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Quantitative trait loci for resistance against powdery mildew in a segregating wheat × spelt population

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Abstract Powdery mildew is one of the major diseases of wheat in regions with a maritime or semi-continental climate and can strongly affect grain yield. The attempt to control powdery mildew with major resistance genes (*Pm* genes) has not provided a durable resistance. Breeding for quantitative resistance to powdery mildew is more promising, but is difficult to select on a phenotypic basis. In this study, we mapped and characterised quantitative trait loci (QTLs) for adult-plant powdery mildew resistance in a segregating population of 226 recombinant inbred lines derived from the cross of the Swiss wheat variety Forno with the Swiss spelt variety Oberkulmer. Forno possibly contains the *Pm5* gene and showed good adult-plant resistance in the field. Oberkulmer does not have any known *Pm* gene and showed a moderate susceptible reaction. Powdery mildew resistance was assessed in field trials at two locations in 1995 and at three locations in 1996. The high heritability ($h^2 = 0.97$) for powdery mildew resistance suggests that the environmental influence did not affect the resistance phenotype to a great extent. QTL analysis was based on a genetic map containing 182 loci with 23 linkage groups (2469 cM). With the method of composite interval mapping 18 QTLs for powdery mildew resistance were detected, explaining 77% of the phenotypic variance in a simultaneous fit. Two QTLs with major effects were consistent over all five environments. One of them corresponds to the *Pm5* locus derived from Forno on chromosome 7B. The other QTL on 5A, was derived from the spelt variety Oberkulmer and did not correspond to any known *Pm* gene. In addition, five QTLs were consistent over three environments, and six QTLs over two environments. The QTL at the *Pm5* locus showed a large effect, although virulent races for *Pm5* were present in the mixture of isolates. Molecular markers linked with QTLs for adult-plant resistance offer the possibility of simultaneous marker-assisted selection for major and minor genes.

Key words *Erysiphe graminis* · Powdery mildew resistance · QTL · *Triticum aestivum* · *Triticum spelta*

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Introduction

Powdery mildew in wheat (*Triticum aestivum* L.), caused by the obligate biotrophic ectoparasitic fungus *Erysiphe graminis* DC. f. sp. *tritici* (Em. Marchal) (syn. *Blumeria graminis* f. sp. *tritici*), is of economic importance in regions with high rainfall and with a maritime or semi-continental climate. With the spread of irrigation, the use of semi-dwarf cultivars, chemical growth regulators and the increased application of nitrogen fertilisers, powdery mildew has also moved into areas with hotter and drier climates (Bennett 1984). Plants can be infected from the first leaf stage until senescence. Early infection reduces tillering and increases the resorption of tillers. Late infection of the upper leaves and of the ears can heavily reduce grain yield (Heyland et al. 1979), because the fungus is able to divert the nutrients

towards the colonisation site. In regions of Eastern USA with a temperate climate and rainfall of about 1000 mm per year, grain yield losses of 20% (Pearce et al. 1996) and 33% (Fried et al. 1981) were observed.

Much of the earlier work on breeding for resistance to powdery mildew was based on the exploitation of race-specific major genes (*Pm* genes) that are expressed in seedlings and throughout the life cycle of the host plant. *Pm* genes confer complete resistance, caused by a hypersensitive reaction including papilla formation and cell death at the site of entry of the fungal penetration peg (Kunoh 1995), after infection with isolates carrying the corresponding avirulence gene. Up to now, 25 *Pm* loci are known (http://probe.nalusda.gov:8300/cgi-bin/query), and many of them were derived from wild relatives of wheat. Some *Pm* genes have been mapped with molecular markers, as e.g. *Pm1* on chromosome 7AL (Ma et al. 1994; Hartl et al. 1995; Nelson et al. 1995; Hu et al. 1997), *Pm2* on 5DS (Ma et al. 1994; Hartl et al. 1995; Nelson et al. 1995; Mohler and Jahoor 1996), *Pm3* on 1AS (Hartl et al. 1993; Ma et al. 1994), *Pm4a* introgressed from *Triticum monococcum* and *Pm4b* from *Triticum carthlicumon* 2AL (Ma et al. 1994), *Pm12* introgressed from *Aegilops speltoides* on 6BS (Jia et al. 1996), *Pm13* introgressed from *Aegilops longissima* on 3B and 3D (Donini et al. 1995), *Pm18* on 7A (Hartl et al. 1995), *Pm21* introgressed from *Haynaldia villosa* on 6AL (Qi et al. 1996) and *Pm25* introgressed from *T. monococcum* on 1A (Shi et al. 1998).

In virulence analysis, many varieties used in important wheat growing regions of the world seemed to have the same few *Pm* genes. In German wheat cultivars the most common resistance genes are *Pm2*, *Pm4b*, *Pm5*, *Pm6* and *Pm8* (Heun and Fischbeck 1987 a, b; official variety list of Germany 1998), in Czechoslovakian cultivars *Pm4b*, *Pm5* and *Pm8* (Lutz et al. 1992), in French cultivars *Pm2*, *Pm4b* and *Pm6* (Zeller et al. 1993 a), in North American cultivars *Pm3a*, *Pm5* and *Pm6* (Leath and Heun 1990), in Japanese cultivars *Pm10* and *Pm15* (Tosa et al. 1995), and in Chinese cultivars *Pm2*, *Pm4a*, *Pm4b*, *Pm5*, *Pm6* and *Pm8* (Xia et al. 1995; Huang et al. 1997). In contrast to wheat, limited information is available for spelt (*Triticum spelta* L.) concerning the presence of *Pm* genes. Zeller et al. (1994) found a known *Pm* gene in only one of 20 European spelt varieties.

