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Microsatellite markers identify three additional quantitative trait loci for resistance to soybean sudden-death syndrome (SDS) in Essex × Forrest RILs

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Abstract Resistance to the sudden-death syndrome (SDS) of soybean (*Glycine max* L. Merr.), caused by *Fusarium solani* f. sp. *glycines*, is controlled by a number of quantitatively inherited loci (QTLs). Forrest showed a strong field resistance to SDS while Essex is susceptible to SDS. A population of 100 recombinant inbred lines (RILs) derived from a cross of Essex × Forrest was used to map the loci effecting resistance to SDS using phenotypic data obtained from six environments. Six loci involved in resistance to SDS were identified in this population. Four of the QTLs identified by BARC-Satt214 (P = 0.0001, R^2= 24.1%), BARC-Satt309 (P = 0.0001, R^2 = 16.3), BARC-Satt570 (P = 0.0001, R^2 = 19.2%) and a random amplified polymorphic DNA (RAPD) marker $OEO2_{1000}$ (P = 0.0031, R^2=12.6) were located on linkage group (LG) G (Satt309 and $OEO2_{1000}$ were previously reported). Jointly the four QTLs on LG G explained 50% of the variation in SDS disease incidence (DI). All the QTLs on LG G derived the beneficial allele from Forrest. Two QTLs, BARC-Satt371 (P = 0.0019, R^2 = 12%) on LG C2 (previously reported) and BARC-Satt354 (P = 0.0015, R^2 = 11.5%) on LG I, derived their beneficial allele from Essex and jointly explained about 40% of the variation in SDS DI. Two-way and multi-way interactions indicated that gene action was additive among the loci underlying resistance to SDS. These results suggest that cultivars with durable resistance to SDS can be developed via gene pyramiding.

Keywords Soybean sudden-death syndrome (SDS) · Quantitative trait loci · Microsatellite markers · Recombinant inbred lines

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Introduction

The sudden-death syndrome (SDS) of soybean [*Glycine max* (L.,) Merr.] is a soil-borne disease caused by *Fusarium solani* (Mart.) Sacc. f. sp. *glycines* (Burk.) Snyd. & Hans., and results in severe crop losses in the United States (Gibson et al. 1994; Wrather et al. 1996). Classical genetic studies have predicted a heterogeneity of resistance to SDS above 90% and a few major loci underlying resistance to SDS (Mathews et al. 1991; Njiti et al. 1996). A few major quantitative trait loci (QTLs) determine partial resistance to SDS (Hnetkovsky et al. 1996; Chang et al 1997). Meksem et al. (1999) used microsatellite markers to separate one QTL on LG G into a cluster of genes underlying resistance to SDS, *F. solani* root infection and soybean cyst nematode (SCN), which behave as a qualitative locus during fine mapping in NILs (Meksem et al. 1999).

The amount of variation explained by four previously identified QTLs was less than the heritability of the trait. In addition, all of the linkage groups were not studied due to the lack of suitable markers and polymorphisms between cultivars. With the availability of integrated microsatellite maps of soybean (Cregan et al. 1999a), it is possible to study a greater percentage of the linkage groups for the presence of all possible QTLs responsible for resistance to SDS. Moreover, the interaction among all known QTLs for resistance to SDS is not fully understood. The objective of the present study was to use microsatellite markers to identify all the loci underlying resistance to SDS in Essex × Forrest recombinant inbred lines (RILs).

Materials and methods

Plant material

A cross of ''Essex'' (Smith and Camper 1973) × ''Forrest'' (Hartwig and Epps 1973) was used to generate an F_5 derived population of 100 recombinant inbred lines (RILs) as described by Hnetkovsky et al. (1996) and Chang et al. (1997). The RILs have

been advanced to the $F_{5:13}$ generation from never less than 300 plants per RIL per generation. Essex is susceptible and Forrest is resistant to SDS (Hnetkovsky et al. 1996; Chang et al. 1997).

Field study

The lines were evaluated for SDS resistance to leaf scorch in six environments over a period of 4 years (Hnetkovsky et al. 1996; Chang et al. 1997). A partially balanced simple-lattice design was used at each environment (Gomez and Gomez 1984). There were 100 lines, eight repetitions of each parent, and four checks, totaling 120 entries comprising a 11×11 square lattice. Each plot consisted of two rows 0.75-m apart and 3-m long, with about 17 plants per meter.

