

QTL analysis of resistance to *Fusarium* head blight in wheat using a 'Wangshuibai'-derived population

M. MARDI¹, H. BUERSTMAYR², B. GHAREYAZIE¹, M. LEMMENS², S. A. MOHAMMADI³, R. NOLZ² and P. RUCKENBAUER²

¹Department of Genomics, Agricultural Biotechnology Research Institute of Iran, Mahdasht Road, PO Box 31535-1897, Karaj, Iran, E-mail: mardi@abrii.ac.ir; ²Department for Agrobiotechnology Tulln, Institute for Plant Production Biotechnology, University of Natural Resources and Applied Life Sciences Vienna, Konrad Lorenz Strasse 20, A-3430 Tulln, Austria; ³Department of Crop Production and Breeding, Faculty of Agriculture, University of Tabriz, Tabriz 51664, Iran

With 2 figures and 2 tables

Received November 16, 2004/Accepted December 9, 2004

Communicated by R. A. McIntosh

Abstract

Fusarium head blight (FHB) is a devastating disease that reduces the yield, quality and economic value of wheat. For quantitative trait loci (QTL) analysis of resistance to FHB, F₃ plants and F_{3:5} lines, derived from a 'Wangshuibai' (resistant)/'Seri82' (susceptible) cross, were spray inoculated during 2001 and 2002, respectively. Artificial inoculation was carried out under field conditions. Of 420 markers, 258 amplified fragment length polymorphism and 39 simple sequence repeat (SSR) markers were mapped and yielded 44 linkage groups covering a total genetic distance of 2554 cM. QTL analysis was based on the constructed linkage map and area under the disease progress curve. The analyses revealed a QTL in the map interval *Xgwm533–Xs18/m12* on chromosome 3BS accounting for up to 17% of the phenotypic variation. In addition, a QTL was detected in the map interval *Xgwm539–Xs15/m24* on chromosome 2DL explaining up to 11% of the phenotypic variation. The QTL alleles originated from 'Wangshuibai' and were tagged with SSR markers. Using these SSR markers would facilitate marker-assisted selection to improve FHB resistance in wheat.

Key words: *Triticum aestivum* — *Fusarium* head blight — AFLP — SSR — QTL mapping

Fusarium head blight (FHB) or scab is one of the most important fungal diseases of wheat worldwide (Bai et al. 1999). The disease may cause severe yield reductions, grain quality losses and mycotoxins in humans and domestic animals. Although several species of *Fusarium* are capable of inciting FHB in wheat, *Fusarium graminearum* and *F. culmorum* are the dominant species (Parry et al. 1995). Breeding resistant cultivars is the method of choice for disease control. Currently a key strategy for successful FHB resistance breeding involves the identification of resistance genes and their introduction into regionally adapted varieties (Bai and Shaner 1994). However, breeding for FHB resistance is difficult for several reasons: (1) the most resistant germplasm is of exotic origin and poorly adapted to local growing conditions, (2) the inheritance is oligogenic or polygenic and (3) screening for FHB resistance is difficult.

Arthur (1891) first reported the differential response of various wheat genotypes to FHB. Most of the studies on the genetics of FHB resistance reported oligogenic or polygenic control with additive gene effects (Snijders 1990). Some studies, however, also indicated a role of epistasis and

dominant gene actions (reviewed by Bai and Shaner 1994). As the majority of common wheat cultivars are susceptible to FHB (Mesterhazy 1995), the few available resistance sources are poorly adapted. Sources originating from China (e.g. 'Sumai-3' and 'Wangshuibai'), South America (e.g. 'Frontana' and 'Encruzilhada') and Europe (e.g. 'Arina' and 'Praag-8') were used in different studies (Ruckenbauer et al. 2001). Schroeder and Christensen (1963) classified FHB resistance as type I (resistance against initial infection) and type II (resistance to the spread of pathogen within a spike) and Mesterhazy (1995) described additional types of FHB resistance in wheat. Due to the poor agronomic characters of many FHB-resistant varieties, genotype × environment interaction and the high cost and tediousness of field screening, progress in breeding for FHB resistance using traditional methods has been slow (Miedaner 1997, Buerstmayr et al. 2002).

