Breeding Barley for Multiple Disease Resistance in the Upper Midwest Region of the USA

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Abstract: The Upper Midwest is one of the largest barley production areas in the USA. In this region, diseases can markedly reduce both the yield and quality of the crop. Molecular and classical breeding techniques are being employed to develop cultivars with resistance to five different diseases in the Minnesota barley improvement program. Stem rust and spot blotch have been successfully controlled for many years through the deployment of the major gene *Rpg1* and a major effect QTL, respectively. A sequence characterized amplified region (SCAR) marker developed from the sequence of *Rpg1* has made marker-assisted selection (MAS) for stem rust resistance highly effective. The major QTL controlling durable adult plant spot blotch resistance was first identified in the Steptoe/Morex population. This QTL was completely suppressed in the Harrington/Morex and Dicktoo/Morex populations, highlighting the importance of genetic background for the expression of resistance. The onset of Fusarium head blight (FHB) in 1993 led to dramatic changes in the focus of the breeding program. Significant resources have been expended to develop populations for mapping resistance QTL and identify closely linked markers for MAS. This is a difficult challenge because FHB resistance is controlled by many QTL with small effects. Sources of resistance to net blotch and Septoria speckled leaf blotch (SSLB) have been identified in a number of barley accessions. These resistances are simply inherited and are being introgressed into elite lines via phenotypic and MAS. Continued progress toward multiple disease resistance will require efficient phenotypic screening, MAS, and utilization of discoveries in barley genomics to manage numerous resistance genes and desirable gene complexes assembled over decades of breeding.

Keywords: barley; *Hordeum vulgare* L.; disease resistance; marker-assisted selection

The Upper Midwest region of the USA is one of the most productive cereal-growing regions in the northern Great Plains and the major source of barley for the malting and brewing industries. This region includes the states of Minnesota, North Dakota, and South Dakota and produces over 2000 Mt of barley annually, most of which is intended for malting. In recent years, economic factors and disease pressure have pushed six-rowed malting barley production from an area centred in northwestern Minnesota and eastern North Dakota to north central North Dakota and southern Canada. Diseases that commonly impact barley production in this region include stem rust (caused by *Puccinia graminis* f.sp. *tritici*), spot blotch (caused by *Cochliobolus sativus* [anamorph: *Bipolaris sorokiniana*]), FHB (caused primarily by *Fusarium graminearum* [teleomorph: *Gib-*

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berella zeae]), net blotch (caused by *Pyrenophora teres* f. *teres* [anamorph: *Drechslera teres* f. *teres*]), and Septoria speckled leaf blotch (SSLB) (caused by both *Septoria passerinii* and *Phaeosphaeria avenaria* f.sp. *triticea* [anamorph: *Stagonospora avenae* f.sp. *triticea*]) (Fetch *et al.* 2003; Steffenson 2003). The deployment of host resistance is often the preferred method of control for these diseases because it is an effective, economical, and environmentally sound strategy.

One of the great challenges in breeding malting barley is to incorporate multiple disease resistance while maintaining favourable gene complexes responsible for regional adaptation and acceptable malting and brewing characteristics. Approval of a barley cultivar for use in malting and brewing is based on about 25 different quality traits (Wych & Rasmusson 1983). Additionally, an approved cultivar must also pass taste tests after it is malted and made into beer. These specific requirements have forced breeders to cross closely related parents that already possess superior malting and brewing characteristics. As a result, the Minnesota barley germplasm base has been drastically narrowed to the extent in which 50% of the parentage traces back to only five ancestors (Martin *et al.* 1991). Introgression of genes from exotic sources, such as in the case of disease resistance, requires a process of parent building or cyclical breeding. In this process, the most desirable progenies from crosses in early cycles of breeding are used as parents in subsequent breeding cycles. After several breeding cycles, progenies will be suitable for crosses that will potentially lead to new cultivar candidates. The number of breeding cycles necessary will depend on the ease and reliability of the screening methods and whether the trait exhibits simple or complex inheritance. For more challenging diseases under complex genetic control (e.g. FHB), it is likely that at least 4–5 breeding cycles will be necessary to generate breeding lines that can be used as parents to ultimately produce a new cultivar. Parent building is generally used to improve a single trait. Therefore, breeding for multiple disease resistance can be viewed as a multiple parent building enterprise that will ultimately lead to the combination of desired resistances in a single cultivar. The objective of this paper is to review current and past efforts in breeding six-rowed malting barley cultivars for multiple disease resistance in the Upper Midwest region of the USA. Successes and continuing challenges in this endeavour are discussed as well as prospects for the future.

