysis

Phenotypic data were analysed on three parameters – antixenosis (percentage of spikelets infested with eggs), antibiosis (difference between percentage eggs and percentage pupae as a percentage of the original egg count) and on pupal counts alone (recorded separately in QTL analysis). Before undergoing statistical analysis, RIL data for each measure were blocked according to the day of testing and year, and when no significant day × RIL or year × RIL interactions were recorded, ANOVA was used to predict means using common effects estimated across blocks and years. Predicted means for all three measures were then used in the QTL analysis.

QTL analysis

MAPQTL (Van Ooijen and Maliepaard 1996) was used for the QTL analysis in this study. Non-parametric mapping and interval mapping were conducted separately on the phenotypic data to identify genomic regions associated with different mechanisms to midge resistance. For the non-parametric mapping, a significant level of 0.005, as suggested by the authors of the programme, was applied to identify loci with genetic effects on midge resistance. For interval mapping, a LOD threshold of 2.4, which corresponds to the same significant level of 0.005, was used to claim the presence of a putative QTL in a given genome region.

Results

Genetic map of ICSV745 × 90562

A total of 235 RFLP probes and eight SSR primers produced 271 polymorphic markers in this cross, and these were used for further progeny screening. The level of DNA polymorphism is 20.6%, which is slightly lower than that observed in the other RI population derived from a cross of QL39 × QL41, which was mapped previously (Tao et al. 1998a). Among the 271 loci, 264 were mapped onto 12 linkage groups and cover a genetic distance of 1,472 cM. As approximately half of these loci have been mapped on other populations, these can be used as reference loci to align this map with other published maps. The 12 linkage groups were eventually reduced to

