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Inheritance of field resistance to *Stagonospora nodorum* leaf and glume blotch and correlations with other morphological traits in hexaploid wheat (*Triticum aestivum* L.)

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Abstract Breeding for wheat varieties resistant to *Stagonospora nodorum* blotch (SNB) is the most sustainable strategy for controlling the disease. In order to map quantitative trait loci (QTLs) for SNB resistance we analysed 204 recombinant inbred lines of the cross between the winter wheat (*Triticum aestivum* L.) variety Forno and the winter spelt (*Triticum spelta* L.) variety Oberkulmer. We determined the level of resistance of adult plants to leaf blotch (SNL) and glume blotch (SNG) as well as morphological traits for 2 years after artificial inoculation with *S. nodorum*. Using composite interval mapping and LOD > 3.7, we detected ten QTLs for SNG blotch resistance (six inherited from the susceptible parent Forno) and 11 QTLs for SNL resistance (four inherited from Forno) across 2 years. Both resistance traits were moderately correlated ($r = 0.52$) and had only one common QTL. For SNL resistance, seven QTLs were not associated with QTLs for morphological traits. Among them, *QSnl.eth-2D*, *QSnl.eth-4B* and *QSnl.eth-7B3* had major effects ($R^2 > 13\%$) and were potential candidates for marker-assisted selection. For SNG, the major QTL on chromosome 5A, explaining 36% of the phenotypic variance for resistance, was associated with the *q* locus conferring the spelt morphology (long lax ear, long culm and hard glumes). Only *QSng.eth-1BS*, which explained 7% of the variance for resistance to SNG blotch, was not associated with QTLs for morphological traits. The consequences for breeding programmes are discussed.

Keywords *Triticum* · *Stagonospora nodorum* · Field resistance · QTL

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Introduction

Stagonospora nodorum leaf blotch (SNL) and glume blotch (SNG) are major diseases of wheat (*Triticum aestivum* L.). With increasing latitude and precipitation during the grain filling period, the incidence and severity of the pathogen increases as well (Leath et al. 1993). Severe symptoms of *S. nodorum* blotch are found in wheat growing areas in Europe, North Africa, the Near East, the United States, Australia and Canada (Duczek et al. 1999; Halama 2002) as well as in developing countries (Rajaram 1999; Van Ginkel and Rajaram 1999). Yield losses can amount to 30% (Eyal et al, 1987) and are due mainly to reduced seed set and seed filling as a result of reduced photosynthesis. Moreover, shrivelled kernels are often lost during harvest and their milling and baking quality is poor (Rosielle and Brown 1980; Eyal et al. 1987).

The fungal agent of *S. nodorum* blotch (SNB) was identified as *Phaeosphaeria nodorum* (E. Müller) Hedjaroude, anamorph *S. nodorum* (Berk.) Castellani & Germano [= Syn. *Septoria nodorum* (Berk.) Berk. in Berk. & Broome]. The most ecological and economical

approach to controlling this disease is to cultivate varieties with a high level of resistance. In the available wheat gene pool complete resistance has not been found yet, whereas genetic variation for partial resistance has been reported (Polley and Thomas 1991; Ma and Hughes 1993; Cunfer and Johnson 1999; Loughman et al. 1999; Wicki et al. 1999). Nevertheless, the breeding process using traditional methods is rather slow. This is due mainly to the complex inheritance of resistance, which is controlled quantitatively (Parlevliet 1979; Cunfer et al. 1988) and because resistance of the ear is inherited independently from resistance of the leaf (Fried and Meister 1987; Bostwick et al. 1993; Van Ginkel and Rajaram 1999; Wicki et al. 1999).

In this paper we have dissected the inheritance of resistance to SNB in a wheat (*T. aestivum* L.) × spelt (*Triticum spelta* L.) population derived from the cross of Forno and Oberkulmer. The spelt gene pool has not been exploited in wheat breeding because of the lower yield potential and unfavourable harvest properties such as the hulled seed of spelt. Nevertheless, the spelt gene pool may be an important source for the adaptation of wheat to marginal conditions as well as for resistance to several diseases, among them to *S. nodorum*.

Wide crosses and subsequent backcrossing are often used to introduce important genes (disease resistance, quality characters, etc.) into commercial varieties. Although spelt is closely related to common wheat (Siedler et al. 1994; Bertin et al. 2001), it differs to a great extent in several important physiological and phenotypical traits (Schmid and Winzeler 1990). With marker-assisted selection, spelt genes can be introgressed into wheat germplasm with a minimum number of genes with undesirable agronomic traits of spelt.

Several studies of wheat × spelt crosses have been conducted to elucidate the inheritance of important agronomic traits such as resistance to leaf rust (Messmer et al. 2000) and to powdery mildew (Keller et al. 1999a), pre-harvest sprouting (Zanetti et al. 2000), bread-making quality (Zanetti et al. 2001), lodging resistance (Keller et al. 1999b) and cold and flooding tolerance (Burgos et al. 2001). Thus, we have a general overview of the basis of inheritance of several agronomic traits and their correlations. This also allows establishing general breeding strategies based on the localisation of resistance genes and the co-localisation of QTLs for other traits.

Five papers have presented QTLs for resistance to SNB. Schnurbusch et al. (2003) investigated the inheritance of ear resistance to SNB at adult plant level under natural conditions of infection, whereas Toubia-Rahme et al. (2003) dissected ear resistance in the greenhouse, using artificial inoculations. Czembor et al. (2003), Arseniuk et al. (2004) and Liu et al. (2004) studied the inheritance of resistance of seedling leaves under artificial inoculations. The present paper is the first that reports QTL for resistance to SNL and to SNG in the same population using artificial infection of adult plants. Five major QTLs were assessed for durable SNB resistance, two for ear resistance (*QSng.eth-5A2* and *QSng.eth-5B1*), which overlapped with QTLs for morphological traits, and three for leaf resistance (*QSnl.eth-2D*, *QSnl.eth-4B* and *QSnl.eth-7B3*), which were not associated with other morphological traits.

