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QTL mapping of resistance to *Sporisorium reiliana* in maize

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Abstract We mapped and characterized quantitative trait loci (QTL) for resistance to *Sporisorium reiliana*. A population of 220 F_3 families produced from the cross of two European elite inbreds (D32, D145) was evaluated with two replications at a French location with high natural incidence of *S. reiliana* and at a Chinese location employing artificial inoculation. The 220 F_3 families were genotyped with 87 RFLP and seven SSR markers. Using composite interval mapping, we identified two different sets of 3 and 8 QTL for the French and the Chinese locations explaining 13% and 44% of respectively. Individual QTL explained up to 14% of $\hat{\sigma}^2_p$. The 11 QTL mapped to eight maize chromosomes and displayed mostly additive or partial dominant gene action. Significant digenic epistatic interactions were detected for one pair of these QTL. Only a few QTL for *S. reiliana* were in common with QTL for resistance to *Ustilago maydis* and *Puccinia sorghi*, identified at a German location for the same population. Consequently, in our materials resistance to these three fungal pathogens of maize seems to be inherited independently.

Keywords Maize · Head smut · *Sporisorium reiliana* · QTL · RFLP · *Ustilago maydis* · *Puccinia sorghi*

Introduction

Head smut is a soil-borne and systemic disease of maize (*Zea mays* L.) and sorghum (*Sorghum bicolor* L. Moench) caused by host-specific strains of *Sporisorium reiliana* (Kühn) Langdon & Fullerton [*Sphacelotheca reiliana* (Kühn) Clint] (Frederiksen 1977). Economic losses resulting from the infection of susceptible varieties are due to stunting and the sori of head smut replacing inflorescences. In individual environments, up to 80% smutted maize plants have been observed (Frederiksen 1977). Head smut occurs in most of the maize growing regions of the world and is an increasing problem in seed production areas as well as in grain maize production in the southwestern region of France (Bernardo et al. 1992). Hitherto, *S. reiliana* incidence has appeared only sporadically in Germany, but it remains a potential threat to hybrid maize seed production in the upper Rhine valley (Dutzmann and Duben 1993).

Chemical control of head smut in maize is possible by the in-furrow application of fungicides (Meinert 1997; Simpson and Fenwick 1971). However, for ecologic and economic reasons the cultivation of resistant varieties is the preferred way of disease control. Identification of *S. reiliana*-resistant genotypes can be accomplished by direct selection at locations with a high natural incidence of head smut or using artificial inoculation methods (Frederiksen 1977). Both approaches can be hampered by either varying infection conditions in field experiments or limited capacities for seedling assays. Hence, indirect selection based on molecular markers might be a valuable complementary tool to improve resistance to *S. reiliana*.

Race-specific as well as race-unspecific resistance to *S. reiliana* has been reported for sorghum (Craig and Frederiksen 1992). Differences in pathogenicity of isolates of *S. reiliana* affecting sorghum can be evaluated using differential host varieties (Mehta et al. 1967). A major gene involved in race-unspecific head smut resistance was mapped to sorghum linkage group A by bulked segregant analysis (Oh et al. 1994). In contrast, no race-specific resistance to *S. reiliana* has been reported so far for maize (Ali and Baggett 1990; Bernardo et al. 1992; Stromberg et al. 1984; Whythe and Gevers 1988) as holds true for resistance to common smut caused by *Ustilago maydis* (Lübberstedt et al. 1998a). The mode of gene action of resistance to *S. reiliana* was found to vary between predominantly additive and domi-

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nant depending on the materials and experimental conditions employed.

In the study presented here, we evaluated 220 F_3 families from the cross of two European maize inbred lines at a French location with high natural incidence of *S. reiliana* and at a Chinese location with artificial inoculation. The objectives of our research were to (1) determine the number, genomic positions, and gene effects of quantitative trait loci (QTL) involved in head smut resistance, (2) assess the importance of digenic epistatic interactions, (3) compare results from natural and artificial infection, and (4) investigate phenotypic correlations between resistance to head smut, common smut, and common rust as well as the number of QTL in common among these traits.

