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Identification of Quantitative Trait Loci for Resistance to Soybean Cyst Nematode Race 5

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콩 Cyst 선충 Race 5에 대한 저항성 QTL 탐색

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ABSTRACT : The objectives of this study were; (1) to identify and localize QTLs for resistance to soybean cyst nematode(SCN) race 5 on RAPD map, (2) to identify the magnitude and mode of inheritance for each QTL, and (3) to identify the best combinations of QTLs for resistance to SCN race 5. Based on the univariate regression analysis, we detected 26 markers(22 RAPD and 4 RFLP) which showed significant association($P<0.05$) with resistance to SCN race 5. From MAPMAKER/QTL analysis, we identified two regions (LGC-20 and Group 2) for resistance to SCN race 5. The QTL that was localized at 8.0 cM from pK418C on LGC-20 showed a recessive mode of inheritance and the QTL that was localized between W03 and $E02^3$ on Group 2 showed a dominant mode of inheritance. Two pairs of flanking markers ($E02^3$ and W03, pK418C and $pK418E_1$) and one unlinked RAPD marker, $G10^1$ were used for multiple regression analysis. Marker combination which was composed of 4 markers, $E02^3$, $G10^1$, W03, and $pK418E_1$, explained the highest amount of phenotypic variation by SCN (35.2%). Further research for the identification of QTLs for resistance to SCN race 5 to explain larger portion of phenotypic variation is needed.

Key words : Soybean, SCN, Race 5, RAPD, RFLP, QTLs.

INTRODUCTION

The soybean cyst nematode(SCN), *Heterodera glycines* Ichinohe, is one of the most serious pests in soybean [*Glycine max*(L.) Merr.]. Damage due to SCN has been estimated to be 634,000 metric tons in the Southern US alone, which amounts to a loss of 143.23 million dollars in 1991[20]. Soybean yield losses caused by SCN throughout the Southeastern US and up to the upper Midwest are around 5% of the total soybean production[16]. Managing the SCN can increase soybean yield by more than 1.5 bushels per

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acre. However, the present management tactics for SCN including the use of resistant cultivars, nematicides, rotation with non-host crops, and biological control, are limited in effectiveness and practicality. The existence of biological races within *H. glycines* has complicated the study of relationships between soybean and soybean cyst nematode parasitism[12].

Using genetic resistance is one of the most efficient and economical methods of SCN control[25]. Long-term production stability of soybean is dependent upon cultivar development that will have SCN resistance and high yield. Caldwell et al.[4] reported that resistance in 'Peking' was determined to be conditioned by three recessive genes designated as *rhg1*, *rhg2*, and *rhg3*. A dominant gene *Rhg4* closely linked to the *i* locus, which determines the distribution of pigments on the seed coat was suggested to be necessary for conditioning resistance in Peking[14]. Sugiyama & Katsumi[23] proposed that black seed trait was linked with recessive gene for resistance in Peking.

The plant introduction PI 437654 has been reported to be resistant to all known races of SCN[1]. Myers & Anand[15] conducted SCN resistance study in the cross of PI 437654 × Essex. For race 5, they obtained the segregation of 25 resistant and 321 susceptible plants, and suggested the presence of 2 dominant and 2 recessive genes.

However, Anand et al.[3] suggested that PI 437654 must have more genes since it rarely had any cysts when infected with different populations of SCN. Luedders[13] proposed that more genes probably conditional resistance to SCN than have been described by conventional genetic analysis. At least ten genes have been hypothesized to govern resistance to SCN in soybeans[18].

Results from several genetic studies indicated that genes for resistance to some races of SCN may be linked, or multiple alleles at a single locus may be involved[2,9,10]. Loci differences among cultivars have been reported for race 5 resistance[2]. Race 3 resistance was presumed to be a requisite for conditioning resistance to any other race[24]. PI 399061, PI 424595, and PI 438342 have been reported to have race 5 resistance but susceptibility to race 3[26]. This suggests that race 3 resistance is not necessary for all SCN resistance and that race 3 also has functional alleles for parasitism. The possibility of resolving problems of allelism or linkages will be possible if individual loci for SCN resistance are mapped and intralocus interactions are determined[9]. Explanation of the complicated nature of resistance to SCN races requires more detailed study of the mechanisms and knowledge of the individual genes contributing resistance.

The objectives of this study were; (1) to identify and localize QTLs for resistance to SCN race 5 on RAPD map developed in our previous study[5,7], (2) to identify the magnitude and mode of inheritance for each QTL, and (3) to identify the best combinations of QTLs for resistance to SCN race 5.

MATERIALS AND METHODS

Seventy-nine $F_{2:3}$ progenies derived from the cross of 'Essex' × PI 437654 were used for this study. One hundred sixty-four RAPD markers, which have been selected by RAPD marker selection strategies, and 41 RFLP markers identified from 123 probes, were utilized for mapping of resistance to SCN[6,7,22].