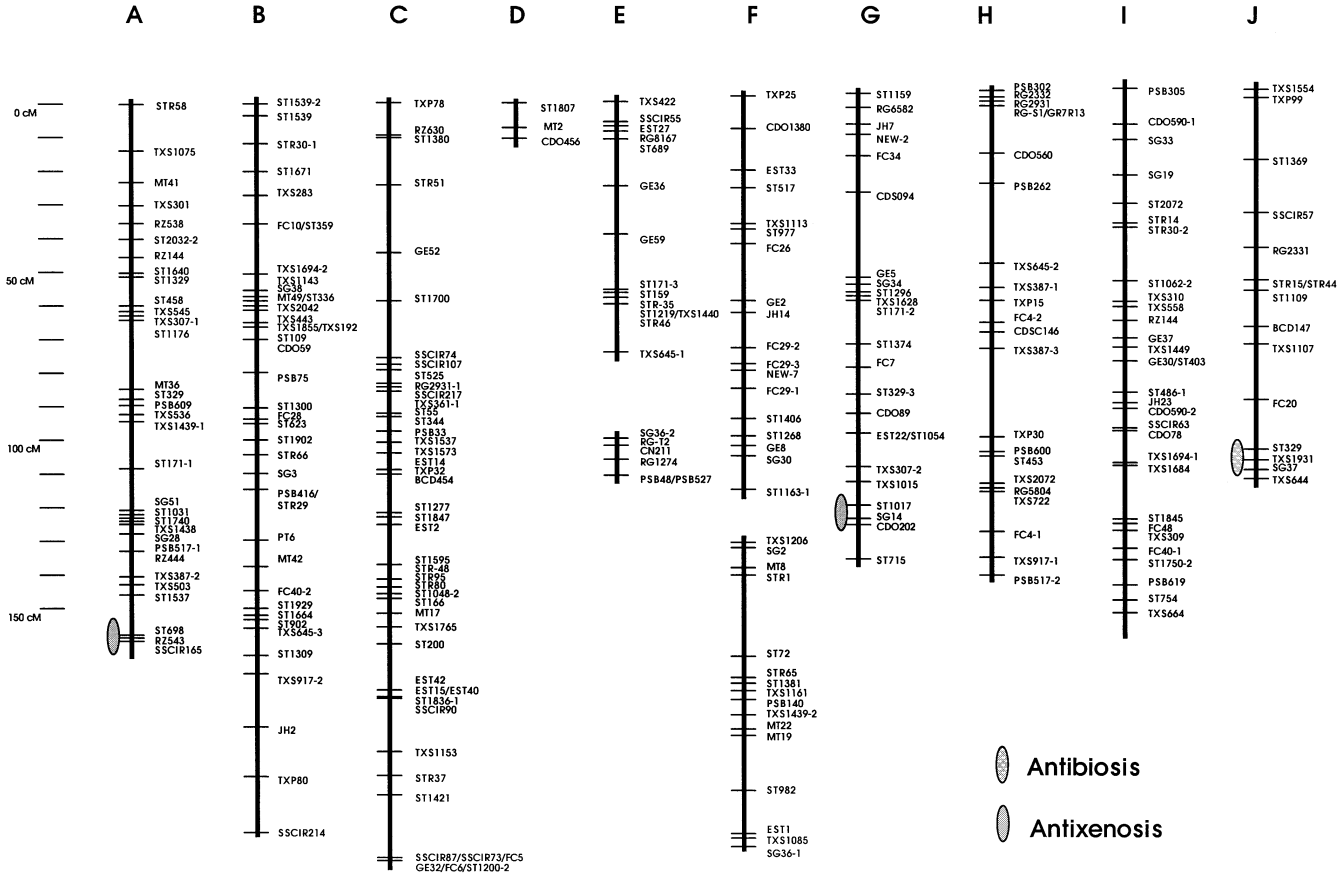

Fig. 1 Genetic map of the sorghum recombinant inbred population derived from the cross ICSV745 × 90562. Regions associated with antixenosis and antibiosis are indicated

ten linkage groups, and these correspond to the consensus sorghum map previously reported by Tao et al. (2000) (Fig. 1).

Phenotypic score for eggs, pupae and the difference between the score of the eggs and pupae

Phenotypic scores for antixenosis and antibiosis were taken from three to five panicles of each RIL. The level of antixenosis and antibiosis in most RI lines fell somewhere between the levels of the parental lines ICSV745 and 90562, while a small percentage of RI lines had either levels of antibiosis above that found in line ICSV745 or had levels of antixenosis higher than that in line 90562 (Table 1).

Antixenosis was measured by recording the percentage of spikelets with eggs. While lines ICSV745 and 90562 averaged 75% and 35% of spikelets infested with eggs, respectively, the scores for the 120 RILs ranged from 5% to 98%. However, in contrast to the egg counts, only 22% of spikelets of line ICSV745 were infested with pupae, whereas 36% of spikelets of line 90562 were infected, and pupal counts for the RI population ranged from 12% to 98%.

Table 1 Summary of phenotypic scoring for egg count, pupal count and the difference between the two counts for each RI and two parental lines

Scores	Range of variation in RILs	ICSV745	90562	Significance
Egg (%)	4.5–98	74.6	34.7	Yes
Pupae (%)	12.3–98	22.2	36.3	No
Difference (%)	0–86	61.4	0	Yes

When antibiosis was recorded as the percentage difference between the egg and pupal infestation (egg – pupal/ egg × 100), line ICSV745 averaged 61% antibiosis, while no antibiosis was recorded in line 90562. All RILs similarly recorded between 0 and 86% antibiosis.

Identification of markers associated with antixenosis and antibiosis

Both non-parametric mapping and interval QTL mapping were performed to identify the association between markers and three parameters – egg counts (antixenosis), the difference between egg and pupal infestation (antibi-

Table 2 Levels of significance of associations between genetic regions and different scores for midge resistance identified through non-parametric mapping (LG linkage group)

LG	Loci	Egg counting	Pupae counting	Difference	Mechanism
A	RZ543	*****	*****		Antixenosis
	ST698	******	******		
G	ST1017	******	*****		Antixenosis
	SG14	*****	****		
J	TXS1931		*******	*******	Antibiosis
	SG-37		*******	*******	

****$P \leq 0.005$, *****$P \leq 0.001$, ******$P \leq 0.0005$, *******$P \leq 0.0001$

Table 3 LOD scores and percentage of variation explained (in brackets) by QTLs associated with different scores for midge resistance identified through interval mapping

LG	Interval	Egg counting	Pupae counting	Difference of two counts
A	RZ543			
	ST698	3.27 (12%)	2.40 (8.8%)	
G	ST1017			
	SG14	4.58 (15%)	3.07 (15%)	
J	TXS1931			
	SG-37		10.6 (33.9%)	10.8 (34.5%)

osis), and pupal infestation alone. Two genetic regions were identified which were significantly associated with egg counts. These are the intervals between loci ST698 and RZ543 of linkage group (LG) A and loci ST1017 and SG14 of LG G, respectively. For all loci of these regions, the means of egg infestation in genotypes with line 90562-derived alleles were lower than for those with ICSV745-derived alleles. This indicated that the genetic effect for the antixenosis came from line 90562, as expected. The degree of phenotypic variation explained by each region was 12% and 15%, respectively.