Materials and Methods

Inbred line evaluation

In 1995, 20 inbred lines including KWA, KWB, KWC, 106589, RZ01, D32, D145, and D408 were evaluated for resistance against *S. reiliana* and *U. maydis* at Chantonnay and Colmar (France) using a randomized complete block design with six replications. Plots consisted of single rows, 0.7 m apart and 3 m long. Plots were overplanted and later thinned to a final plant density of 9 plants m^{-2} with a total of 20 plants. Individual plants were scored for presence or absence of *S. reiliana* as well as *U. maydis* galls 6 weeks after mid-silking by cutting open each ear. The percentage of plants infected by *S. reiliana* (SPO) and *U. maydis* (UST) was the subject of the subsequent analyses.

Mapping population

Two early-maturing European inbred lines, D145 (flint) and D32 (dent) were crossed in both reciprocal forms to produce a random set of 220 F_3 families (population D32×D145). A subset of 122 F_3 families had the cytoplasm of line D145 and the remaining 98 F_3 families that of D32. For each F_3 family, a pool of 20 F_3 plants was chosen to generate an "immortalized F_2" (Gardiner et al. 1993) population (IF_2) by randomly crossing 10 F_3 plants as females and 10 F_3 plants as males. Lines D145 and D32 were chosen for this and a companion study (Xia et al. 1999) because of their complementary performance against sugarcane mosaic virus (SCMV) (Kuntze et al. 1997), *U. maydis*, and *S. reiliana* (Table 1). Both lines are proprietary public inbreds of the University of Hohenheim, Germany.

Marker assays and linkage maps

The restriction fragment length polymorphism (RFLP) and simple sequence repeat (SSR) assays and the segregation and linkage analyses of the marker loci have been described in detail elsewhere (Xia et al. 1999). The RFLP data and marker linkage map given by these authors were also used in the QTL analyses presented here. A total of 87 RFLP and 7 SSR marker loci well distributed over the maize genome were used to genotype the parental F_2 individuals of all 220 F_3 families.

Agronomic trials

Population D32×D145 was evaluated at Chantonnay in 1996 and 1997 for resistance against *S. reiliana* and *U. maydis*. The experiment included 200 entries in a 20×10 alpha design (Patterson and Williams 1976) with two replications: 188 F_3 families, randomly chosen out of the 220 families from population D32×D145, in 1996 and the respective IF_2 families in 1997, both parent lines and their F_1 hybrid, included as duplicate entries each, and six European check inbred lines with relatively high (KWB, KWC) or low (D408, 106589, KWA, RZ01) *S. reiliana* incidence at Chantonnay in 1995 (Table 1). In addition, population D32×D145 was evaluated at Gongzhuling (Jilin Province, China) in 1998 for resistance to *S. reiliana* after artificial inoculation. This experiment included 230 entries in a 23×10 alpha design (Patterson and Williams 1976) with two replications: 220 IF_2 families, quintuple entries of the F_1 hybrid of D32 and D145, as well as five Chinese checks. Moreover, all 220 F_3 (1996) or IF_2 (1997) families were evaluated for resistance to SCMV (Xia et al. 1999), *U. maydis*, and rust (*Puccinia sorghi*) at Eckartsweier 1996 and Hohenheim 1997, respectively. These experiments consisted of 230 entries in a 23×10 alpha design with two replications: the 220 F_3 or corresponding IF_2 families in 1996 and 1997, respectively, and inbred lines D32 and D408, included as quintuple entries each. Plots consisted of single rows, 0.7 m apart and 3 m long. Plots were overplanted and later thinned to a final plant density of 9 plants per square meter with a total of 20 plants.