Materials and methods

Plant materials

For the genetic analysis of *S. nodorum* blotch resistance we used 226 F_5 recombinant inbred lines (RILs) of a cross between the Swiss winter wheat (*T. aestivum* L.) variety Forno and the Swiss winter spelt (*T. spelta* L.) variety Oberkulmer (Messmer et al. 1999). Oberkulmer has a high level of resistance against SNB on the ear and a medium resistance on the leaf, whereas Forno is highly susceptible to SNB on both the ear and the leaf.

Field trials

The field trials to determine SNB resistance were performed at the Swiss Federal Research Station for Agroecology and Agriculture (FAL), Reckenholz (450 m a.s.l.), in the vegetation periods 1994/1995 and 1995/1996. The 226 RILs of Forno × Oberkulmer were grown together with three replicated entries of the parental lines Forno and Oberkulmer and the reciprocal F_1 and 16 standard varieties in a rectangular lattice design with two replications and ten genotypes per incomplete block. The material was sown as naked kernels after seed treatment (Panoctin, Shell Agrar) in five-row plots (200 kernels per 2.5 m^2) on 13 October 1994 and 11 October 1995. To prevent natural infection with foot rot disease, powdery mildew, stripe rust and *Septoria tritici*, 1 l/ha of Tiptor (Maag, Switzerland) was applied at the beginning of April at growth stage decimal code (DC) 25 (Zadoks et al. 1974). Lodging was prevented by mounting a plastic net over the plots below the flag leaves in early May. Oat slugworms were controlled by spraying 1.5 l/ha Zolone (Maag) at growth stage 50–55. The plant material was artificially inoculated with a *S. nodorum* pycnidia spore suspension (1×10^6 spores/ml, 70 ml/plot) four times in 4 weeks, as described by Fried (1989). For the inoculation we used a mixture of a broad spectrum of 40 different isolates collected in Switzerland. The leaves were first inoculated at the booting stage (DC7–DC49). The second to fourth inoculations were applied to the ears. To achieve maximum infection pressure during flowering, the second inoculation was carried out when a third of the genotypes were at DC59–DC61, the third application when two thirds of the genotypes reached this stage and the last application when the remaining plants reached this stage. During the vegetative periods 1994/1995 and 1995/1996 there were 1,042 mm and 690 mm rainfall, respectively, and a relative humidity of 77%.

Phenotypic assessment

The severity of *S. nodorum* leaf blotch was assessed six (1996) and seven (1995) times for each genotype over 31 days (DC50–DC80). For each assessment of each five-row plot, the percentage of necrotic tissue was estimated separately for the flag leaves (F), the leaf layer below the flag leaf (F-1) and the two layers below the flag leaf (F-2). Severity of *S. nodorum* glume blotch was assessed six (1996) and eight (1995) times within 20–24 days (DC60–DC85) by estimating the percentage of necrotic ear tissue. To determine resistance to *Stagonospora* glume blotch, the individual scores (in percentages) of all the sampling dates were averaged. For leaf resistance, percentages of the different assessments were averaged for each leaf layer (F, F-1 and F-2), and a mean for the entire plant was calculated by averaging the percentage means of the three leaves. This average was highly correlated ($r = 0.95$) to the area under the disease progress curve used by Campbell and Madden (1990) and Jeger and Viljanen-Rollinson (2001). Furthermore, we assessed for each genotype heading date (HD) and flowering date (FD) as days from 1 January, the colour of the ear (EC:1 = green, no wax layer to 6 = bluish, strong wax layer) at growth stage DC60, the culm length (CL) and ear length (EL) in centimetres before the harvest.

Table 1 Distribution of mapped SSR markers among the seven homoeologous groups added to the genetic map of Forno × Oberkulmer (Messmer et al. 1999)

SSR marker	Chromosome	Map position (cM) on chromosome
Xgwm131	Chrom 1B	49.8
Xgwm261	Chrom 2A	9.0
Xgwm359	Chrom 2A	118.1
Xgwm614	Chrom 2D	22.6
Xgwm614a	Chrom 2D	29.5
Xgwm161	Chrom 3A	5.9
Xgdm063	Chrom 3A	43.0
Xgwm493	Chrom 3B	0.0
Xgwm526d	Chrom 3B	8.3
Xgwm383	Chrom 3B	86.7
Xgwm114	Chrom 3DL	34.9
Xgwm526	Chrom 4A	88.7
Xgdm126	Chrom 4D	41.9
Xgwm169	Chrom 4D	140.4
Xgwm099	Chrom 5A	14.8
Xgwm443a	Chrom 5A	45.3
Xgwm205	Chrom 5A	68.1
Xgwm186	Chrom 5A	112.9
Xgwm272	Chrom 5DL	90.2
Xgwm219	Chrom 6B	53.9
Xgwm153	Chrom 7A	46.6
Xgwm344	Chrom 7A	146.4
Xgwm573	Chrom 7B	37.4
Xgwm297	Chrom 7B	52.1
Xgwm350	Chrom 7DL	44.6
Xgwm645	Chrom 7DS	15.0

Statistical analysis

The lattice analysis of single environments and the analysis of variance (ANOVA) across the environments were performed with the computer programme PLABSTAT (Utz 1995). The adjusted entry means obtained from the lattice analysis were used for a combined ANOVA across environments to estimate the genotypic (σ^2_G) and the genotype × environment interaction ($\sigma^2_{G\times E}$) variance components. Heritability values (h^2) were based on the variance components of the ANOVA and calculated according to Hallauer and Miranda (1981). For the calculation of the F_5 population mean and the correlation between different traits and different environments, the parental lines and standard lines were excluded, as well as 22 F_5 RILs, which had a higher level of heterogeneity/heterozygosity (> 10%) or for which the molecular data suggested outcrossing (Messmer et al. 1999). Adjusted mean values of the remaining 204 RILs of Forno × Oberkulmer of the single environments as well as the overall mean were used for the QTL analysis. Multiple regression models for *S. nodorum* resistance with all the morphological traits as variables were calculated across the environments with the stepwise procedure of SAS (SAS Institute).

The QTL analysis was based on the genetic map constructed by Messmer et al. (1999) and was improved by adding 27 SSR primer pairs (Table 1) developed at IPK Gatersleben (Institute of Plant Genetics, Germany) (Röder et al. 1998; Pestsova et al. 2000). These new marker loci were added to the genetic map (Table 1) using MAPMAKER, version 3.0 (Lander et al. 1987). Although 121 SSR primers were tested in order to fill larger gaps, the map coverage on chromosomes 1D, 2D, 5D, 6A, 6D and 7B is incomplete because of a lack of polymorphism. The final map consisted of 257 marker loci and spans 2,753 cM (Aguilar 2004).