1. Soybean cyst nematode bioassay

A homogeneous isolate of SCN race 5 was obtained from Dr. Rao-Arelli (University of Missouri, MO). The isolates were maintained and increased in the growth chamber and greenhouse for 12 generations, and tested with differential lines(Peking, 'Pickett', PI 88788, PI 90763, PI 437654, and Essex) for race classification. The isolates were reproduced on PI 88788.

SCN bioassays were conducted in the greenhouse where temperature was maintained at 28℃. Pregerminated seedlings(72 hrs at 27℃) were transplanted into plastic cups filled with sterile sand. Seven seedlings of each $F_{2:3}$ mapping population, differential lines for race classification, and both parents(Essex and PI 437654) were included in bioassay. The seedlings were well watered before inoculation. In about 2~3 days after transplanting, each seedling was inoculated with approximately 2000 nematode eggs. Irrigation was suspended for 24 hours post-inoculation to prevent the draining of the inoculum. Subsequently, the plants were watered 1 to 2 times a day depending on weather conditions. Thirty days after inoculation, the roots were carefully dislodged and washed with a powerful jet of water. Cysts were collected on a 75μm sieve and counted under the stereoscope. Female Index(FI) was used to estimate plant response to SCN race 5. FI was calculated for each $F_{2:3}$ genotype by average number of cysts on each genotype devided by the average number of cysts on susceptible check Essex, and it was expressed as a percentage.

2. Statistical analysis

Genotypic classes of molecular and morphological markers were contrasted with phenotypic variation(FI values) of SCN race 5. Association between markers and resistance to SCN race 5 was tested by linear regression analysis and analysis of variance (ANOVA) using the general linear fixed effect model. The homogeneity within- and between-genotypes was estimated by analysis of variance. Statistical analysis was conducted using PC-SAS version 6.0[19].

MAPMAKER/QTL (version 1.1)[11,17] was used to identify the association of markers with phenotypic variation at each marker position. Map distances, orders of each linkage group, and phenotypic data were the input for MAPMAKER/QTL analysis. Each linkage group was scanned at every 2.0 cM for the detection of presence of QTL. A LOD threshold of 2.0 was used to declare the presence of a putative QTL in a given genome region. "QTL likelihood plots" was performed using the MAPMAKER/QTL covering the entire genome to estimate the following information: (1) regions in the genome which are likely to contain putative QTLs, (2) the strength of the data supporting the hypothesis that particular QTL exists, and (3) the likely position of putative QTLs. All possible modes of inheritance(dominant, additive, and recessive) were tested at each QTL position, and compared with the mode of inheritance in genetics 'free' model. The mode of inheritance at each QTL that had similar χ^2 value, log-likelihood, and variance-explained in comparison with genetics 'free' model was determined as a possible mode of inheritance for the QTL. Interactions among flanking markers and identification of the best marker combinations for resistance to SCN race 5 were estimated by multiple regression analysis and analysis of variance.

RESULTS AND DISCUSSION

1. SCN bioassay

When considering the average number of cysts on susceptible Essex as 100%, the FI values of resistance sources to SCN race 5 were 1.3% and 1.0% for Peking and PI 90763, respectively(Table 1). The FI values of susceptible genotypes to SCN race 5 were 43% and 91.5% for Pickett and PI 88788. PI 88788 had very similar degree of susceptibility to Essex in response to race 5(91.5%). These results confirmed that the nematode isolate we used for bioassay was SCN race 5. PI 437654 showed complete resistance to race 5, which had 0 cyst in every seedling(Table 1).

Fig. 1 shows the distribution of FI values in response to SCN race 5 in $F_{2:3}$ progenies of Essex × PI 437654. Two genotypes were complete resistance, and no genotype had FI value of over 100%. The mean FI value of the $F_{2:3}$ population was 23.1(Table 2). In consideration of resistance as FI < 10%, 24(31.2%) genotypes were resistant.