At Chantonnay, individual plants were scored for presence or absence of *S. reiliana* as well as *U. maydis* galls 6 weeks after mid-silking by cutting open each ear. The percentage of plants infected by *S. reiliana* (SPO) and *U. maydis* (UST) was the subject of the subsequent analyses. Artificial inoculation was applied at Gongzhuling by sowing maize kernels together with *S. reiliana*

Table 1 Means of eight European maize inbred lines evaluated for resistance to *S. reiliana* and *U. maydis* at Colmar in 1995 (Col95) and at Chantonnay from 1995 to 1997 (Cha95, Cha96, Cha97), least significant differences (LSD 5%) between means, and repeatabilities

Inbred	*S. reiliana* (%)					*U. maydis* (%)				
	Col95	Cha95	Cha96	Cha97	Mean	Col95	Cha95	Cha96	Cha97	Mean
D32 (P1)	2.61	38.72	40.20	2.90	21.11	0.00	1.96	0.09	0.00	0.51
D145 (P2)	0.00	11.95	1.90	0.05	3.48	2.89	4.09	8.67	2.71	4.59
D408	0.09	0.00	0.13	0.00	0.03	0.23	7.37	6.92	3.13	4.41
106589	0.00	0.00	0.15	0.05	0.05	0.00	0.00	0.03	0.03	0.02
KWA	0.00	0.88	0.07	0.26	0.30	0.00	0.00	0.00	0.01	0.00
KWB	8.16	55.09	7.40	15.21	21.47	3.26	1.91	6.21	2.52	3.48
KWC	2.68	86.08	89.16	9.66	46.90	0.00	9.46	6.58	7.14	5.80
RZ01	1.62	4.05	3.29	0.23	2.30	1.06	9.47	0.00	2.51	3.26
Mean	1.90	24.60	17.79	3.55	11.96	0.93	4.28	3.56	2.26	2.76
LSD5%	3.70	10.30	19.30	7.62		3.80	7.40	12.09	7.52	
Repeatability[a]	0.27	0.87	0.69	0.20		0.44	0.56	0.47	0.22	

[a] Repeatability was calculated on a single-plot basis

spores bulked from the previous field season. At Gongzhuling for SPO and for UST at both German environments, symptoms were recorded without opening ears by cutting. Plants were scored for symptoms of rust (PUC) 3 weeks after mid-silking on a 1–9 scale for the portion of leaf area covered with naturally occuring rust. Similar to the disease scoring (1–7 scale) by Kim and Brewbaker (1977), ratings of 1, 3, 5, 7, and 9 were scored when the diseased leaf area covered was 0, 10%, 20%, 30%, and more than 40%, respectively, of the leaf surface of representative plants in each plot, excluding the youngest and oldest leaves.

Statistical analyses

For individual environments, analyses of variance were conducted on non-transformed (SPO, UST, PUC) or transformed (TSPO=arcsin $\sqrt{SPO/100}$; TUST=arcsin $\sqrt{UST/100}$) phenotypic data. Entry means adjusted for incomplete block effects in the lattice design and effective error mean squares were calculated as described by Cochran and Cox (1957). The contrast of F_3 means based on the cytoplasmic origin (D145 versus D32) resulted in non-significant differences for resistance to *S. reiliana* and *U. maydis*, but significantly ($P<0.01$) higher PUC values for F_3 families with cytoplasm derived from D32 compared to D145. Hence, all 220 F_3 families were subjected to subsequent analyses irrespective of their cytoplasm. The sums of squares for entries were partitioned into sums of squares due to F_3 families and checks (parent lines, F_1, check inbreds). Estimates of variance components for σ^2 (error variance) and σ_g^2 (genotypic variance) of the F_3 families and their standard errors as well as repeatabilities and phenotypic ($\hat{r}_p$) correlations among traits were calculated by applying standard procedures (Searle 1971) implemented in software PLABSTAT (Utz 1991).

Adjusted entry means and effective error mean squares were used to compute combined analyses of variance across environments for each population as described by Bohn et al. (1996). Heritabilities (h^2) on an entry-mean basis were estimated as described by Hallauer and Miranda (1981). However, because infection levels in individual environments differed considerably and mostly caused non-significant estimates of $\hat{\sigma}_g^2$ and low heritabilities ($\hat{h}^2$), results from the analyses across environments were neither presented here nor employed for QTL analyses.

