Comparative Quantitative Trait Loci Mapping of Partial Resistance to *Puccinia sorghi* Across Four Populations of European Flint Maize

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ABSTRACT

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We mapped and characterized quantitative trait loci (QTL) for partial resistance to *Puccinia sorghi* and investigated consistency across different European flint maize populations. Four independent populations, containing 280 F₃ lines (A×B^I), 120 F₅ lines (A×B^{II}), 131 F₄ lines (A×C), and 133 F₄ lines (C×D), were produced from four European elite flint inbreds (A, B, C, and D) and genotyped at 89, 151, 104, and 122 restriction fragment length polymorphism marker loci, respectively. All F_n lines were evaluated in field trials with two replications in three or five (A×B^I) environments. Genotypic variance was highly significant for rust ratings in

all populations, and heritabilities exceeded 0.64. Between 4 and 13 QTL were detected in individual populations using composite interval mapping, explaining between 33 and 71% of the phenotypic variance. Twenty QTL were distributed over all ten chromosomes, without preference to chromosomes 3, 4, 6, and 10, which harbor qualitatively acting *Rp* loci. In most cases, gene action was additive or partially dominant. Four pairs of QTL displayed significant digenic epistatic interactions, and QTL-environment interactions were observed frequently. Approximately half of the QTL were consistent between A×B^I and A×B^{II} or A×C and C×D; fewer were consistent between A×B^I and A×C or C×D. In European flint maize germ plasm, conventional selection for partial rust resistance seems to be more promising than marker-assisted selection.

Common rust, caused by *Puccinia sorghi* Schwein., is a major disease of sweet corn in the United States and is responsible for damages in grain and forage maize, seed-production fields, and breeding nurseries (11,28). Under the cooler climatic conditions of Central and Western Europe, common rust becomes economically significant only in regions with milder climates, such as the Upper Rhine Valley, where hybrid seed is produced. Fungicides that control *P. sorghi* are available, but for economic and ecological reasons, application is not feasible (14). Resistance within adapted elite breeding materials and cultivation of resistant cultivars are the most appropriate alternative solutions for disease control.

There are two types of resistance against *P. sorghi*: partial and race-specific. More than 25 dominant resistance (*Rp*) genes are involved in race-specific resistance and organized in complex loci at chromosomes 3, 4, and 10 (14). Within these complex loci, novel resistance specificities are generated by genetic reassortment events, such as unequal crossing-over or gene conversion (29). Recently, another *Rp* locus was detected on chromosome 6 (4). Single race-specific resistance genes confer high levels of resistance to specific rust biotypes and can be employed for control of prevalent rust isolates. Because of high selection pressure, race-specific resistance frequently is overcome within a short time (14). Pyramiding of multiple closely linked genes into "compound" genes has been proposed as a possible means of constructing more durable race-specific resistance inherited by complex loci against common rust in maize or powdery mildew in barley (15).

Partial resistance against common rust reduces the rate of fungal development on leaves of mature plants and is believed to be race-nonspecific and, therefore, more durable than race-specific resistance (36). Partial rust resistance is highly heritable and inherited by a few

to several genes. Depending on the materials employed in generation mean analyses, the prevalent mode of gene action varies between additive and dominant for resistance against *P. sorghi* (17,28).

In this study, we used four European flint maize populations to characterize quantitative trait loci (QTL) for partial resistance to *P. sorghi* identified after natural infections in Germany. The objectives of our research were to (i) determine the number, genomic position, and gene effects of QTL involved in rust resistance; (ii) assess the importance of QTL-environment (QTL×E) and digenic epistatic interactions; and (iii) investigate the consistency of QTL across populations.