The development of DNA-based markers provides a powerful alternative method for the dissection of complex traits, including FHB resistance in wheat (Gupta et al. 1999). DNA markers linked to genes governing resistance to FHB have been identified in several studies using different mapping populations (Bai et al. 1999, Waldron et al. 1999, Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Somers et al. 2003, Lin et al. 2004, Paillard et al. 2004, Steiner et al. 2004). The objectives of this study were to map FHB-resistant QTLs from 'Wangshuibai' and to identify simple sequence repeat (SSR) and/or amplified fragment length polymorphism (AFLP) markers which can be applied in marker-assisted selection to improve FHB resistance in wheat.

Materials and Methods

Plant materials: A total of 180 F₃ plants, one from each F₂ individual, and their derived F₃ lines obtained from a cross between 'Wangshuibai', a highly resistant Chinese landrace of wheat, *Triticum aestivum* L., and 'Seri82', a susceptible Mexican spring cultivar, were used in this study. The population was developed through single seed descent. The pedigree of 'Seri82' is 'Kavkaz'/Buho-sib//Kalyansona'/Bluebird'. The lines of the population together with the parents were evaluated for severity of FHB in field experiments at the Department for Agrobiotechnology Tulln (IFA-Tulln), Austria, during 2001 and 2002. The average temperatures and annual precipitations were 9.9°C and 639.6 mm and 10.1°C and 671.8 mm in 2001 and 2002, respectively. The soil type is meadow-czernosem and the preceding crop was maize

(*Zea mays* L.). To control seed-borne diseases, the seeds were treated with 'Rovral-TS' (Rhone-Poulenc, Lyon, France) seed dressing at a rate of 1.5 g/kg of seed. The experiments were sown in the first half of March. The plants were fertilized at late tillering with 90 kg/ha N, 14 kg/ha P, 23 kg/ha K and 12 kg/ha Mg in both years. In 2001, 180 space-planted F₃ plants were evaluated in a non-replicated trial with 25 cm distances between the plants. Five to ten seeds from each single F₃ plant were multiplied in the greenhouse during the winter of 2001/02. In 2002, 180 F_{3.5} lines were sown in a randomized complete block design with three blocks. Each plot consisted of two rows (1 m per row) planted with 2 g seeds per row and a 17 cm row spacing. Blocks were sown at time intervals of about 1 week.

Inoculation procedures and disease assessment: The inoculation of plants with *F. graminearum* was carried out using the spray inoculation method. IFA65, a pathogenic strain of *F. graminearum*, was used for artificial inoculation as described by Buerstmayr et al. (2002). Conidia concentrations were adjusted to 50 000 conidia per ml. In the 2001 experiment, three heads in each F₃ plant were marked with self-adhesive paper labels and artificially inoculated at anthesis by spraying 1 ml of conidial suspension using a manual atomizer. To provide high humidity, the infected heads were covered with a transparent plastic shelter after inoculation. After 20 h, the covers were removed and the inoculation was repeated after 2 days. From each F₃ plant, non-infected heads were protected to develop F_{3.4} lines. In the 2002 experiment, the inoculation was performed on each plot at 50% anthesis by spraying 50 ml of spore suspension with a motor-driven back-pack sprayer in the evening. Inoculations were repeated after 2 days on the same plots. To facilitate successful infection after inoculation, the nurseries were irrigated with overhead misters for 20 h by using a leaf wetness control for activating mist irrigation. Disease symptoms were assessed on three heads of each F₃ plant and by visually averaging whole plots in 2001 and 2002, respectively. The percentage of infected spikelets was estimated 14, 18, 22 and 26 days after inoculation. In addition, the area under the disease progress curve (AUDPC) was calculated for each inoculated spike in 2001 and each plot in 2002 using the following formula:

$$\text{AUDPC} = \sum_{i=1}^n \left\{ \left[\frac{(y_i + y_{i-1})}{2} \right] (x_i - x_{i-1}) \right\},$$

where y_i is the percentage of visibly infected spikelets ($y_i/100$) at the i th observation and x_i is the day of the i th observation, and n indicates the total number of observations (modified from Shaner and Finney 1977).