We used the method of composite interval mapping (CIM) (Zeng 1994) for mapping of QTL and estimation of their effects as described by Bohn et al. (1996). A LOD threshold of 2.5 was chosen for declaring a putative QTL significant. For each population and environment and also for the joint analyses across environments, cofactors were selected by stepwise regression (Draper and Smith 1981). Final selection was for the model that minimized Akaike's information criterion with penalty=3.0 (Jansen 1993). QTL positions were determined at the local maxima of the LOD ($\log_{10}$ odds ratio) plot curve in the regions under consideration. The proportion of the phenotypic variance ($\hat{\sigma}_p^2$) explained by an individual QTL was calculated as the square of the partial correlation coefficient (Melchinger et al. 1998). Putative QTL were examined for the presence of digenic epistatic interactions (Lübberstedt et al. 1997). All computations were performed with the software package PLABQTL (Utz and Melchinger 1996).

Results and discussion

Agronomic trait analysis

The choice of parents is crucial in QTL mapping studies because the chances for detection of QTL increase if the parents are extremes for the traits of interest substitute by: (Lander and Botstein 1989). In previous studies, 137 European maize inbred lines were evaluated in 1993 and 1994 for resistance to corn borer (Schulz et al. 1997), *Setosphaeria turcica* (Welz et al. 1998), sugarcane mosaic virus (SCMV) (Kuntze et al. 1997), *Ustilago maydis*, *Sporisorium reiliana*, and *Puccinia sorghi* (Lübberstedt, unpublished data). Twenty of these inbreds were re-evaluated for resistance to *S. reiliana* and *U. maydis* at two locations in 1995 in order to choose parents with an extreme and opposite resistance reaction to different pathogens for the development of a mapping population. Data from a subset of eight inbreds evaluated across four environments are presented in Table 1. Much higher levels of natural infection across these eight inbreds were found for both fungal diseases at Chantonnay 1995 compared to Colmar 1995 (Table 1). SPO ranged between 0.0% to 86.1% with an arithmetic mean of 24.6% at Chantonnay compared to a range of 0.0% to 8.2% with a mean of 1.9% at Colmar. Likewise, means for UST were 4.3% at Chantonnay and 0.9% at Colmar. While D32 and D145 differed substantially but were not extremes with regard to resistance against *S. reiliana* and *U. maydis*, they were chosen as the parents of our mapping population due to complementary resistance against other pests of maize.

Several biotic and abiotic stress factors are characterized by an unstable expression across environments. While the infection level of the checks for *S. reiliana* and *U. maydis* was similar in 1994 to 1996, it was much lower in 1997 (Table 1). More than 86.0% of all plants of inbred line KWC were infected with *S. reiliana* between 1994 (Lübberstedt, unpublished data) and 1996, but only 9.7% in 1997 (Table 1). Likewise, infection of inbred D32 exceeded 38.7% between 1994 and 1996, but was only 2.9% in 1997. The mean for SPO across the 188 F_3 families of population D32×D145 dropped from 15.7% in 1996 to 2.1% in 1997 (Table 2). For UST, 6.1% of all F_3 individuals were infected in 1996, whereas in 1997 only 2.6% showed symptoms. Consequently, repeatabilities for SPO and UST at Chantonnay were much lower in 1997 compared with 1995 and 1996 (Table 1). Important reasons for the low infection level in 1997 might have been the comparatively low temperatures in June and more than twofold higher precipitation during May and June 1997 compared with 1994–1996, causing suboptimal conditions for seedling infection by *S. reiliana* that requires warm and dry weather. Interestingly, other fungal pathogens with different life cycles and infection pathways, such as *U. maydis* and *P. sorghi* also showed a rather low incidence at German locations in 1997 (Table 2; Lübberstedt, unpublished data). Another environment with a low incidence of *S. reiliana* was Colmar 1995 (Table 1), a result of the low infection pressure in the Upper Rhine valley. Consequently, Chantonnay 1997 and Colmar 1995 were dropped from further analyses.

