

Identification and location of *Stb9*, a gene for resistance to septoria tritici blotch in wheat cultivars Courtot and Tonic

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This study reports the discovery of a gene for resistance to septoria tritici blotch (STB) in two spring wheat cultivars, Courtot and Tonic. The gene, named *Stb9*, confers resistance to *Mycosphaerella graminicola* isolate IPO89011. It was mapped by quantitative trait loci (QTL) analysis using an existing map of Courtot × Chinese Spring and was located between markers *Xfbb226* (3.6 cM) and *XksuF1b* (9 cM) on the long arm of chromosome 2B. Markers linked to *Stb9* in Courtot were then shown to be linked to resistance to IPO89011 in F₃ families of Tonic × Longbow. Allelism tests in which Tonic was crossed with Courtot confirmed that Tonic has a gene for resistance to IPO89011 at or very close to the *Stb9* locus. SSR markers flanking *Stb9* may be used in marker-assisted selection to introgress this gene into winter cultivars or in spring wheat breeding programmes outside Europe.

Keywords: isolate-specific resistance, *Mycosphaerella graminicola*, septoria tritici blotch, *Stb9*, *Triticum aestivum*, wheat

Introduction

Genetic dissection of complex agronomic traits requires the use of well-characterized populations and good mapping data. In wheat, several populations have been developed and molecular maps generated in order to identify and locate genes or quantitative trait loci (QTL) for complex agronomical traits (Gale *et al.*, 1995; Nelson *et al.*, 1995a,b,c; Parker *et al.*, 1999; Pestsova *et al.*, 2000). Cadalen *et al.* (1997) developed such a population of 187 doubled-haploid lines from a cross between the French cv. Courtot and the landrace Chinese Spring. A complete genetic map of this cross including 659 markers and covering 95% of the genome has been developed, allowing extensive QTL analysis of bread-making quality as well as developmental traits (Sourdille *et al.*, 2003). Courtot is not widely grown, but is related to many of the most commercially important European spring wheats (Martynov *et al.*, 2006).

A trait which is difficult to assess accurately in field trials, but which is economically important in wheat breeding, is resistance to septoria tritici blotch (STB), caused by the ascomycete fungus *Mycosphaerella graminicola* (anamorph *Septoria tritici*). It is the most important foliar disease of wheat in Europe and several other regions of the world (Polley & Thomas, 1991; van Ginkel & Rajaram, 1993; Cowger *et al.*, 2000; Hardwick *et al.*, 2001). The use

of fungicides to control STB is expensive and not fully reliable. Strobilurin fungicides (Qo inhibitors) were commonly used to control STB in the late 1990s and early 2000s, but in 2002, *M. graminicola* isolates resistant to strobilurins were identified in much of Europe (Fraaije *et al.*, 2003). Isolates of *M. graminicola* with reduced sensitivity to triazole fungicides were also discovered recently (Fraaije *et al.*, 2007). Breeding for resistance to STB is therefore of great interest to farmers, and breeders have increased their efforts to breed for resistance to STB since the early 1980s. Breeding for resistance to STB now makes use of marker-assisted selection (MAS) and there is significant demand for new resistance genes to STB that can be selected using genetic markers (Goodwin, 2007). Chartrain *et al.* (2004a) showed that several sources of resistance to STB have many resistance genes, suggesting that 'stacking' or 'pyramiding' individual resistance genes may be useful in breeding. Hence it is useful to tag resistance genes within the current gene pool, to enable simultaneous selection of several genes.

The genetics of resistance to STB are complex and less well studied than for other diseases of wheat such as rusts and powdery mildew. Resistance to STB may be isolate-specific or quantitative. Isolate-specific resistance is near-complete, oligogenic (Somasco *et al.*, 1996; Arraiano *et al.*, 2001a; McCartney *et al.*, 2002) and follows a gene-for-gene relationship (Brading *et al.*, 2002), whereas quantitative or partial resistance is incomplete, polygenic (Jlibene *et al.*, 1994; Simon & Cordo, 1998; Zhang *et al.*, 2001) and isolate non-specific (Chartrain *et al.*, 2004b). Twelve isolate-specific

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