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The genetics of non-host disease resistance in wheat to barley yellow rust

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Abstract Non-host resistance is investigated as a potential source of durable resistance. However, the genetics of non-host resistance between closely related plant species and their corresponding pathogens would indicate that in these interactions, non-host resistance primarily involves major genes that operate on a gene-for-gene principal similar to that seen in host resistance. Wheat is a non-host of the barley-attacking form of the fungus responsible for yellow rust, i.e. *Puccinia striiformis* f. sp. *hordei*. While *P. striiformis* f. sp. *hordei* is generally unable to infect wheat, a partial susceptibility was exhibited by the wheat variety Chinese 166. Consequently, in the cross Lemhi × Chinese 166 two major QTLs for resistance to *P. striiformis* f. sp. *hordei* were identified: one on chromosome 1D and a second on 2B. These two QTLs accounted for 43.5% and 33.2% of the phenotypic variance for resistance to barley yellow rust, respectively. In addition, two QTLs of smaller effect were also identified: one on chromosome 5A, contributing 5.1% of the variance and a second on chromosome 6A, contributing 10.9% to the phenotype. The QTL on 6A was derived from the susceptible variety, Chinese 166. In all cases the resistance towards *P. striiformis* f. sp. *hordei* was associated with a visual chlorosis/necrosis response typical of race-specific host resistance.

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Introduction

Non-host resistance, defined as the ability of most genotypes of a plant species to resist infection by most isolates of a potential pathogen (Heath 1981; Niks 1988), encompasses the full spectrum of non-host/non-pathogen interactions, from a pathogen of cereals unable to infect dicotyledonous species to the situation where a pathogen of barley is unable to infect wheat. The biology and genetic complexity behind the non-host interactions in these extremes is likely to be very different.

In cases of non-host interactions where the host species of the pathogen is taxonomically very distant from the non-host species under study, resistance is thought to be due to the absence of a basic compatibility between the plant and the pathogen (Heath 1981, 1991). The pathogen lacks the required pathogenicity factors to achieve colonisation of the plant species, being unable to overcome the plant's basic defence mechanisms, and the plant does not provide the environment required by the pathogen for successful infection (Heath 2000). Where a basic compatibility has been established between a parasitic organism and a plant species, allowing the parasite to infect and reproduce, the plant species has now become a host and the parasite its pathogen. Superimposed upon this basic compatibility is a pathogen-recognition system that allows the plant to fight back. This host resistance is genotype-specific, and usually race-specific, effective only against certain races of the pathogen (Heath 1981; Niks 1988).

In cases of non-host/non-pathogen interactions involving formae speciales, the genetics and biology of resistance appears to resemble that of race-specific host resistance (Niks 1988; Tosa 1996). In these interactions

plant and pathogen responses are seen which are commonly associated with race-specific host resistance, including post-haustorial retardation of the pathogen and death of the invaded plant cell (Niks 1988). In *Arabidopsis*, a non-host resistance gene (R-gene), *Rac4*, effective against the *Brassica oleracea* pathogen *Albugo candida*, the causal agent of white rust, has been isolated (Holub 2002). The structure of *Rac4* is similar to that of host R-genes belonging to the NBS-LRR class of R-genes cloned from a wide range of plant species. This supports the theory that the biology of non-host resistance against pathogens of closely related plant species is similar to the biology of race-specific host resistance and will involve R-genes that belong to the classes of host R-genes already cloned.

The causal agent of yellow rust in cereals is a biotrophic fungus, *Puccinia striiformis*. Different formae speciales of this fungus are responsible for infection on different cereal species. On barley, yellow rust is caused by *P. striiformis*. f. sp. *hordei* and on wheat by *P. striiformis* f. sp. *tritici*. Both formae speciales, by definition, are non-pathogens of each other's host, with most isolates of each pathogen being unable to infect most genotypes of the other's host species. Population studies using DNA markers show that *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* isolates fall into two very distinct groups (Chen et al. 1995), and attempts to produce somatic hybrids between isolates of these two formae speciales have been unsuccessful (Newton et al. 1986), indicating that barley and wheat yellow rust are caused by two distinct pathogens. However, occasionally varieties of wheat and barley are found that can be partially infected by isolates of *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici*, respectively (Johnson and Lovell 1994). These rare cases of susceptible non-host genotypes allow studies of the genetics of non-host resistance.