The strategy to control powdery mildew by the use of major genes has been ephemeral (Roberts and Caldwell 1970; Shaner 1973; Brown et al. 1997). The *Pm* genes employed so far for resistance breeding provide little protection against the contemporary pathogen populations (Limpert et al. 1987). Lack of durability results from a rapid build up of powdery mildew populations with virulence against the *Pm* genes in response to the selection pressure exerted by resistant varieties (Roberts and Caldwell 1970). Virulent clones are dispersed over hundreds of kilometres. Even pyramiding several *Pm* genes in one variety has not provided durable resistance (Brown et al. 1997). Niewoehner and Leath (1998) found an increase in virulence frequencies and complex combinations of virulence patterns in powdery mildew isolates in the Eastern United States which was correlated with the resistance genes carried by the varieties grown in a particular area. From 1980 to 1983 the spring wheat variety Walter with a combination of *Pm1*, *Pm2*, *Pm4b*, and *Pm9* (Heun and Fischbeck 1987b) reached 5% of the wheat growing area in Switzerland. Winzeler et al. (1990) reported a marked increase of powdery mildew isolates virulent on these genes from 1981 to 1984 in Switzerland. As a result, the resistance of Walter under natural infection was significantly reduced. In contrast, the varieties Probus, Zenith and Arina, which were the major varieties of Switzerland during the last decades, showed stable intermediate resistance but do not carry any known *Pm* genes (Winzeler et al. 1990). Similar examples of durable resistance of wheat varieties without any *Pm* genes were reported from USA (Roberts and Caldwell 1970), Germany (Chae and Fischbeck 1979) and UK (Bennett 1984).

Incomplete resistance that retards infection, growth, and reproduction of powdery mildew in adult plants has been called 'slow mildewing' (Roberts and Caldwell 1970), 'adult-plant resistance' (Gustafson and Shaner 1982), or 'partial resistance' (Hautea et al. 1987). Partial powdery mildew resistance is supposed to extend the latent period and to reduce the sporulation of the fungus (Royer et al. 1984). Partial resistance has been detected in wheat cultivars that either have no identified major genes or in which major genes have been overcome (Bennett 1984). Shaner and Finney (1975) showed that partial resistance to powdery mildew behaves as a quantitative trait. Chae and Fischbeck (1979) found 14 genomic regions that were involved in the expression of partial resistance in Diplomat using monosomics of the wheat varieties Chinese Spring and Caribo; whereas Das and Griffey (1994 a) and Griffey and Das (1994) estimated that two to three genes were governing adult-plant resistance in different winter wheat crosses. Additive effects were predominant in the inheritance of this trait (Das and Griffey 1994 b). However, Das and Griffey (1995) also found dominance effects and digenic epistasis.

As it is difficult to reliably assess different levels of partial resistance in the field (Gustafson and Shaner 1982), molecular markers linked with the corresponding resistance genes would provide an important tool for breeding durable powdery mildew resistant wheat varieties. With the possibility of establishing genetic maps and to calculate the most likely positions of quantitative trait loci (QTLs) on these maps, molecular markers for quantitative disease resistance can be found (Geiger and Heun 1989). There are three studies which localised QTLs for powdery mildew resistance in barley doubled-haploid populations (Heun 1992; Backes et al. 1995, 1996). Only a few studies have been

published so far which localised QTLs for resistance against pathogens in wheat, e.g. QTLs for resistance against tan spot (Faris et al. 1997), leaf rust (Nelson et al. 1997) or karnal bunt (Nelson et al. 1998). To our knowledge, no QTLs for powdery mildew resistance have been localised in wheat or spelt. In the present study we mapped and characterised QTLs for adult-plant powdery mildew resistance in a segregating population of 226 recombinant inbred lines (RILs) derived from the cross of the Swiss wheat variety Forno with the Swiss spelt variety Oberkulmer. Messmer et al. (1999) established a comprehensive genetic map of this population which was used as a basis for the QTL analysis. The objectives of our study were: (1) to estimate the number and genomic positions of QTLs with significant effects on adult-plant powdery mildew resistance, and (2) to estimate the stability of the detected QTLs over environments. Knowledge about the inheritance of powdery mildew resistance will lead to a more efficient breeding strategy for durable resistance.

Materials and methods

Plant material

The investigated plant population consisted of 226 F_5-recombinant inbred lines (RILs) originating from a cross between the winter wheat Forno and the winter spelt Oberkulmer (Messmer et al. 1999). The parental varieties differed in their resistance against powdery mildew by about one scoring point on a scale from 1 (no colonies = resistant) to 9 (leaf area totally covered with colonies = highly susceptible). There is strong evidence that the more resistant parent Forno contains the recessive major resistance gene *Pm5*. This is based on the following three facts. (1) Seedling tests with 11 defined isolates revealed the potential presence of *Mli* (M. Heun, TUM, Weihenstephan, personal communication). *Mli* and *Pm5* are considered to be identical (Heun and Fischbeck 1987 b; Hovmoller 1989). (2) Forno is derived from a cross with Kormoran (Siedler et al. 1994), which is known to carry *Pm5* (Heun and Fischbeck 1987 a). (3) Forno shows the same resistance pattern in seedling tests as Hope (Schachermayr, personal communication) which is used as the differential variety for *Pm5* (Lebsock and Briggle 1974). Inoculated with a mixture of six Swiss isolates with the virulence/avirulence formula *Pm1*, *Pm2*, *Pm3a*, *Pm3c*, *Pm3d*, *Pm3f*, *Pm4a*, *Pm4b*, *Pm5*, *Pm6*, *Pm7*, *Pm8*, *Pm9*, *Mld*/*Pm3b*, *Pm17 MlAx*, *MlSo*, *MlTo*, Forno showed adult-plant resistance in the field, whereas Hope was susceptible (Schachermayr, personal communication). Oberkulmer, the more susceptible parent, is not known to possess any *Pm* genes causing a hypersensitive reaction (Zeller et al. 1994) and showed a moderate susceptible reaction when treated with a mixture of Swiss powdery mildew isolates (see above) in the field.