SDS disease scoring

Disease incidence (DI) and disease severity (DS) of SDS were rated weekly as described by Njiti et al. (1996). The last score before and the first score after the R6 (full pod) growth stage (Fehr et al. 1971) were used to standardize DI and DS to the R6 growth stage (Njiti et al. 1996). The trait data were normalized by arcsine transformation to increase the variability among the genotypes with low scores (resistant). Therefore, variability was equalized across the progeny population.

DNA isolation

RILs were grown in the greenhouse; 3 g of leaves were collected from 5 to 6 2-week-old seedlings and immediately frozen in liquid nitrogen. The leaves were ground in liquid nitrogen into a very fine powder and DNA was extracted after Paterson et al. (1993). DNA concentration was measured by a flourometer and diluted to 15 ng/µl for further use in PCR reactions.

Microsatellite amplifications

Microsatellite markers from all 20 linkage groups were selected at 25-cM intervals from the soybean genetic map (Cregan et al. 1999a). The primer pairs were purchased from Research Genetics, Inc., Huntsville, Ala., USA. Amplifications were carried out in PE 9600 thermal cycler as described by Akkaya et al. (1995). Two negative controls (with no template DNA), along with the two parents DNA as positive controls, were run in all the amplifications.

Loci associated with quantitative resistance

To detect genomic regions associated with SDS resistance, the recombinant inbred lines were classified as Essex (E) type or Forrest (F) type for each marker. Marker data were compared with SDS DI and DS scores by a one-way analysis of variance (ANOVA) performed with SAS (SAS Institute Inc., Cary, N.C.; Wang et al. 1994). The probability of association of each marker with each trait was determined and a significant association was declared if $P \leq 0.005$, to maximize the detection of associations (Lander and Botstein 1989).

Interactions between quantitative resistance loci

Pair-wise comparisons among markers with significant association to SDS used a two-way ANOVA to detect additive and non-additive interactions between the unlinked QTLs (Lark et al. 1995; Chang et al. 1997). Non-additive interactions between markers which were significantly associated with SDS response were excluded when $P > 0.05$. All markers associated with SDS were analyzed by multi-way ANOVA to estimate joint heritabilities for traits associated with multiple QTLs and to evaluate the effects of gene pyramiding. Joint heritability was determined from the R^2 term for the joint model in a multi-way ANOVA.

Mapping quantitative resistance loci

Mapmaker-EXP 3.0 (Lander et al. 1987) was used to calculate map distances as centimorgans (cM) in Haldane units between linked markers and to construct a linkage map (heterozygous lines were excluded). The recombinant inbred line (ri-self) genetic model was used. The $\log_{10}$ of the odds ratio (LOD) for grouping markers was set at 2.0, the maximum distance was 30 cM. Conflicts were resolved in favor of the highest LOD score after checking the raw data for errors. Marker order within groups was determined by comparing the likelihood of many map orders. A maximum-likelihood map was computed with error detection. Groups were assigned to linkage groups by anchored microsatellite markers (Shoemaker and Specht 1995; Cregan et al. 1999a).

The map and disease data were simultaneously analyzed with Mapmaker/QTL 1.1 (Paterson et al. 1988) using the F_2-backcross genetic model for trait segregation (Webb et al. 1995; Chang et al. 1996, 1997; Hnetkovsky et al. 1996). Quantitative trait loci were inferred when LOD scores exceeded 2.0 at some point in each interval since this was found empirically to be equivalent to a single marker, $P < 0.005$, the criterion used in one-way ANOVA. The positions of the QTLs were inferred from the interval peak LOD score.

The microsatellite markers used in this study have earlier been mapped (Cregan et al. 1999a) in other soybean populations. Therefore, markers were anchored on the linkage groups on the basis of their known locations. This is important for the comparison of the known QTL locations from other populations (Njiti et al. 2000) as well in the same population (Chang et al. 1997) with dominant markers.