QTL analysis

Simple interval mapping (SIM) and composite interval mapping (CIM) were performed with the computer package PLABQTL (Utz and Melchinger 2000). In order to avoid biased results because of non-normal distributed data, phenotypic means across environments were tested for normality with the Shapiro–Wilk test ($P < 0.05$). Natural log, log 10, square root and cubic root transformations were tested for their ability to improve normality. For the strongly distorted traits SNG and SNL, the best results were obtained by log 10-transformation which was used for QTL analysis. The HD and FD were normally distributed, whereas EC, CL and EL showed only minor deviation from normal distribution and could not be improved by transformation. Therefore, untransformed data of these traits were used for QTL analysis. For the QTL detection with SIM and CIM, the LOD threshold was determined by a permutation test for each trait with 1,000 permutations and a type-one error rate of $\alpha = 0.25$ (Beavis 1998). The LOD

thresholds for the different traits ranged from 2.15 to 2.21 for SIM and from 3.75 to 3.83 for CIM. For the final analysis, CIM with the covariate selection statement (*cov select*) was used.

Only additive effects and additive epistatic effects (statement *model AA* in the PLABQTL programme) were considered in the model, because the level of heterozygosity in a F_5 population is low. Additive effect estimates were based on log 10-transformed data only for SNG and SNL. The percentage of phenotypic variance, explained by a single QTL (R^2), was estimated as were the squared partial correlation coefficient (part. R^2), the additive effects (a) and the total phenotypic variance explained by all QTL applying a simultaneous fit. Additive effects were negative when the Forno allele increased the measurements and positive when the Oberkulmer allele increased the measurements. The QTL with support intervals, which did not overlap, were assumed to be different.

The effect of sampling on calculation of QTL was tested by a fivefold cross-validation run with the statement *cross-validate* of the programme PLABQTL. Finally, the QTL × environment interaction was tested using the PLABQTL programme.

Results

Phenotypic trait analysis

SNL and SNG resistance

Disease assessment for SNL and SNG after artificial infection were consistent between years, the correlation coefficients between the 2 years were $r = 0.75$ and 0.91 for disease assessment on the leaf and on the ear, respectively. The percentage of necrotic ear tissue averaged over all RILs and all scoring dates was 21.24% and 19.38% in 1995 and 1996, respectively. The percentage of diseased leaf area averaged across all RILs, the three leaf layers and all the scoring dates were 27.22% and 36.02% for 1995 and 1996, respectively (Table 2).

Figure 1a, b shows the phenotypic distribution of the RILs for SNL and SNG averaged across years. Both traits showed a continuous but non-normal distribution. The incidence of SNG in the Forno parent was five times higher than in the resistant parent Oberkulmer. The F_1 and F_5 means differed slightly and were shifted towards the more resistant parent Oberkulmer. The RILs F_5-242 and F_5-69 were more resistant than Oberkulmer, whereas the lines F_5-223 and F_5-22 were more susceptible than Forno. The incidence of SNL in the Forno parent was 1.4 times higher than in Oberkulmer. The F_1 and F_5 means were more or less the same and shifted towards the resistant parent Oberkulmer. About 75 RILs were more resistant than Oberkulmer and five lines were more susceptible than Forno. None of the lines were fully resistant to SNB at ear or leaf level.

Figure 1c shows the phenotypic correlation between SNG and SNL for the 204 RILs, considering the percentage of necrotic tissue across years. The rank correlation between the two types of resistance to SNB was $r = 0.52$ ($P < 0.01$). When considering the adjusted means over years the broad-sense heritabilities of SNB resistance of the ear and on the leaf were about 0.92 and 0.90, respectively, indicating that the phenotypic data for both traits were a reliable basis for QTL mapping.

Ear emergence, flowering date, ear colour, culm length and ear length

When considering adjusted means over years, the FD and the HD showed a normal distribution, while EC, CL and EL showed a non normal distribution. All the traits showed transgressive segregation (Table 3). The HD and FD were highly correlated ($r = 0.91$ with $P < 0.01$, Table 4). Twenty-two RILs flowered earlier than the earliest parent Forno (157 days after 1 January) and 28 RILs flowered later than Oberkulmer (162 days after 1 January).

CL and EL were significantly correlated ($r = 0.54$ with $P < 0.01$). Only three RILs showed transgression for CL, i.e. they were taller than the spelt parent Oberkulmer (160 cm). Sixteen lines had a shorter ear than the parent Forno (9.4 cm), and four lines had longer ears than the parent Oberkulmer (18 cm). Transgression in EC was observed for 63 RILs showing a more intensive wax layer than the parental line Oberkulmer. EL and EC had the highest rank correlation with *S. nodorum* resistance of the ear compared to other traits (Table 4). The RILs with long ears and a thick wax layer were more resistant to SNG than short genotypes with short ears and no

Table 2 *Stagonospora nodorum* glume blotch (*SNG*) and *S. nodorum* leaf blotch (*SNL*) of the parental wheat line Forno, the spelt line Oberkulmer, the F_1 and the 204 recombinant inbred lines (*RILs*) of Forno × Oberkulmer

Year	Traits	Forno	Oberkulmer	Parental mean	F_1	Mean 204 RILs	SD	F_5–RILs range	
								Min	Max
1995	SNG	60.35	9.12	34.74	18.64	21.24	9.92	8.15	64.43
	SNL	37.95	26.43	32.19	28.43	27.22	5.10	15.22	40.92
1996	SNG	43.85	10.75	27.30	17.44	19.38	6.91	7.35	47.18
	SNL	48.30	33.01	40.66	35.54	36.02	5.82	21.76	54.95
Average	SNG	52.10	9.94	31.02	18.04	20.31	8.22	8.10	55.80
Across years	SNL	43.12	29.72	36.42	31.99	31.89	5.41	18.60	50.80