MATERIALS AND METHODS

Plant materials. Four early maturing, homozygous European flint maize lines, KW1265, D146, D145, and KW1292 (subsequently named A, B, C, and D, respectively), were crossed to produce a random set of 280 F₃ lines of cross A×B, an independent sample of 120 F₅ (F_{4,5}) lines of cross A×B, 131 F₄ (F_{3,4}) lines of cross A×C, and 133 F₄ (F_{3,4}) lines of cross C×D. The four resulting populations were designated A×B^I, A×B^{II}, A×C, and C×D, respectively. Test-crossed progenies of the families used in this investigation were employed in previous studies on forage yield and quality traits (22–24). Lines A and D are private inbreds developed by KWS Kleinwanzlebener Saat-zucht AG (Einbeck, Germany); lines B and C are public inbreds proprietary to the University of Hohenheim (Stuttgart, Germany).

Restriction fragment length polymorphism assays and linkage maps. The restriction fragment length polymorphism (RFLP) assays of all four populations and the segregation and linkage analyses of the RFLP marker loci have been described in detail elsewhere (22,32). The RFLP data and marker linkage maps published by Lübberstedt et al. (22) and Schön et al. (32) were used in the QTL analyses presented here. A total of 89, 151, 104, and 122 RFLP marker loci, well distributed over the maize genome, were used to genotype 275 F₃ A×B^I, 113 F₅ A×B^{II}, 131 F₄ A×C, and

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133 F₄ C×D lines. The 275 F₃ A×B^I lines used in this study represent a random sample of the 344 F₃ lines used by Schön et al. (1994). Populations A×B^I and A×B^{II} were derived from independent samples of the A×B cross.

Agronomic trials. Population A×B^I was evaluated in five environments in Germany (1993: Eckartsweier, Hochburg, Krozingen, and Zell; 1994: Eckartsweier). The experiment included 300 entries in a 30 × 10 alpha design (27) with two replications: 280 F₃ lines and both parent lines 10 times each. Field experiments with progenies of the three smaller populations were conducted in adjacent trials in three environments in Germany (1993: Eckartsweier, Hochburg, and Krozingen). Each experiment included 150 entries: 131 or 133 F₄ lines of A×C and C×D, respectively, and 120 F₅ lines of A×B^{II} plus each parent line up to 15 times each. The experimental design in each environment was a 15 × 10 alpha design with two replications. Plots consisted of single rows, 0.7 m apart and 4 m long. Plots were overplanted and later thinned to a final plant density of 9 plants per m² for a total of 27 plants.

Plants were scored for disease symptoms 3 weeks after mid-silking on a 1 to 9 scale based on the proportion of leaf area covered with naturally occurring rust. Similar to the disease-scoring scale of Kim and Brewbaker (17), ratings of 1, 3, 5, 7, and 9 were given when the diseased area covered 0, 10, 20, 30, and >40%, respectively, of the leaf surface of representative plants in each plot, excluding the youngest and oldest leaves.

Data analyses. Analyses of phenotypic data and QTL mapping were performed as described by Bohn et al. (1). Briefly, adjusted entry means and effective error mean squares were used to compute combined analyses of variance across environments for each population. The sums of squares for entries were subdivided into variation among F_n lines (F₃ A×B^I, F₄ A×C and C×D, and F₅ A×B^{II}) and orthogonal contrasts among means of both parent lines (P1 and P2) and F_n lines. A corresponding subdivision was conducted on the entry-environment interaction sums of squares. Estimates of variance components σ_g^2 (genotypic variance), σ_{ge}^2 (genotype-environment interaction variance), and σ^2 (error variance) of F_n lines, and their standard errors were calculated as described by Searle (33). To test significant differences of σ_g^2 across populations, *F* tests were employed according to the approximation given by Satterthwaite (31). Heritabilities (*h*²) on an entry mean basis were estimated as described by Hallauer and Miranda (12):

$$\hat{h}^2 = \frac{\hat{\sigma}_g^2}{\hat{\sigma}_g^2 + \hat{\sigma}_{ge}^2 / e + \hat{\sigma}^2 / r \cdot e}$$

where *r* = number of replications and *e* = number of environments. Exact 90% confidence intervals of *h*² were calculated according to Knapp et al. (18). Significance of transgressive segregation in each population was tested using a *t* test (10).