Results

Polymorphism of SSR markers in Essex and Forrest and linkage distribution

A total of 400 markers covering all the 20 linkage groups of soybean were tested in Essex and Forrest DNA to identify polymorphic markers that could be mapped in the population. One hundred and thirty three SSRs were polymorphic between the two parents which were then scored and mapped in the 100-RIL population. One hundred and seven markers were found to be linked representing 18 known linkage groups. On average six markers were placed on each linkage group. The actual number of markers ranges from three, as in linkage group C1, D1a and J, to 16, as on G. The total map coverage excluding unlinked markers was 2823.1 cM with an average of 26.4 cM between loci. This coverage is comparable to the known recombination distance of about 3000 cM encompassing the 20 linkage groups of soybean (Cregan et al. 1999a).

Markers associated with SDS

Six independent genomic regions associated with SDS were identified on the basis of a one-way analysis of variance (ANOVA) at $P \leq 0.005$; four on linkage group G, one on C2 and one on I (Table 1, Fig. 1). Three microsatellite markers, BARC-Satt570, BARC-Satt309 and

Table 1 DNA markers associated with the QTLs underlying resistance to SDS in the Essex × Forrest population. L.G. = linkage group

Marker	L.G.	Trait	R^2	$P > F$	LOD[a]	QTL var.[b]	Mean	
							Essex	Forrest
BARC-Satt214	G	DI	24.1	0.0001	5.56	23.6	59.8 ± 3.4	36.3 ± 3.0
BARC-Satt309	G	DI	16.3	0.0001	3.96	16.9	57.9 ± 3.1	38.8 ± 3.5
BARC-Satt570	G	DI	19.2	0.0001	4.25	18.1	60.0 ± 3.0	39.0 ± 3.5
$OEO2_{1000}$	G	DI	12.6	0.0031	2.36	15.3	55.0 ± 3.7	39.0 ± 3.6
BARC-Satt371	C2	DI	12.0	0.0019	2.32	12.4	39.2 ± 4.4	56.1 ± 3.1
BARC-Satt354	I	DI	11.5	0.0015	2.20	12.0	41.3 ± 3.2	57.0 ± 3.4

[a] LOD is an indicative of the probability based on the presence of a locus, not on the absence, LOD threshold = 2.0

[b] Amount of variability in the trait explained by the marker loci based on MapMaker QTL

