ROTHAMSTED RESEARCH ASSOCIATION

RRA Newsletter

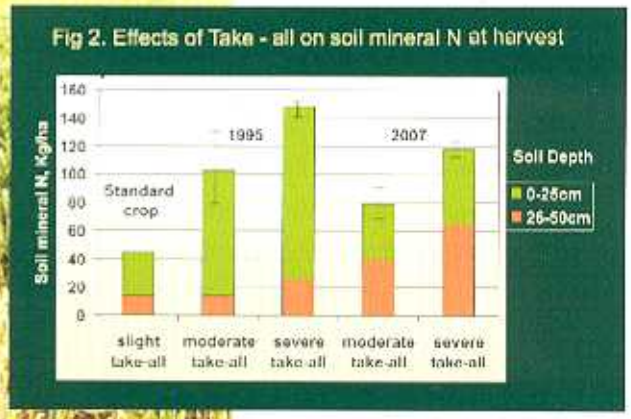
Rothamsted Research Association

Issue 27 (www.rothra.org) June 2008

Take-all and the Wheat Genetic Improvement Network (WGIN)

The name take-all was used in Australia in the 1870s, but was recognised as early as 1852 as a devastating disease of wheat. It is a damaging root disease and if severe symptoms occur around anthesis, then the crop often shows as stunted, prematurely ripening plants (Fig.1).

The disease severely reduces yield, because the grain from the infected plants is often small and shrivelled. As the plant dies prematurely it does not make full use of the available soil nitrogen. Work at Rothamsted has shown that a severely infected crop can leave as much as three times more mineral N in the top 50cm layer of soil, compared to a crop with only slight infection (Fig 2).



Take-all is a patchy disease and being able to predict when a severe outbreak will occur has always been difficult. However, there are known factors which can encourage the disease. Therefore, it is important to prepare for crops at risk. For example, soils low in either phosphate or potash (i.e. below index 2) will increase the risk and any amendments should be made before the break-crop. Cereal volunteers and a number of wild and cultivated grasses can act as carriers of the take-all fungus and should be rigorously controlled during the break-crop year. Cereal volunteers between crops (so called 'green bridge') should not be allowed to develop as this will maintain soil inoculum longer and lead to a higher risk. Cultivation method can affect disease severity in short rotations. Ploughing, where the most infective inoculum is buried and less infective inoculum is brought to the surface, tends to develop less disease than minimal tillage, where the infective inoculum remains in place.

The Wheat Genetic Improvement Network (WGIN) project funded by defra, which started in 2003 (for further information see www.wgin.org.uk), has two overall aims. Firstly, to generate pre-breeding material carrying novel traits for the use by breeding companies, and secondly, to deliver accessible technologies thereby ensuring the means are available to produce new, improved cultivars. Within this project a series of experiments, started in the autumn of 2003, were designed to study the nitrogen use efficiency of different NW European commercial winter wheat cultivars. Most of the cultivars chosen had good bread making qualities, and included both current and former elites.

The experiments, all grown as first wheats, tested 22 cultivars at three or four nitrogen rates (0-350Kg/ha), in randomised block designs of three replicates. In the 1980s, trials at Rothamsted suggested that cultivars may differ in their ability to build up the take-all fungus at the start of an epidemic. These large experiments, of five years duration, provided an opportunity to further test this hypothesis. The take-all infectivity of the soil (i.e. inoculum) was measured by a soil core bioassay, using wheat seedlings as bait. Research at Rothamsted has shown that the amount of inoculum in the soil after harvest was positively, and linearly, related to the severity of disease in the following wheat crop. The regression coefficient (i.e. slope of the line) was not, however, consistent between crops or years so it has limited predictive value. This is probably because the relationship is dependent on many variables such as weather, soil nutrients (especially phosphate), cultivation practices and the sowing date of the subsequent crop.