Stem rust

Stem rust has historically been one of the most devastating diseases of barley in the Upper Midwest region. Since 1942, losses to stem rust in barley have been minimal due to the planting of cultivars with the durable resistance gene *Rpg1* (Steffenson 1992). Pathotypes with virulence for *Rpg1* have been reported periodically in the Upper Midwest region since 1942 (Steffenson 1992). In 1989, a pathotype (QCCJ) with virulence for *Rpg1* became widespread in the Upper Midwest and damaged some barley fields (Roelfs *et al.* 1991). Pathotype QCCJ is still a threat to barley production in the region. To obtain stable stem rust control in the future, breeders may have to combine into cultivars *Rpg1* and gene(s) for resistance to pathotype QCCJ. The retention of *Rpg1* in new cultivars is essential because this gene has proven durable to many pathotypes of *P. g.* f.sp. *tritici* in the region for over 60 years. Resistance to pathotype QCCJ was identified in barley accession Q21861 (PI 584766) and is conferred by a single recessive gene *rpg4* (Jin *et al.* 1994). Prior to the appearance of pathotype QCCJ, breeding for stem rust resistance was easy because it only required the introgression of *Rpg1*. Since all of the elite parents carried *Rpg1*, stem rust resistance was maintained in the program without any phenotypic selection. The transfer of an additional gene (i.e. *rpg4*) for resistance to pathotype QCCJ will complicate the breeding effort. A significant advance for the high-throughput detection of *Rpg1* in the breeding program would be the development of a molecular marker in the gene itself. *Rpg1* was recently isolated by a map-based approach (Brueggeman *et al.* 2002). By exploiting sequence variation in the gene, Eckstein *et al.* (2003) developed a robust, allele specific SCAR marker that can differentiate between lines with the functional resistance gene and those that lack the gene or contain one of several susceptibility alleles. This *Rpg1* marker was 92% accurate in detecting stem rust resistance in a historical set of 100 Minnesota breeding lines and Midwestern cultivars (Condon *et al.* 2004). Development of a molecular marker within the *rpg4* gene is in progress (Kleinhofs

Table 1. Breeding scheme and timeline for disease screening in the Minnesota barley improvement program

Year	Breeding generation	Time/location	Disease screening[1]
1	parent selection	autumn greenhouse	SB(G), SR(M)
	F_1	winter greenhouse	
	F_2	summer field	FHB(M), SR(M), SSLB(M)
2	F_3	autumn greenhouse	
	F_4	winter nursery (NZ)	SSLB(G), NB(G)
	$F_{4:5}$	summer field	FHB(F), SSLB(F), NB(F)
3	$F_{5:6}$	winter nursery (NZ)	
	$F_{5:7}$ preliminary yield	summer field	FHB(F), SSLB(F), NB(F)
4	$F_{5:8}$ intermediate yield	summer field	FHB(F), SSLB(F), NB(F)
5	$F_{5:8}$ advanced yield	summer field	FHB(F), SSLB(F), NB(F)

[1]SB = spot blotch; SR = stem rust; FHB = Fusarium head blight; SSLB = Septoria speckled leaf blotch; NB = net blotch; (G) = greenhouse disease screen; (M) = DNA marker screen; (F) = field disease screen; (NZ) = New Zealand

et al., unpublished) and when completed it will allow multiplexing molecular markers for the two stem rust resistance genes on parents and in early generation (F_2) segregating populations, thereby increasing the efficiency and throughput of stem rust resistance breeding (Table 1). Still, stem rust phenotyping (Jin *et al.* 1994) must be done to verify the presence of the genes and their expression, since the *Rpg1* marker has not proven infallible.