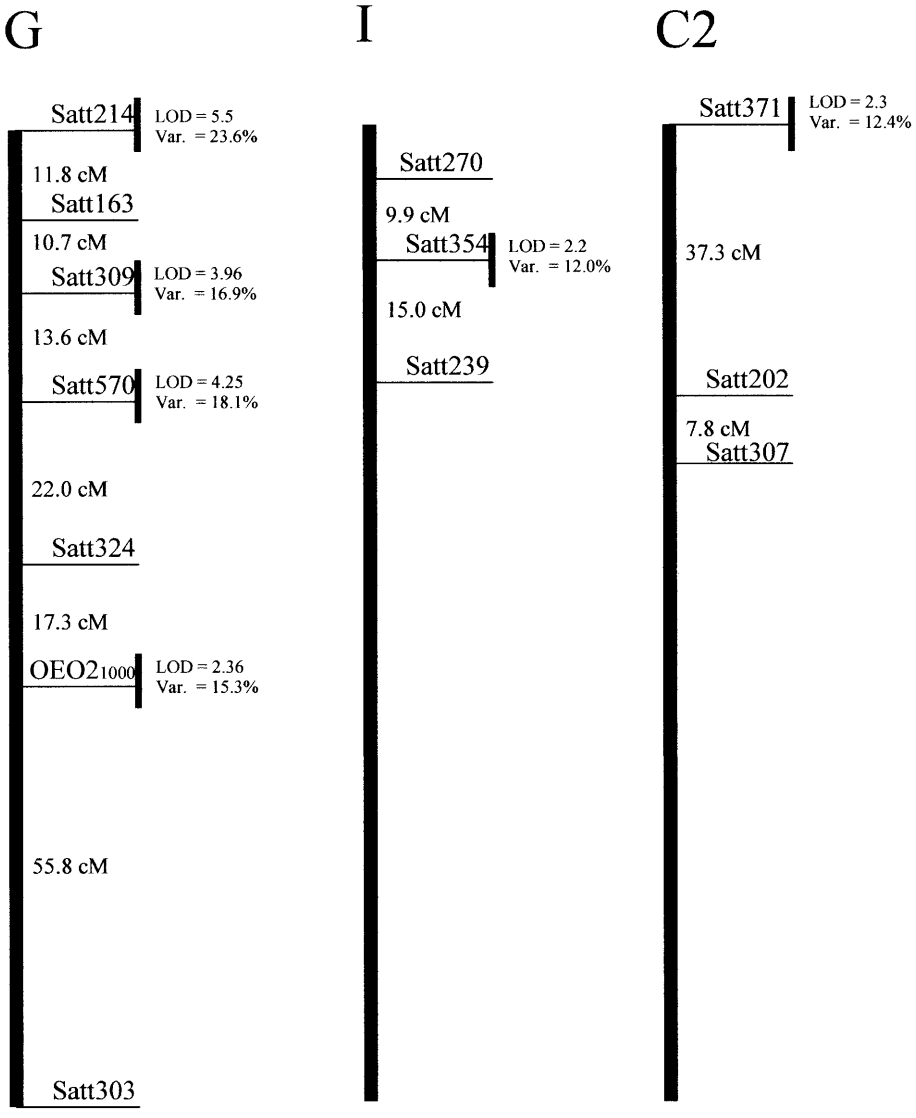

Fig. 1 Location of DNA markers and the six QTLs underlying resistance to the sudden-death syndrome (SDS) of soybean. Markers were assigned to the linkage groups G, C2 and I on the basis of the soybean genetic linkage map (Cregan et al. 1999a). The position of the QTLs is estimated on the basis of interval mapping using MAPMAKER/QTL 1.1. The QTL LOD score is the peak-LOD score of the particular marker associated with the QTLs

BARC-Satt214, and a random amplified polymorphic DNA marker, $OEO2_{1000}$, on linkage-group G, identified separate QTLs for resistance to SDS disease incidence.

The region on LG G identified by BARC-Satt214 was significantly ($P = 0.0001$ and $R^2 = 24.1\%$) associated with SDS DI and derived the beneficial allele from Forrest. The interval between Satt214 and Satt163 (Fig. 1) was 11.8 cM and the QTL peak-LOD score was 5.55. This peak was right at the marker Satt214 and explained 23.6% of the total variation in DI. This was a novel region.

A second region identified by BARC-Satt309 was significantly ($P = 0.0001$, $R^2 = 16.3$) associated with

Table 2 Interaction among various combination of SDS QTLs from LG G, C2 and I calculated by four-way analysis of variance in E × F RIL population. **a** Interaction of the top-half of the QTL on LG G with LG C2 and the I QTL, **b** bottom-half of the LG G QTL with LG C2 and I, **c** new QTLs identified on LG G with LG C2 and I. A = Essex allele, B = Forrest allele

a

Sr. no.	SATT214	SATT309	SATT371	SATT354	# of RILs	Mean DI ± SEM[a]
1	A	A	A	A	5	45.0 ± 8.8
2	A	A	A	B	2	50.8 ± 17.5
3	A	A	B	A	10	61.9 ± 5.5
4	A	A	B	B	7	81.0 ± 4.3
5	A	B	A	A	1	7.8
6	A	B	A	B	2	46.6 ±36.8
7	A	B	B	A	1	51.7
8	A	B	B	B	2	65.9 ± 11.3
9	B	A	A	A	1	40.8
10	B	A	A	B	1	45.0
11	B	A	B	B	1	19.0
12	B	B	A	A	9	26.4 ± 8.0
13	B	B	A	B	3	41.0 ± 4.8
14	B	B	B	A	8	40.9 ± 4.8
15	B	B	B	B	7	43.8 ± 6.8