Results from the first three years of the WGIN trials showed that cultivars could apparently build up take-all inoculum in the soil differentially during the growth of the first crop. The cultivars could be grouped according to their ability to build up the fungus;

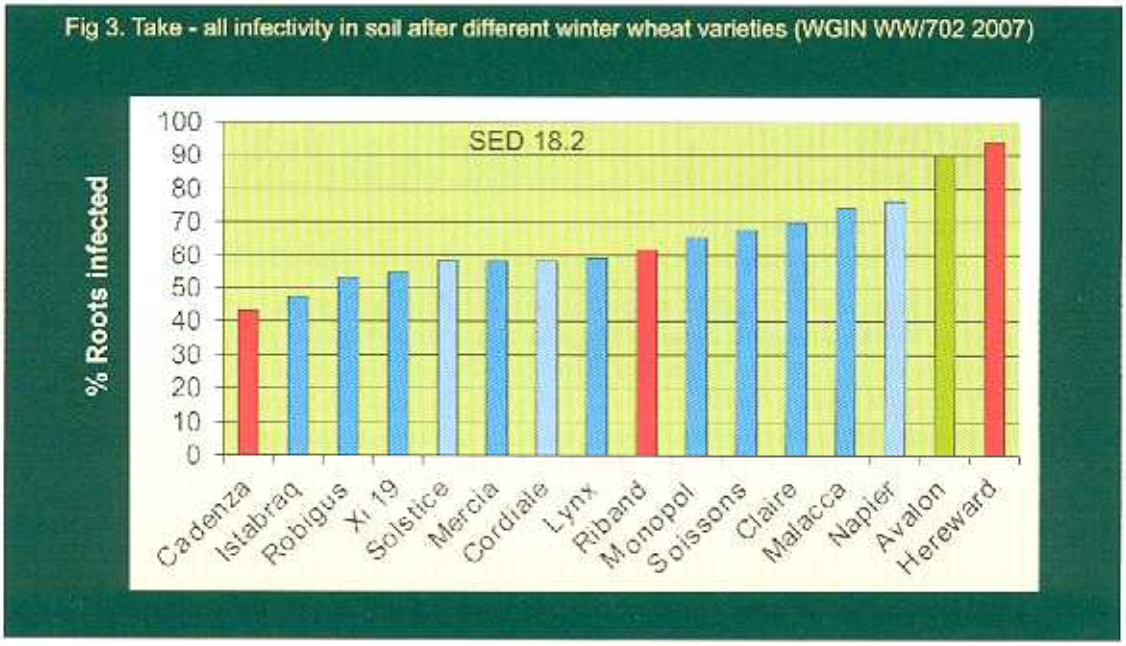
Low: Cadenza, Cordiale, Mercia;

Medium: Riband, Monopol, Soissons;

High: Hereward, Avalon, Equinox.

The grouping of the cultivars in 2007 was in broad agreement with previous years (Fig.3).

Fig 3. Take - all infectivity in soil after different winter wheat varieties (WGIN WW/702 2007)



The current bioassay approach for measuring take-all inoculum in the soil is not commercially viable. However, a molecular technique for measuring take-all DNA in the soil has been developed in Australia (called PreDicta B) and has recently been successfully tested in New Zealand. The test, which is based on real time PCR, is now being evaluated at Rothamsted through a HGCA/Bayer project.

Triticum monococcum, an ancient relative of wheat, is a rich source of resistance to septoria tritici blotch disease

Hai-Chun Jing, Richard Gutteridge, Kim Hammond-Kosack, Rothamsted Research

Septoria tritici blotch is an important foliar disease of wheat caused by the fungal pathogen *Septoria tritici* (teleomorph *Mycosphaerella graminicola*). In the July 2007 RRA newsletter a full description of this disease and its economic impact is given.

Breeding for resistance is one of the most efficient and economical approaches for disease control. However, the current genepool of hexaploid wheat has failed to provide adequate and durable protection against septoria tritici blotch. It is therefore necessary to expand the repertoire of available resistance genes. *Triticum monococcum* is a diploid einkorn wheat ($2n=2x=14$, A^mA^m) closely related to the cultivated hexaploid wheat. It was domesticated ~8000 years ago in Fertile Crescent and dominated early human farming activity. However, *T. monococcum* has been infrequently used in wheat breeding. This distinct history of *T. monococcum* as well as the availability of many land races makes this species potentially useful for genetic improvement of cultivated wheat.