We employed the composite interval mapping method (40) for mapping QTL and estimating the effects, as described by Bohn et al. (1). A LOD (log₁₀ odds ratio) threshold of 2.5 was chosen for declaring a putative QTL significant. For each population and environment and for joint analyses across environments, cofactors were selected by stepwise regression (6). Final selection was based on the model that minimized Akaike's information criterion with a penalty = 3.0 (16). For each population, QTL positions were determined at the local maxima of the LOD (log₁₀ odds ratio) plot curve in the regions studied. The phenotypic variance ($\hat{\sigma}_p^2$) explained by a single QTL was obtained by the square of the partial correlation coefficient. Putative QTL were examined for the presence of QTL×E and digenic epistatic interactions (22). Each QTL was represented by a 20-centimorgan (cM) interval, with the local LOD maximum as the center. For comparison of QTL across two populations, the positions (20-cM intervals) of one population were adjusted relative to the nearest flanking RFLP markers in common with the other population, adopting the procedure described by Lübberstedt et al. (22). When two populations lacked common RFLP markers for some genomic regions, positions (20-cM intervals) were compared relative

to closely linked RFLP markers that were polymorphic in only one of the populations. QTL with overlapping 20-cM intervals were considered common, irrespective of additive or dominance effects. All computations were performed using PLABQTL software (35).

RESULTS

Agronomic trait analysis. For each of the four F_n populations, means for rust ratings differed significantly among environments, ranging from 3.92 (A×B^I: Eckartsweier 1994) to 7.03 (A×B^I: Zell 1993). Means across the three environments common to all four segregating populations were greatest for both A×B populations (Table 1). Means of both parent lines differed significantly (*P* < 0.05) for A×B^I, A×B^{II}, and A×C rust ratings but not C×D. Means of F_n lines significantly (*P* < 0.05) transgressed the means of both parent lines in all four populations. The means of the F_n lines were significantly (*P* < 0.05) greater than the midparent value for rust incidence in A×C and C×D but not different in both A×B populations.

Repeatability of rust ratings in individual environments ranged from 0.46 (A×B^I: Zell 1993) to 0.78 (A×B^{II}: Hochburg 1993), except for a low value (0.05) for Krozingen 1993 (A×B^I). Genotypic variance among F_n lines and estimates of σ_{ge}^2 was highly significant (*P* < 0.01) in each population (Table 1). In all four populations, $\hat{\sigma}_{ge}^2$ was smaller than $\hat{\sigma}_g^2$. Genotypic variance in A×B^I was significantly smaller than in the other three populations. Heritability for rust ratings was highest in A×C and C×D (*h*² = 0.82) and substantially greater than in A×B^I (*h*² = 0.65) for the three environments common to all four populations.

QTL analyses. Except for population A×B^I, a detailed presentation of results from QTL analyses is given for means across environments, because (i) these QTL are the most important for breeding purposes; (ii) a test of QTL×E interactions can be included; and (iii) the results are easier to survey. (A complete list of the number and designation of the selected cofactors used for each population is available upon request from Th. Lübberstedt.)

TABLE 1. Estimates of means, variance components, and heritabilities for common rust ratings (1 to 9 scale) of parent lines (P1 and P2) and F_n lines (cross of P1 and P2) from maize populations (A×B^I, A×B^{II}, A×C, C×D)^a evaluated in three common environments^b