b

Sr. no.	SATT570	OE02	SATT371	SATT354	# of RILs	Mean DI ± SEM[a]
1	A	A	A	A	1	70.1
2	A	A	A	B	1	6.7
3	A	A	B	A	3	47.9 ± 5.5
4	A	A	B	B	4	51.1 ± 12.6
5	A	B	A	A	4	35.6 ± 10.6
6	A	B	A	B	2	45.3 ± 12.0
7	A	B	B	A	3	68.0 ± 7.7
8	A	B	B	B	1	62.6
9	B	A	A	A	1	69.5
10	B	A	A	B	1	83.4
11	B	A	B	A	5	51.5 ± 5.8
12	B	A	B	B	4	67.3 ± 8.8
13	B	B	A	A	6	12.9 ± 3.1
14	B	B	A	B	2	25.5 ± 15.7
15	B	B	B	A	3	36.6 ± 9.8
16	B	B	B	B	4	44.6 ± 5.0

c

Sr. no.	Satt214	Satt570	Satt354	A Satt371	# of RILs	MDI ± SEM[a]
1	A	A	A	A	4	48.6 ± 10.5
2	A	A	A	B	8	67.8 ± 3.8
3	A	A	B	A	2	50.8 ± 17.5
4	**A**	**A**	**B**	**B**	**7**	**81.7 ± 4.4**
5	A	B	A	A	1	7.8
6	A	B	A	B	3	42.9 ± 10.6
7	A	B	B	A	2	46.6 ± 36.8
8	A	B	B	B	3	71.8 ± 8.8
9	B	A	A	A	2	28.2 ± 12.6
10	B	A	A	B	1	37.7
11	B	A	B	A	2	35.0 ± 10.0
12	B	A	B	B	3	43.9 ± 13.0
13	**B**	**B**	**A**	**A**	**8**	**27.8 ± 8.9**
14	B	B	A	B	7	46.7 ± 5.0
15	B	B	B	A	3	41.0 ± 4.8
16	B	B	B	B	6	40.8 ± 7.2

[a] The SEM could not be calculated on groups that had only one line represented in the mean

SDS DI and was located 22.5 cM from Satt214 (Fig. 1). This region represents a second QTL for resistance to SDS. The interval (8.5 cM) between Satt275 and Satt610 (markers around Satt309, data not shown in Fig. 1) containing the QTL had a peak-LOD score of 3.96 and explained about 16.9% of the total variation in DI.

The third QTL on LG. G was identified by BARC-Satt570 (13.6 cM from Satt309) and was significantly (P = 0.0001, R^2 = 19.2%) associated with SDS DI. The interval (9.2 cM) between Satt122 and Satt130 (markers around Satt570, data not shown in Fig. 1) containing the QTL had a peak-LOD score of 4.4 and explained 19.7% of the total variation in SDS DI.

The fourth region on LG G associated with SDS had earlier been identified by RAPD marker $OEO2_{1000}$ and was confirmed by the same marker in this analysis. Three microsatellite markers were mapped around the OEO2 region. Satt130 and Satt324 spanned the 39.3 cM

between Satt570 and OEO2, while Satt303 was 55.8 cM below $OEO2_{1000}$ (Fig. 1). The QTL identified by $OEO2_{1000}$ ($P = 0.0031$, $R^2 = 12.6$) had a peak-LOD score of 2.36 and explained 15.3% of the trait variation. All the QTLs identified on LG G derived the beneficial allele from Forrest.

A new QTL for SDS resistance, identified on LG I by Satt354, was significantly ($P = 0.0015$, $R^2 = 11.5\%$) associated with SDS DI and derived the beneficial allele from Essex. The adjacent markers Satt270 (9.9 cM) and Satt239 (15 cM) were not significantly associated with DI. The interval containing this QTL had a peak-LOD score of 2.2 and explained 12% of the total variation in SDS DI.

A region on LG. C2 identified by Satt371 was also significantly ($P = 0.0019$, $R^2 = 12\%$) associated with SDS DI and derived the beneficial allele from Essex, the SDS susceptible parent. The closest polymorphic marker to this region was BARC-Satt202 (37.3 cM) and was not associated with SDS DI.

Interaction among QTLs conferring resistance to SDS

A multiway analysis of variance involving all the loci for resistance to SDS in this population indicated that these six loci explained about 91% of the total variation in SDS DI. A four-way ANOVA involving all the loci for SDS resistance on LG G indicated that linkage group G alone acounted for about 50% of the total variation in SDS DI. This implies that the other two loci accounted for about 40% of the total variation in SDS DI.