Studies in wheat identified a *P. striiformis*. f. sp. *hordei* isolate, BWR80/1, that was able to partially infect the wheat variety Chinese 166 (Johnson and Lovell 1994). This susceptibility of Chinese 166 allowed the identification of genes that prevented *P. striiformis* f. sp. *hordei* infection in Lemhi, a wheat variety which is highly susceptible to wheat yellow rust, with few known host R-genes (Garrood 2001). Two genes with major effect that prevented *P. striiformis* f. sp. *hordei* from infecting Lemhi were identified by QTL mapping. Two loci, having a minor effect on non-host resistance in the Lemhi × Chinese 166 cross were also found.

Materials and methods

Plant material and mapping population

Wheat varieties Chinese 166 and Lemhi and barley variety Berac were used as controls. F_2 populations were obtained by crossing Chinese 166 and Lemhi in reciprocal crosses, using plants that had first been checked for their disease reaction to *P. striiformis* f. sp. *hordei* isolate BWR80/1. Cross CP8 used Lemhi as the male parent, and cross CP9 used Chinese 166 as the pollen donor. F_3 families were obtained after self-pollination of each F_2 individual.

Pathogenicity of the barley yellow rust isolate BWR80/1 was tested on the wheat and barley varieties listed in Table 1. For the genetic analysis 282 F_2 plants were tested for their disease reaction towards BWR80/1 (cross CP8, 139 F_2 plants; CP9, 143 F_2 plants). The analysis was carried out in three separate tests using 100 F_2, 91 F_2 and 91 F_2 plants, respectively. For the mapping population another 118 F_2 plants and their F_3 families were screened for their disease reaction towards BWR80/1.

Pathogen isolates and yellow rust disease tests

The *P. striiformis* f. sp. *hordei* isolates used in this study are part of a rust collection currently held at John Innes Centre. Isolate BWR80/1 [avirulent on Lemhi, infection type (IT) ;, virulent on Chinese 166,

Table 1 Disease reactions to *Puccinia striiformis* f. sp. *hordei* isolate BWR80/1 on wheat and barley varieties. *IT* Infection type

Variety[a]	First leaf IT[b]	Second leaf IT
Wheat:		
Carstens V (*YrCV*)	0^n	;
Chinese 166 (*Yr1*)	2^{cn}–3	1–2^{cn}
Clement (*Yr2*, *Yr9*, *Yr25*, *YrCle*)	;	;
Compair (*Yr8*, *Yr19*)	;–0	;
Heines VII (*Yr2*, *Yr25*, *YrHVII*)	0^c	;
Heines Kolben (*Yr2*, *Yr6*)	0^n	;–0^n
Heines Peko (*Yr2*, *Yr6*, *Yr25*)	;–0^c	;
Hybrid 46 (*Yr4b*)	;–0^c	;
Lee (*Yr7*)	0^n	;
Lemhi (*YrLem*)	;	;
Moro (*Yr10*, *YrMor*)	;	;
Nord Desprez (*Yr3a*, *Yr4a*)	;–0	;
Reichersberg 42 (*Yr7*, *Yr25*)	;–0	;
Spaldings Prolific (*YrSp*)	0^c	;
Strubes Dickkopf (*YrSd*, *Yr25*)	0^c	;
Suwon 92/Omar (*Yr4*, *YrSu*)	;–0	;
T. spelts (*Yr5*)	0^c	;
Vilmorin 23 (*Yr3a*, *Yr4a*)	0^n	;
Barley:		
Berac	4^c	4^c
Bigo (*BYr2*)	0^c	0^c
Delibes (unknown)	0	;
Derkado (unknown)	0^c	;
Golden Promise	4	4
Igri (unknown)	0^c	0–1^c
Midas	4	4
Sultan	4	4
Triumph (*BYr3*)	0^c	0^c

[a]The host resistance genes to *P. striiformis* present in each variety are given in *parentheses* (Boshoff et al. 2002; Meadway and Hutton 1998)

[b]IT disease reaction: *0* no visible symptoms, *;* small necrotic flecks, *n;* necrotic regions greater than 1 mm in diameter, 0^n necrotic regions greater than 2 mm in diameter, 0^{nn} spreading necrotic regions greater than 4 mm in diameter, *1* small sporulating uredia surrounded by necrotic tissue, *2* moderately sized sporulating uredia surrounded by necrotic tissue, *3* moderately sized sporulating uredia surrounded only by chlorotic tissue, *4* large sporulating uredia surrounded by green tissue, *n* necrotic tissue, *c* chlorotic tissue