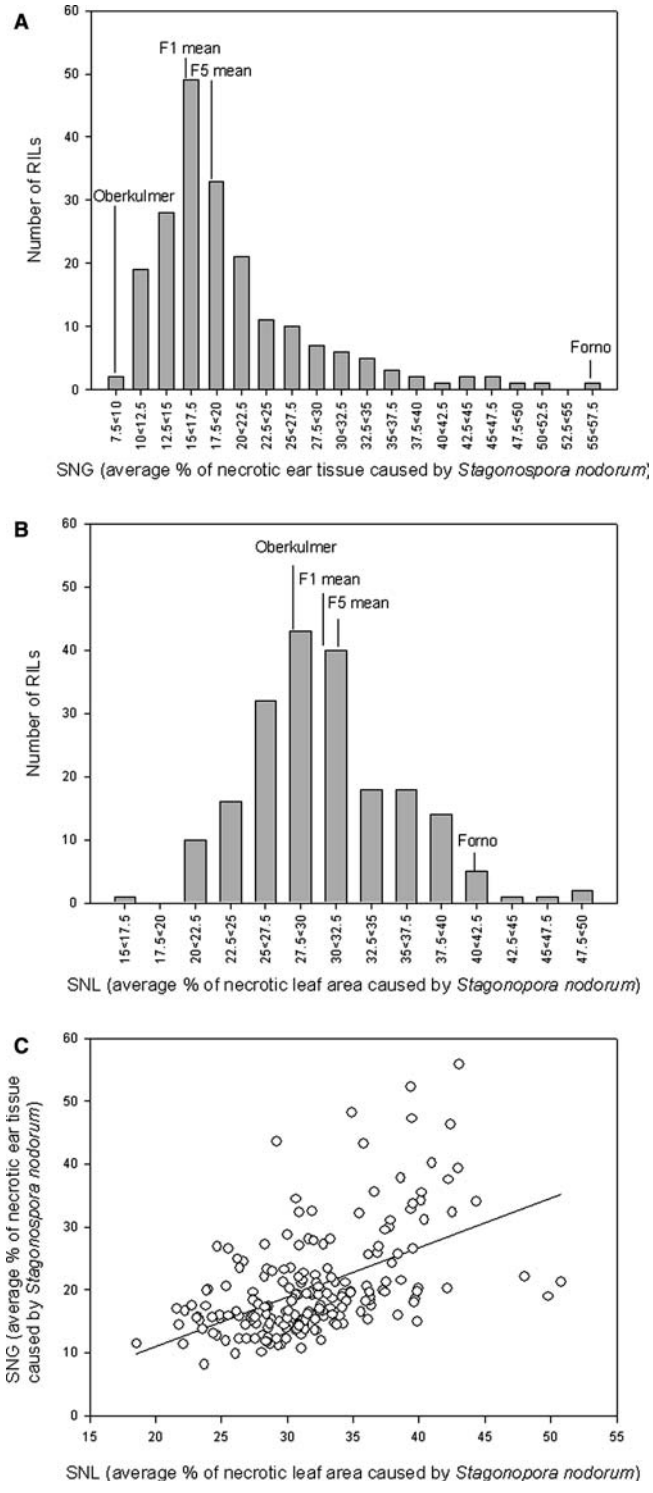

Fig. 1 Phenotypic distribution of the 204 RILs from the cross Forno × Oberkulmer, their F_1 hybrid mean and F_5 mean for *S. nodorum* glume blotch [(*SNG*) **a**], *Stagonospora nodorum* leaf blotch [(*SNL*) **b**] and their phenotypic correlation (**c**) across the different years

wax layer. Table 4 lists rank correlations showing that early flowering genotypes with short culm were more susceptible to SNL than later flowering and taller genotypes.

To elucidate the numerous correlations between the different morphological and resistance traits, multiple regression-models were calculated. FD and CL explained 44.5% of the phenotypic variance of SNL. In the case of SNG resistance, EL explained 47% of the phenotypic variance and together with EC, CL and HD up to 71.3%.

QTLs for SNG

The assessment of SNG adjusted over the two years and log 10-transformed enabled the detection of ten QTLs with LOD scores ranging from 3.8 to 27.5 and corresponding R^2 ranging from 0.3% to 36% (Table 5). The QTL with the strongest additive effects for improved resistance, *QSng.eth-5A2* (LOD = 27.5, $R^2 = 35.8\%$, $a = -0.103$), originated from Oberkulmer, whereas *QSng.eth-5B1* (LOD = 10, $R^2 = 6.3\%$, $a = +0.044$) originated from Forno. Both QTLs were consistent in the analysis of single environments. This result was confirmed by the fivefold cross-validation done on the averaged percentage of SNG disease across environments, where *QSng.eth-5A2* and *QSng.eth-5B1* were detected on each of the five cross-validation splits. Of the ten QTLs detected for the averaged percentage of diseased ear tissue, four were detected in only one of the two environments (*QSng.eth-2B*, *QSng.eth-3A1*, *QSng.eth-4A1* and *QSng.eth-7AL*) and four were detected only across experiments (*QSng.eth-1BS*, *QSng.eth-3A2*, *QSng.eth-4A2* and *QSng.eth-4DL2*).

A significant digenic epistatic effect was found between *QSng.eth-1BS* and *QSng.eth-5B1*, explaining 3.5% of the phenotypic variance for ear resistance.

When considering means across years for SNG, only *QSng.eth-1BS* did not overlap with QTLs for morphological traits. *QSng.eth-5A2*, with major effects on SNG, was located in the same chromosomal region as QTLs for CL, EL, FD, HD and EC. Thus, the allele from Oberkulmer conferring SNG resistance was associated with longer ear, later HD and FD and a stronger wax layer.

QTLs for SNL

Assessment of SNL, adjusted over the 2 years and log 10-transformed, enabled the detection of 11 QTLs with LOD scores ranging from 3.8 to 14 and R^2 ranging from 0.9% to 21% (Table 5). *QSnl.eth-2D* (LOD = 9, $R^2 = 20.8\%$, $a = -0.024$), with the more resistant allele originating from Oberkulmer, and *QSnl.eth-7B3* (LOD = 7, $R^2 = 12.8\%$, $a = +0.019$), with the more resistant allele originating from Forno. Both were consistently detected in single-environment analysis. *QSnl.eth-2D* was detected in each of the five cross-validation splits and *QSnl.eth-4B* was detected in four splits.

