Genetic analysis of resistance to barley scald (*Rhynchosporium secalis*) in the Ethiopian line ‘Abyssinian’ (CI668)

S. Grønnerød1, A.G. Marøy1, J. MacKey2, A. Tekauz3, G.A. Penner3 & A. Bjørnstad1,∗

1Agricultural University of Norway, Department of Horticulture and Crop Sciences, P.O. Box 5022, N-1432 Ås, Norway; 2Department of Plant Breeding Research, Swedish University of Agricultural Sciences, S-75007 Uppsala, Sweden; 3Cereal Research Centre, Agriculture Canada, Winnipeg, Canada; (∗author for correspondence)

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Summary

A doubled haploid barley (*Hordeum vulgare* L.) population from a cross between the cultivar ‘Ingrid’ and the Ethiopian landrace ‘Abyssinian’ was mapped by AFLP, RFLP, SSR and STS markers and tested for resistance to isolates ‘4004’, ‘2’, ‘16-6’, ‘17’, ‘22’ and ‘WRS 1872’ of *Rhynchosporium secalis* (Oudem.) J.J. Davis, the causal agent of leaf scald. Resistance tests were conducted on parents, DH-lines, a near-isogenic line of ‘Abyssinian’ (NIL) into ‘Ingrid’, and an F2 population descended from the same F1 plants as the DHs. The DH population segregated for at least two major *R. secalis* resistance QTL. All isolates tested identified a major QTL on chromosome 3 (3H) associated with *R. secalis* resistance, in a 4 cM support interval between the co-segregating markers Bmac0209/Falc666 and MWG680. The QTL was linked with the markers Falc666 (2.3 cM), YLM/ylp (0.3 cM), MWG680 (1.7 cM), cttaca2 (2.5 cM) and agtc17 (9.8 cM). The second QTL was located on chromosome 1 (7H). However, this QTL was only detected by one isolate and was located in an interval of 16 cM in the distal part of the chromosome. At this QTL the allele for improved scald resistance originated from the parent ‘Ingrid’. There were a number of minor QTL on chromosomes 2 (2H), 4 (4H) and 6 (6H) that were not repeatable either across replications or analysis methods. The importance of checking QTL-models by cross-validation is stressed.

Introduction

Leaf scald of barley (*Hordeum vulgare* L.) is caused by the haploid imperfect fungal pathogen *Rhynchosporium secalis* (Oudem.) J.J. Davis. Scald is a serious foliar disease that occurs in all of the major barley growing regions of the world. The pathogen is characterised by an extensive genetic variability (Hansen & Magnus, 1973; Tekauz, 1991; Salamati et al., 2000). A sexual stage has not been reported for *R. secalis*, but Salamati et al. (2000) suggest that *R. secalis* populations in Norway, Finland, and Australia undergo regular sexual recombination, though a teleomorph has not been recognized.

Resistance of barley to the pathogen is often controlled by a gene-for-gene interaction, a gene for resistance in the plant corresponding to a gene for avirulence in the pathogen. Studies on the inheritance of resistance to *R. secalis* in barley led to the definition of several major and minor resistance genes. At least 14 different resistance genes, called *Rh* to *Rh14* have been reported (more correctly *Rrs* instead of *Rh* in some reports (Søgaard & Wettstein-Knowles, 1987; Abbott et al., 1992, 1995; Garvin et al., 1997, 2000), but there is still much confusion as to the number of loci. On the one hand, the *Rh-Rh3-Rh4* locus complex on chromosome 3 (3H) (Dyck & Schaller, 1961a; Wells & Skoropad, 1963; Habgood & Hayes, 1971) is regarded as a single gene with several alleles (Habgood & Hayes, 1971). Others maintain that at least two different, but closely linked genes exist (Dyck & Schaller, 1961b). Most of the varieties listed in the literature possess a resistance factor on chromosome 3 (3H).

A single gene with incomplete dominance controls the resistance in both ‘Abyssinian’ (CI668) and
‘Kitchin’ (CI1296) (Baker & Larter, 1963). In the homozygous state, this gene, designated Rh9, confers full resistance to either variety, whereas in a heterozygous state it expresses a delayed and intermediate reaction with small lesions on the margins of leaves and leaf axils (Baker & Larter, 1963). The Rh9 gene was assigned to chromosome 4 (4H) by trisomic analysis (Bockelman et al., 1977). Bockelman et al. (1977) also found that rh6 from ‘Jet’ was located on chromosome 4 (4H).

