

Disease resistance mapping in spring barley

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Introduction

Scald (*Rhynchosporium secalis*) is difficult to control chemically. Apart from Doyen, there are currently no UK spring barleys recommended cultivars with good resistance. Spring barleys with major-gene resistance have failed to establish as their malting quality is not the best.

Major-gene resistance may only be a short-term solution so durable resistance combined with the highest malting quality is required. Associations of reduced quantitative resistance with reduced height and the Mlo mildew resistance have been observed.

We tested these associations using a mapping population that segregated for the *mlo11* allele against a range of *R. secalis* isolates in detached leaf tests, which we compared with results from natural field infection.

We also studied the same population for resistance to the rice blast pathogen (*Magnaporthe grisea*), physiological spotting, and the post-anthesis spotting complex thought to be due to *Ramularia collo-cygni*.



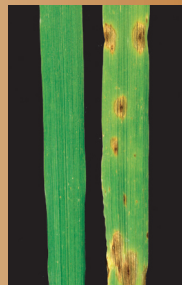
Physiological Spots



RLS



Scald



Rice Blast

Materials & Methods

The Derkado x B83-12/21/5 spring barley mapping population was grown in field nurseries and trials to collect scores of natural scald infection and post-anthesis leaf spotting, which we have termed "Ramularia-Like Spots" (RLS).

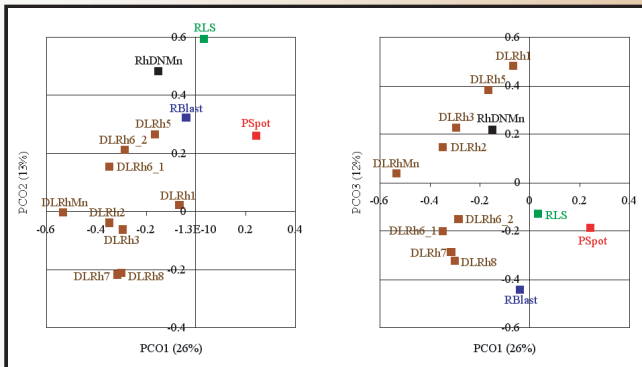
Clear physiological spotting (PSpot) is apparent in some trials and scored. The population was inoculated with isolate 96-1-27 of rice blast at the seedling stage and scored for infection (RIBlast).

Detached leaves of the population were inoculated with different races of *R. secalis* and scored for lesion development.

Principal components analysis was used to investigate the relationships between the variables.

A revised map incorporating 22 EST derived markers (Ctig, SNP and abc) and two genomic SSRs was used in QTL analysis.

Results



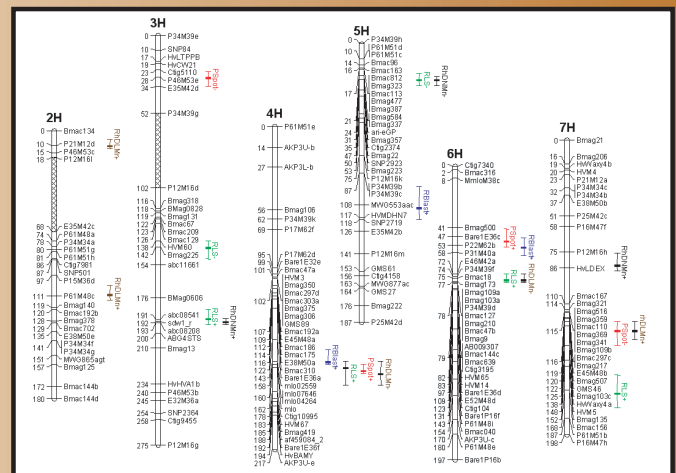
QTLs accounting for between 72 and 13% of the phenotypic variation were found on six of the seven barley chromosomes. The *mlo* locus was associated with each character apart from RhDNMn.

The dwarfing loci, *sdw1* and *ari-eGP* had large effects upon RhDNMn and the other QTL (near HvLDEX on 7H) was co-located with a height QTL. There was no co-location of QTL loci for RhDLMn with RhDNMn. QTLs for RLS are not co-located with PSpot, apart from the region of *mlo* on 4H, suggesting independent control.

The *mlo11* allele was associated with increased infection of RIBlast, RLS and PSpot but decreased RhDLMn. Epistasis was detected for PSpot between the loci on 4H and 6H.

PSpot is clearly separated from the rest of the variables by PCO1 and is opposite to RhDNMn. The detached leaf scores are variable with different loadings on PCO2 and PCO3 but similar on PCO1.

RhDNMn is clearly separated from RhDLMn by PCO2 and the two measures of scald infection do not appear to be highly related. PSpot and RLS are separated by both PCO1 and PCO2 and do not appear to be related. RIBlast does not appear to be highly related to any other character.



Conclusions

Field scores of apparent quantitative resistance to scald reflect escape rather than true resistance but there was no evidence for *mlo11* increasing susceptibility to scald.

Detached Leaf tests can be used to identify potential quantitative resistance to scald but there was some co-location of resistance QTLs with increased physiological spotting.

Co-location of QTLs for increased RIBlast and PSpot with the *mlo* locus is consistent with previous reports.

The epistatic interaction between the QTLs for PSpot on 4H and 6H that increases expression of symptoms, suggests that allelic variation at the locus on 6H can modify expression of the *mlo* QTL.

Acknowledgements

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