Genomic DNA extraction: Healthy leaves harvested from the parents ('Wangshuibai' and 'Seri82') and 180 F₃ individual plants were used for DNA extraction. Total genomic DNA was isolated using a CTAB method (Saghai-Marouf et al. 1984) with minor modifications. DNA quantity and quality were measured with a UV-Photometer.

AFLP and SSR markers: The AFLP analysis was performed in a similar way to that described by Hartl et al. (1999) using the enzyme combination *Sse8387I* and *MseI*. Fifty-one *Sse8387I* + *NN/MseI* + *NN* primer combinations with two selective nucleotides on the 3'-end of either primer were used for selective PCR amplification. The SSR markers were used as landmarks to locate AFLP markers in the genetic map. A total of 110 SSR primer pairs (Roeder et al. 1998) were used to assay parental polymorphism as described by Buerstmayr et al. (2002). The polymorphic SSR primers were used for genotyping the entire population. The *Sse8387I* and SSR forward primers were 5' end-labelled with the fluorescent dye IRD-800 or IRD-700. A DNA analyser system (LI-COR 4200 DNA analyzer; Lincoln, NE, USA) was used to detect the amplified fragments as described by Buerstmayr et al. (2002). The resulting images were printed and scored manually and independently by two people.

Analysis of phenotypic variation: The analysis of variance was performed for data obtained from the 2 year experiments using the

general linear model (GLM) procedure of the SAS/STAT software (SAS Institute Inc. 1990). Error mean squares across the two data sets were homogenous as determined by Hartley's test (Hartley 1950), thus pooled analysis of the 2 year experiments was performed using a mixed model considering genotypes and experiments as random variables. The different sources of variation were tested depending on their expected mean square. Due to unequal replications in the two year experiments, the degrees of freedom for Rep. (year) and error term were calculated as follows: $(r_1-1) + (r_2-1)$ and $[(r_1-1)(g_1-1)] + [(r_2-1)(g_2-1)]$, respectively, where r_1, r_2, g_1 and g_2 are the numbers of replications and genotypes in years 1 and 2, respectively. In order to estimate the reproducibility of the disease evaluations, the Pearson correlation coefficient was calculated for mean FHB severity (AUDPC) between single F₃ plants evaluated in 2001 and their F_{3.5} lines evaluated in 2002.

Construction of the linkage map: Segregating AFLP markers were scored for each individual of the mapping population with reference to parental genotypes. Scoring for polymorphic SSR markers was carried out as homozygous or heterozygous loci. A linkage map was constructed by using the 'group', 'order', 'ripple' and 'try' commands of the computer program MAPMAKER 3.0b (Lander et al. 1987). The polymorphic AFLP markers were assigned to linkage groups with reference to SSR markers with known chromosomal locations. A minimum logarithm of the odds ratio (LOD) score of 3 and a maximum genetic distance of 30 cM were used for pair-wise linkage analysis. The Kosambi mapping function (Kosambi 1944) was used to convert recombination frequencies into genetic distances.

QTL analysis: The QTL analysis was performed using the genotypic data from the F₃ plants and the phenotypic data obtained from the F₃ plants in 2001, the F_{3.5} lines in 2002 and the combined data. Interval mapping was conducted with the PLABQTL software (Utz and Melchinger 1996). To account for the masking effects of other QTLs on the detected QTL, composite interval mapping (CIM) was performed using a stepwise regression analysis with the following options: additive genetic model and selection of markers as cofactors to declare the presence of a putative QTL in a given genomic region (Zeng 1994). The location of a QTL is defined as the position where the LOD score value exceeds 3.5.