Spot blotch

Spot blotch was one of the most devastating foliar diseases of barley in the Upper Midwest region. The disease has been successfully controlled for over 40 years through the use of host resistance and is one of the great success stories in breeding barley for resistance. This durable spot blotch resistance was derived from the breeding line NDB112 and has been incorporated into all of the major six-rowed malting cultivars grown in the region (Steffenson *et al.* 1996). To elucidate the genetic basis of durable spot blotch resistance in six-rowed malting cultivars, we studied the Steptoe/Morex (S/M) population. Morex is a resistant six-rowed malting cultivar derived from NDB112, and Steptoe is a susceptible six-rowed feed cultivar. A single gene (designated *Rcs5*) located at the telomeric region of chromosome 1(7H) was found to confer spot blotch resistance at the seedling stage (Steffenson *et al.* 1996). Two quantitative trait loci (QTL) conferred adult plant resistance in the S/M population: one of major effect on chromosome 5(1H) explaining 62% of the variance and the other of minor effect on chromosome 1(7H) explaining 9% of the variance (Table 2). The QTL on chromosome 1(7H)

Table 2. Summary of major QTL (chromosomal location and % phenotypic variance explained) contributing to adult plant spot blotch resistance in three mapping populations derived from resistant parent Morex

Population	Chrom 1(7HS) *iEst5-ABC158*	Chrom 3(3HS) *saflp119-saflp54*	Chrom 3(3HL) *saflp35-saflp53*	Chrom 5(1HL) *ABG500A-ABG452*
S/M	12	–[1]	–	62
D/M	20	36	11	–
H/M	75	–	–	–

[1]No significant QTL detected in this region

mapped to the same region as *Rcs5*. Thus, durable spot blotch resistance in six-rowed malting barley cultivars is conferred mostly by a single QTL of major effect on chromosome 5(1H). To corroborate these findings, the same analysis was conducted on the two- × six-rowed cross of Harrington/Morex (H/M). Harrington is a susceptible two-rowed malting cultivar. As in the S/M population, a single gene (presumably *Rcs5*) on chromosome 1(7H) conferred spot blotch resistance at the seedling stage. However, a different and quite unexpected result was obtained for adult plant resistance in the H/M population: no chromosome 5(1H) effect was detected. Instead, a single gene mapping at or near *Rcs5* on chromosome 1(7H) conferred resistance. When the disease severity data were subjected to quantitative analysis, a single major effect QTL explaining 75% of the variance was identified, again at or near *Rcs5* (Table 2) (Steffenson 2000; Bilgic *et al*. 2006). One additional population involving Morex (Dicktoo/Morex [D/M]) was tested for its reaction to spot blotch. In this case, the susceptible parent was the six-rowed feed cultivar Dicktoo; thus, the D/M population was used to test whether the Morex-derived chromosome 5(1H) adult plant resistance QTL first identified in the S/M population would again be expressed in a different six- × six-rowed cross. Three QTLs were detected at the adult plant stage in the D/M population: one on the short arm of chromosome 3(3H) explaining 36%, the second on the long arm of chromosome 3(3H) explaining 11%, and the third near *Rcs5* on the short arm of chromosome 1(7H) explaining 20% of the phenotypic variation (Bilgic *et al*. 2006). No effect whatsoever was detected in the chromosome 5(1H) region where the adult plant resistance QTL was first discovered in the S/M population (Table 2). Over the past 40 years, breeders have been very successful in retaining the chromosome 5(1H) resistance QTL in their six-rowed malting germplasm, presumably by fixing the resistance allele in elite parents and practicing occasional phenotypic selection. It appears that this resistance is highly expressed in the six-rowed genetic backgrounds of the major malting barley breeding programs in the Midwest. This resistance QTL may, however, be completely suppressed when introgressed into more diverse two- or six-rowed genetic backgrounds (e.g. H/M and D/M populations). Molecular markers for the chromosome 5(1H) spot blotch resistance QTL are being developed. Their utility in MAS for the chromosome 5(1H) QTL may be limited given the suppression that occurs in crosses with both two- and six-rowed susceptible parents. In the future, we will employ MAS to verify that parents used in the breeding program carry the resistance allele at the 5(1H) QTL (Table 1) and continue to screen advanced breeding lines in the field to ensure that the resistance is expressed in the current breeding background.