Parameter ^c	A×B ^I	A×B ^{II}	A×C	C×D
Mean ^d				
P1	6.75 ± 0.07	6.21 ± 0.12	6.22 ± 0.09	4.67 ± 0.12
P2	6.20 ± 0.07	5.47 ± 0.12	4.34 ± 0.09	4.69 ± 0.12
$\bar{P}$	6.48 ± 0.05	5.84 ± 0.08	5.28 ± 0.06	4.68 ± 0.09
F _n	6.40 ± 0.02	6.06 ± 0.05	5.65 ± 0.05	5.56 ± 0.06
F _n EWE	5.75 ± 0.03	4.79 ± 0.11	4.14 ± 0.07	4.60 ± 0.09
F _n HOCH	6.88 ± 0.03	6.97 ± 0.09	6.34 ± 0.07	5.92 ± 0.06
F _n KROZ	6.56 ± 0.02	6.41 ± 0.05	6.46 ± 0.05	6.15 ± 0.04
Range ^e	4.75–7.35	3.89–7.81	3.65–7.06	3.43–7.15
Variance component ^f				
$\hat{\sigma}_g^2$	0.10** ± 0.01	0.35** ± 0.06	0.26** ± 0.04	0.35** ± 0.05
$\hat{\sigma}_{ge}^2$	0.07** ± 0.01	0.25** ± 0.04	0.06** ± 0.02	0.10** ± 0.02
$\hat{\sigma}^2$	0.20 ± 0.02	0.32 ± 0.02	0.20 ± 0.02	0.26 ± 0.02
Heritability ($\bar{F}_n$ lines)				
<i>h</i> ²	0.65	0.73	0.82	0.82
90% CI ^g	(0.57; 0.71)	(0.62; 0.80)	(0.76; 0.87)	(0.75; 0.86)

^a Populations contained 280 F₃ lines, 120 F₅ lines, 131 F₄ lines, and 133 F₄ lines, respectively.

^b F_n EWE: Eckartsweier 1993; F_n HOCH: Hochburg 1993; F_n KROZ: Krozingen 1993.

^c $\bar{P}$ = mean of P1 and P2; F_n = means of F₃ (A×B^I), F₄ (A×C, C×D), and F₅ lines (A×B^{II}), respectively.

^d Means ± standard errors.

^e Minimum to maximum.

^f ** indicates value is significant at *P* < 0.01.

^g Confidence intervals of *h*² were calculated by the method of Knapp et al. (18).

For the three environments common to all four populations, a total of 9, 4, 7, and 13 QTL were detected in A×B^I, A×B^{II}, A×C, and C×D, respectively. The QTL were distributed over all 10 chromosomes (Table 2). A simultaneous fit of all putative QTL explained between 33.4% (A×B^I) and 70.9% (C×D) of $\hat{\sigma}_p^2$. Individual QTL explained between 4.2 and 25.5% of $\hat{\sigma}_p^2$ (A×C: chromosome 2). All QTL displayed significant additive gene effects, except for three QTL (Table 2). The sum of absolute additive effects was 1.07, 0.93, 1.46, and 2.56 score units in A×B^I, A×B^{II}, A×C, and C×D, respectively, representing ≈13% of diseased leaf area in C×D. In three cases, significant dominance effects were detected (A×B^I and A×B^{II}: chromosome 1; C×D: chromosome 2). In each population, alleles that increased rust resistance were contributed by both parents.

In total, 11 QTL displayed significant ($P < 0.05$) QTL×E interactions (Table 2). For A×B^I, most of the QTL detected across the three common environments were significant in only one to three of four environments each (Table 3). No QTL was detected for A×B^I in Krozingen 1993. The signs of additive effects of QTL common across environments in A×B^I in most cases were identical. Significant ($P < 0.05$) digenic epistatic interactions of type dominance and dominance ($\hat{d}d$) were detected in C×D between both QTL at chromosome 3 ($\hat{d}d = -2.35$) and QTL located on chromosomes 3 (Bin region 3.04) and 8 ($\hat{d}d = -1.58$). Significant ($P < 0.05$) digenic epistatic interactions of type additive and dominance ($\hat{a}d$) were detected in A×B^I between the QTL at chromosomes 3 and 1 (Bin region 1.03) ($\hat{a}d = 0.39$) and between the QTL on chromosomes 8 and 3 ($\hat{a}d = 0.85$).