Results

FHB resistance

The level of infection 26 days after inoculation was 80 and 75% on the susceptible parent 'Seri82' in 2001 and 2002, respectively. The difference in FHB severity between the parental genotypes at the end of the observation period ranged from 5% in 'Wangshuibai' to 80% in 'Seri82'. A highly significant correlation coefficient was observed between the two individual experiments for AUDPC means ($r = 0.75$, $P < 0.0001$). The distribution of mean AUDPC of the F₃ and F_{3.5} lines is shown in Fig. 1. The pooled analysis of variance revealed a highly significant influence of the factor genotypes on the variation for AUDPC (Table 1).

Genetic linkage map

A total of 110 SSR primer pairs and 51 AFLP primer combinations were screened to assess parental polymorphism. Forty-one SSR primer pairs produced scorable polymorphic bands. Analysis of the mapping population using polymorphic AFLP primer combinations resulted in 379 polymorphic bands with an average of 7.6 bands per primer pair. Of 420 markers, 258 AFLP and 39 SSR markers were mapped and yielded 44 linkage groups. These linkage groups covered a total genetic

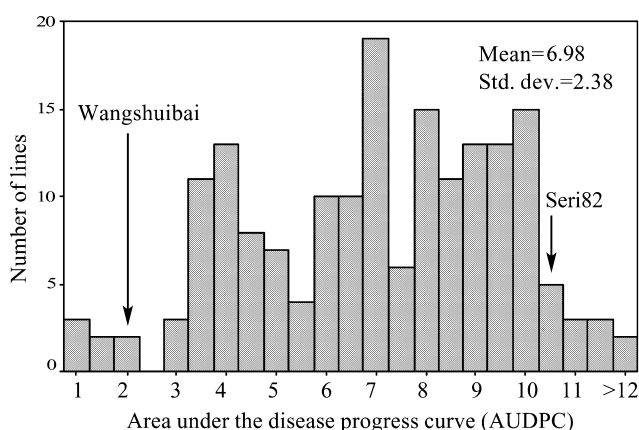


Fig. 1: Frequency distribution of 180 lines derived from a 'Wangshuibai'/'Seri82' cross for area under the disease progress curve (AUDPC) based on combined data across 2 years

Table 1: Pooled analysis of variance across 2 years for area under the disease progress curve (AUDPC)

Source	DF	MS	P-value
Year	1	2.06	< 0.0004
Rep. (year)	2	4.64	< 0.0001
Genotype (year)	337	0.97	< 0.0001
Error	319	0.16	

distance of 2554 cM providing partial linkage groups for all chromosomes except 4D and 7B.

Quantitative trait mapping

A total of 420 polymorphic markers were used to associate markers and phenotypes in the mapping population. CIM analysis detected two QTLs mapping to chromosomes 3BS and 2DL for AUDPC (Fig. 2). The chromosomal locations of marker intervals, effect of each QTL on AUDPC and coefficients of determination are presented in Table 2. A QTL in the map interval *Xgwm533-Xs18/m12* on chromosome 3BS was detected in both years and accounted for up to 17% of the phenotypic variation. Based on the QTL analysis of the $F_{3:5}$ lines and the combined data, a second QTL was detected in the map interval *Xgwm539-Xs15/m24* on chromosome 2DL. This QTL explained up to 11% of the phenotypic variation. Both QTL alleles conferring resistance were contributed by 'Wangshuibai' and were tagged with flanking SSR markers.