Phenotypic assessment in the field

The 226 RILs of Forno × Oberkulmer were cultivated in five different environments: in 1995 in Ruemlang (Ru95) and Reckenholz (SN95) and in 1996 at two locations in Reckenholz (Re96; SN96) and one location in Eschikon (Es96). All environments were located near Zurich, Switzerland, at 450 to 550 m above sea level on loamy soils. The RILs were grown together with 18 standard cultivars and three replicated entries of the parental varieties (250 entries) in a rectangular lattice design with two replications and ten genotypes per incomplete block. In Ru95, SN95 and SN96 the material was sown in 5-row plots (1.5 m^2) with 130 naked kernels/m^2, in Re96 in 6-row drill plots (6 m^2) and in Es96 in 7-row drill plots (6 m^2) with 350 naked kernels/m^2. In Re96 and Es96 between the tracks of the 250 plot 6-row tracks (7-row tracks in Es96) a mixture of the Swiss winter wheat varieties Arina and Bernina was planted. This prevented the genotypes from dragging down each other when lodging occurred. These isolation tracks, but not the 250 genotypes, were treated with 0.5 l/ha of the growth regulator Moddus at DC 33 (decimal code according to Zadoks et al. 1974). Fertilisation and chemical plant protection were done according to standard agricultural practice in Switzerland. Nitrogen fertilisation was at 100 kg N/ha. Foot-rot diseases were prevented by applying 1 l/ha of Tiptor (Maag) at DC 25 in Ru95, SN95 and SN96, and by applying 1 l/ha of Sportak (Bayer) between DC 31 and 33 in Re96 and Es96.

There was medium pressure of powdery mildew due to natural or artificial infection with *E. graminis* in all environments. However, in neighbouring fields (< 100 m away) of SN95, Re96, and SN96, and in the neighbourhood of Ru95 (about 1 km away), there was artificial inoculation with a mixture of six Swiss powdery mildew isolates with the virulence/avirulence formula *Pm1*, *Pm2*, *Pm3a*, *Pm3c*, *Pm3d*, *Pm3f*, *Pm4a*, *Pm4b*, *Pm5*, *Pm6*, *Pm7*, *Pm8*, *Pm9*, *Mld*/*Pm3b*, *Pm17 MlAx*, *MlSo*, *MlTo*. In the region of Es96 there was no artificial inoculation. The powdery mildew resistance (PMR) phenotype was recorded for the 226 RILs of Forno × Oberkulmer and the parental lines on a scale from 1 to 9 on a field-plot basis. PMR was scored twice in SN95 (22.5., 8.6.), Re96 (28.5., 10.6.) and SN96 (28.5., 10.6.), and once in Ru95 (6.6.) and Es96 (4.6.). In SN95, Re96 and SN96, single scorings were averaged to reduce the scoring error.

Statistical analysis of field data

Lattice analysis of single environments and analysis of variance over environments were performed with the program PLABSTAT (Version 2M, Utz 1995). Adjusted entry means were used to compute the analysis of variance across environments. Components of variance were computed considering the effects of the environment and genotype as random. Estimates of variance components σ_g^2 (genetic variance), σ_e^2 (environment variance), σ_{ge}^2 (genotype × environment interaction variance) and σ^2 (error variance) were calculated. Heritabilities (h^2) were calculated on an entry mean basis according to Hallauer and Miranda Fo (1981). The segregation of the 226 RILs for powdery mildew resistance was tested for normality. Phenotypic correlation coefficients of PMR scores between the five environments were calculated on an entry mean basis.

QTL analysis

The marker genotype of the 226 RILs was assessed with 176 RFLP probes and nine wheat microsatellites. For the construction of the genetic map, linkage analysis was performed with the program MAPMAKER (Lander et al. 1987) using the Haldane mapping function (Haldane 1919) as described by Messmer et al. (1999). After the removal of closely linked marker loci (< 1 cM) the genetic map used for QTL mapping comprised 182 marker loci (2469 cM) with an average marker density of 13.6 cM. This covers about 2/3rds of the wheat genome (Messmer et al. 1999). Genotypes with more than 10% of the markers being heterozygous, or deviating bands indicating outcrossing, were excluded from the QTL data set. The QTL analysis was performed with 204 genotypes by the software package PLABQTL (Utz and Melchinger 1996) based on composite interval mapping (CIM). Co-factors were obtained by the procedure cov SELECT. The threshold for the detection of a QTL was fixed at a LOD value of 3.0. The explained phenotypic variance of each QTL

and of multiple regression models with all detected QTLs were calculated. Due to the small degree of heterozygosity of our population we estimated additive effects but no dominance effects. The occurrence of QTL × QTL interactions was tested by adding digenic epistatic effects to the additive effects in the model. The significance of QTL × environment interaction was tested by fitting a model to the adjusted entry means of each environment which included all QTLs detected in the analysis across environments, as described by Bohn et al. (1996). To check the consistency of QTLs across environments, QTL analysis was done for single environments, for the average of the environments in 1995 and in 1996, as well as for the average over all environments. Based on the QTL analysis of 1995 (average of Ru95 and SN95), predicted values for every genotype were calculated. A simple regression was calculated between these predicted values and the observed values in 1996 (average of Re96, Es96 and SN96). Since the assignment of linkage groups to physical chromosomes was not in every case clear, it is advisable for the localisation of QTLs to refer to flanking markers rather than to physical chromosomes (Messmer et al. 1999).