Four QTLs for SNL (*QSnl.eth-1BS2*, *QSnl.eth-2A2*, *QSnl.eth-4B* and *QSnl.eth-5B*) were detected in only 1 of

Table 3 Means of the parental wheat line Forno (*Fo*), the parental spelt line Oberkulmer (*Ob*), the F_1 and the 204 RILs derived from their cross; plus standard deviations (*SD*) and heritability (h^2) for heading date (*HD*), flowering date (*FD*), ear colour (*EC*), culm length (*CL*) and ear length (*EL*) averaged over 2 years

Trait	Score	Fo	Ob	Parental mean	F_1	Mean 204 RILs	SD	Max.	Min.	h^2
HD	Days	151.9	158.6	155.3	154.5	155.8	2.6	163.8	149.5	0.96
FD	Days	156.8	161.7	159.2	158.4	159.7	2.1	167.6	154.4	0.88
EC	1–6	1	4.3	2.7	3.9	3.8	1.2	6	1	0.94
CL	cm	97.5	159.8	128.7	145.0	136.0	12.2	161.8	103.7	0.98
El	cm	9.4	18.0	13.7	11.0	13.5	2.4	18.7	7.8	0.97

the 2 years. When considering means across experiments, four QTLs were detected (*QSnl.eth-7B1*, *QSnl.eth-2B1*, *QSnl.eth-2B2* and *QSnl.eth-3B*), which were not detected in one of the two environments. No epistatic effect was detected. The QTLs for SNG and SNL on chromosome 2BS (*QSng.eth-2B* and *QSnl.eth-2B1*) showed overlapping support intervals, whereas the QTLs *QSng.eth-1BS* and *QSnl.eth-1BS2* were located on the same chromosome arm 1BS.

For SNL, 4 of 11 detected QTLs overlapped with QTLs for other morphological traits. Overlapping QTLs were *QSnl.eth-1BS2* (LOD = 3.79, $R^2 = 7.3\%$, $a = +0.016$) with CL, *QSng.eth-2A2* (LOD = 3.8, $R^2 = 1.8\%$, $a = -0.016$) with EC and EL, *QSnl.eth-2B1* (LOD = 3.9, $R^2 = 8.1\%$, $a = -0.015$) with FD, HD and EL and *QSnl.eth-7B1* (LOD = 4.6, $R^2 = 0.9\%$, $a = +0.016$) with FD (Table 5).

QTLs for HD and FD

Averaged over 2 years the traits HD and FD were highly correlated ($r = 0.91$, $P < 0.01$). Eleven and ten QTLs (LOD > 3.8) were detected for each trait, respectively, and nine were co-localised for both traits. For HD 11 QTLs explained individually 4.4–15.8% of the phenotypic variance in composite interval mapping. A model fitting all QTLs explained 71.3% of the phenotypic variance. Six QTLs for HD were co-localised with QTLs for SNG (Table 5). For these QTLs, alleles for later HD were inherited from the same parents conferring also the alleles for increased SNG resistance. One QTL for HD was co-localised with *QSnl-2B1* for SNL blotch; the alleles inherited from Oberkulmer conferring a later HD and increased resistance.

For FD, the ten detected QTLs explained individually between 5.6% and 16.8% of the phenotypic variance in CIM. A model fitting all QTLs explained 61.1% of the phenotypic variance. Six QTLs for FD were co-localised with QTLs for SNG. In all these cases the alleles for later flowering were inherited from the same parent, which also conferred increased resistance towards SNG.

QTLs for EL

Averaged over 2 years, ten QTLs were detected for EL, explaining between 3.2% and 54.7% of the phenotypic variance in composite interval mapping and totally 78.3% in the final simultaneous fit. A major QTL for EL was found on chromosome 5A (LOD = 45.4, $R^2 = 54.7\%$, $a = +1.73$). At this chromosomal region we detected also QTLs for SNG ($R^2 = 35.8\%$, $a = -0.103$), EC ($R^2 = 53.4\%$, $a = +0.7$), CL ($R^2 = 9.9\%$, $a = +2.46$), FD ($R^2 = 23.3\%$, $a = -0.77$) and HD ($R^2 = 15.8\%$, $a = -1.1$). Besides the QTL on chromosome 5A, three QTLs for EL on chromosome 2B, 3A and 4A were coincident with a QTL for SNG. In all these cases SNG-resistant alleles were inherited from the same parent conferring alleles for longer ear. One significant epistatic effect for EL was detected between *QEl.eth-5A1* and *QEl.eth-5A3* explaining 7% of the phenotypic variance for EL.

QTLs for EC

Averaged over 2 years, four QTLs were detected explaining individually between 4.2% and 62.2% of the

Table 4 Number of significant (LOD > 3.77) quantitative trait loci (QTLs) and explained genotypic variance (*diagonal*), number of common QTLs (*above diagonal*) and Spearman rank correlation (*below diagonal*) of SNG, SNL, HD, FD, EC, CL and EL across 2 years (1995 and 1996) for the 204 F_5-RILs of Forno × Oberkulmer

	SNG	SNL	HD	FD	EC	CL	EL
SNG	**10 (70%)**	1	7	6	2	5	4
SNL	+0.52**	**11 (56%)**	1	2	1	1	2
HD	−0.21**	−0.33**	**11 (42%)**	8	2	5	4
FD	−0.14*	−0.35**	+0.91**	**10 (48%)**	1	4	3
EC	−0.48**	+0.01	−0.05	−0.20**	**4 (85%)**	1	2
CL	−0.39**	−0.42**	−0.30**	−0.29**	−0.04	**13 (74%)**	5
EL	−0.63**	−0.26**	−0.18*	−0.17*	+0.27**	+0.54**	**10 (76%)**

Significant at $*P < 0.05$ and $**P < 0.01$, respectively

Table 5 Identification of significant (LOD > 3.7) QTLs for SNG and SNL, their chromosomal location, support interval, flanking markers, explained phenotypic variance (R^2) and overlapping QTLs with other traits based on the composite interval mapping of the adjusted means of 204 RILS of Forno × Oberkulmer across 2 years (95, 96)