Discussion

FHB evaluation

Although several *Fusarium* species infect cereals, FHB resistance is non-specific and horizontal (Van Eeuwijk et al. 1995). Therefore, one well defined and highly aggressive *Fusarium* isolate was used for resistance evaluation in the trials. The inoculations were made by spraying inoculum directly on the heads at anthesis as described by Lemmens et al. (1993) and Buerstmayr et al. (2000). Environmental factors such as temperature and humidity influence FHB severity in wheat

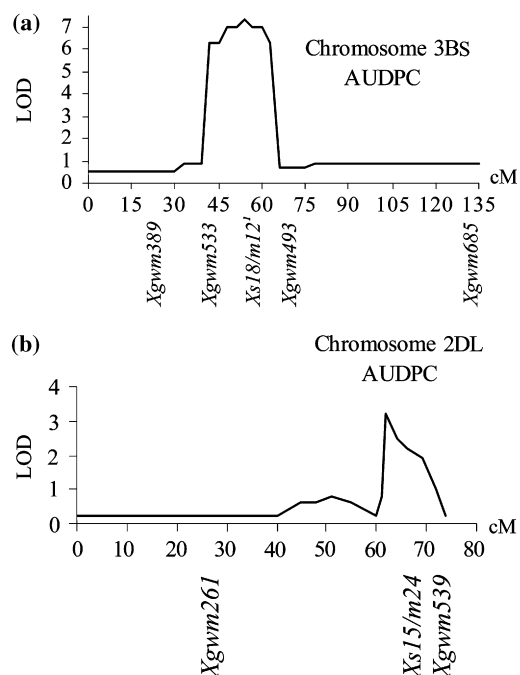


Fig. 2: The LOD score plots of two QTLs for area under the disease progress curve (AUDPC) on linkage groups corresponding to parts of chromosomes 3BS (a) and 2DL (b). Only selected microsatellite and AFLP markers are presented. AFLP marker names are abbreviated according to the standard nomenclature of AFLPs proposed by KeyGene

Table 2: The map intervals, chromosomal locations, logarithm of odds (LOD), the percentage of explained phenotypic variance (VE) and effects of quantitative trait loci (QTL) detected for area under the disease progress curve (AUDPC) using F_3 and $F_{3:5}$ populations derived from a 'Wangshuibai'/'Seri82' cross. QTL analysis was carried out by composite interval mapping (CIM)

Generations	Map interval	Chr.	LOD	VE	Effect ¹
F_3	<i>Xgwm533-Xs18/m12</i>	3BS	3.96	9.80	-1.48
$F_{3:5}$	<i>Xgwm533-Xs18/m12</i>	3BS	7.21	17.11	-0.93
	<i>Xgwm539-Xs15/m24</i>	2DL	4.04	11.7	-0.78
Combined analysis	<i>Xgwm533-Xs18/m12</i>	3BS	7.03	16.70	-0.90
	<i>Xgwm539-Xs15/m24</i>	2DL	3.20	8.14	-1.25

¹Negative effects indicate the direction of the QTL response on AUDPC for alleles contributed by 'Wangshuibai'.

(Parry et al. 1995) and thus complicate resistance evaluations. Bagging of infected heads with polyethylene bags in the 2001 trial (Mesterhazy 1983) and the use of a mist irrigation system in the 2002 trial (Buerstmayr et al. 2000) provided the required humidity for disease development. The severe and uniform infection of the susceptible parent 'Seri82' and the significant correlation coefficient between AUDPC mean values from the 2001 and 2002 trials indicated that these inoculation methods resulted in reproducible disease evaluations of the genotypes under investigation. The percentage of infected spikelets, reflecting combined type I and type II resistance, was used for estimation of the resistance level (Schroeder and Christensen 1963, Wilcoxson et al. 1992, Buerstmayr et al. 2003). Furthermore, the calculation of an AUDPC provided an integrated measure of FHB resistance. This result was in agreement with the results of Bai et al. (1999) and Buerstmayr

et al. (2002, 2003). The large difference between parental genotypes for FHB response led to significant genetic variation in the mapping population. The continuous distribution of the AUDPC values with a few peaks (Fig. 1) suggested an oligogenic or polygenic control of FHB resistance. Waldron et al. (1999) and Buerstmayr et al. (2002) stated that FHB resistance in wheat may be controlled by a few major genes and a number of minor genes.