Analysis of variance to estimate the homogeneity between- and among-genotypes is presented in Table 3. Variance between F_2 progenies was highly significant(P = 0.0001), indicating genotypic differences in segre-

Table 1. Reaction to soybean cyst nematode race 5 of parental lines 'Essex' and PI 437654, and SCN race classification differential lines

Genotype	Race 5	
	Range[1]	F.I.[2](%)
Essex	130~189	100
PI 437654	0	0
Peking	0~5	1.3
PI 88788	118~186	91.5
Pickett	59~83	43
PI 90763	0~4	1.0

1 : Number of cysts
2 : Female index

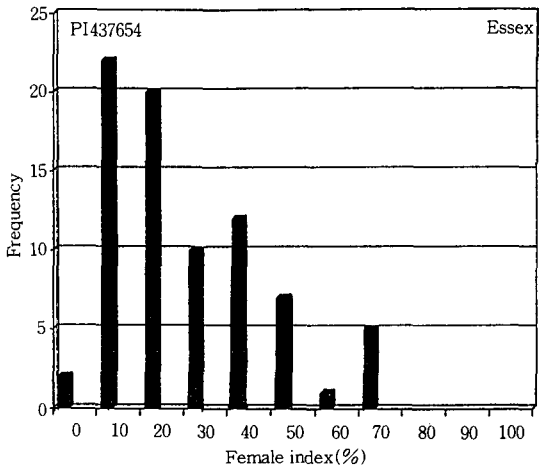

Fig. 1. Distribution of female index of response to soybean cyst nematode race 5 in $F_{2:3}$ progenies of 'Essex' × PI 437654.

Table 2. Mean value and distribution of female index(FI) in response to soybean cyst nematode race 5 in $F_{2:3}$ progenies of 'Essex' × PI 437654 cross

SCN race	Mean	SD[1]	F. I. value	
			Minimum	Maximum
Race 5	23.1	18.3	0	66.9

1 : Standard deviation

Table 3. Analysis of variance within- and between-genotypes in response to soybean cyst nematode race 5 in $F_{2:3}$ progenies of 'Essex' × PI 437654 cross

Mean value of female index ; 38.677 cysts/plant

	d. f.	F-value	Pr > F
Between genotypes	76	23.9	0.0001**
Within genotypes	6	3.0	0.0063**

** ; Significant at 99% confidence level.

gating population. F-value was 23.9 in reaction to SCN race 5. In consideration of the wide range of FI of $F_{2:3}$ progeny(0~66.9)(Table 2), this result was not surprising. Within-genotypic analysis of variance indicated that FI of $F_{2:3}$ individuals were significantly different(Table 3). The standard deviation in response to SCN race 5 was 79.2%(18.3/23.1) of FI mean(Table 2).

2. Regression analysis for molecular marker association with SCN

Based on the univariate regression analysis to identify the associations of selected RAPD markers and unlinked markers for resistance to SCN race 5, we detected 26 markers(22 RAPD and 4 RFLP) which showed significant association($P < 0.05$) with resistance to SCN race 5. Table 4 shows probability, F-value, and R^2 value for each marker. Two markers, G10[1] and $pK418E_1$, showed highly significant association. These markers explained 14.2% and 14.4% of phenotypic variation by SCN, respectively.

Table 4. RAPD markers and RFLP markers that significantly associated with resistance to soybean cyst nematode race 5 from linear regression analysis in $F_{2:3}$ progenies of 'Essex' × PI 437654 cross.

Marker		LG[1]	Select[2]	F-value	Pr > F	R^2 value
RAPD	A02[2]	Group 1	S	5.52	0.021*	0.067
	A07[1]	Group 2	S	4.25	0.043*	0.052
	A07[3]	Group 2	S	6.19	0.015*	0.075
	A11[4]	Group 2	S	5.86	0.018*	0.071
	B15[1]	Group 1	S	5.86	0.018*	0.071
	C08[5]	Group 1	S	5.78	0.019*	0.070
	E02[1]	Group 2	S	4.55	0.036*	0.056
	E02[3]	Group 2	S	4.63	0.035*	0.057
	E04[2]	C-1		5.00	0.028*	0.061
	G10[1]	unlinked		12.74	0.001**	0.142
	G10[2]	Group 2	S	5.52	0.021*	0.067
	G10[4]	Group 2	S	5.52	0.021*	0.067
	G13[2]	Group 2	S	5.52	0.021*	0.067
	G15	Group 2	S	5.52	0.021*	0.067
	H04[2]	Group 2	S	4.42	0.039*	0.054
	H07[1]	Group 2	S	5.14	0.026*	0.063
	H15	Group 1	S	4.71	0.033*	0.058
	K01	C-1		5.39	0.023*	0.065
	N11[1]	unlinked		4.15	0.045*	0.051
	N18	unlinked		6.07	0.016*	0.073
	W03	Group 2	S	4.49	0.037*	0.056
	W13	C-20		4.90	0.030*	0.060
RFLP	pA112A	C-1		4.77	0.032*	0.061
	pA112B	C-1		3.23	0.046*	0.086
	pK418C	C-20		4.11	0.048*	0.079
	$pK418E_1$	C-20		12.23	0.001**	0.144

[1] : Linkage group
[2] : Markers from RAPD marker selection strategies
* : Significant at 95% confidence level
** : Significant at 99% confidence level

Twenty-six markers that showed significant associations with resistance to SCN race 5 exceeded the number of suggested loci. Several reasons can cause the high number of detected markers. Type 1 error can be one of the reasons for overestimating number of association. The occurrance of Type 1 error has been discussed in the previous studies[8,21]. We used low significance level at this step in order to compare molecular marker association with those identified by MAPMAKER/QTL analysis. This strategy might minimize the possible Type II error. Another reason can be explained by the fact that some of these markers might provide information for the same chromosomal region.