QTL position for *S. nodorum* resistance				SNG across replications			SNL across replications			Overlapping QTLs found for other traits
Chromosome	QTL detected	Support interval (cM)	Flanking markers	LOD	R^2 (%)	Additive effects	LOD	R^2 (%)	Additive effects	
1BS	*QSng.eth-1BS*	14 (8–22)	*Xglk301, Xglk317*	5.12	7.4	0.026				
1BS	*QSnl.eth-1BS2*	36 (32–40)	*Wgwm18, Xglk483*				3.79	7.3	0.016	CL
2A	*QSnl.eth-2A2*	90 (84–92)	*Xpsr131, Xglk687*				3.96	1.8	−0.016	EC, EL
2B	*QSng.eth-2B*	82 (78–86)	*Xpsr961, Xglk687*	5.13	3.8	−0.031				HD, FD, EL, SNL
2BS	*QSnl.eth-2B1*	86 (82–94)	*Xglk407, xpsr924*				3.87	8.1	−0.015	HD, FD, EL, SNB
2BS	*QSnl.eth-2B2*	164 (158–168)	*Xpsr644b, Xpsr956a*				6.59	4.7	−0.019	
2D	*QSnl.eth-2D*	122 (120–124)	*Xpsr932, Xpsr331a*				8.6	20.8	−0.024	
3A	*QSng.eth-3A1*	50 (36–52)	*Xpsr304, Xpsr598*	3.95	0.3	−0.032				CL
3A	*QSng.eth-3A2*	94 (82–96)	*Xglk118, Xglk577*	3.79	6.4	0.026				HD, FD, CL, EL
3B	*QSnl.eth-3B*	32 (30–36)	*Xglk538, Xpsr902*				5.63	9.9	−0.018	
4A	*QSng.eth-4A1*	48 (38–52)	*CD16.2, Xglk331*	5.18	1.4					CL, EL
4A	*QSng.eth-4A2*	182 (178–188)	*Xglk128, Xgwm710b*	5.51	4.3	0.029				HD, FD
4B	*QSnl.eth-4B*	4 (0–12)	*Xglk348, Xpsr921*				14.3	17	−0.032	
4DL	*QSng.eth-4DL2*	98 (90–100)	*Xglk302, Xpsr1101a*	4.11	1.3	0.024				HD, FD
5A	*QSng.eth-5A2*	218 (214–220)	*Xpsr1194, Xpsr918b*	27.5	35.8	−0.103				HD, FD, CL, EL, EC
5B	*QSng.eth-5B1*	22 (16–32)	*Xpsr128, Xglk614*	9.72	6.3	0.044				HD, FD, CL
5B	*QSnl.eth-5B2*	74 (70–80)	*Xglk163, Xpsr426*				5.82	1.6	−0.018	
7A	*QSng.eth-7AL*	16 (12–20)	*Xglk165, pwir232a*	9.34	5.2	−0.052				HD, EC
7B	*QSnl.eth-7B1*	14 (4–26)	*Xpsr952, Xgwm46*				4.55	0.9	0.016	FD
7B	*QSnl.eth-7B3*	188 (184–188)	*Xmwg710a, Xglk576*				6.72	12.8	0.019	
7S	*QSnl.eth-7S*	8 (6–26)	*Xglk61, Xpsr563b*				4.03	1.4	0.014	
Digenic effects Q × Q interaction (%)	QTL1BS × QTL5B				3.5					
Final simultaneous fit (adj. R^2 and final LOD)				45.20	60.0		30.58	44.1		

Additive effects for SNG and SNL are obtained from the simultaneous fit of all putative QTLs by multiple regressions based on log 10-transformed data. Positive additive effects indicate that Forno contributed to a higher level of resistance, whereas negative additive effects indicate that the allele for resistance was contributed by Oberkulmer

phenotypic variance. The QTL with the highest explained phenotypic value on chromosome 2B had no overlapping support intervals with QTLs for other traits.

For all four QTLs for EC the alleles for a thicker wax layer were inherited from Oberkulmer. One QTL for EC was coincident with *QSnl.eth-2A* and 2 QTLs for EC were coincident with *QSng.eth-5A2* and *QSng.eth-7AL*. At these loci, the alleles for increased resistance were inherited from Oberkulmer. One significant epistatic effect was detected between *QEc.eth-2B* and *QEc.eth-5A*, explaining 12.5% of the phenotypic variance.

QTLs for CL

Averaged over 2 years, 13 QTLs were detected explaining individually between 5.7% and 25.4% of the phenotypic variance. Three QTL × QTL interactions were detected with weak effects (explaining 2.0–4.8% of the phenotypic variance). Four QTLs for CL on chromosomes 3A, 4A, 5A and 5B were co-localised with QTLs for SNG. For these four QTLs the alleles conferring increased resistance to SNB were inherited from the parent, which conferred alleles for longer culms. One QTL for CL was coincident with *QSnl.eth-1BS*, and again the allele for increased resistance was inherited from the parent conferring longer culms.

Discussion

S. nodorum resistance

The parental lines Forno and Oberkulmer of the analysed population differed significantly with regard to SNG and SNL. Therefore, the RILs showed extreme variability for these traits. We found very high heritabilities for SNL and SNG, indicating that the genotypic value of individual RILs was accurately assessed by repeated scorings during disease progress.

Resistance to SNG and SNB showed continuous phenotypic distribution in our population, confirming their quantitative nature. Moreover, 21 QTLs were detected for SNB distributed across the genome, indicating polygenic inheritance. None of the RILs had a fully resistant phenotype, and alleles from the susceptible parent also contributed to improved resistance. These findings confirm several other genetic studies of resistance to SNB (Fried and Meister 1987; Wicki et al. 1999; Schnurbusch et al. 2003).

For SNG and SNL, disease ratings of the F_1 derived from the cross Forno × Oberkulmer were similar to the ratings of the F_5 mean, indicating that dominance is of minor importance, in accordance with previous studies (Karjalainen et al. 1983; Wilkinson et al. 1990; Ma and Hughes 1995; Wicki et al. 1999).

Resistance to SNG was moderately correlated with SNL, with only one common QTL. Therefore, SNG and SNL had to be treated as two genetically independent traits.