TABLE 2. Parameters associated with putative quantitative trait loci (QTL) significantly affecting common rust ratings (1 to 9 scale) in F_n families of maize populations (A×B^I, A×B^{II}, A×C, C×D) across three common environments^a

Bin ^b	A×B ^I				A×B ^{II}				A×C				C×D			
	<i>a</i> ^c	<i>d</i> ^c	<i>R</i> ^{2d}	Env. ^e	<i>a</i>	<i>d</i>	<i>R</i> ²	Env.	<i>a</i>	<i>d</i>	<i>R</i> ²	Env.	<i>a</i>	<i>d</i>	<i>R</i> ²	Env.
1.03	-0.13*	0.01	8.7	H**												
1.05-06																
1.08-10	0.08	0.57*	5.5	E	0.06	-0.87*	13.1	K	-0.15*	0.07	8.6	H	-0.20**	0.06	17.9	E,H**
2.05-06									0.19*	0.27	11.8	E,K				
2.08	-0.10*	0.12	5.1						-0.29*	0.09	25.5	E,H,K*	0.23**	-0.12	14.5	E,H,K
2.09	-0.14*	-0.03	9.0	E,H**												
3.04	0.17*	-0.12	5.6	H									0.17**	0.36*	12.4	E
3.07													0.25**	-0.18	15.3	E,H**
4.03-04													0.19**	-0.22	13.9	E,K**
4.08									-0.23*	-0.08	10.7		0.16**	-0.42	11.3	E,H
5.01													0.18**	0.46	9.8	
5.04-05	-0.14*	-0.07	7.7	E,H**									0.24**	-0.26	19.6	E,H**
6.04													-0.32**	-0.14	9.4	E
6.07													0.19**	-0.18	8.7	E
7.01-02					0.30*	-0.05	14.0	E,H**	-0.25*	0.09	23.6	E,H,K	0.07	-0.58	8.8	E,H
7.04	-0.15*	0.02	6.9	E,H*												
8.02-05	0.08*	0.08	4.2	E,H					0.22*	0.30	12.4	E,H	-0.20**	-0.14	15.9	E
9.01													-0.16**	0.08	9.2	E
9.06-07					0.20*	-0.32	10.7	E	0.13*	-0.12	10.0	E,H				
10.05-06	-0.12*	0.04	4.3	E,H	-0.37*	0.51	10.8	E,H**								
Total	1.07	1.13	40.6		0.93	1.75	33.4		1.46	1.02	53.4		2.56	3.20	70.9	

^a Eckartsweier 1993, Hochburg 1993, and Krozingen 1993.

^b Chromosomes dissected into Bin regions and QTL positions based on the marker linkage maps published in Lübberstedt et al. (22) and the Maize Genetics Newsletter (vol. 70, pp. 123-132).

^c *a* = additive gene effect; *d* = dominance effect of QTL. Values significant at $P < 0.05$ are indicated by *. Positive additive effects were assigned to parent A (A×B^I, A×B^{II}, A×C) or C (C×D).

^d Percent phenotypic variance explained by QTL.

^e QTL-environment (Env.) interactions significant at $P < 0.05$ and 0.01 are indicated by * and **, respectively. Letters E, H, and K indicate detection of the QTL in Eckartsweier 1993, Hochburg 1993, and Krozingen 1993, respectively.

TABLE 3. Quantitative trait loci (QTL) that significantly affect common rust ratings in maize population A×B^I in four German environments (Eckartsweier 1993, Hochburg 1993, Zell 1993, Eckartsweier 1994) and across three environments (series) in common with populations A×B^{II}, A×C, and C×D

Chrom. ^a	Series			Eckartsweier			Hochburg			Zell			Eckartsweier		
	Pos. ^a	<i>a</i> ^b	<i>d</i> ^b	Pos.	<i>a</i>	<i>d</i>	Pos.	<i>a</i>	<i>d</i>	Pos.	<i>a</i>	<i>d</i>	Pos.	<i>a</i>	<i>d</i>
1	68	-0.13*	0.01	26	0.06	0.64*	66	-0.20*	-0.03	66	-0.17*	0.05	68	-0.19*	0.18
1							166	0.12*	0.25						
1	204	0.08	0.57*	204	0.01	0.69							216	0.26*	1.40*
2							86	-0.10*	0.33*				78	-0.25*	0.56*
2	146	-0.10*	0.12							136	-0.24*	-0.03			
2	178	-0.14*	-0.03	172	-0.32*	0.26	170	-0.24*	0.07				180	-0.44*	-0.06
3	76	0.17*	-0.12										58	0.57*	-0.75
3				104	0.13*	-0.22	102	0.14*	-0.04	122	0.13	-0.50*			
4										66	-0.27*	0.18	58	-0.25*	-0.11
5	82	-0.14*	-0.07	86	-0.16*	0.04	100	-0.21*	-0.19	102	-0.19*	-0.40*	96	-0.28*	0.43
6				124	0.16*	-0.12	126	0.16*	-0.04				136	0.32*	0.00
7				38	-0.19*	0.25									
7	62	-0.15*	0.02				84	-0.21*	-0.07	62	-0.20*	0.14			
8	68	0.08*	0.08	64	0.09*	0.00	62	0.13*	0.04						
8							140	-0.17*	-0.23				132	-0.24	-0.01
9							0	0.13*	0.14						
10	112	-0.12*	0.04	94	-0.20*	0.08	96	-0.19*	-0.30						

