

Short Communication

Mapping wheat powdery mildew resistance derived from *Aegilops markgrafii*

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Abstract

The pattern of inheritance of powdery mildew resistance expressed by two bread wheat (cultivar ‘Alcedo’)/*Aegilops markgrafii* introgression lines was explored using F₂ populations bred from crosses made with the powdery mildew-susceptible cultivar ‘Kanzler’. Disease reaction was tested at both seedling and adult plant stages. Two resistance loci, designated *QPm.ipk-1A* and *QPm.ipk-7A*, were identified as mapping to the distal ends of chromosome arms 1AS and 7AL, respectively. Whereas *QPm.ipk-1A* was expressed throughout the plant’s life, *QPm.ipk-7A* was only effective at the seedling stage. The map location of both resistance loci indicated that resistances originated from *A. markgrafii*. The possible genetic relationship of these disease-resistant genes to known *Pm* genes is discussed in the context of synteny.

Keywords: *Aegilops caudata*; *Blumeria graminis*; genetic mapping; interspecific introgression lines; *Puccinia recondita*; *Triticum aestivum*

Powdery mildew (causative pathogen: *Blumeria graminis* f. sp. *tritici*) is an important foliar disease of bread wheat in cooler environments. To date, a large number of both major genes (denoted *Pm*) and quantitative trait loci associated with powdery mildew resistance have been described in the literature (McIntosh *et al.*, 2008, 2009, 2010, 2011), and one (*Pm3*) of the former genes has been successfully isolated (Yahiaoui *et al.*, 2004). While the majority of these genes have been identified within the primary gene pool represented by *Triticum* spp., some have also originated from the closely related genera *Secale* and *Aegilops*. The potential of the more distantly related species *Aegilops markgrafii* (syn. *Aegilops caudata* L.; $2n = 2x = 14$) as a donor for powdery mildew resistance prompted Schubert (1991) to construct

a series of bread wheat (cultivar ‘Alcedo’)/*A. markgrafii* (accession ‘S 740-69’) single chromosome addition lines. Spontaneous genetic rearrangements occurring during the necessary crossing programme later allowed for the selection of powdery mildew-resistant euploid derivatives having a largely cultivar ‘Alcedo’ background. Genetic analysis of some of these materials led to the conclusion that resistance factors had been transferred to both chromosomes 1A and 7A (Junghans, 1995). Here we deployed a standard genetic mapping approach to characterize the genomic location of powdery mildew resistance present in two of the best performing introgression lines.

Experimental

The two powdery mildew-resistant introgression lines ‘EgIII’ and ‘EgIV’ were each crossed with the powdery mildew-susceptible cultivar ‘Kanzler’, and the resulting F₂ populations, along with the parental material and

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the cultivar 'Alcedo', were raised in a greenhouse. The first fully expanded seedling leaf was removed from each plant for use in a detached leaf *in vitro* resistance test. The distal centimetre of the leaf was discarded, and the remaining 3 cm laid on agar containing benzimidazol (35 ppm) and silver nitrate (1.5 ppm). The leaves were then inoculated with a mixed population of the pathogen (composed of spores produced by isolates carrying virulences against *Pm1*, *Pm2*, *Pm3a*, *Pm3b*, *Pm3c*, *Pm3d*, *Pm4b*, *Pm5*, *Pm6*, *Pm8*, *Pm9* and *Pm17*) and held at 16°C under a 16h/8h light/dark photoperiod (light intensity >10,000 lx). At 10 d after the inoculation, a disease score was assigned to each segment, ranging from 0 (no visible symptoms) to 4 (clearly defined powdery mildew pustules), following McIntosh *et al.* (1995). For the characterization of the adult plant response, the same set of seedlings was first fully vernalized (8 weeks at 4°C), and then planted outside in plots of 20 plants each, with each plot surrounded by similarly sized, earlier sown plots comprising the fully susceptible cultivars 'Pallas' and 'Kanzler'. Disease spread was achieved by including within the plots pot-grown plants of the cultivar 'Kanzler' which had been artificially inoculated in a greenhouse. The same scale of resistance/susceptibility was applied as for the seedling test. The F₂ segregation of disease scores among both seedling and adult plants and plant numbers investigated are shown in Fig. 1.