IT 1^{cn}–3 and virulent on Berac, $IT4^{c}$] was used to screen the CP8 and CP9 F_2 and F_3 populations. *P. striiformis* f. sp. *hordei* isolates used to screen Chinese 166 and Lemhi are listed in Table 2. All yellow rust disease tests were carried out on 12- to 14-day-old seedlings grown under spore-free conditions, using 10–12 seedlings per variety for each test. The inoculation procedure is as described in Boyd and Minchin (2001). Disease reactions were scored 14–16 days after inoculation using the following IT scale (Fig. 1):

- 0 No visible symptoms
- ; Small necrotic flecks
- n; Necrotic regions greater than 1 mm in diameter
- 0^n Necrotic regions greater than 2 mm in diameter
- 0^{nn} Spreading necrotic regions greater than 4 mm in diameter
- 1 Small sporulating uredia surrounded by necrotic tissue
- 2 Moderately sized sporulating uredia surrounded by necrotic tissue
- 3 Moderately sized sporulating uredia surrounded only by chlorotic tissue
- 4 Large sporulating uredia surrounded by green tissue
- n Necrotic tissue
- c Chlorotic tissue

For QTL mapping a numerical disease score was given to each F_2 plant. F_2 plants were given a disease score based on the phenotype of the individual plant, using a 1–6 scale or a 1–9 scale based on the disease reaction of the F_2 plant and the disease-reaction types segregating in the F_3 family:

IT	Disease score	
	1–6 scale	1–9 scale
F_2 IT;/F_3 all IT;	1	9
F_2 ITn;/F_3 not segregating for IT4 or $IT0^{nn}$	2	8
F_2 ITn;/F_3 segregating for IT4 and $IT0^{nn}$	2	7
F_2 $IT0^{n}$/F_3 not segregating for IT4 or $IT0^{nn}$	3	6
F_2 $IT0^{n}$/F_3 segregating for IT4 and $IT0^{nn}$	3	5
F_2 $IT0^{nn}$/F_3 not segregating for IT4	4	4
F_2 $IT0^{nn}$/F_3 segregating for IT4	4	3
F_2 IT4/F_3 all IT4	6	1

SSR and AFLP mapping

After scoring F_2 seedlings for their disease reaction to BWR80/1, seedlings were grown until the eighth leaf had emerged. Plant DNA was extracted from uninfected leaf material using the CTAB protocol (Saghai-Maroof et al. 1984). Polymorphic AFLP markers were identified between Chinese 166 and Lemhi as described by Smith et al. (2002). A total of 256 +2/+2 AFLP primer pair combinations were screened.

Polymorphic SSR markers were identified between Chinese 166 and Lemhi as described by Boyd et al. (2002), using the psp (Bryan et al. 1997), the gwm (Röder et al. 1998) and the barc (http://www.scabusa.org) wheat SSR markers. Eight-eight SSR markers were screened, of which 41 were polymorphic (46.6% polymorphism). The wheat varieties Chinese Spring, Cappelle Desprez and the Chinese Spring nullisomic and nulli-tetrasomic lines were used as controls to confirm the SSR marker pattern and chromosomal location.

Linkage map construction

JoinMap version 3.0 for Windows (van Ooijen and Voorrips 2001) was used to create a linkage map of an F_2 population from the cross Lemhi × Chinese 166 (118 F_2 individuals), using 172 AFLP and 41 SSR markers. Markers with more than 40% of the data points missing or showing a χ^2 probability of >0.5% (P=0.05) were removed from further analysis. Linkage groups were determined using a minimum LOD score of 3.0/3.5 and a maximum recombination frequency (REC) of 0.45, with most linkage groups holding a range of LODs from 3.0 to 7.0. The recombination values were converted into genetic distances using the Kosambi (1994) mapping function. Twenty-two linkage groups were obtained, of which 18 formed linkage maps. The linkage maps used for QTL analysis were the ones derived from the first round of mapping (i.e. the map generated under the most stringent mapping conditions).

