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Identification of molecular markers associated with partial resistance to powdery mildew, leaf rust and stripe rust in bread wheat line Saar

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SUMMARY – The CIMMYT bread wheat line Saar has a high level of partial resistance to powdery mildew (PM), leaf rust (LR) and stripe rust (YR). Saar is known to carry *Lr34/Yr18*, and experiments were set up to test whether the same genetic factor could also be involved in the resistance to PM. A population of 113 recombinant inbred F₆ lines from a cross between Saar and the susceptible line Avocet-YrA was tested for LR and YR in Mexico and PM in Norway and China. There was a strong association among the disease scores, and they were all strongly correlated with leaf tip necrosis (LTN). A bulked segregant analysis with SSR markers was conducted to identify molecular markers associated with the resistance to PM. Two major QTLs were identified, one on chromosome 7D and the other on chromosome 1B, corresponding to the adult plant rust resistance loci *Lr34/Yr18* and *Lr46/Yr29*, respectively.

Introduction

Powdery mildew (PM, caused by *Blumeria graminis* f. sp. *tritici*), leaf rust (LR, caused by *Puccinia triticina*) and stripe rust (YR, caused by *P. striiformis* f. sp. *tritici*) are among the most important diseases of bread wheat (*Triticum aestivum*). The CIMMYT bread wheat line Saar confers partial and race non-specific resistance to all three diseases and represents a highly valuable source of resistance in wheat breeding. Previous work has shown that part of the complex adult plant resistance to LR and YR is controlled by the tightly linked or pleiotropic genes *Lr34* and *Yr18* in combination with other minor genes for resistance (Navabi *et al.*, 2003; Navabi *et al.*, 2004). Despite never having been exposed to powdery mildew during its development, the line exhibits a high level of partial resistance to PM, which is controlled by at least three genes (Lillemo *et al.*, 2005). Leaf Tip Necrosis (LTN) is a widely used phenotypic marker for *Lr34/Yr18* (Singh *et al.*, 1992), and a strong correlation with PM indicated that *Lr34* might also be involved in the PM resistance of Saar (Lillemo *et al.*, 2007).

Field testing

A population of 113 recombinant inbred F₆ lines from a cross between Saar and the susceptible line Avocet-YrA was used for field testing and QTL analysis. PM testing was conducted at two locations in Norway (Hamar and Ås) and Beijing, China. In addition, the population was tested for LR and LTN in Ciudad Obregón, Mexico and YR in Toluca, Mexico. All field testing was conducted in 2005 by use of a randomized complete block design with two replications at each location. Further disease testing is being conducted in the 2006 growing season.

Bulked segregant analysis

Resistant and susceptible bulks were constructed based on the PM data from the two locations in Norway by mixing equal amounts of DNA from the five most resistant and five most susceptible lines, respectively. At the time of writing, 572 SSR markers have been tested on the two parents, and resulted in the identification of 334 polymorphic marker loci. 56 of these marker loci showed differentiation when tested on the resistant and susceptible bulks.

Map construction and QTL analysis

The 56 differentiating marker loci were used to create linkage groups in JoinMap v. 3.0 that were subsequently used for QTL analysis with PlabQTL v. 1.2. Simple interval mapping detected two major QTLs for PM resistance on chromosomes 1B and 7D. Although the initial markers were selected based on differentiation between the resistant and susceptible bulks for PM resistance only, the same QTLs also showed large effects on LR and YR. The R-square values of the two QTLs for selected disease parameters are shown in Table 1.

Table 1. Chromosomal location and R-square values of the two QTLs showing effects on LTN, PM, LR and YR in the Avocet-*YrA* x Saar population

QTL	Marker interval	PM Hamar	PM Ås	PM Beijing	LR	YR	LTN
1B	wmc40-barc80	30.8	23.3	13.3	29.5	18.6	11.2
7D	gwm295-gwm1220	30.3	37.7	24.3	51.0	29.9	71.7

PM Hamar = Powdery Mildew, last assessment date, Hamar, Norway (0-100); PM Ås = Powdery Mildew, last assessment date, Ås, Norway (0-100); PM Beijing = Powdery Mildew, Beijing, China (0-9); LR = Leaf Rust, Cd. Obregon, Mexico (0-100); YR = Stripe Rust, Toluca, Mexico (0-100); LTN = Leaf Tip Necrosis, Cd. Obregon, Mexico (absence/presence, 0-1).

Indications are strong that the QTL on chromosome 7D could be due to *Lr34*. It showed a strong association with LTN (Table 1) and mapped to the known marker interval for *Lr34* (Schnurbush *et al.*, 2004). Associations between *Lr34* and PM resistance have also been reported in other studies (Spielmeyer *et al.*, 2005; Lillemo *et al.*, 2007).

The QTL on 1B is located in the same area as a previously reported QTL for partial resistance to PM in the winter wheat variety Massey (Liu *et al.*, 2001). The very similar effect of this QTL on all three diseases in the present study indicates that it could be caused by the tightly linked or pleiotropic genes *Lr46* and *Yr29*, which are located in the same region (Rosewarne *et al.*, 2006), and has recently been shown to be associated with resistance PM as well as the previously known effects on LR and YR (Lillemo *et al.*, 2007).

Further work

Several of the 56 markers that showed differentiation between the resistant and susceptible bulks remained unlinked in the initial map construction, but showed significant association with resistance to PM, LR and/or YR (data not shown). Further work includes adding additional markers to build linkage groups around these significant marker loci, as well as fine mapping of the already detected QTLs on 1B and 7D.

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