SNG

The multiple-regression model calculated for SNG showed that 77% of the phenotypic variance for ear resistance can be explained by morphological traits. This was reflected by the fact that of ten QTLs for SNG, nine overlapped with QTLs for FD (six coincidences), HD (seven coincidences), CL (five coincidences), EL (four coincidences) and EC (two coincidences). Significant correlation between plant height and SNG resistance of wheat was also found by Fried and Meister (1987), Bostwick et al. (1993) Schnurbusch et al. (2003) and Wicki et al. (1999), whereas Scott et al. (1982) and Wicki et al. (1999) found a relationship between resistance of the ear and late-maturing genotypes. Unique to our wheat × spelt population was the phenotypic correlation of resistance with long lax ear and hard glumes.

QSng.eth-5A2, the most important QTL for ear resistance ($R^2 = 35.8\%$), was inherited from the spelt parent Oberkulmer and co-localised with QTLs for all the morphological traits assessed. This position corresponds to the *q* locus responsible for the long lax ear, brittle rachis, hard glumes and hulled kernels of spelt. Therefore, the resistance conferred by this locus is probably because of these morphological traits rather than to defence mechanisms. Hard glumes and thick wax layer are mechanical barriers preventing the penetration of the pathogen. Furthermore, the long ear and culm conferred by the spelt parent results in a less humid microclimate, which is less favourable for the spread and development of *S. nodorum*. Selection for this QTL will lead to tall plants with the spelt ear type, which cannot be used for the development of more resistant wheat varieties.

Other QTLs for SNG resistance associated with late flowering might be exploitable for wheat breeding. Genotypes with the Forno allele at *QSng.eth-4A2* conferring increased resistance flowered 0.92 days later. This was also the case for *QSng.eth-4DL2* overlapping with QTLs for HD and FD, where the alleles of Forno increased resistance but delayed flowering by 1.06 days. For *QSng.eth-7AL* overlapping with QTLs for HD and EC, the Oberkulmer alleles improved SNG and delayed heading by 1.4 days.

Schnurbusch et al. (2003) studied 240 single seed-descent lines derived of an Arina × Forno $F_{5:7}$ population to identify and map QTLs for SNG resistance under natural infestation. Results from three environments conducted over 2 years led to the detection of seven QTLs for SNG. Because of different markers used in both studies, it is rather difficult to compare the exact location of these QTLs with our results. In both studies QTLs for SNG resistance were found on chromosomes 4B, 5A, 5B and 7B. On chromosomes arms 4BL, 5AL and 7BS the resistance alleles were inherited in both

studies from Forno (a common parent), whereas on chromosome 5AL the resistance alleles were inherited from Oberkulmer in our case and by the wheat parent Arina in the population analysed by Schnurbusch et al. (2003).

In a previous report Toubia-Rahme et al. (2003) analysed a population of 164 F_1-derived doubled haploid (DH) lines from the cross Frontana × Remus to identify QTLs for SNG resistance at adult plant stage in the greenhouse. The genetic map comprised 579 markers and enabled the detection of QTLs on chromosomes 5A, 5B and 3B. The precise location was not yet published, but common chromosomes carrying resistance alleles with our work were 5A and 5B.

Correlations between resistance to SNG and resistance to powdery mildew (Keller et al. 1999a) and leaf rust (Messmer et al. 2000) were also studied in the frame of this work. No significant correlations were found for SNG resistance and leaf rust, and only a weak negative correlation ($r = -0.17$, $P < 0.05$) between ear resistance to *S. nodorum* and resistance on the leaf to powdery mildew. It is noteworthy that four QTLs for SNG resistance (*QSng.eth-1BS*, *QSng.eth-3A1*, *QSng.eth-4A2* and *QSng.eth-5A2*) overlapped with QTLs for resistance to powdery mildew (Keller et al. 1999a) and that in both studies the resistance loci were inherited from the same parent.

SNL

Multiple-regression model applied for SNL showed that 46% of the phenotypic variance for leaf resistance can be explained by other traits. The highest correlation with SNL blotch resistance was with CL, HD and FD, in this order.

Four of 11 QTL for SNL blotch resistance coincided either with QTLs for CL (*QSnl.eth-1BS2*), EC and EL (*QSnl.eth-2A2*), with QTLs for FD (*QSnl.eth-7B1*) or with QTLs for HD/FD (*QSnl.eth-2B1*), thus reflecting correlations between these traits. *QSnl.eth-1BS2* was associated with the undesirable agronomic trait CL, which increases the susceptibility for lodging and is, thus, unsuitable for breeding purposes.

Seven QTLs for SNL resistance did not overlap with QTLs for other traits. Among them *QSnl.eth-2D* ($R^2 = 20.8\%$, $a = -0.024$) and *QSnl.eth-4B* ($R^2 = 17\%$, $a = -0.032$) inherited from Oberkulmer, and *QSnl.eth-7B3* ($R^2 = 12.8\%$, $a = +0.019$), inherited from Forno, explained the highest phenotypic variance and were considered to be major loci for SNL resistance. The alleles of *QSnl.eth-2A2*, *QSnl.eth-2D*, *QSnl.eth-3B*, *QSnl.eth-4B* and *QSnl.eth-5B2*, derived from the spelt parent, improved SNL resistance without undesirable morphological affects and are therefore interesting candidates for introgression of spelt resistance genes into wheat.

QSnl.eth-2B2 and *QSnl.eth-2D* had several markers in common in their support interval (*Xpsr956a* on chromosome 2B and *Xpsr956b* on chromosome 2D or *Xglk610b* on 2B and *Xglk610c* on 2D), indicating that these QTLs for leaf resistance to *S. nodorum* were duplicated or homoeologous loci. Interestingly, *Xglk610* in chromosome 2A was 14 cM away from a QTL for SNG resistance (*QSng.sfr-2A*) in the study of Schnurbusch et al. (2003), in which natural infection pressure was used. *Xglk610c* and *Xglk753*, in the support interval of *QSnl.eth-2D*, are 6 cM and 14 cM away respectively from the flanking marker *Xcfd276a* of *QSng.sfr-2AL*. *Xpsr902* is 10 cM away from *QSnl.eth-3B* and 7 cM from the flanking marker *Xcfa2134b* of *QSng.sfr-3BL*.