QTL mapping analysis

The QTL mapping package MapQTL version 4.0 for Windows (van Ooijen et al. 2002) was used to locate QTLs for resistance to *P. striiformis* f. sp. *hordei* in the Lemhi × Chinese 166 F_2 mapping population. The Kruskal-Wallis test, interval mapping and MQM mapping, using both F_2 data sets (scale 1–6 and 1–9), were applied.

The F_2 data set based on the 1–6 scale gave a normal distribution (Fig. 2a). The data set based on the scale 1–9 gave a good approximation to a normal distribution (Fig. 2b), with a correlation of R=0.9914 and a P>0.10 that the data fit a normal distribution (Ryan-Joiner test for normality; Minitab, version 13.1 for Windows). No mathematical transformation of this data set improved the normality of the distribution; therefore, a Kruskal-Wallis test was also applied to this data set. The Kruskal-Wallis test is a non-parametric test in which no assumptions are made about the probability distribution of the quantitative trait (after fitting the QTL genotype).

The criterion for detecting a QTL was set at a significance level of 0.05 in the Kruskal-Wallis test. For QTL mapping an imposed significance value of 5% gave an upper LOD significance threshold of 4.2 as calculated by van Ooijen (1999), while a permutation analysis (1,000 permutations, MapQTL) gave an upper LOD threshold of 3.9. The markers with the highest LOD values in the region of each putative QTL were used as co-factors for MQM mapping (Jansen and Stam 1994).

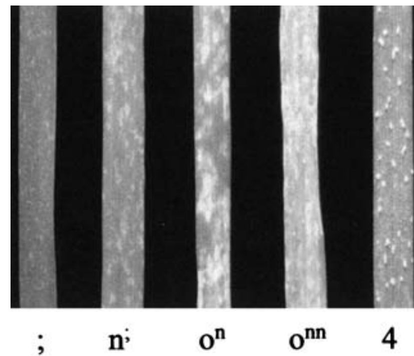

Fig. 1 Disease-infection phenotypes seen on F_2 progeny from the cross Lemhi × Chinese 166, following inoculation with the *P. striiformis* f. sp. *hordei* isolate BWR80/1. Infection types shown are: *;* small necrotic flecks, *n;* necrotic regions greater than 1 mm in diameter, 0^n necrotic regions greater than 2 mm in diameter, 0^{nn} spreading necrotic regions, greater than 4 mm in diameter and *4* large, sporulating uredia surrounded by green tissue

Table 2 Disease reactions on wheat and barley varieties to eight *P. striiformis* f. sp. *hordei* isolates. IT infection type

P. striiformis f. sp. *hordei* isolates	Chinese166		Lemhi		Berac	
	First leaf IT[a]	Second leaf IT	First leaf IT	Second leaf IT	First leaf IT	Second leaf IT
BYR84/4	0–1^{cn}	1–2^{cn}	;	;	4^{c}	4^{c}
BYR84/3	1–2^{cn}	1–2^{cn}	;	;	4^{c}	4^{c}
BYR82/6	0–2^{cn}	2^{cn}	;	;	4^{c}	4^{c}
BWR81/1	1–2^{cn}	2^{cn}–3	;	;	4^{c}	4^{c}
BWR80/1	1–2^{cn}	2^{cn}	;	;	4^{c}	4^{c}
BYR79/2	0–1^{cn}	1–2^{cn}	;	;	4^{c}	4^{c}
BYR61/31	0–1^{cn}	1–2^{cn}	;	;	4^{c}	4^{c}
BYR60/7	0	;	;	;	4^{c}	4^{c}

[a]IT disease reaction: 0 no visible symptoms, ; small necrotic flecks, n; necrotic regions greater than 1 mm in diameter, 0^{n} necrotic regions greater than 2 mm in diameter, 0^{nn} spreading necrotic regions greater than 4 mm in diameter, 1 small sporulating uredia surrounded by necrotic tissue, 2 moderately sized sporulating uredia surrounded by necrotic tissue, 3 moderately sized sporulating uredia surrounded only by chlorotic tissue, 4 large sporulating uredia surrounded by green tissue, n necrotic tissue, c chlorotic tissue