Septoria speckled leaf blotch

Septoria speckled leaf blotch (SSLB) is a disease complex caused by two different pathogens. In the Upper Midwest region, *S. passerinii* is the most common SSLB pathogen, although *P. a*. f.sp. *triticea* is also frequently isolated from symptomatic barley tissue (Krupinsky & Steffenson 1999). In recent years, SSLB has re-emerged as one of the most important diseases of barley in the Upper Midwest region due to the increased use of minimum tillage and high rainfall during the growing season. Yield losses of 23–38% were reported on barley due to *S. passerinii* infection (Toubia-Rahme & Steffenson 2004). All of the major malting and feed barley cultivars in the Upper Midwest region are highly susceptible to SSLB (Toubia-Rahme *et al*. 2003). Fortunately, many sources of resistance to *S. passerinii* have been identified in both cultivated (Rasmusson & Rogers 1963; Legge *et al*. 1996) and wild barley (*H. vulgare* subsp. *spontaneum* and *H. bulbosum*) (Fetch *et al*. 2003; Toubia-Rahme *et al*. 2003). In the Minnesota barley improvement program, two sources of resistance are being used: CIho 4780 (an accession from northern China) and PC84 (a breeding line from the ICARDA/CIMMYT program in Mexico). Both accessions exhibit high levels of resistance in the field. Resistance in CIho 4780 is conferred by a single dominant gene *Rsp2* (Rasmusson & Rogers 1963), which was recently mapped to the short arm of chromosome 5(1H) (Zhong *et al*. 2006). A SCAR marker cosegregating with *Rsp2* was developed and evaluated for MAS of SSLB resistance. Selection of F_2 plants homozygous for the resistance allele of the SCAR marker in two segregating populations was 96–100% effective in identifying SSLB resistant F_5 lines. Resistance in PC84 is thought to be under the control of a single dominant gene that is different from the one present in CIho 4780 (Steffenson & Smith, unpublished). Our goal is to increase

the diversity of SSLB resistance by incorporating both genes into new cultivars.

Net blotch

Net blotch is perhaps the most important foliar pathogen of barley in the Upper Midwest on an annual basis given the sporadic nature of SSLB epidemics and the success attained in controlling stem rust and spot blotch by host resistance. The disease is widely distributed and is often found in high severities in commercial fields (Steffenson, unpublished). Many sources of net blotch resistance have been described in cultivated and wild barley (Shipton *et al.* 1973; Fetch *et al.* 2003). The Canadian cultivar Heartland is currently being used as a source of net blotch resistance in the Minnesota program. Preliminary studies indicate that this resistance is simply inherited. We have initiated work to identify markers that will be useful in MAS for net blotch resistance. Currently, we screen for net blotch resistance in segregating populations during single seed descent using remnant F_4 seed in a greenhouse seedling assay (Table 1). Resistant lines ($F_{4:5}$) are advanced to a field screen on adult plants where selection is based on disease resistance as well as other traits (i.e. lodging, stem strength, height, maturity, etc.).