At Rothamsted Research, *T. monococcum* has been explored for resistance to septoria tritici blotch disease through the defra-funded Wheat Genetic Improvement Network (WGIN) (www.WGIN.org.uk). We assembled a global collection of *T. monococcum* accessions and selected thirty for detailed assessment of resistance to septoria tritici blotch. Since 2004 field experiments were carried out for four consecutive years at multiple sites and the results indicate that *T. monococcum* has a high level of host resistance. This is manifested as the complete inhibition of the formation of necrotic lesions and asexual pycnidia-derived spores (Figure 1 top panel). Cytological analysis showed that *T. monococcum* was very effective in blocking the fungal hyphal growth and development when the fungus entered leaf tissue through the natural openings (stomata) (Figure 1 bottom right panel). This resistance was found in all the accessions tested suggesting that it is a common trait in *T. monococcum*. Thus, *T. monococcum* is a good source of resistance to *Septoria tritici*.

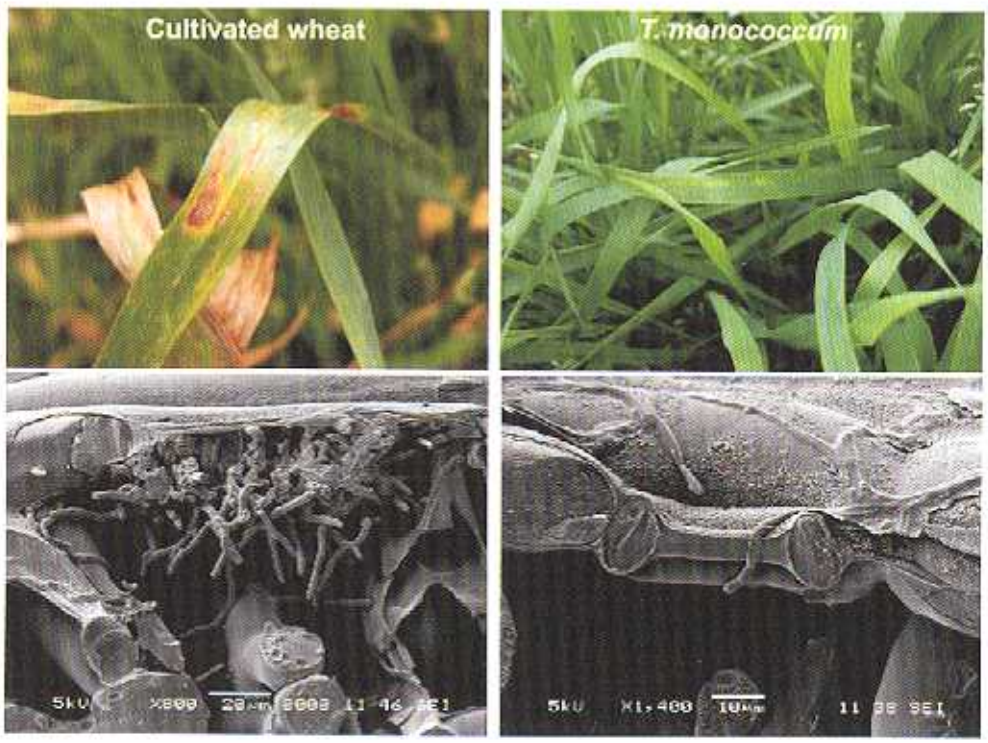


Figure 1. A high level of resistance to septoria tritici blotch disease is found in *T. monococcum* accessions. Top panel shows the severe disease symptoms in the field plots of hexaploid wheat variety Hereward, whereas no disease lesions were found in all of the *T. monococcum* plots. Bottom panel shows extensive colonisation by hyphae of the leaf breathing pores in hexaploid wheat which was absent in *T. monococcum*. This result has been confirmed in 4-years of field trials at Rothamsted Research. The two high magnification level views shown in the bottom two panels were taken using a Scanning Electron Microscopy at the Biomaging Unit at Rothamsted Research.

To understand the genetic basis of the resistance in *T. monococcum*, we characterised in detail the responses of *T. monococcum* accessions to *Septoria tritici* isolates in controlled environments using a seedling attached-leaf assay. Under such high disease pressure, the majority of the *T. monococcum* accessions still exhibited high resistance and inhibited disease development (e.g. Figure 2 MDR043). However, disease symptoms were observed in a few accessions, for example MDR002 in Figure 2. We then made a cross between the resistant accession MDR043) and the susceptible accession MDR002. The resultant F_1 seedlings showed resistant phenotypes suggesting that the resistance is dominant or semi-dominant. Next, we selfed the F_1 plants and made an F_2 populations as well as tested F_2 individual plants for their responses to *Septoria tritici*. At the same time, we developed a series of molecular markers called Simple Sequence Repeat (SSR) markers, which were spaced along the seven *T. monococcum* chromosomes (Figure 2b). By combining these two pieces of information, we could complete a genetic analysis and show that there is a genetic locus *TmStb1* on chromosomal $7A^m$ (Figure 2c) conferring resistance to *Septoria tritici* isolate IPO323. This isolate was chosen for the study because its full genome has recently been sequenced (http://www.jgi.doe.gov/jamborees/mycosphaerella/index.html).