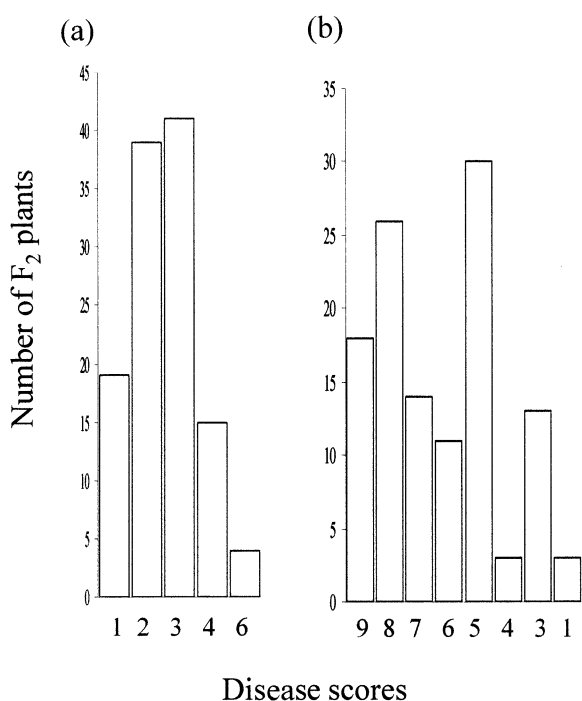

Fig. 2a, b Distribution of the disease scores given to F_2 individuals of the Lemhi × Chinese 166 mapping population. Two disease score scales were used, one based on a 1–6 scale (**a**) and the other on a 1–9 scale (**b**)

Results

Non-host yellow rust disease resistance tests

P. striiformis f. sp. *hordei* isolate BWR80/1 was inoculated onto a range of wheat and barley varieties to confirm the isolate as forma specialis *hordei* (Table 1). All wheat varieties, except Chinese 166, were fully resistant to BWR80/1, and only a weak susceptible reaction was seen on Chinese 166 (IT 1^{cn}–3). BWR80/1 was able to infect four of the nine barley varieties tested, giving fully susceptible, IT4 disease reactions. The resistance response on the remaining barley varieties would indicate a host resistance towards BWR80/1 in these varieties.

To confirm that susceptibility of Chinese 166 to *P. striiformis* f. sp. *hordei* was not specific to isolate BWR80/1, eight *P. striiformis* f. sp. *hordei* isolates, collected in the United Kingdom between the years 1960 and 1984, were tested on Chinese 166 (Table 2). All *P. striiformis* f. sp. *hordei* isolates, except BYR60/7, produced a similar susceptible reaction in Chinese 166 to that seen with BWR80/1.

Genetics of resistance to *P. striiformis* f. sp. *hordei* in the cross Lemhi × Chinese 166

Reciprocal crosses were made between Lemhi and Chinese 166. An F_2 population of 282 plants (cross CP8, 139 F_2 plants; CP9, 143 F_2 plants) was screened for resistance to *P. striiformis* f. sp. *hordei* isolate BW80/1. Another 118 F_2 individuals selected as a mapping population were also tested for resistance to isolate BWR80/1. No significant differences were seen in the number of resistant and susceptible plants from the two crosses CP8 and CP9, indicating that there was no maternal effect on the resistant phenotype (data not shown).

Previous analysis had indicated that two major genes were contributing to the resistance in Lemhi to BWR80/1 (Johnson and Lovell 1994). χ^2 analysis of the 282 F_2 population (observed R=269, S=13; χ^2=1.29, df=1, P=0.5–0.25) and the 118 F_2 mapping population (Fig. 2; χ^2=1.65, df=1, P=0.25−0.1) indicated that the data gave a reasonable fit to a model for the segregation of two unlinked dominant genes (15R:1S). However, in both F_2 populations there were fewer susceptible plants than expected. This would suggest that in addition to the two major genes, one or more genes of possible minor effect for

resistance towards BWR80/1 were segregating in this cross.

Linkage map construction

After χ^2 analysis of the 213 segregating loci, 11 loci were excluded from further analysis, either because of a high χ^2 value (P>0.05, five loci) or a large number of missing data points (six loci). From the 202 remaining loci, 18 linkage maps were constructed. Eighty-six markers remained ungrouped, including 27 SSR markers; the 18 linkage maps being composed of 116 loci. The map spanned 680 cM, with an average marker coverage of one marker every 6 cM. Eight of the 18 linkage maps were assigned to six wheat chromosomes (1D, 2B, 3A 5A, 6A and 6B) using SSR markers, the chromosomal location of which had been confirmed by nullisomic and nulli-tetrasomic analysis.