Natural infection and artificial inoculation

Artificial inoculation can help to ensure a high infection level and has been successfully established for *S. reiliana* (Baggett and Koepsell 1983; Mehta et al. 1967). In fact, the percentage of infected F_3 plants of population

Table 2 Means, variance components, and repeatabilities of parent lines D32 and D145, their F_1 hybrid, and 188 or 220 F_3 (IF_2) families of cross D32×D145 evaluated at Chantonnay in 1996 and 1997 (Cha96, Cha97) for the percentage of plants infected by *S. reiliana* (SPO) or *U. maydis* (UST), at Gongzhuling in 1998 (Gon98) for SPO, and at Eckartweier in 1996 (Ewe96) for UST and rust (*P. sorghi*) ratings

Parameters	*S. reiliana* (%)			*U. maydis* (%)			*P. sorghi* (1–9 scale)
	Cha96	Cha97	Gon98	Cha96	Cha97	Ewe96	
Means[a]							
D32	40.20±6.78	2.90±2.69	–	0.09±4.25	0.00±2.68	3.23±6.11	5.53±1.16
D145	1.90±6.78	0.05±2.69	–	8.67±4.25	2.71±2.68	–	–
F_1	17.00±6.78	2.16±2.69	35.72±6.60	3.09±4.25	0.03±2.68	–	–
F_3	15.73±1.18	2.11±0.38	40.31±1.29	6.11±0.61	2.55±0.09	12.47±0.72	3.29±0.15
Variance components							
$\hat{\sigma}_g^2$	212.3±28.41**	3.60±2.93**	255.4±37.13**	33.21±7.63**	4.12±0.81**	75.92±11.34**	3.68±0.50**
$\hat{\sigma}^2$	91.9 10.18	14.49±1.61	217.6±22.62	36.22±4.01	14.42±1.60	74.71±7.11	2.71±0.26
Repeatability	0.77	0.25	0.68	0.63	0.36	0.74	0.70
90% C.I.[b]	(0.70; 0.83)	(0.01; 0.44)	(0.58; 0.75)	(0.51; 0.73)	(0.15; 0.52)	(0.66; 0.80)	(0.61; 0.77)

*, ** Significant at the 0.05 and 0.01 probability levels, respectively
[a] Standard errors are attached
[b] Confidence intervals (C.I.) of repeatabilities were calculated by the method of Knapp et al. (1985)

Table 3 QTL detected for resistance to *S. reiliana* and *U. maydis* for 188 F_3 lines of cross D32×D145 evaluated at Chantonnay in 1996

Disease chromosome	Position[a] (cM)	LOD	$R^{2[b]}$ (%)	Gene effect	
				Additive	Dominant
S. reiliana					
6	162	2.7	7.2	–0.86	16.19
8	6	2.5	6.0	–3.47*	–5.63
9	70	3.1	7.3	–3.56**	4.36
Total		5.6	12.7	7.89	26.18
U. maydis					
2	80	2.8	6.7	–3.46*	1.32
2	98	5.0	11.7	5.89**	–4.51
5	172	2.8	7.0	2.90**	11.00**
Total		7.5	16.9	12.25	16.83

*, ** Significant at the 0.05 and 0.01 probability levels, respectively
[a] Map position according to Xia et al. (1999)
[b] Percentage phenotypic variance explained by QTL

D32×D145 was more than two times greater at Gongzhuling (Jilin province, China) in 1998 than at Chantonnay in 1996. The mean across all 220 F_3 families of cross D32×D145 evaluated for resistance to *S. reiliana* after artificial inoculation at Gongzhuling was 39.7% (Table 2). It is a crucial question for breeders, whether results from artificial inoculation are consistent with those obtained under natural infection conditions. Since our method of artificial inoculation exclusively increased the concentration of *S. reiliana* spores in close proximity to seeds, no principal difference compared to natural infection was expected. However, no QTL for TSPO or SPO were in common between Chantonnay in 1996 and Gongzhuling in 1998 (Tables 3 and 4), which was in agreement with a poor phenotypic correlation ($\hat{r}_p$=0.03) among entry means in these two environments. Possible reasons are QTL-by-environment interactions caused by (1) different races of *S. reiliana* prevalent at both locations, interacting with race-specific resistance genes, or (2) different growing conditions both for maize and the pathogen at the two locations.