^a Position (Pos.) on chromosomes (Chrom.) 1 to 10 from the terminus (in centimorgans) of the short arm according to the linkage map published by Schön et al. (32).

^b *a* = additive gene effect; *d* = dominance effect of QTL. Values significant at $P < 0.05$ are indicated by *.

None of the QTL were consistently detected across all four populations. Of the four QTL detected in $A \times B^{\text{II}}$, two were identified in $A \times B^{\text{I}}$. Two QTL were common between $A \times B^{\text{I}}$ and $A \times C$, four were common between $A \times B^{\text{I}}$ and $C \times D$, and five were common between $A \times C$ and $C \times D$ (Table 2). The signs of additive effects of QTL in most cases were consistent across populations (Table 2).

DISCUSSION

There are no reports on the economic significance of common rust in maize in Central Europe, probably because severe rust incidence is limited to a few years and areas with conditions favorable to fungal development. However, because of the unknown ability of *P. sorghi* to adapt to cooler climates and the need to prevent high incidence in years favorable to common rust, increasing the level of resistance in adapted germ plasm is important agronomically. Relatively high natural rust incidence in 1993, especially in the Rhine Valley, allowed us to perform rust ratings on four flint populations of F_n lines grown to evaluate other agronomic traits.

In most QTL studies of partial resistance, highly resistant parents have been crossed with susceptible parents to increase the chance of QTL detection (20,38). In contrast, selection of the four flint inbred lines used in this study was based on agronomic performance rather than rust resistance. According to our rust ratings method, parent lines C and D had intermediate scores, whereas lines A and B had slightly increased susceptibility. In crosses $A \times B^{\text{I}}$ and $C \times D$, differences between parent lines were not significant (Table 1). Nevertheless, intermediate ($\hat{h}^2 > 0.64$) to high broad-sense heritabilities for all four F_n populations indicate their usefulness for our QTL-mapping study. Partial rust resistance was also highly heritable in previous studies (17,28). Moreover, significant transgressive segregation was found in all four populations, indicating the pres-

ence of positive alleles in each parent line. For a class width of 0.3 units (Fig. 1), rust ratings were approximately normally distributed in all four F_n populations, which is comparable to earlier reports on partial resistance to *P. sorghi* (17).

Previous estimates of the number of genes involved in partial resistance against *P. sorghi* varied from two (17) and unknown high numbers (28), depending on the materials investigated. The estimate in the first study was obtained by the Castle-Wright formula (3), which tends to underestimate true gene numbers (39). In a review on QTL mapping of quantitative disease resistance in plants, in most cases 2 to 10 effective factors were detected (38), suggesting the number of loci involved in quantitative resistance generally is lower than for other agronomically important traits, such as grain or forage yield. In contrast, our study detected a minimum of 20 QTL involved in partial resistance against common rust across all four populations, which is similar to the number detected for forage yield and quality traits with test-cross progenies of the same populations (22–24). A comparably complex inheritance of quantitative resistance was reported by Bubeck et al. (2) for gray leaf spot in maize. High gene numbers for at least some partial resistances might be due to complex developmental (mature-plant resistance), morphological (growth habit; leaf surface, shape, and posture; and stomatal behavior), and chemical (cell content of acids and sugars; concentration of chitinases and glucanases; and lignification of cell wall) characters rather than race-specific interactions with a pathogen (14,26).