QTL mapping

The high multiplex ratio of AFLPs integrated with the known genomic positions of SSR markers proved highly efficient in generating a large number of markers in a relatively short time (Gupta et al. 1999, Buerstmayr et al. 2002). The resultant map, despite being incomplete, facilitated the mapping of QTL for FHB resistance in the 'Wangshuibai'/'Seri 82' population. Two QTLs significantly associated with FHB resistance were found, mapping to 3BS and 2DL, respectively. The major QTL, *Qfhs.ndsu-3BS*, from 'Sumai-3' was detected in several mapping populations (Waldron et al. 1999, Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Zhou et al. 2002). The known pedigree of 'Sumai-3' indicated that 'Sumai-3' was not related to 'Wangshuibai'. In addition, Liu and Anderson (2003) showed by SSR marker analysis that both lines possess different haplotypes for several SSR markers around *Qfhs.ndsu-3BS*. It is considered unlikely therefore that 'Wangshuibai' and 'Sumai-3' possess the same alleles at *Qfhs.ndsu-3BS*, but both confer quantitative FHB resistance. The second QTL identified mapped to chromosome 2DL. Somers et al. (2003) also detected a QTL for FHB resistance in a similar region using a 'Wuhan-1'/'Maringa' population which is possibly the same QTL or a closely related allele. 'Maringa' was determined to be closely related to the Asian lines 'Nyuuubi' and 'Yanggangfangzhu', based on a haplotype study using microsatellite markers (McCartney et al. 2004). Compared with 'Sumai-3' and its relatives, 'Wangshuibai' most likely carries a common QTL for FHB resistance on chromosome 3BS (*Qfhs.ndsu-3BS*) and a different QTL on 2DL. In the present study, phenotypic evaluations were carried out using individual F₃ plants and F_{3.5} lines. The considerable heterozygosity in the F₃ generation leads to heterogeneity within the F_{3.5} lines. In addition, the genetic map was partly based on dominant AFLP markers that cannot assess heterozygosity. Although the use of later generation recombinant inbred lines would have partly overcome this problem, the experimental protocol used did identify two QTLs in regions of the genome where FHB-resistant QTLs had been previously detected (Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Somers et al. 2003, Lin et al. 2004). This result underlines the feasibility of FHB-resistant QTL detection by genotyping in the F₃ generation and phenotyping in advanced F₃-derived generations.

The combination of several quantitative resistance genes is necessary to achieve a high level of FHB resistance in a wheat genotype. Therefore, improvement for FHB resistance should be made by combining resistance genes from different sources and simultaneous selection for resistance and desirable agronomic traits. In this study, QTL analysis of FHB resistance of the Chinese landrace 'Wangshuibai' led to the detection of QTLs on chromosomes 3BS and 2DL that can be used in marker-assisted selection to improve FHB resistance in wheat. The fact that 'Sumai-3' and 'Wangshuibai' have their major

QTL in common limits the merits of combining these two resistance sources.

Acknowledgements

The authors offer special thanks to Dr H.F. Utz (University of Hohenheim, Germany) for his contribution in QTL analysis. They also appreciate the help of B. Steiner for sharing her experience in all the laboratory skills and M. Fidesser (IFA-Tulln) for excellent technical assistance. This study was funded by an international grant from the Biosciences Department, Technology Cooperation Office (TCO), Presidency of Iran, and Division of International Affairs, Federal Ministry of Education, Science and Culture of the Republic of Austria.