Three genetic regions were also found to be associated with pupal infestation. These regions are located on LG A, LG G and LG J, respectively. The regions on LG A and LG G are the same as the ones associated with egg counts, and similar trends of low pupal infestation corresponding to low egg infestation were observed. The levels of phenotypic variations explained by each region are 8.8% and 15%, respectively (Table 2, 3). The other region associated with pupal counts is the interval between loci TXS1931 and SG37 on LG J. In contrast to the other two regions, a reduction in the number of pupae was associated with the presence of the ICSV745 marker loci genotype in this region. It explained 33.9% of total variation of pupal counts. This region on LG J is also significantly associated with the difference between egg and pupal counts and explained 34.5% of the total variation observed. As expected, the reduction of pupae in the spikelet is associated with the antibiosis presented in ICSV745. The locations of QTLs for antixenosis (the egg counts) and antibiosis (the difference of egg and pupal counts) are shown in Fig. 1.

Discussion

Different mechanisms for midge resistance

Franzmann (1993) reported that the primary mechanism of resistance in genotypes developed in Australia is ovipositional antixenosis, which means few eggs are laid in the resistance genotypes. This is the case in most midge-resistant sorghum lines, and high levels of antibiosis to larvae have been identified in only a few genotypes, including TAM2566 and DJ6514 and the related lines ICSV197 and ICSV745. (Teetes and Johnson 1978; Wuensche 1980; Sharma et al. 1993).

Several studies have attempted to examine the mechanisms underlying resistance to sorghum midge in these lines. Waquil et al. (1986) assessed the resistance mechanisms of sorghum midge-resistant hybrids and determined that resistance responses were characterised by adult visitation and oviposition non-preference for resistant sorghum and by a low level of antibiosis to larvae. Ovipositional non-preference could result from oviposition inhibition caused by floret morphology, the absence of a required stimulus or the presence of a deterrent. Female midges on panicles of resistant sorghum were more active in moving over the panicle and in ovipositional probing but laid fewer eggs than on panicles of susceptible sorghum.

Spikelet morphology, as suggested by Teetes (1985), seems more likely to be related to the reduction of eggs laid in spikelets of resistant genotypes. Sharma (1985) showed a significant correlation between short glumes and resistance in terms of egg infestation. Glume length, glume breadth and glume area were positively associated with susceptibility to sorghum midge in both Kenya and India (Sharma et al. 1999). In addition to being short, glumes of resistant genotypes appear to be more tightly held together.

Another possible cause of the ovipositional antixenosis mechanism of resistance to sorghum midge was examined in the study of Diarisso et al. (1998) by comparing the time of oviposition with the times that spikelets of resistant and susceptible sorghum flowered. They found that under Texas summer conditions most spikelets of resistant sorghum flowered and closed early in the morning before peak periods of midge oviposition, thus avoiding damage. By altering the flowering time of resistant lines to coincide with that present in susceptible lines, these authors produced similar levels of midge egg lay across lines. These results support the hypothesis that resistance is caused by asynchrony between the time of sorghum spikelet flowering and the presence of sorghum midge. However, results from a similar experiment conducted in Australia on the same lines failed to support this hypothesis (Hardy and Franzmann, unpublished).

Antibiosis, or mortality of the immature stages of midge development, has been identified in a number of lines, including line DJ6514, and the related line ICSV745, both of which produced over 60% larval mortality (Sharma et al. 1993).

We have studied here two different mechanisms of midge resistance in the same segregating population as evidenced from both phenotypic screening and QTL mapping results. Evidence of the two mechanisms was demonstrated in our phenotypic results where most RI lines recorded levels of antixenosis and antibiosis between the levels presented in both parents. Line ICSV745 recorded egg counts that were double that present in line 90562; however, the pupal counts in both parents were similar. All eggs laid in the spikelets of line 90562 developed through to pupae, while only 40% of the eggs survived through to pupation in line ICSV745. Consequently, line 90562 contains high antixenosis (low eggs) and no antibiosis, while line ICSV745 contains low antixenosis (high eggs) and high antibiosis (high eggs/pupae mortality).

QTL mapping results have further clarified the contribution of both mechanisms of resistance within the RI population. Since larvae feeding on the developing seed cause midge damage, the pupal counts in this study should give the most accurate measure of the level of midge resistance. If the antixenosis mechanism of resistance alone conferred resistance to midge, a clear correlation or co-segregation would be observed between egg counts and pupal counts. If this was the case, all regions associated with pupal counts should correspond exactly to the regions associated with egg counts. This was not observed in this study. QTL mapping results indicated that a correlation between egg counts and pupal counts only occurred on LG A and LG G but not on LG J. Even though a significant association was identified between that region and pupal counts, no such association was identified between LG J and egg counts, highlighting the role of a second mechanism of resistance (antibiosis) in the RI population.