QTL identification

Interval mapping identified two QTLs (Fig. 3): one on chromosome 1D and the other on 2B. These two QTLs were detected using both F_2 data sets, although when the scale 1–6 scores were used, the QTL on chromosome 1D had a LOD value of 3.32, below the LOD threshold of 4.2.

The F_2 disease-reaction scores based on the 1–9 scale were used for MQM mapping. The two markers with the highest LOD values associated with each of the QTLs detected by interval mapping (chromosome 1D, S13 M15_170 and GWM106 and chromosome 2B, PSP3034 and GWM120) were used as co-factors (Fig. 3). The presence of the QTLs on chromosomes 1D and 2B were supported by MQM mapping (Table 3). Both QTLs originated from the non-host resistant parent Lemhi. The QTL on chromosome 1D accounted for most of the phenotypic variance (43.5%) and was designated *QPsh.jic-1D*. The QTL on chromosome 2B accounted for 33.2% of the phenotypic variance and was designated *QPsh.jic-2B*.

MQM mapping also identified two minor QTLs which contributed to the *P. striiformis* f. sp. *hordei* resistance in

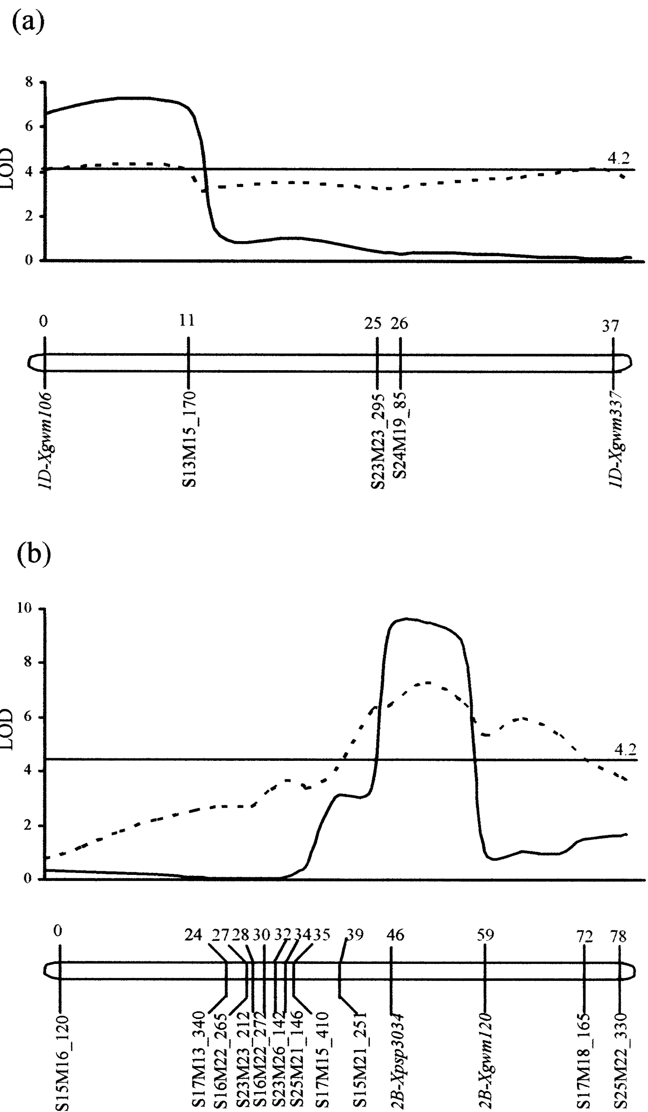

Fig. 3 LOD profiles of QTLs on chromosome 1D (**a**) and chromosome 2B (**b**). *Dotted lines* are based on interval mapping and *solid lines* on MQM analysis. The marker linkage map corresponding to each LOD profile shows markers below and the position of the markers (in centiMorgans) above the line. The LOD threshold is that obtained from van Ooijen (1999)

Table 3 Summary of QTLs for non-host resistance to barley yellow rust in the wheat Lemhi × Chinese 166 cross. *AA* The estimated mean of the distribution of the quantitative trait associated with the Chinese 166 genotype, *AB* the estimated mean of the distribution of the quantitative trait associated with the heterozygous genotype, *BB* the estimated mean of the distribution of the quantitative trait associated with the Lemhi genotype