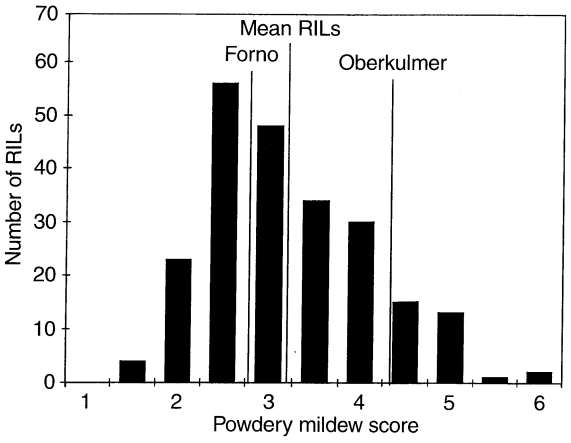

Fig. 1 Distribution of powdery mildew scores for the 226 RILs derived from the cross Forno × Oberkulmer averaged over five environments (Ru95, SN95, Re96, Es96, SN96). The means of the two parents Forno and Oberkulmer as well as the mean of the 226 RILs are shown. The distribution deviated significantly from normality (skewness: 0.60, kurtosis: − 0.23)

Results

Phenotypic analysis of powdery mildew resistance

The segregation of powdery mildew resistance (PMR) in the population was in the same range in all five environments, but the population mean was clearly lower in Es96 than in the other environments (Table 1). The powdery mildew disease started very late in Es96 causing a low disease pressure. The F_1 of Forno × Oberkulmer was more susceptible than the parental mean (Table 1) indicating dominance for susceptibility. In all environments, there was a two-sided transgressive segregation of PMR (Table 1). The distribution of PMR averaged over all five environments was skewed to the right side and deviated significantly from a normal distribution (Fig. 1). Therefore, the population mean did not correspond to the parental mean but was shifted towards the more resistant parent Forno.

The PMR scores of the five environments were highly significantly ($P < 0.01$) correlated to each other (Table 2). The correlations between locations of the same year were not higher than between locations of different years. However, the PMR scores of Es96, which was outside the area of artificial PM inoculation, were clearly more weakly correlated ($r < 0.80$) to all other environments. The three environments SN95, Re96 and SN96 were all located in Reckenholz where breeding material in neighbouring fields was artificially inoculated with the same mixture of powdery mildew isolates in both years. The correlations between these environments were very high ($r > 0.85$). Due to artificial inoculation in neighbouring fields in Ru95, SN95, Re96 and SN96 there were virulent races against *Pm1*, *Pm2*, *Pm3a*, *Pm3b*, *Pm3c*, *Pm3d*, *Pm3f*, *Pm4a*, *Pm4b*, *Pm5*, *Pm6*, *Pm7*, *Pm8*, *Pm9*, *Pm17*, while in Es96 there was probably the same spectrum of virulences present which was predominant in this region of Switzerland. In 1996 there were high frequencies (> 50%) of isolates virulent against *Pm1*, *Pm3c*, *Pm3f*, *Pm4a*, *Pm4b*, *Pm5*, *Pm6*, and *Pm7* in eastern Switzerland (Schachermayr, personal communication). Estimates for σ_g^2 and for σ_{ge}^2 among the RILs were highly significant ($P < 0.01$) across environments. However, genotype × environment interactions ($\sigma_{ge}^2 = 0.05$) were small compared to the genotypic variance components ($\sigma_g^2 = 0.74$). The heritability estimate of PMR was 0.97. We consider our phenotypic data of single environments, as well as the average over environments, as a reliable basis for QTL mapping.

Table 1 Mean of powdery mildew scores of the parental lines Forno (Fo) and Oberkulmer (Ok), their F_1 hybrid and 226 RILs derived from their cross, plus standard deviations (SD), minima (Min.) and maxima (Max.) for single environments (Ru95, SN95, Re96, Es96, SN96) and for the average over all environments

Environment	Fo	Ok	Parent. mean	F_1 (Fo × Ok)	Mean 226 RILs	SD	Min.	Max.
Ru95	2.80	5.45	4.13	5.00	3.47	1.19	0.72	7.21
SN95	2.78	4.43	3.61	3.94	3.43	0.94	1.38	6.26
Re96	2.94	3.50	3.22	–	3.15	0.86	1.36	5.68
Es96	2.42	3.69	3.06	–	2.74	0.88	1.16	5.52
SN96	2.83	3.74	3.29	4.21	3.16	0.92	1.52	5.94
Average	2.76	4.16	3.46	4.38	3.19	0.88	1.46	5.90

Table 2 Phenotypic correlation coefficients between powdery mildew scores in five environments (Ru95, SN95, Re96, Es96 and SN96) for 226 RILs of the cross Forno × Oberkulmer