The genotypic analysis initially involved 30 simple sequence repeat (SSR) loci, of which 15 are known to map to chromosome 1A and 15 to chromosome 7A. The polymerase chain reaction conditions applied were those detailed by Röder *et al.* (1998). Following the

outcome for the cultivars 'Kanzler', 'EgIII' and 'EgIV', the SSR assays GWM1223, GWM0033b (chromosome 1A) and GWM0698, GWM1061 (chromosome 7A) were applied to DNA extracted from each of the F₂ individuals. When the phenotypic (powdery mildew resistance score) and genotypic (*Xgwm1223*, *Xgwm0033b*, *Xgwm0698* and *Xgwm1061*) scores were inserted into the QGENE program (Nelson, 1997), the resulting analysis indicated the presence of two loci, denoted *QPm.ipk-1A* and *QPm.ipk-7A*, mapping to the distal ends of chromosome arms 1AS and 7AL, respectively (Fig. 2). The LOD score associated with the former locus was 6.8 at the seedling stage and 5.2 at the adult stage, while the latter locus was only expressed significantly (LOD 13.4) at the seedling stage. The alleles *Xgwm1223*, *Xgwm0033b*, *Xgwm0698* and *Xgwm1061* present in 'EgIII' and 'EgIV' were inherited from 'S 740-69', so the assumption is that both resistances were derived from *A. markgrafii*.

Discussion

Various *Aegilops* species have been identified as potential sources of disease resistance in bread wheat (McIntosh *et al.*, 2008). The *A. markgrafii* parent of 'EgIII' and 'EgIV' was also resistant to leaf rust (Schubert, 2001; Weidner, 2004), and a leaf rust-resistant locus derived from the donor was successfully mapped to chromosome arm 2AS by Iqbal *et al.* (2007). Here we have confirmed that powdery mildew resistance present in 'EgIII' and 'EgIV' relies on gene(s) located on chromosomes 1A and 7A, as suggested by Junghans (1995), and shown that