Abbott et al. (1992) reported linkage between scald resistance and the isozyme locus Acp2 on chromosome 4 (4H). This linkage is of general importance for the use of wild barley germplasm (Abbott et al., 1992). This isozyme locus is highly polymorphic in *H. vulgare* sp. *spontaneum* with at least nine alleles (Brown, 1983). The locus is much less polymorphic in *H. vulgare* sp. *vulgare* where only three alleles have been described (Nielsen & Johansen, 1986). The exact position of Acp2 is not known.

The objective of the present study is to determine the genetic control of resistance in ‘Abyssinian’ using a double haploid population from a cross between ‘Ingrid’ and ‘Abyssinian’, and molecular markers to map QTL for *R. secalis* resistance. The study also describes a near-isogenic line (NIL) of ‘Abyssinian’ into ‘Ingrid’.

**Material and methods**

**Plant materials**

The genetic material used in this study was various generations derived from a cross between the *R. secalis* resistant cultivar ‘Abyssinian’ (CI668) and the *R. secalis* sensitive cultivar ‘Ingrid’. A population of 50 doubled-haploid (DH) lines of ‘Ingrid’ × ‘Abyssinian’ (I × A) was produced from F1-plants by the *Hordeum bulbosum* L. method using embryo culture (Jensen, 1976) followed by chromosome doubling through colchicine treatment. A near-isogenic BC7, F8 line (NIL of ‘Abyssinian’, identified in a disease test done in 1996) of ‘Ingrid’ carrying alleles from ‘Abyssinian’, developed by Dr James MacKey and Dr Håkon A. Magnus, was also studied. Resistance tests were conducted on parents, DH-lines, NIL, and the F2 population descended from the same F1 plants as the DHs.

**Pathogen isolates and resistance tests**

Plants were tested with six different *R. secalis* isolates, including isolate ‘4004’ of Danish origin (Lyngs Jørgensen, 1992), four isolates of Norwegian origin (Salamati & Tronsmo, 1997) and one isolate from Canada (Graner & Tekauz, 1996). Table 1 outlines the *R. secalis* isolates used in the study, their virulence patterns based on octal numbers (Goodwin et al., 1990) and experiments done.

The barley plants were grown in fertilized peat in 30 × 20 cm plastic boxes with 55 ‘pots’ (Jiffy strips) in each box and 3–4 seeds per pot. The plants were inoculated at the two- to three-leaf stage (Z 12–13) (Zadoks et al., 1974) 14 days after sowing. Inoculum for each isolate was produced according to Salamati & Tronsmo (1997). The concentration of the inoculum was adjusted to 2 × 10^5 spores ml^-1. The spore suspension was sprayed as evenly as possible onto the plants with an electric jet vaporiser until leaves were moist by droplets. A single inoculum preparation was used on all seedlings in a given experiment. To maintain a high humidity, the inoculated plants were kept in a mist chamber or covered with plastic hoods for 48 hours incubation in the dark (Table 1). The temperatures were 17–18 °C. After incubation, the plants in September 1996 and March 1997 tests were grown in a growth chamber for two weeks, whereas the other test plants were grown in a greenhouse or phytotron, with temperatures 18 °C day/15 °C night and 18 hours light/6 hours dark. Plants were assessed visually for signs of scald symptoms on the lamina of the second leaf of single seedlings 14 days after inoculation. The following scale, modified from Lyngs Jørgensen (1992), was used:

0 No visible symptoms.
1 One or a few small discrete lesions, often with a dark margin, covering less than 5% of the lamina.
2 Somewhat larger lesions covering less than 10% of the lamina. Often not a typical lesion with a dark margin, but an irregular wilted greyish-green area without this margin.
3 Relatively large lesions about 10–40% of the lamina. Symptoms more often as described for type 2.
4 Partial collapse of the lamina. About 40–80% covered with greyish-green wilted areas with dark margins.
5 Total collapse of the lamina, 80–100% with symptoms as type 4.