Environment	Ru95	SN95	Re96	Es96
SN95	0.82**			
Re96	0.81**	0.85**		
Es96	0.74**	0.74**	0.76**	
SN96	0.80**	0.86**	0.87**	0.79**

** Correlation was significant at the 0.01 probability level

QTLs for powdery mildew resistance

In single environments, six QTLs (Ru95), 14 QTLs (SN95), six QTLs (Re96), 11 QTLs (Es96) and 13 QTLs (SN96) for PMR were detected with a LOD > 3.0 in composite interval mapping (Fig. 2). Individual QTLs explained between 6.8 and 36.6% of the phenotypic variance in composite interval mapping comprising the cofactors, and had a partial R^2 between 5.0 and 26.3% in the simultaneous fit. Models fitting all QTLs explained 44.4% (Ru95), 71.5% (SN95), 43.6% (Re96), 48.1% (Es96) and 72.0% (SN96) of the phenotypic variance. Averaged over all five environments 18 QTLs were detected explaining 77.2% of the phenotypic variance (Fig. 2). Individual QTLs explained between 7.5 and 31.8% of the phenotypic variance across environments in composite interval mapping comprising the co-factors, and had a partial R^2 between 2.9 and 29.8% in the simultaneous fit. At ten QTLs the allele for improved PMR was from the more resistant parent Forno, whereas at eight QTLs the improved resistance was derived from Oberkulmer (Table 3). There were 153 possible digenic epistatic effects between these 18 QTLs. Only one of them, corresponding to 0.65% [between the QTLs on 5A (198–216 cM) and on 6B (0–8 cM)], was significant. This is less than the number expected to occur by chance. It explained only a partial R^2 of 1.1% of the phenotypic variance for PMR in a multiple-regression model with all QTLs as variables. We therefore consider additive effects as the predominant factor of inheritance in the analysed population.