Classic studies on quantitative resistance, including slow rusting resistance, indicated the prevalence of additive or partial dominant gene action. Moderate heterosis was found in the direction of resistance as well as susceptibility (8). In agreement with previous studies (17,28), most QTL displayed additive or partially dominant gene action (Table 2), which is in contrast to the dominant gene

Fig. 1. Mean rust ratings (1 to 9 scale) of F_n lines of flint maize populations $A \times B^{\text{I}}$, $A \times B^{\text{II}}$, $A \times C$, and $C \times D$ evaluated in three common environments. The overall mean of F_n lines ($\bar{F}_n$) is indicated by the solid line. Arrows indicate means of the parent lines.

action of race-specific resistance genes against *P. sorghi* (14). Approximately half of the partial to overdominant alleles caused increased susceptibility; the other half increased resistance. However, especially for the $F_{3:4}$ ($A \times C$ and $C \times D$) and $F_{4:5}$ ($A \times B^I$) populations, the statistical power of the test for dominance effects was low. Consequently, only three QTL displayed significant dominance effects. Estimates of dominance effects and the degree of dominance might be biased due to the limited database, however. Only 25% of the parental genotypes of $F_{3:4}$ and 12.5% of $F_{4:5}$ families were expected to be heterozygous for a given marker locus and informative with regard to estimating dominance effects. In addition, dominance effects estimated in selfing progenies of individuals used for genotyping need to be doubled. This might explain the opposite signs of dominance effects of an overdominant QTL detected in both $A \times B$ populations on chromosome 1 (Table 2) and the prevalence of overdominant QTL in $A \times B^I$.

In this study, QTL for resistance to *P. sorghi* displayed moderate to poor consistency across maize populations. Similar findings were reported for complex forage maize traits (22) and resistance to gray leaf spot (2). Possible reasons for inconsistency in QTL across populations include (i) different sets of polymorphic and detectable QTL in each of the three crosses; (ii) low QTL detection ability; (iii) QTL $\times$ E interactions; and (iv) epistasis. The best QTL agreement was found between $A \times C$ and $C \times D$, with 5 common QTL of 7 ($A \times C$) and 13 ($C \times D$) QTL identified in each population, probably due to the closer pedigree relationship of lines A and D, with a coancestry coefficient (f) (25) of 0.13 compared with lines B and C ($f = 0.06$); lines A and D are unrelated to lines B and C. As a result, at least some of the QTL alleles common between $A \times C$ and $C \times D$ might be identical by descent, while others might be common only because of similar chromosomal positions, such as the common QTL on chromosome 1 with opposite signs of additive effects for the allele of line C in $A \times C$ and $C \times D$ (Table 2). Fewer QTL were common between unrelated crosses $A \times B$ and $C \times D$. Presumably, a large number of QTL are involved in partial resistance against common rust with specific detectable and polymorphic subsets in individual crosses.

In small populations of ≈ 100 individuals or families, the level of QTL detection, even for traits with high heritabilities, was low, and estimates of the proportion of σ_p^2 explained by detected QTL might have been severely inflated (34). For a trait with $h^2 = 0.7$ and a population of 100 F_2 individuals, the level of QTL detection that explained 6% of σ_p^2 was 31%. Therefore, simultaneous detection of the same QTL in two populations of 100 F_2 individuals was only $\approx 10\%$. Because most QTL detected in our study explained $< 15\%$ of σ_p^2 , consistency of QTL across populations might be severely impaired by the low level of QTL detection in small populations. In addition to sampling effects, this also might explain inconsistencies between the two $A \times B$ populations studied.