References

- Anderson, J. A., R. W. Stack, S. Liu, B. L. Waldron, A. D. Fjeld, C. Coyne, B. R. Moreno-Sevilla, J. Mitchell, Q. J. Song, P. B. Cregan, and R. C. Frohberg, 2001: DNA markers for a *Fusarium* head blight QTL in two wheat populations. *Theor. Appl. Genet.* **102**, 1164–1168.
- Arthur, J. C., 1891: Wheat scab. *Indiana Agric. Exp. Stn Bull.* **36**, 129–132.
- Bai, G., and G. Shaner, 1994: Scab of wheat: prospects for control. *Plant Dis.* **78**, 760–766.
- Bai, G., F. L. Kolb, G. Shaner, and L. L. Domier, 1999: Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling scab resistance in wheat. *Phytopathology* **89**, 343–348.
- Buerstmayr, H., B. Steiner, M. Lemmens, and P. Ruckenbauer, 2000: Resistance to *Fusarium* head blight in two winter wheat crosses: heritability and trait associations. *Crop Sci.* **40**, 1012–1018.
- Buerstmayr, H., M. Lemmens, L. Hartl, L. Doldi, B. Steiner, M. Stierschneider, and P. Ruckenbauer, 2002: Molecular mapping of QTLs for *Fusarium* head blight resistance in spring wheat. I. Resistance to fungal spread (type II resistance). *Theor. Appl. Genet.* **104**, 84–91.
- Buerstmayr, H., B. Steiner, L. Hartl, M. Griesser, N. Angerer, D. Lengauer, T. Miedaner, B. Schneider, and M. Lemmens, 2003: Molecular mapping of QTLs for *Fusarium* head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. *Theor. Appl. Genet.* **107**, 503–508.
- Gupta, P. K., R. K. Varshney, P. C. Sharma, and B. Ramesh, 1999: Molecular markers and their applications in wheat breeding. *Plant Breeding* **118**, 369–390.
- Hartl, L., V. Mohler, F. J. Zeller, S. L. K. Hsam, and G. Schweizer, 1999: Identification of AFLP linked to the powdery mildew resistance genes *Pm1c* and *Pm4a* in common wheat (*Triticum aestivum*). *Genome* **42**, 322–329.
- Hartley, H. O., 1950: The maximum F-ratio as a short-cut test for heterogeneity of variance. *Biometrika* **37**, 308–312.
- Kosambi, D. D., 1944: The estimation of map distances from recombination values. *Ann. Eugen.* **12**, 172–175.
- Lander, E. S., P. Green, J. Abrahamson, A. Barlow, M. J. Daley, S. E. Lincoln, and L. Newburg, 1987: MAPMAKER: an interactive computer package of constructing primary genetic linkage maps of experimental and natural populations. *Genomics* **1**, 174–181.
- Lemmens, M., H. Buerstmayr, and P. Ruckenbauer, 1993: Variation in *Fusarium* head blight susceptibility of international and Austrian wheat breeding material. *Die Bodenkultur* **44**, 65–78.
- Lin, F., Z. X. Kong, H. L. Zhu, S. L. Xue, J. Z. Wu, D. G. Tian, J. B. Wei, C. Q. Zhang, and Z. Q. Ma, 2004: Mapping QTL associated with resistance to *Fusarium* head blight in the Nanda2419 x Wangshuibai population. I. Type II resistance. *Theor. Appl. Genet.* **109**, 1504–1511.
- Liu, S., and J. A. Anderson, 2003: Marker assisted evaluation of *Fusarium* head blight resistant wheat germplasm. *Crop Sci.* **43**, 760–766.