In a previous study of mapping QTLs to SNL, Czembor et al. (2003) used 111 DH lines from a cross between the resistant *T. aestivum* Liwilla and the susceptible *T. aestivum* Begra. The DH lines were inoculated with a mixture of 15 *S. nodorum* strains at stage GS15 (Zadoks et al. 1974) in the greenhouse. Three different resistance components were evaluated on the fifth leaf. The results indicate the presence of four QTLs for partial resistance on chromosomes 2B, 3B, 5B and 5D. Arseniuk et al. (2004) studied a population of 131 F_1-derived DH lines from the cross Alba × Begra to identify QTLs for SNL seedling resistance. The DH lines were inoculated in the greenhouse at stage GS15 using 15 *S. nodorum* strains, and the same resistance components as described by Czembor et al. (2003) were assessed. Two QTLs were detected in chromosomes 6AL and 6D. In the present study of adult plant resistance to SNL no QTL was detected on chromosome 5D chromosomes 6A and 6D, but resistance was found on chromosomes 2B, 3B and 5B. Further comparisons of the exact location of resistant loci were not possible because of differences in the markers used.

Liu et al. (2004) studied seedling resistance to SNL in a population of 106 RILs from the cross of synthetic W-7984 × spring wheat Opata 85. Plants were inoculated in the greenhouse at the two-to-three-leaf stage with isolate Sn2000. Disease reaction scored 5, 7 and 10 days after inoculation at the second leaf revealed a major QTL on chromosome arm 1BS which was also detected in our study as carrying alleles for resistance to SNL. Liu et al. (2004) also reported minor QTLs, which were not significant for all reading dates, on chromosome 3AS, 3DL, 4AL, 5DL, 6AL and 7BL. Chromosomes 3A, 4AL, 4B and 7BL were common to our study as carrying alleles for SNL resistance at the adult stage under field conditions.

When comparing resistance to SNL and resistance to powdery mildew in the same population (Keller et al. 1999a) as well as resistance to leaf rust (Messmer et al. 2000), no significant correlations were found. Nevertheless, the supporting interval of *QSnl.eth-1BS* overlaps with *QLr.sfr-1BS* ($R^2 = 3.3\%$) for leaf rust resistance, both alleles being inherited from Forno. Schnurbusch et al. (2004) also mapped in the Arina × Forno population a major QTL for leaf rust resistance on chromosome 1B (*QLr.sfr-1BS*, $R^2 = 28$ %) with the resistance alleles inherited from the Forno parent. *Xpsr642* map-

ped in the Arina × Forno population less than 2 cM from *QLr.sfr-1BS* and 2.8 cM from *QSnl.eth-1BS* in our population. Two overlapping QTLs were detected between SNL resistance and powdery mildew: *QSnl.eth-2A2* was co-localised with *QPm.sfr-2A* ($R^2 = 7.7\%$), but the alleles were inherited from different parents. *QSnl.eth-2D* was co-localised with *QPm.sfr-2D* ($R^2 = 10\%$) (Keller et al. 1999a); both resistance alleles were inherited from Oberkulmer. However, it will be difficult to determine whether resistance to various diseases is because of pleiotropic effects of one gene or clusters of genes involved in plant defence (Spielmeyer et al. 1998; Li et al. 1999).

Consequences for wheat breeding

We concluded from our data that SNG and SNL resistance were controlled by different genes, and that they should be considered to be two independent traits in breeding programmes, as suggested by Fried and Meister (1987), Bostwick et al. (1993), Van Ginkel and Rajaram (1999) and Wicki et al. (1999). With regard to SNG, only one of ten QTLs was not associated with a change in EL, CL or FD. Therefore, these QTLs do not trigger defence reaction but act as mechanical barriers or escape mechanisms. The high level of resistance to SNG observed in the spelt parent Oberkulmer was due mainly to one QTL on 5AL conferring the long lax ear of spelt, tall culm and late flowering. Therefore, spelt is of limited value for introgression of resistance genes to SNG into wheat breeding lines. However, improved SNG resistance can be obtained by indirect selection for strong wax layer and delayed flowering. In addition, Arina, which was used as resistant parent in the study of Schnurbusch et al. (2003) because of the high and durable resistance level against SNG, has a strong wax layer and delayed flowering compared to Forno.

With regard to SNL resistance, it must be emphasised that resistance of the leaf is a difficult trait to assess reliably, because the symptoms are similar to *S. tritici* and can be masked by other disease symptoms, such as symptoms of leaf rust and powdery mildew. Studies using natural infections did not lead to QTLs for this trait. In our case the successful assessment of leaf resistance was because of: (1) a reliable screening technique carried over the three upper leaves with seven assessments dates, which conferred 21 data points for each plant; (2) severe infection pressure obtained by four inoculations of the wheat plants within 4 weeks; and (3) the use of a mixture of isolates of *S. nodorum* containing all virulences occurring in a particular region. In the present study, 7 of 11 QTLs for SNL resistance were detected, which were not co-localised with morphological traits. These QTLs may, therefore, represent genes directly involved in the host–pathogen interaction. Among them, *QSnl.eth-2D* is the most interesting one, because it explained the highest phenotypic variance ($R^2 = 21\%$) and was associated with *QPm.sfr-2D* for powdery mildew resistance. Selection for the Oberkulmer allele at this QTL can be achieved with the closely linked marker *Xglk653*. Furthermore, this marker was common to *QSng.sfr-2A* for SNG resistance, found by Schnurbusch et al. (2003) in one of four environments. Another interesting candidate for the introgression of resistance genes from Oberkulmer was *QSnl.eth-4B*, which explained 17% of the phenotypic variance for this trait, with flanking markers *Xpsr921* and *Xglk3488*. Finally, *QSnl.eth-7B3* from the wheat parent Forno can be directly selected in wheat breeding programmes with the flanking markers *Xmwg710a* and *Xglk576*. For efficient and cost-effective introgression of the two resistance loci derived from Oberkulmer into wheat breeding material and the marker-assisted selection for resistance loci derived from Forno, it will be necessary to convert the linked RFLP markers into PCR markers. In addition, the effectiveness of these resistance alleles should be verified in different genetic backgrounds.

Results of our study highlight the importance of the simultaneous assessment of different agronomic traits and underline the power of molecular genetic analysis for the elucidation of the genetic basis of phenotypic correlations between traits. In summary, the dissection of resistance for SNB and morphological traits into individual QTLs allows for the first time identifying resistance genes which are not related to undesirable traits and therefore are interesting candidate genes for marker assisted selection in breeding programs.

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