A two-way analysis of variance revealed no significant ($P \leq 0.01$) interaction between pairs of markers that were individually associated with SDS DI except for one pair (Satt371 and $OEO2_{1000}$) that had a significant interaction.

Due to the limited number of lines, gene action among the loci was determined by three separate four way ANOVAs (Table 2a,b,c). Two QTLs on the top of LG G (Satt214 and Satt309) when combined with QTLs on LG I (Satt354) and LG C2 (Satt371) showed no significant multi-way interaction. When the two QTLs on the bottom of LG G (Satt570 and $OEO2_{1000}$) were combined with the QTLs on LG I and C2, there was no significant four-way interaction. All interaction analysis indicated that lines that accumulated the beneficial alleles from all loci were most-resistant to SDS, and lines that accumulated none of the beneficial allele were most-susceptible to SDS. Lines that accumulated some beneficial alleles had an intermediate response to SDS.

Discussion

The low frequency of polymorphisms in this population was expected due to the close relatedness of the two parents as described earlier (Allen and Bhardwaj 1987; Keim et al. 1992; Skorupska et al. 1994; Hnetkovsky et al. 1996), which enforces the need for further SSR development within gaps in the map. The large number of markers on LG G was due to earlier saturation mapping of SDS and soybean cyst nematode (SCN) resistance loci (Meksem et al. 1999).

The present study identified six QTLs for resistance to SDS in the E × F RIL population. Four QTLs were mapped to linkage group G. Two of these QTLs (Satt214 and Satt570) were new discoveries while the rest mapped to regions that were previously identified as containing QTLs for resistance to SDS (Hentkovsky et al. 1996; Chang et al. 1997) and more-likely confirmed the presence of these QTLs in the population. Satt309 has previously been identified as 1G, and $OEO2_{1000}$ identified as 2G. Satt214 has also been identified in the Pyrimad × Douglas population (Nijiti et al. 2000). One locus thought to contain a QTL on LG N (Hentkovsky et al. 1996) was shown not to be related to SDS resistance.

The six QTLs identified in this population appeared to represent a complete picture of the loci underlying resistance to SDS in the E × F RILs. In this population, the heritability of SDS DI has been estimated to be above 90% on a line mean basis (Hentkovsky et al. 1996; Chang et al. 1997). The QTLs for resistance from all loci jointly explained about the same (91%) variation in SDS DI. Based on this observation we conclude that all possible QTLs have been identified in this population. We note that the amount of variation explained by these QTLs may be over-estimated due to the small number of lines that were used in the multi-way analysis of variance. Therefore, it is still possible that with increased saturation of the microsatellite map and with the use of more RILs and NILs, additional QTLs for resistance to SDS may be detected or existing QTLs can be divided into discrete loci.

There was a significant interaction between Satt371 and $OEO2_{1000}$ which resulted in the explanation of an additional amount of variation in SDS DI. However, this was probably due to the fact that these markers derived the beneficial allele from opposite parents. The most-resistant lines were those with the Forrest allele at $OEO2_{1000}$ and the Essex allele at Satt371. Since each of these alleles by themselves contributed to resistance to SDS, it is clear that the gene action observed here was predominantly additive rather than being due to an epistatic interaction. All the other interactions behaved primarily in an additive manner, with the level of resistance increasing with the increased accumulation of benificial alleles. This type of gene action was also observed in the Pyramid × Douglas RIL population (Njiti et al. 2000). These results suggest that cultivars with strong resistance to SDS can be developed by gene-pyramiding via back crossing, marker-assisted selection, plant transformation etc. The concentration of SDS resistance QTLs on LG G close to a major disease resistance cluster of genes, including *rfs1* (Prabhu et al. 1999) and *rhg1* (Meksem et al. 1999, Cregan et al. 1999b), suggests that resistance to other diseases can be improved simultaneously with resistance to SDS. Marker-assisted selection for LG G alone has a more than 50% chance of identifying lines with strong resistance to SDS, whereas selection for SCN resistance would recover approximately 20% resistance with consistency.

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Note added in proof: BARC-Satt--- and Satt--- in text, tables and figures represent the same DNA marker.