Fusarium head blight

FHB is one of the most devastating and insidious diseases of barley. In addition to causing yield loss, the primary pathogen, *F. graminearum*, produces various mycotoxins (most notably deoxynivalenol or DON) that are hazardous to humans and animals (Steffenson 2003). FHB has been a relatively minor and sporadic disease problem of barley in the United States for many years. Over the past decade, however, it has re-emerged as the most important factor reducing the yield and quality of the crop in the Upper Midwest. The head blight epidemics of the 1990's were particularly devastating and caused severe economic losses, grain processing problems for producers and end-users alike, food/feed safety concerns, and human hardship (Steffenson 2003). These epidemics also forced breeders to make drastic changes in their programs. Today, a significant portion of the breeding effort is focused on breeding for resistance to FHB and the accumulation of DON. A number of conventional and molecular mapping studies have been made on the genetics of FHB resistance in barley (reviewed in Steffenson 2003). All have reported complex inheritance for the trait. The molecular mapping studies indicate that FHB resistance is a complex quantitative trait controlled, in most cases, by a number of loci with relatively small effects that are scattered across the barley genome. From these genetic studies, it is evident that FHB resistance in barley is under polygenic control and its heritability can vary greatly. Given the great importance of this disease, the numerous challenges in quantifying FHB severity, and the complex genetics of resistance, we have developed a modified FHB breeding strategy in the Minnesota program. The large experimental error and environmental effects on FHB severity have dictated that our early generation screening efforts employ multiple locations and replications. For other diseases such as net blotch, it is possible to do greenhouse screening on seedlings using remnant seed from early generations (F_3, F_4) during single seed descent, followed by a single F_5 head row evaluation in the field for a number of traits. For FHB, we cannot effectively conduct greenhouse screening in early generations. In year two (F_5 generation), we evaluate FHB reaction in misted and inoculated field nurseries (Table 1). Each new breeding line is replicated twice at two locations and evaluated for FHB severity. We harvest grain from resistant lines and checks for quantification of DON. In addition, we grow a fifth row in a non-inoculated nursery and harvest the grain for malting quality evaluation. Because FHB resistance is linked to maturity and plant morphology traits, we have emphasized selection for resistance prior to selection for other traits in the early cycles of breeding.

The need for replication in early generations and the desire to work with more homozygous material (F_4-derived) have forced us to make changes in our single seed descent program. The initial protocols, however, are the same. We make most crosses in the autumn, grow F_1's in the winter greenhouse, and F_2's in a summer field trial (Table 1). We then plant the F_3 generation immediately after harvest in early August to allow for an off-season F_4 generation in New Zealand. The F_4 generation is planted as spaced single plants to allow the harvest of sufficient $F_{4:5}$ seed for growing five 1.8 m rows in the disease and quality nurseries described above. This laborious screening effort has forced us to reduce the number of crosses and

new lines that we can evaluate each year, but has given us much more confidence in our early generation selection. In year three, we evaluate lines selected from year two in five disease nurseries with three replications per nursery. These same lines are evaluated in preliminary yield trials at two locations. Lines that continue on in year four are evaluated in three location trials in Minnesota. The best lines from the advanced yield trials (year five) are evaluated in a collaborative regional FHB nursery with eight locations in Minnesota, North Dakota, and Canada.

Recently, we have begun to evaluate MAS for FHB resistance. We evaluated markers linked to two major QTLs for FHB resistance discovered (De La Pena *et al*. 1999) and validated (Canci *et al*. 2003) from the Chevron source of resistance. The Chevron alleles at the QTL on chromosome 2(2H) reduced FHB by 43% and increased HD by two days as was predicted by the mapping studies (Gustus & Smith 2001). Selection for the Chevron alleles at the chromosome 6(6H) region reduced FHB by 22%, but also increased grain protein by 14 g/kg. We are continuing to evaluate these and other markers to increase the efficiency of FHB selection. MAS is generally used to select lines homozygous for the resistance marker allele in the F_2 generation prior to single seed descent (Table 1).

CONCLUSIONS AND FUTURE DIRECTIONS

The successful development of malting barley cultivars with multiple disease resistance requires the introgression of resistance alleles that function in the target genetic background and are free of linkage to undesirable traits. Past progress has relied on parent building after fixing genes for resistance or by exploiting individual segregating populations using phenotypic selection. For several diseases, markers now allow breeders to track resistance alleles in the broad arrays of breeding lines within the program, thereby reducing the need for expensive and sometimes variable phenotypic screening. In the future, it may be possible to exploit phenotypic variation in the complex pedigree structure of breeding germplasm to identify new QTL through the use of association genetics (Jannink *et al*. 2001). This approach exploits the tremendous amount of phenotypic data generated by breeding programs and the relatively inexpensive DNA genotyping technologies currently available to study important traits. By routinely genotyping breeding lines with a strategic set of DNA markers, it will be possible to validate QTL in the relevant germplasm, identify new QTL for important breeding traits, and determine if alleles introgressed into breeding lines perform as predicted by genetic studies. The rapidly advancing field of genomics is providing information on the location, expression profile, and function of genes that will be important for continued progress in breeding as well as new tools for manipulating them in breeding programs. All of this new technology and information will facilitate the management of multiple disease resistance in barley.

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