QTL	LOD	Expl[a] (%)	Lines having the alleles		
			Chinese 166 (AA)	Heterozygote (AB)	Lemhi (BB)
QPsh.jic-1D	7.1	43.5	3.85	6.76	7.80
QPsh.jic-2B	9.5	33.2	4.54	6.15	7.86
QPsh.jic-5A	2.1	5.1	5.96	7.34	6.58
QPsh.jic-6A	4.2	10.9	5.76	6.92	5.20

[a]The proportion of the explained variance for the single QTL model

the Lemhi × Chinese 166 cross (Table 3). A QTL on chromosome 5A (*QPsh.jic-5A*) contributed 5.1% of the phenotypic variance, but had a LOD value below the threshold, while a QTL on chromosome 6A (*QPsh.jic-6A*) contributed 10.9% of the variance.

As expected from the χ^2 analysis (15*R*:1*S*) of the F_2 genetic segregation, the two major QTLs showed dominance (Table 3). However, the minor QTLs had larger means for the heterozygous genotypes than either parental genotype (Table 3), indicating an interaction between the alleles from Lemhi and Chinese 166 at these loci.

The Kruskal-Wallis test was carried out using the F_2 disease-reaction scores rated using the 1–9 scale. The Kruskal-Wallis test did not identify any markers associated with the resistant phenotype that had not been identified by MQM mapping.

Discussion

Non-host resistance, by its very definition, represents a form of durable resistance. Many have studied non-host resistance with the view of using such forms of resistance in crop species of the pathogen under study (Heath 2001; Niks 1988). The genetics underlying most cases of non-host resistance is likely to be very complex, reflecting the inability of the pathogen to form a basic, compatible association with the non-host plant. However, for closely related plant species, e.g. wheat and barley, non-host resistance may be controlled by a genetic system similar to host-pathogen, gene-for-gene resistance (Heath 1991, 2001; Tosa 1996).

In wheat, Tosa (1996) identified four genes that conferred non-host resistance to the powdery mildew forma specialis of Agropyron (*Blumeria graminis* f. sp. *agropyri*) and four genes in the pathogen required to trigger resistance conferred by each of the wheat resistance genes. In an accession of the wild lettuce, *Lactuca saligna*, one major race-specific QTL was identified, plus three minor QTLs, to the cultivated lettuce (*L. sativa*) pathogen for downy mildew, *Bremia lactucae* (Jeuken and Lindhout 2002). Similarly, we have found two major QTLs for non-host resistance to barley yellow rust in the wheat cross Lemhi × Chinese 166, and two minor QTLs; one originating from Chinese 166. This supports the genetic segregation study of Johnson and Lovell (1994) in which genes of minor effect, originating from both Lemhi and Chinese 166, were suggested. While the major QTLs may represent a resistance-triggering system similar to the gene-for-gene interaction common in host resistance, the minor QTLs may encode for products that prevent a basic compatibility from being established between the non-host and the non-pathogen.

The response of Lemhi to *P. striiformis* f. sp. *hordei* isolates involved a classic hypersensitive cell death (Table 2) typical of race-specific host resistance. Both *QPsh.jic-1D* and *QPsh.jic-2B* conferred this hypersensitive cell death-associated resistance to barley yellow rust. These QTLs may therefore operate through recognition of an avirulence factor present in the *P. striiformis* f. sp. *hordei* isolates that triggers a classic host-type resistance. However, the minor QTL *QPsh.jic-6A*, with a significant effect originating from the susceptible parent Chinese 166, can also trigger a visible cell necrosis in Chinese 166, following inoculation with *P. striiformis* f. sp. *hordei* isolates (Table 2).

In phytophthora, hypersensitive cell death appears to be a common feature in all non-host interactions. The isolation of elicitors that trigger this cell death appears to indicate that multiple layers of gene-for-gene interactions form the initial defence barrier to phytophthora in non-host plants (Kamoun 2001). Further evidence that common defence systems operate in host and non-host interactions comes from the *Arabidopsis*-*Pseudomonas* bacterial system (Kang et al. 2003). A glycerol kinase gene (*NHO1*) that is required for non-host resistance to bean and tobacco pathovars of *P. syringae* is suppressed in compatible host interactions with the virulent isolate *P. syringae* pv. *tomato* DC3000. However, inoculation with an avirulent isolate, carrying *avrB* in DC3000, results in induction of *NHO1* expression. It would therefore appear that the gene-for-gene host-incompatible interaction between the avirulence factor *avirB* and its corresponding R-gene, *RPM1*, utilises a component of non-host resistance, or vice versa, in this bacterial-plant system.