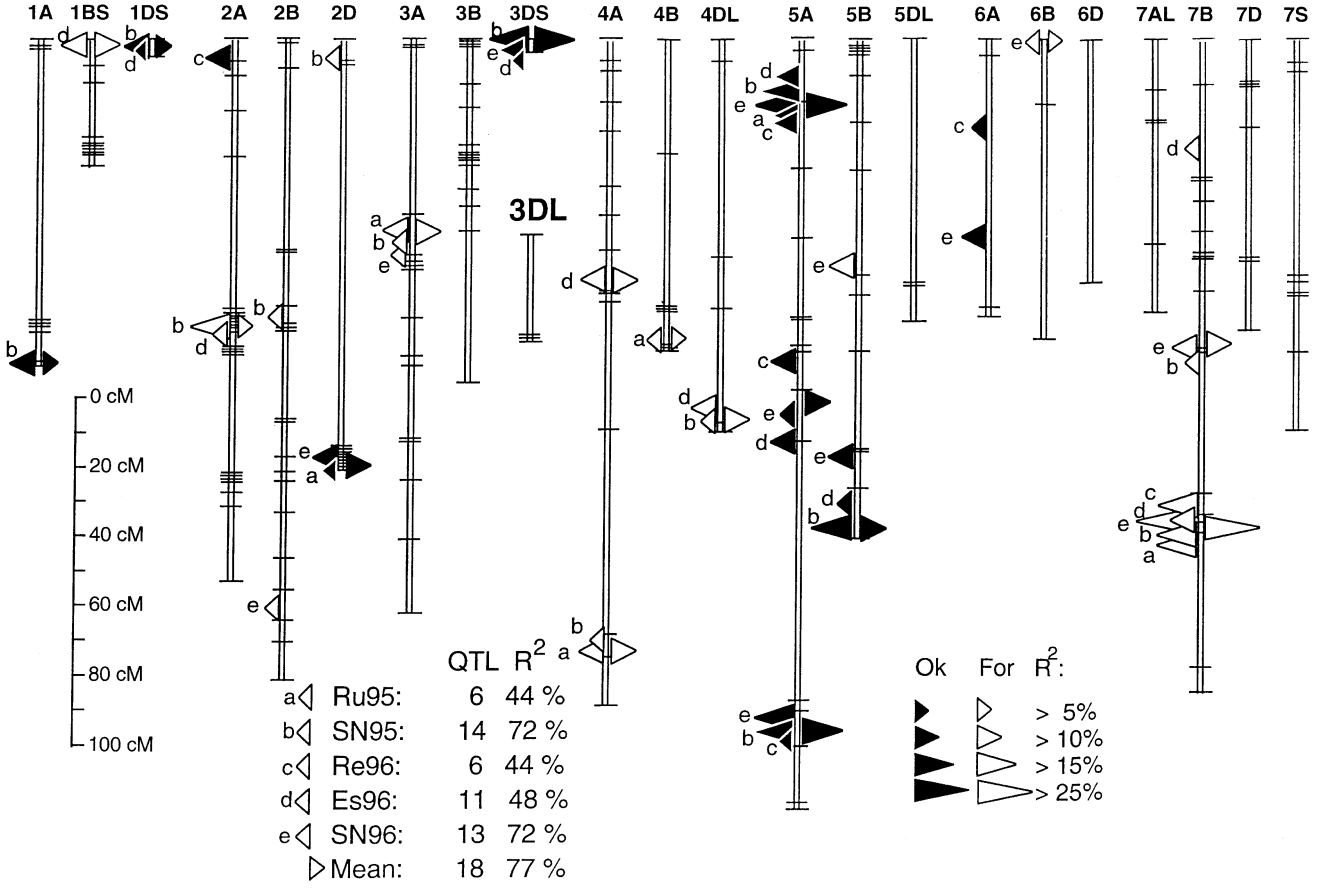

Fig. 2 Positions of significant (LOD > 3.0) QTLs for powdery mildew resistance on the genetic map of 204 RILs derived from the cross Forno × Oberkulmer. QTLs for powdery mildew resistance in single environments are indicated by *triangles to the left side* of the chromosomes. QTLs for mean powdery mildew resistance over all environments are indicated by *triangles to the right side* of the chromosomes. The size of the triangles indicates the explained phenotypic variance (R^2) of single QTLs. *White or black triangles* indicate that the allele for improved resistance was inherited from Forno or from Oberkulmer, respectively

Table 3 QTLs for powdery mildew resistance across environments and coinciding QTLs for single environments (Ru95, SN95, Re96, Es96, SN96) for 204 RILs from the cross Forno × Oberkulmer with the position on chromosomes, the support interval (cM), the explained phenotypic variance (R^2) of single QTLs, additive effects in scoring points (Add.) and the significance of QTL × environment interaction (QTL × E)

Chromosome	QTLs across environments		QTLs for single environments								QTL × E[d]
	Flanking markers		Ru95		SN95		Re96		Es96		SN96		
		R^2 Add.[b]	R^2	Add.	R^2	Add.	R^2	Add.	R^2	Add.	R^2	Add.	
1A	Xpsr1201b–Xpsr941	7.7% − 0.08			11.3% − 0.14								n.s.
cM[c]		86-**94**-94			90-**94**-94								
1B	CD9b–Xpsr593a	11.6% 0.11							11.7% 0.10				n.s.
cM		0-**0**-8							0-**0**-4				
1D	Xpsr168–Xglk558b	9.5% − 0.14			13.7% − 0.30				8.1% − 0.15				n.s.
cM		0-**2-4**			0-**2**-4				0-4-4				
2A	Xpsr380–Xglk293b	7.7% 0.09			18.1% 0.24				6.8% 0.12				**
cM		78-**82-86**			78-**82**-84				78-**82**-86				
2D	Xpsr932–Xpsr331a	10.0% − 0.17	8.2%	− 0.41							12.2%	− 0.22	*
cM		120-**122**-124	120-**124**	− 124							120-**124**-124		
3A	Xpsr598–Xpsr570	10.4% 0.19	11.0%	0.32	8.5% 0.22						7.5%	0.18	n.s.
cM		40-**54**-62	36-**54**-60		38-**56**-64						30-**56**-64		
3D	Xpsr1196a–Lrk10_6	15.7% − 0.19			22.3% − 0.31				9.8% − 0.25		14.4%	− 0.25	n.s.
cM		0-**0**-2			0-**0**-2				0-**0**-2		0-**0**-2		
4A	Xgwm111c–Xpsr934a	14.7% 0.16							10.3% 0.19				n.s.
cM		68-**74**-78							66-**74**-80				
4A	Xmwg710b–Xglk128	14.3% 0.17	10.8%	0.28	7.1% 0.17								n.s.
cM		176-**180**-188	176-**180**-190		158-**178**-184								
4B	Xpsr593b–Xpsr1112	7.5% 0.17	8.1%	0.23									n.s.
cM		84-**88**-88	82-**88**-88										
4D	Xglk302b–Xpsr1101a	14.4% 0.16			7.3% 0.19				10.9% 0.20				n.s.
cM		104-**112**-114			102-**112**-114				98-**110**-114				
5A	Xpsr644a–Xpsr945a	22.9% − 0.27	10.0%	− 0.47	24.9% − 0.36		11.4%	− 0.33	11.4%	− 0.12	21.4%	− 0.32	**
cM		16-**20**-26	12-**20**-30		14-**20**-26		14-**22**-30		10-**18**-26		14-**20**-26		
5A	Xpsr911–Xpsr120a	10.5% − 0.13					11.2%	− 0.14	10.1%	− 0.17	7.2%	− 0.17	n.s.
cM[3]		96-**106**-116					90-**94**-100		108-**118**-146		102-**110**-150		
5A	Xpsr1194–Xpsr918b	16.6% − 0.23			24.2% − 0.37		8.9%	− 0.42			16.9%	− 0.37	n.s.
cM		198-**202**-212			198-**202**-206		198-**204**-216				200-**204**-210		
5B	Xpsr580b–Xpsr143	12.6% − 0.25			17.1% − 0.27				7.3%	− 0.23	11.9%	− 0.22	n.s.
cM		138-**144**-144			140-**144**-144				124-**136**-144		108-**120**-126		
6B	Xpsr167b–Xpsr964	8.7% 0.12									7.5%	0.14	n.s.
cM		0-**0**-8									0-**0**-8		
7B	Xpsr593c–Xpsr129c	11.3% 0.18			7.3% 0.21						14.6%	0.23	n.s.
cM		88-**90**-106			88-**94**-112						88-**92**-106		
7B	Xglk750–Xmwg710a	31.8% 0.31	19.0%	0.37	20.7% 0.28		23.3%	0.27	12.7%	0.26	36.6%	0.34	n.s.
cM		138-**142**-152	138-**146**-158		138-**144**-154		134-**136**-138		138-**142**-156		138-**144**-152		