- McCartney, C. A., J. Somers, G. Fedak, and W. Cao, 2004: Haplotype diversity at Fusarium head blight resistance QTLs in wheat. *Theor. Appl. Genet.* **109**, 261—271.
- Mesterhazy, A., 1983: Breeding wheat for resistance to *Fusarium graminearum* and *Fusarium culmorum*. *Z. Pflanzenzüchtg.* **91**, 295—311.
- Mesterhazy, A., 1995: Types and components of resistance to *Fusarium* head blight of wheat. *Plant Breeding* **114**, 377—386.
- Miedaner, T., 1997: Breeding wheat and rye for resistance to *Fusarium* diseases. *Plant Breeding* **116**, 201—220.
- Paillard, S., T. Schnurbusch, R. Tiwari, M. Messmer, M. Winzeler, B. Keller, and G. Schachermayr, 2004: QTL analysis of resistance to Fusarium head blight in Swiss winter wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* **109**, 323—332.
- Parry, D. W., P. Jenkinson, and L. McLeod, 1995: *Fusarium* ear blight (scab) in small grain cereals – a review. *Plant Pathol.* **44**, 207—238.
- Roeder, M. S., V. Korzun, K. Wendehake, J. Plaschke, M. H. Tixier, P. Leroy, and M. W. Ganal, 1998: A microsatellite map of wheat. *Genetics* **149**, 2007—2023.
- Ruckenbauer, P., H. Buerstmayr, and M. Lemmens, 2001: Present strategies in resistance breeding against scab (*Fusarium* spp.). *Euphytica* **119**, 121—127.
- Saghai-Marouf, M. A., K. Soliman, R. A. Jorgensen, and R. W. Allard, 1984: Ribosomal DNA spacer-length polymorphisms in barley: mendelian inheritance, chromosomal location, and population dynamics. *Proc. Natl Acad. Sci. USA* **81**, 8014—8018.
- SAS Institute Inc., 1990. SAS/STAT User's Guide, Version 6, Vol. 2, 4th edn. SAS Institute Inc., Cary, NC.
- Schroeder, H. W., and J. J. Christensen, 1963: Factors affecting the resistance of wheat to scab caused by *Gibberella zeae*. *Phytopathology* **53**, 831—838.
- Shaner, G., and R. A. Finney, 1977: The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. *Phytopathology* **67**, 1051—1056.
- Snijders, C. H. A., 1990: Genetic variation for resistance to *Fusarium* head blight in bread wheat. *Euphytica* **50**, 171—179.
- Somers, D. J., G. Fedak, and M. Savard, 2003: Molecular mapping of novel genes controlling *Fusarium* head blight resistance and deoxynivalenol accumulation in spring wheat. *Genome* **46**, 555—564.
- Steiner, B., M. Lemmens, M. Griesser, U. Scholz, J. Schondelmaier, and H. Buerstmayr, 2004: Molecular mapping of resistance to Fusarium head blight in the spring wheat cultivar Frontana. *Theor. Appl. Genet.* **109**, 215—224.
- Utz, H. F., and A. E. Melchinger, 1996: PLABQTL: a program for composite interval mapping of QTL. *J. Agric. Genomics* <http://www.ncgr.org/research/jag/papers96/paper196/indexp196.html>
- Van Eeuwijk, F. A., A. Mesterhazy, Ch. I. Kling, P. Ruckenbauer, L. Saur, H. Buerstmayr, M. Lemmens, L. C. P. Keizer, N. Maurin, and C. H. A. Snijders, 1995: Assessing non-specificity of resistance of wheat to head blight caused by inoculation with European strains of *Fusarium culmorum*, *F. graminearum* and *F. nivale*, using a multiplicative model for interaction. *Theor. Appl. Genet.* **90**, 221—228.
- Waldron, B. L., B. Moreno-Sevilla, J. A. Anderson, R. W. Stack, and R. C. Froberg, 1999: RFLP mapping of QTL for *Fusarium* head blight in wheat. *Crop Sci.* **39**, 805—811.
- Wilcoxson, R. D., R. H. Busch, and E. A. Ozmon, 1992: *Fusarium* head blight resistance in spring wheat cultivars. *Plant Dis.* **76**, 658—661.
- Zeng, Z. B., 1994: Precision mapping of quantitative trait loci. *Genetics* **136**, 1457—1468.
- Zhou, W. C., F. L. Kolb, G. H. Bai, G. Shaner, and L. L. Domier, 2002: Genetic analysis of scab resistance QTL in wheat with microsatellite and AFLP markers. *Genome* **45**, 719—727.