QTL analysis

For Chantonnay 1996, histograms for SPO and UST were strongly skewed to the right. Snedecor's (1956, p 200) measure of skewness (g_1) was highly significant (P<0.01) for both SPO (g_1=1.76) and UST (g_1=1.61). Therefore, QTL analyses were performed for SPO and UST as well as for TSPO and TUST. Three QTL were detected for TSPO (chromosomes 6, 8, 9) and TUST (Chromosomes 2, 2, 5) explaining in total 12.7% and 16.9% respectively, of $\hat{\sigma}_p^2$ (Table 3). Individual QTL accounted for 6.0–11.7% of $\hat{\sigma}_p^2$. For comparison, only 1 QTL (chromosome 6) and 2 QTL (both on chromosome 2) were detected for SPO and UST, respectively. This in contrast to almost identical QTL results for SPO and TSPO at Gonzhuling as well as a good agreement of QTL detected for UST and TUST in another study (Lübberstedt et al. 1998a). Additive gene effects were significant for 5 out of the 6 QTL, whereas only 1 QTL (TUST, chromosome 5) displayed significant dominant gene action. Digenic epistatic interactions of type additive×dominant were found for TUST between

Table 4 Parameters associated with putative QTL significantly affecting percentage of plants infected by *S. reiliana* at Gongzhuling (Jilin, China) in 1998 after artificial inoculation, for percentage of plants infected by *U. maydis*, and for rust ratings evaluated at Eckartsweier in 1996 for 220 F_3 lines of maize population D32×D145

Chrom.[a]	*Sporisorium reiliana*				*Ustilago maydis*				*Puccinia sorghi*			
	a[b]	d[b]	R^2[c]	Position[d]	a	d	R^2	Position	a	d	R^2	Position
	%				%				%			
1									0.74**	–0.18	11.7	12
1	5.72**	10.84	6.3	52								
1					–4.67**	–3.30	6.3	132	0.40**	–0.08	5.2	120
1					5.06**	10.50*	7.0	212	–0.45**	–0.18	8.1	228
2					–4.89**	–0.16	10.5	42				
2	6.31**	6.48	6.3	100								
3	–6.13*	–23.86**	6.5	110	5.08**	–2.97	7.1	90				
3									–0.52**	1.38*	9.5	144
4	–9.00**	2.46	13.9	78	–4.89**	–5.22	11.8	88				
5									–0.77*	–1.08	6.2	42
5	–10.75**	–5.20	13.3	112					–0.88**	0.01	19.3	114
6	–5.68**	0.96	6.9	16	4.38**	–8.97**	7.0	20				
6									–0.64**	–0.03	9.6	70
6	–7.67**	–3.25	12.4	114								
7					4.57**	7.46	6.7	134				
8									0.47**	0.21	9.5	16
8	5.06**	2.21	5.6	74								
9					4.33**	–2.94	6.6	120				
10					4.05**	0.32	6.3	6				
Total	56.32	55.26	44.0		41.92	41.84	41.1		4.87	3.15	47.1	

*,** Significant at the 0.05 and 0.01 probability levels, respectively
[a] Maize chromosome
[b] a=additive gene effect and d = dominance effect of QTL
[c] Percentage phenotypic variance explained by QTL
[d] Map position according to Xia et al. (1999)

one pair of QTL on chromosome 2 at position 98 cM and Chromosome 5.

At Gongzhuling in 1998, 8 QTL were detected for SPO on chromosomes 1, 2, 3, 4, 5, 6 (2 QTL), and 8, with these 8 QTL explaining altogether 44.0% of $\hat{\sigma}_p^2$ (Table 4). Individual QTL accounted for 5.6–13.9% of $\hat{\sigma}_p^2$. All 8 QTL displayed significant additive gene effects, whereas only 1 QTL (chromosome 3) displayed dominant gene action. No significant digenic epistatic interactions were found for SPO in this environment.