[a] The flanking markers refer to the QTLs across environments

[b] Additive effects were estimated from the simultaneous fit of all QTLs. Negative effects indicate that the Forno allele contributed to a lower powdery mildew score, i.e. improved resistance

[c] The first and the third numbers indicate the support interval, the number printed in bold indicates the position of the highest LOD score for the respective QTL. QTLs with overlapping support intervals and/or with less than 20 cM between the peaks cannot definitively be separated from each other and are therefore set in the same line

[d] *,** value was significant at the 0.05 and 0.01 probability level, respectively

Stability of QTLs over environments

Summarised over all five environments, 26 genomic regions were detected to have an influence on the expression of PMR (Fig. 2). Two QTLs on chromosome 5A (10–30 cM) and on 7B (134–158 cM), each explaining more than 20% of the phenotypic variance for PMR, were consistent over all five environments. Additionally, five QTLs (on 3A, 3DS, 5A, 5AL and 5B) were consistent over three environments and six QTLs over two environments. Five QTLs reached significance in only one environment but were confirmed in the QTL analysis across environments. In general, the additive effects of the 13 QTLs detected in more than one environment did not vary much (Table 3). However, at four QTLs (1D, 2A, 2D, 5A) the additive effects varied by a factor of two or more. Three of these QTLs (2A, 2D, 5A) showed significant QTL × environment interactions (Table 3). Three QTLs (1D, 2A, 5A) showed markedly lower additive effects in Es96 than in the other environments.

To analyse the predictability of PMR of the RILs from one year to the other based on QTL data, we calculated correlations between predicted and observed PMR in 1995 and 1996. The predicted PMR of the RILs based on the QTLs detected in 1995 (average between Ru95 and SN95) explained 67.9% of the observed phenotypic variance in 1996 (average between Re96, Es96 and SN96). This value was almost as high as that part of the phenotypic variance in 1996 that was explained by the simultaneous fit of the QTLs detected in the same year ($R^2 = 69.8\%$).

Discussion

Genetic basis of quantitative resistance to powdery mildew

Across all environments, we found 18 QTLs for powdery mildew resistance distributed across the whole genome. Therefore, adult-plant resistance to powdery mildew under field conditions has to be considered as a polygenic trait. This is in agreement with Chae and Fischbeck (1979) who found 14 genomic regions for powdery mildew resistance using monosomic lines. They reported that chromosomes 4D, 5A, 5B, 5D and 7A of the wheat variety Diplomat increased adult-plant resistance, while chromosomes 2D and 6D were responsible for more intensive powdery mildew attack. We also found QTLs on 2D, 4D, 5A, and 5B but not on 5D, 6D and 7A. In contrast to our study in wheat, Heun (1992) and Backes et al. (1995, 1996) found only up to two significant QTLs for powdery mildew resistance in barley doubled-haploid lines. Possibly, Backes et al. (1995, 1996) did not detect all the QTLs involved in the expression of adult-plant resistance in their field study because of the low infection rate. Heun (1992) used leaf segments of 7-day-old seedlings inoculated with one single isolate virulent on the resistance gene *Mla12*. With this approach, one might detect only a subset of genes involved in resistance during the seedling stage and not the whole range of genes important for powdery mildew resistance in field plots.

Because both parents of the Forno × Oberkulmer population contributed positive and negative alleles, the distribution of the powdery mildew score showed transgression. Hautea et al. (1987) also found transgressive segregation for PMR and suggested that partial resistance to powdery mildew is a polygenic trait with mainly additive effects. The resistance data from the F_1 of Forno × Oberkulmer indicate that dominance effects for susceptibility might be important in our cross in the early generations. In advanced (F_5) lines additive effects were the predominant source of variation, while digenic epistatic effects were negligible. Similarly, Chae and Fischbeck (1979) found that the positive effect of certain chromosomes on powdery mildew resistance was independent of the genetic background in which they were transferred.

QTL × environment interactions

Only 3 of 18 QTLs showed significant QTL × environment interactions. All three showed markedly lower additive effects in one environment. The variance explained by these interactions was about 20-fold smaller than the variance explained by the 18 QTLs (data not shown). This ratio is in agreement with the ratio between the genotype × environment interaction and the effect of the genotype, which was about 1 : 15. The high heritability estimates and the small number of significant QTL × environment interactions suggest that the environmental effect on the resistance phenotype was small. Likewise, Hautea et al. (1987) and Griffey and Das (1994) did not observe significant effects of the environment on partial resistance to powdery mildew in wheat. As we expected from the high correlations between years and from the good coincidences of QTLs between years, the predicted values based on the QTLs responsible for powdery mildew resistance in 1995 explained nearly as much of the phenotypic variance in 1996 as the detected QTLs in 1996 itself. These results suggest that the QTLs estimated in one year are reliable predictors for the phenotypic values in another year, which is an important prerequiste for marker-assisted selection (MAS).