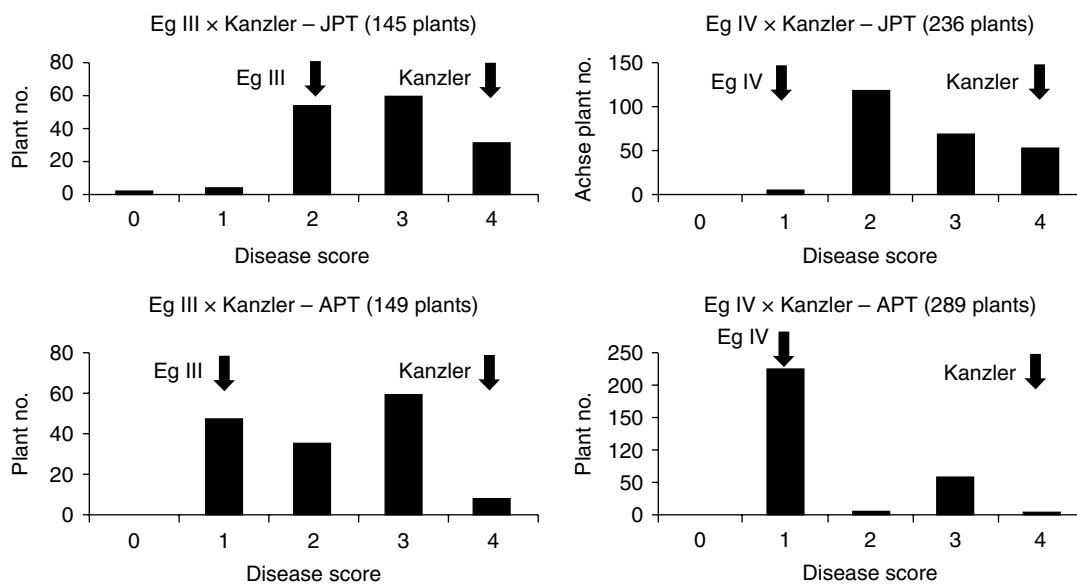


Fig. 1. Segregation for powdery mildew resistance expressed at seedling (JPT) and adult plant (APT) stages in F₂ populations derived from the crosses 'EgIII' x cultivar 'Kanzler' and 'EgIV' x cultivar 'Kanzler' (lower disease scores represent higher levels of resistance).

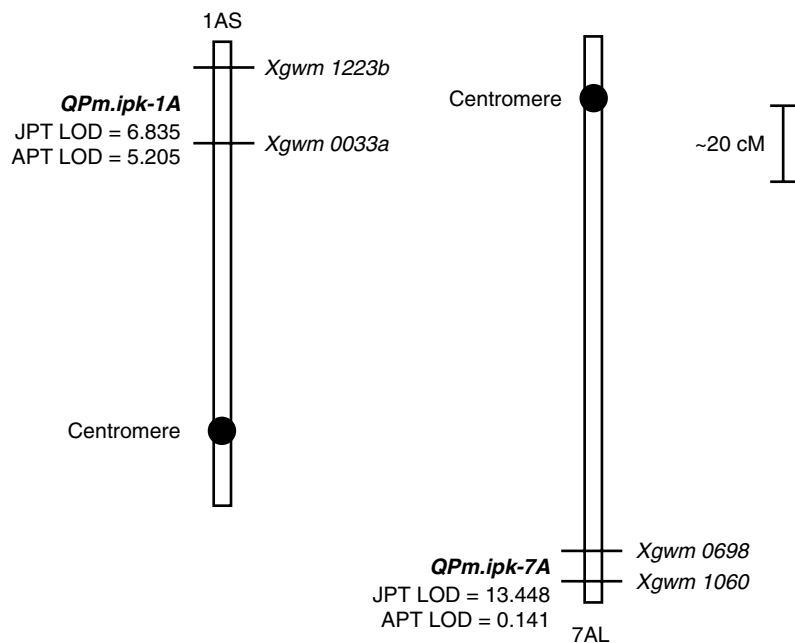


Fig. 2. Map positions of the loci underlying powdery mildew resistance on the chromosome arms 1AS and 7AL. LOD scores associated with both resistances at seedling (JPT) and adult plant (APT) stages are shown.

these resistance factors both map to the distal ends of the two chromosome arms 1AS and 7AL. Both these genomic regions are known to carry genes responsible for powdery mildew resistance. The 1AS-located gene *Pm3* maps 2.3 cM from *Xgdm0033* (Bouget *et al.*, 2002), an SSR locus which is closely linked to *Xgwm1223b* (Ganal and Röder, 2007). The possibility is therefore that *QPm.ipk-1A* is a homoeoallele of the multi-allele locus *Pm3*. It should be noted that, to date, all known *Pm3* resistance alleles have originated from within the primary bread wheat gene pool, so if *QPm.ipk-1A* does prove to be a *Pm3* homoeoallele, this would represent a new departure, as may also be the case for *Pm8* of rye (Keller, pers. commun.). The chromosome arm 7AL is the site of both *Pm1* and *Pm37*, genes which can be traced to *Triticum* spp. donors. The two genes lie within 16 cM of one another, but while *Pm37* is separated from *Xgwm332* by just 0.5 cM (Perugini *et al.*, 2007), *Pm1* co-segregates with *Xpsr687* (Neu *et al.*, 2002). Based on the Paillard *et al.* (2003) linkage map (which includes both *Xgwm332* and *Xpsr687*), *QPm.ipk-7A* is more likely to be related to *Pm1* than to *Pm37*, because *Xpsr687* maps to the distal region of chromosome 7AL, whereas *Xgwm332* is located closer to the centromere.

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