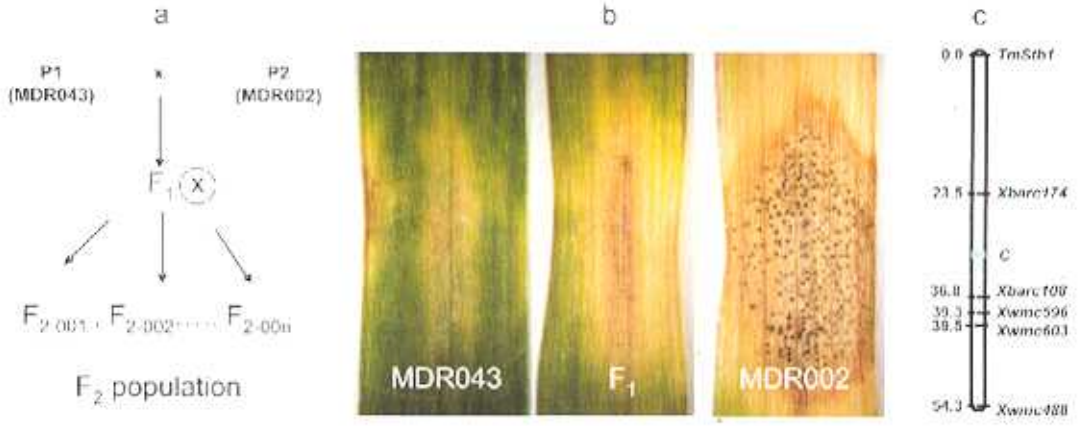


Figure 2. Identification of a genetic locus conferring resistance to septoria tritici disease in *T. monococcum*. (a) A diagram showing the genetic analysis of resistance/susceptibility to septoria tritici blotch disease using an inter-cross F_1 hybrids and an F_2 segregating population. (b) The responses of the resistant (MDR043) and susceptible (MDR002) *T. monococcum* accessions and their F_1 hybrids to infection by *Septoria tritici* isolate IPO323. (c) A diagram showing the chromosomal location of *TmStb1* in relation to known SSR marker loci on $7A^m$.

The location of the *TmStb1* is surprisingly different from all previously known *Stb* genes which are located on 12 other chromosomes in cultivated hexaploid wheat. This result implies that *T. monococcum* may have many unidentified genes conferring resistance to septoria tritici blotch. The result also indicates that another region of the wheat genome has evolved which influences the outcome of this interaction.

In summary, this study at Rothamsted Research has showed that *T. monococcum* deserves more attention for exploiting novel resistance to septoria tritici blotch disease. *T. monococcum* also has many other traits which can be utilised for wheat genetic improvement. Please refer to our recent publications for further details[1,2]. At Rothamsted we are currently generating wheat – *T. monococcum* introgression lines to introduce the novel resistance to cultivated hexaploid wheat.

[1]. Jing et al., 2007. *J. Exp. Bot.* 58(13): 3749-3764

[2]. Jing et al., 2008. *New Phytologist* (in press)

Dates for your diary....

11th & 12th June 2008 - Cereals 2008
26th June - RRA Science Day
14th November - WGIN Stakeholder Meeting

Also at: www.rothra.org

Edited By: Mary-Louise Burnett

RRA is the Members' Association of Rothamsted Research
RRA is a company Limited by Guarantee. Registered in England No. 78931
Registered Charity No. 298226
Registered Office: Rothamsted Research, Harpenden, Hertfordshire. AL5 2JQ

For further information regarding RRA and / or Rothamsted Research
Please Contact: Mary-Louise Burnett
Rothamsted Research, Harpenden
Hertfordshire. AL5 2JQ
Telephone +44 (0) 1582 763133 ext. 2485
Fax +44 (0) 1582 760981
E-mail mary-louise.burnett@bbsrc.ac.uk
Rothamsted Research Website www.rothamsted.ac.uk