The two parent lines produced similar levels of seed set in the pupal sample from different mechanisms – line ICSV745 from antibiosis and line 90562 from antixenosis. Each RI line recorded a combination of the antixenosis and antibiosis presented in each parent and, in a few cases, higher levels of either mechanism of resistance. It is obvious that both parental lines contribute contrasting levels of two mechanisms of midge resistance in this population. The ovipositional antixenosis carried by line 90562 reduces the number of spikelets infested with eggs during flowering, while the larval antibiosis carried by ICSV745 reduces larval feeding damage to the developing seed during the grain fill period.

Gene control for midge resistance

Resistance to sorghum midge has not been well understood due to the difficulty in estimating the level of midge resistance. Although a number of studies have been conducted to discover gene action for midge resistance (Sharma 1985, Sharma et al. 1996), no results show a clear genetic basis for any mechanism of midge resistance in sorghum.

For the first time, QTLs for two different mechanisms of midge resistance were identified from a relatively large segregating population through the accurate glasshouse screening method used in this study. Two genetic regions were found to be associated with antixenosis, while one region was found to be associated with antibiosis (Fig. 1).

It is interesting to note that only one genetic region for antibiosis was identified in QTL analysis and that phenotypic results produced larval antibiosis scores scattered regularly between 0 and 86%. Such results indicate that antibiosis may be a qualitative trait rather than a quantitative trait. Further work is needed to determine whether one or more closely linked genes on group J confer the antibiosis present in ICSV745. What causes the antibiosis, either lethal, chemical or specific enzyme activity, and how such processes work in the resistant host plant are questions that remain unsolved. Further work is underway to determine whether a single antibiotic substance associated with antibiosis exists.

As morphological and physiological factors such as spikelet characteristics and flowering time, which are often influenced by environmental variation, are more likely to be involved in antixenosis, it was not surprising to find that the genetic control of antixenosis is more complicated than that of antibiosis. Two genetic regions on LG A and G associated with antixenosis were identified in this study. These regions are consistent to some extent with the results from another study of QTL mapping for midge resistance on another RI population derived from QL39 × QL41 segregating for antixenosis by field testing (Tao, unpublished). The genetic regions associated with antixenosis on the QL39/41 population were also found on LG A and LG G. The exact locations of the regions were, however, slightly different (data not shown).

Breeding efforts have been successful, and sorghum commercial hybrids with midge resistance levels that approach practical field immunity are now available to farmers in Australia (Henzell, unpublished). However, as resistance is mainly comprised of one component (ovipositional antixenosis), it may break down by the development of a new midge biotype. In addition, a previous study has shown that selection for midge resistance in Australian sorghum hybrids has been associated with a decline in genetic diversity (Jordan et al. 1998).

In order to reduce the risk of resistance breakdown and also to increase levels of midge resistance, sorghum breeders need to explore new sources of midge resistance, to isolate and incorporate alternative mechanisms of resistance and to pyramid different resistance genes into commercial hybrids. This goal can not be achieved through conventional breeding technology, as it is neither an effective nor efficient approach for large-scale selection. Traditionally the most appropriate measure of the level of resistance to sorghum midge is the amount of yield lost following a standard midge infestation (Franzmann et al. 1986). The field trials used to measure yield by scoring the percentages of seed set, however, are very labour-intensive and expensive. Experimental variability is high, and there are significant interactions with environmental factors. Also, genetic effects from different mechanisms of midge resistance can not be distinguished from assessing seed set alone, so it is impossible to discover new sources of midge resistance for gene pyramiding. The weakness is evidenced from the nature of midge resistance in the two parental lines used in this study. Each line contains only one mechanism: 90562, a high level of antixenosis with no antibiosis, while ICSV745 is strongly antibiotic but weak for antixenosis. Phenotype and QTL results indicate that both mechanisms present in RI lines increased the overall seed set in such lines, demonstrating both the significance and potential of gene pyramiding in future midge resistance breeding.

One other method to enable gene pyramiding of midge resistance genes is to use the glasshouse cage method. The method was used to produce phenotypic results in this study. While this method ideally identifies antixenosis and antibiosis, the cage test is even more labour-intensive, facility-dependent and time-consuming than traditional breeding methods, and logistically impossible to use in large-scale breeding practise for direct selection of midge resistance.

Polymerase chain reaction (PCR)-based DNA markers are, therefore, an ideal approach for selecting for this type of trait in a practical breeding programme. We are currently developing more closely linked PCR-based markers. The more user-friendly PCR-based markers will be used directly by breeders for midge resistance selection.

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