In total, we identified 11 QTL for SPO on each of the ten maize chromosomes except for chromosomes 7 and 10 (Tables 3, 4). Additive gene effects were significant for 10 out of 11 QTL, whereas only 1 QTL on chromosome 3 displayed a significant dominant gene effect (Table 3). About one-half of the QTL showed additive or partially dominant gene action, while the other half displayed dominant or overdominant gene action. For 8 out of 11 QTL, the resistance-increasing allele originated from the resistant parent D145. Dominant gene effects both increased and decreased resistance to *S. reiliana* (Tables 3 and 4). Our findings are largely consistent with classical genetic studies (Ali and Baggett 1990, Bernardo et al. 1992; Whythe and Gevers 1988) suggesting a preponderance of additive gene action, especially under a high disease incidence (Ali and Baggett 1990). Accordingly, only 2 out of 8 QTL identified at Gongzhuling 1998 displayed overdominant gene action in contrast to all 3 QTL detected at Chantonnay 1996 (Tables 3, 4).

Different from the pathosystem Sorghum – *S. reiliana* (Craig and Frederiksen 1992; Oh et al. 1994), neither major nor race-specific resistance genes have so far been detected in maize against *S. reiliana*. Likewise, we detected exclusively minor resistance genes individually explaining up to 13.9% of $\hat{\sigma}_p^2$ (Table 4). However, the presence of major resistance genes to *S. reiliana* in maize cannot be ruled out because some inbred lines (D408, 106589, KWA) displayed extreme resistance from 1994 to 1997.

Clustering of resistance genes

Under natural infection conditions at Eckartsweier in 1996, 9 and 8 QTL were detected for UST and PUC, respectively, explaining 41.1% and 47.1% of $\hat{\sigma}_p^2$ (Table 4). Individual QTL accounted for 5.2 to 19.3% of $\hat{\sigma}_p^2$. All QTL for both UST and PUC displayed significant additive gene effects, whereas only 2 QTL for UST (chromosomes 1, 6) and 1 QTL for PUC (chromosome 3) displayed dominant gene action. Digenic epistatic interactions of type additive×dominant were found for PUC between the QTL located on chromosome 7 and 1 at position 12 cM. These findings are in agreement with former studies (Lübberstedt et al. 1998a, b), in which additive gene action or partial dominance prevailed for QTL affecting PUC, while about half of the QTL detected for UST displayed dominant or overdominant gene action.

Largely different sets of QTL were detected for SPO at Gongzhuling and UST at Eckartsweier (Table 4). This is in harmony with the low phenotypic correlation ($\hat{r}_p$=0.21) between both traits in population D32×D145 and is also consistent with the different infection pathways of *S. reiliana* (systemic infection) and *U. maydis* (local infection). However, given that the phenotypic correlation between SPO and UST was much tighter for the set of 20 inbred lines tested in multiple environments ($\hat{r}_p$>0.45) (Lübberstedt, unpublished data), the poor consistency of QTL for SPO and UST might be specific for population D32×D145 or attributable to contrasting environmental conditions. In addition, only few QTL were in common between SPO and PUC and between UST and PUC, which was again in agreement with the findings of Lübberstedt et al. (1998a).

Our results do not support the hypothesis of resistance gene clusters (McMullen and Simcox 1995), at least not for minor resistance genes. However, interestingly those two regions (chromosomes 4, 5), which explained most of $\hat{\sigma}_p^2$ for SPO at Gongzhuling (13.9%, 13.3%), also explained the largest proportion of $\hat{\sigma}_p^2$ for UST (chromosome 4: 11.8%) and PUC (Chromosome 5: 19.3%), respectively, with identical signs of the additive effects. This raises the question of whether a clustering of resistance genes is restricted to major genes and QTL with large effects.

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