Coincidences between QTLs for powdery mildew resistance and *Pm* genes

In the simultaneous fit across environments, two QTLs had large effects ($R^2 > 20\%$) on powdery mildew

resistance indicating the presence of major resistance genes. One QTL was on 7B (134–158 cM) and had major effects (19–36%) in four of the five environments. This QTL was linked with the markers *Xglk750* (0.7 cM), *Xgwm111a* (3.7 cM), *Xpsr547* (5.4 cM) and *Xpsr129* (11.4 cM). According to the published genetic maps of wheat, the powdery mildew resistance gene *Pm5* is located on the long arm of 7B in the same region as the markers *Xglk750*, *Xpsr547* and *Xpsr129* (McGuire and Qualset 1997) which were linked to the QTL in our population. The parental line Forno, which most likely contains the *Pm5* gene, contributed the positive allele at this QTL locus. Therefore, we have strong evidence that the major QTL on 7B represents *Pm5*. *Pm5* was described as a recessive gene causing complete or intermediate resistance (Lebsock and Briggle 1974). Thus, the effect of *Pm5* might partly explain the dominance observed for susceptibility in the F_1 of Forno × Oberkulmer. Although the powdery mildew infection was caused by a mixture of different isolates, including some that can overcome the resistance mechanism of *Pm5*, the detected effect of the *Pm5* resistance gene can be explained by the reduced growth of isolates with the *Pm5* avirulence gene and/or by the delayed spread of isolates with virulence on *Pm5*, the so-called residual effects of defeated major genes. Nass et al. (1981) found residual effects of *Pm3c* and *Pm4* resulting in a strongly reduced number of sporulating colonies, although they were virulent to these genes. However, for *Pm2* and *Pm5* they did not find any residual effects. Martin and Ellingboe (1976), Royer et al. (1984) and Negassa (1987) also found that *Pm* genes which have been overcome by virulent isolates still contribute to partial resistance.

Another major QTL was detected on 5A (16–26 cM), the positive allele inherited from the susceptible parent Oberkulmer. No *Pm* gene has been described on 5A so far (http://probe.nalusda.gov:8300/cgi-bin/query). In addition, Oberkulmer did not show any seedling resistance reaction against different powdery mildew isolates, including virulences against the known wheat *Pm* genes (Zeller et al. 1994). When compared with the effect of the putative *Pm5* gene, one can speculate that the QTL on 5A closely linked to *Xpsr644a* (1.1 cM) might represent a similar resistance gene from spelt not corresponding to any known *Pm* gene.

Besides the supposed *Pm5* gene itself, only eight of the remaining 17 QTLs were on chromosomes where known *Pm* genes are located: on chromosome 1A there are *Pm3* (Briggle and Sears 1966; Hartl et al. 1993; Zeller et al. 1993 b), *Pm17* (Heun et al. 1990) and *Pm25* (Shi et al. 1998). On 1B there is *Pm8* (Ren et al. 1997), on 1D there are *Pm10* (Tosa et al. 1987) and *Pm22* (Peusha et al. 1996). On 2A there is *Pm4* (The et al. 1979; Ma et al. 1994), on 4A *Pm16* (Reader and Miller 1991), on 4B *Pm7* (Discroll and Anderson 1967) and on 6A *Pm21* (Qi et al. 1996). On 6B there are *Pm11* (Tosa et al. 1988), *Pm12* (Jia et al. 1996) and *Pm14* (Tosa and Sakai 1990). For *Pm10*, *Pm11*, *Pm14*, *Pm16* and *Pm22*, a comparison of the locations of QTLs and *Pm* genes is difficult because they have not been mapped precisely on genetic maps to-date. *Pm7*, *Pm8*, *Pm17*, *Pm21*, and *Pm25* are located on translocations which are unlikely to be present in the parental varieties of our population. From the positions that could be compared with coinciding markers, an allelic relationship between the corresponding QTL on 1A and *Pm3* or *Pm12* is unlikely. However, an allelic relationship between the QTL on 2A and *Pm4* is possible. Robertson (1985) suggested that the loci for qualitative and quantitative traits are the same. Consequently, QTLs for powdery mildew resistance could also be less-effective alleles of *Pm* genes. Backes et al. (1996) found a QTL for powdery mildew resistance in barley in the same genomic region as the major resistance gene *mlt* which was not present in the population. However, from our data there is little evidence that QTLs for powdery mildew resistance are, in general, alleles of known *Pm* genes. There were less coinciding chromosomal locations between QTLs and *Pm* genes than could be expected from the high number of detected QTLs and known *Pm* loci. In addition, of the three locations that could be compared by markers only one coincidence is probable.

Perspectives for marker-assisted selection

We conclude from our data that differences in powdery mildew resistance in a wheat × spelt population were controlled by at least 18 loci distributed over the whole genome. These QTLs were very stable over environments and represent a good basis for marker-assisted selection. The major QTL on 5A could be introgressed from spelt into wheat. It is evident that Forno has a good quantitative resistance caused by the combination of about ten loci and possibly *Pm5*. Considering the high adaptability and the fast distribution of virulent isolates over large distances (Limpert et al. 1987), even pyramiding several *Pm* genes in one variety is a less durable strategy than the consequent breeding for quantitative resistance (Brown et al. 1997). However, if *Pm* genes can improve resistance under field conditions by restricting the spread of a subset of pathogen races, their presence in breeding material would be desirable. Therefore, the best strategy would be to combine resistance genes with different resistance mechanisms, major genes based on the recognition of certain races as well as genes causing a non-race-specific delay of the spread of the whole pathogen population. In order to distinguish genes for partial resistance from race-specific major genes the breeding material should be inoculated with a mixture of isolates containing all virulences occurring in a particular region (Winzeler et al. 1991). However, Cox (1995) showed that the combination of resistance genes with both large and small effects in a variety can hardly be achieved by phenotypic

selection, but is feasible when linked markers are available. With our knowledge about the genetic basis of powdery mildew resistance in the field, this approach can in the future be improved by marker-assisted selection. Molecular markers linked with QTLs for adult-plant resistance under field conditions offer the potential for the simultaneous selection of major and minor resistance genes.

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