Genotype-environment and QTL $\times$ E interactions frequently are associated with quantitative resistance (2,5,8). Although only three environments in 1993 were used in our study, all four populations displayed significant σ_{ge}^2 estimates, and one ($A \times C$) to four ($A \times B^I$ and $C \times D$) QTL exhibited significant QTL $\times$ E interactions. However, QTL $\times$ E interactions were unrelated to reversed signs of additive effects in individual environments (Table 3). In contrast, only a few QTL showed QTL $\times$ E interactions in a study of forage traits with test-cross progenies of the same populations evaluated in five environments (22). One explanation for an increased level of QTL $\times$ E interactions for partial resistance against *P. sorghi* compared with forage traits might be race-specific expression of QTL and, as a result, characteristic QTL patterns in each environment, depending on the prevalent rust races. Although partial resistance is often referred to as race-nonspecific, QTL studies of partial resistance against *Phytophthora infestans* (21) uncovered race-specific reactions. Investigations on race specificity of partial resistance to *P. sorghi* in maize are lacking. Alternatively, different growing conditions and developmental stages of maize populations for rust

ratings in individual environments might affect the expression of QTL involved in developmental, morphological, and chemical characters indirectly affecting partial resistance to *P. sorghi*.

Evidence for epistatic interactions in partial resistance of maize to *P. sorghi* was found in one of three crosses investigated by generation means analysis (28). Dissection of partial resistance by QTL mapping revealed digenic epistatic interactions for QTL involved in resistance to rice blast (37) but not for other disease resistance (38). In our study, four cases of significant *ad* and *dd* interactions were detected in $A \times B^I$ and $C \times D$. The comparably large estimate for the absolute *dd* effect (2.35) obtained in $C \times D$ might be biased due to the small number of double heterozygotes in a $F_{3:4}$ population of this sample size.

QTL for partial resistances might be defeated or allelic versions of qualitative resistance genes (30). This hypothesis is supported by the similar map positions of QTL and qualitative resistance genes acting against *Phytophthora infestans* in potato (21), *Pyricularia oryzae* in rice (37), and *Setosphaeria turcica* and *Puccinia polysora* in maize (5,7,13). In contrast, QTL for resistance against common rust detected in this study were not preferentially located in map positions close to *Rp* loci on chromosomes 3L, 4S, 6L, or 10S but were distributed over all 10 maize chromosomes. Only one common QTL in $A \times B^I$ and $C \times D$ on chromosome 3 and another QTL identified in $A \times C$ and $C \times D$ on chromosome 4 might be related to *Rp* loci. In conclusion, different biological mechanisms appear to be involved in quantitative versus qualitative resistance against *P. sorghi* in maize.

Partial resistance to common rust was successfully improved by S1 and S2 recurrent selection and pedigree breeding in sweet corn (9,28). Improvement of rust resistance in European flint maize material by conventional selection methods also seems promising, because significant genetic variation and transgressive segregation were found in each of the four flint populations investigated. Evaluation of line performance with a limited number of plants per family should be adequate because of the high heritabilities and preponderance of additive or partially dominant gene action.

Marker-assisted selection might be useful, if used with the same cross used for QTL mapping. QTL mapping may aid breeders in (i) identifying QTL with large effects consistently expressed across environments; (ii) detecting QTL with dominant alleles, simplifying hybrid breeding; (iii) performing indirect selection in the absence of natural infection without the need for artificial infection; and (iv) selecting in both sexes prior to flowering. In addition, QTL for partial rust resistance might be incorporated into the strategy of constructing compound rust resistance genes (15) using molecular markers. This strategy was proposed for combination of two or more closely linked *Rp* loci in coupling phase, which can be manipulated as single genes in breeding programs. However, the relative efficiency of marker-assisted selection compared with conventional phenotypic selection (19) was < 0.91 in all four populations. The relative efficiency (*RE*) of a single cycle of selection, given the same selection intensity for both selection schemes, was estimated by $RE = (p/h^2)^{1/2}$, where p is the proportion of σ_p^2 explained by the putative QTL (19). Hence, conventional selection should be superior to marker-assisted selection, if environments with high rust incidence are available. Furthermore, because of the poor consistency of QTL across populations, QTL information gathered in one cross appears to be of little use for marker-assisted selection in other crosses.

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