Characterisation of Fusarium Head Blight Resistance Located on Chromosome 4A of *Triticum macha*

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Abstract

Chromosome 4A of *Triticum macha* carries resistance to *Fusarium* head blight (FHB). Double haploid lines (DH) of *T. macha* 4A were used to determine the type of resistance and location of the gene(s). FHB resistance and yield trait data collected over two seasons following spray and point inoculation, indicate that the resistance is of type I and is probably conferred by a single gene. The resistance was mapped with microsatellite markers to a small area of the *T. macha* 4A chromosome flanked by markers gwm 610 and gwm 165. This could greatly facilitate future marker assisted selection work aimed at increasing resistance to FHB in other winter wheat lines.

Keywords: *Triticum aestivum; Triticum macha; Fusarium culmorum*

INTRODUCTION

The predominant causal agent of FHB in the UK is *Fusarium culmorum*. FHB can cause large reductions in yield but potentially more important is the accumulation of mycotoxins in the grain making it unfit for human or animal consumption (JOFFE 1983). *T. macha* has a high level of resistance to FHB that is conferred by several chromosomes (GRAUSGRUBER *et al*. 1998; MENTEWAB *et al*. 2000). The present study has characterised and located the resistance residing on chromosome 4A.

MATERIALS AND METHODS

45 DH of *T. macha* 4A in a Hobbit Sib background were spray and/or point inoculated at mid-anthesis with a single DON producing *Fusarium culmorum* isolate (FU 42). The trials were conducted in a polythene horticultural tunnel to maintain high humidity. Individual plants, grown in 1 litre pots, were randomised in four replicated blocks, 3 or 4 plants per line in each block. Two ears per plant were sprayed inoculated (1×10^5 conidia/ml) until run off (2001, 2002). A single ear per plant was point inoculated with 30 μl of 1×10^6 conidia/ml applied to a central cut spikelet (2002). Point inoculated and non-inoculated control ears were covered with small cellophane crossing bags to prevent contamination.

Visual disease was scored 7, 14 and 21 days post inoculation (dpi). Spray inoculated ears were scored as % of the spike showing disease, later converted to area under the disease progress curve (AUDPC). Point inoculated ears was scored as number of spikelets infected. Ear weights were taken at harvest and Relative Ear Weights (REW) calculated with respect to the control ears.

DNA was extracted from fresh leaf tissue of all the DH recombinant inbred lines using a modified CTAB method (MURRAY & THOMPSON 1980). Microsatellite analysis was conducted as BRYAN *et al*. (1997) using microsatellites obtained from RODER *et al*. (1998).

RESULTS

One-way analysis of variance of AUDPC (Tukey's pairwise comparison), showed a significant difference between DH lines ($P < 0.01$) with a bi-modal frequency distribution centering about the parents (Figure 1). Dunnett's comparison with the Hobbit sib parent as the control revealed that each line had a phenotype similar to one or other of the parents, there were no intermediates.

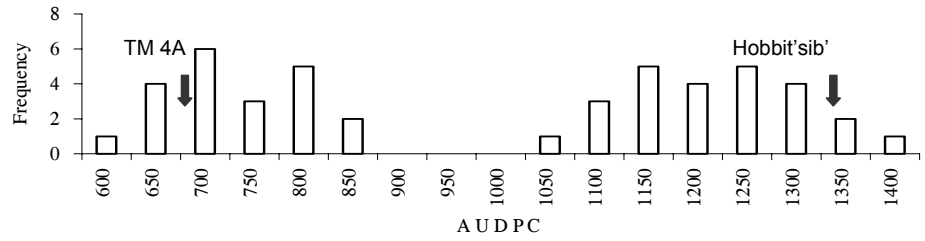

Figure 1. Frequency distribution for *Fusarium* head blight AUDPC (2001)

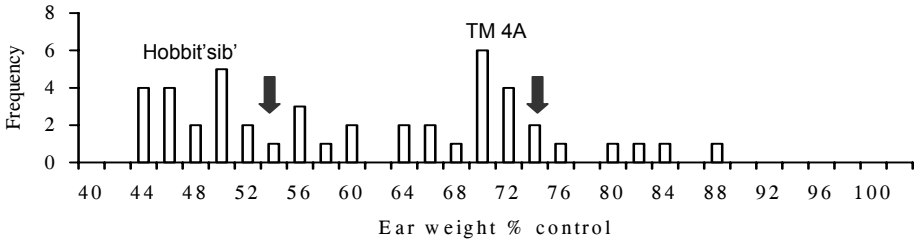

Figure 2. Frequency distribution for ear weight as % of the control (2001)

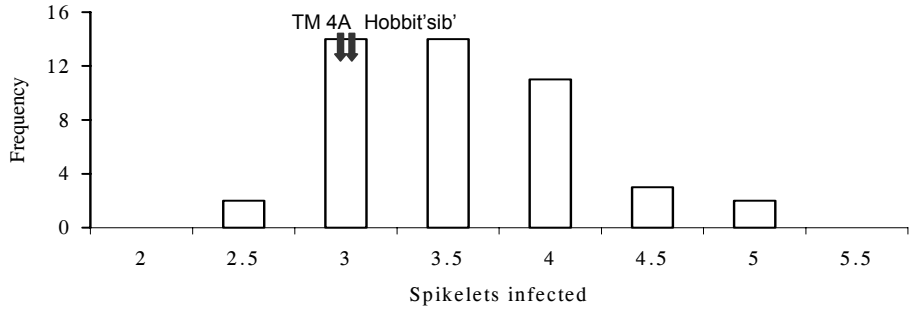

Figure 3. Frequency distribution for *Fusarium* headblight point inoculation (2002)

One-way analysis of variance for REW also showed significant differences between lines at the 0.05% level and distribution was also bi-modal (Figure 2). Those lines with the Hobbit sib/*T. macha* 4A disease resistance (AUDPC) phenotype had the higher REW. In all but four cases Dunnet's comparison (5% level of significance) using the Hobbit sib parent as the control showed that lines significantly different from the Hobbit sib parent for AUDPC were also significantly different for REW. The correlation between AUDPC in 2001 and 2002 was highly significant (0.863), the data from 2002 is not presented in this paper.

One-way analysis of variance showed no significant difference between the parental lines for point score at 14 or 21 dpi ($P > 0.05$). The frequency distribution for point inoculation followed a normal distribution for the DH recombinant lines about the two parents (Figure 3).

Using the JIC consensus microsatellite map in conjunction with the phenotyping and genotyping results for the DH lines, we determined that the *T. macha* resistance is in the centromeric region of 4A flanked by gwm 601 and gwm 610, data not shown.

Conclusion

The bi-modal frequency distribution for AUDPC and REW indicates that the FHB resistance of *T. macha* 4A is conferred by a single gene (or tightly linked group of genes). The combined results from the point and spray inoculation trials provide strong evidence that the resistance to FHB on chromosome 4A of *T. macha* is of Type I (resistance to initial infection). Most resistances reported for FHB to date are predominantly of Type II (ANDERSON *et al*. 2001; BUERSTMAYR *et al*. 2002). The resistance of *T. macha* 4A being of Type I makes it an ideal candidate for pyramiding with resistances such as those found on chromosome 3B in Sumai 3, that are of Type II. The combining of different resistance mechanisms to FHB should provide an effective method for combating the initial infection and spread of the fungus. We are

presently carrying out further back crosses of the *T. macha* 4A fragment into the Hobbit sib background to allow further elucidation of the resistance gene(s) position.

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