A number of genes for yellow rust host resistance have been mapped in wheat. A comparison of the location of yellow rust R-genes revealed that the region of the long arm of chromosome 2B where *QPsh.jic-2B* mapped contains a number of host R-genes. The race-specific seedling-expressed genes *Yr5* and *Yr7* map 10.9 cM distal of *Xgwm120-2B* (Fig. 3; L.A. Boyd, unpublished data). A QTL for adult plant resistance to yellow rust has also been located close to this region in the wheat variety Camp Remy (Boukhatem et al. 2002). *QPsh.jic-2B* mapped approximately 7.9 cM from *Xgwm120-2B*, but without a common flanking marker, we are unable to determine the orientation of *QPsh.jic-2B* in relation to these host yellow rust R-genes. Only one yellow rust R-gene has been assigned to chromosome 1D, *Yr25* and this has yet to be mapped (Calonnec and Johnson 1998). The location of non-host resistance QTL, close to regions containing host R-genes, may indicate a similar structure and function for non-host and host R-genes.

The potential for *QPsh.jic-1D* and *QPsh.jic-2B* to confer a durable source of resistance to barley yellow rust will require further testing. Barley yellow rust has not been a problem in the United Kingdom, either on the barley or the wheat crop for many years (Slater et al. 2002). *P. striiformis* f. sp. *hordei* isolates that can overcome *QPsh.jic-1D* and *QPsh.jic-2B* may therefore not be present in the United Kingdom *P. striiformis* f. sp. *hordei* population. A more extensive screen, including foreign *P. striiformis* f. sp. *hordei* isolates, may, however, reveal a race-specific phenotype for these R-genes.

While these two major QTLs have been identified in the wheat variety Lemhi, it remains to be tested whether these two QTLs are present in all wheat varieties resistant to *P.*

striiformis f. sp. *hordei*, or whether additional, non-host R-genes of major effect exist in wheat. The non-host resistance in Chinese 166 to *P. striiformis* f. sp. *hordei* isolate BYR 60/7 would indicate that additional non-host R-genes to barley yellow rust do exist in wheat.

Each QTL for *P. striiformis* f. sp. *hordei* resistance in Lemhi, when present in isolation, may also prove less effective. Rye is a non-host of wheat yellow rust. Addition lines of individual rye chromosomes added to the genome of hexaploid wheat indicated that more than one rye chromosome conferred resistance to wheat yellow rust. The yellow rust R-gene on rye chromosome 1 has been transferred to many wheat varieties and was designated *Yr9*. Isolated from the rye genome, the *Yr9* resistance was eventually overcome following the appearance of a virulent *P. striiformis* f. sp. *tritici* isolate. Therefore, the rye, non-host R-gene *Yr9* was shown to function in a gene-for-gene manner (Niks 1988). A similar fate may await *QPsh.jic-1D* and *QPsh.jic-2B* if these were to be transferred individually into barley.

Ultimately, isolation of genes conferring non-host resistance is required to determine the factors preventing a pathogen of one plant species from infecting another. The structural similarity of *Rac4* from *Arabidopsis* to host R-genes may indicate that *Rac4* would not remain effective in *B. oleracea* against *A. candida* for long. While the isolation of *QPsh.jic-1D* and *QPsh.jic-2B* may reveal genes similar in structure to host R-genes, *QPsh.jic-5A* and *QPsh.jic-6A* may prove more interesting candidates.

If non-host resistance to pathogens of closely related plant species is controlled solely by major R-genes through a gene-for-gene recognition system, then why are these non-host R-genes not overcome as readily as in race-specific host resistance? The ‘avirulence’ gene carried by the non-pathogen may be such that mutation within the avirulence factor would severely reduce the fitness of the pathogen. The basic compatibility between the non-host and the non-pathogen may also be poor, as seen here in the Chinese 166-*P. striiformis* f. sp. *hordei* infection. Therefore, such an interaction would not successfully out-compete infection by a wheat yellow rust isolate under field conditions (Heath 1991). The barley yellow rust isolate would therefore appear to have further to evolve to become a successful pathogen of wheat.

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