3. Identification of QTLs for resistance to SCN race 5

From MAPMAKER/QTL analysis, we identified two regions for resistance to SCN race 5. These two regions were mapped on LGC-20 and on Group 2(Fig. 2). The QTL mapped to LGC-20 was localized between RFLP markers, pK418C and pK418E_1. This QTL was assigned with high LOD(9.15) and explained 68.9% of variation(Table 5). The second QTL was assigned with LOD 2.73 and explained 44.2% of variation. This locus was positioned 1.0 cM from $E02^3$ on Group 2(Fig. 2 and Table 5).

4. Mode of inheritance at QTLs for resistance to SCN race 5

The QTL that was localized at 8.0 cM from pK418C showed a recessive mode of inheritance. The values of LOD and variance-explained for recessive inheritance mode(7.85 and 65.1%) at this QTL were similar to those of genetics free model(9.15 and 68.9%)(Table 5). The second QTL that was localized between W03 and $E02^3$ showed a dominant mode of inheritance. Genetics free model for this QTL had LOD of 2.73 and

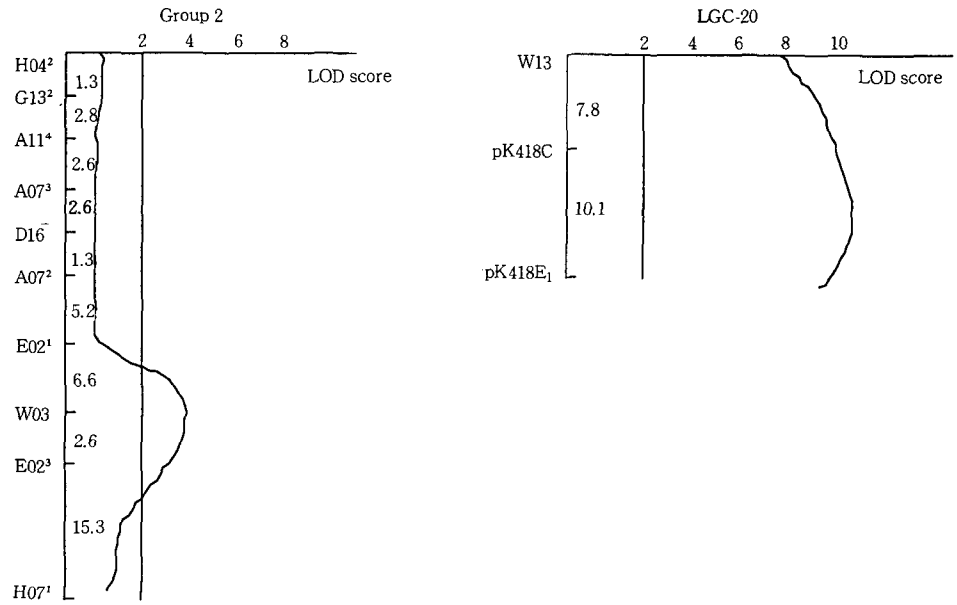

Fig. 2. Localization of quantitative trait loci for resistance to soybean cyst nematode race 5 in $F_{2:3}$ progenies in 'Essex' × PI 437654.

Table 5. Analysis of gene action at the QTL loci for resistance to SCN race 5

(1) LGC-20

Interval	Length[1]	QTL-POS[2]	Genetics
pK418C − pK418E$_1$	10.1	8.0	free
χ^2 = 42.147(2 d. f.)			log-likelihood = 9.15
mean = 1.445	σ^2 = 0.0736		variance-explained = 68.9%

Genetics	mean	χ^2	log-likelihood	variance-explained(%)
Dominant	1.437	5.301	1.15	6.9
Recessive	1.428	39.826	7.85	65.1
Additive	1.492	6.608	1.43	13.4

(2) Group 2

Interval	Length[1]	QTL-POS[2]	Genetics
W03−E02[3]	2.6	1.0	free
χ^2 = 12.556(2 d. f.)			log-likelihood = 2.73
mean = 1.672		σ^2 = 0.077	variance-explained = 44.2%

Genetics	mean	χ^2	log-likelihood	variance-explained(%)
Dominant	1.662	12.391	2.69	44.7
Recessive	1.405	4.351	0.94	5.4
Additive	1.508	6.116	1.33	11.8

[1] : Unit ; centimorgan(cM)
[2] : QTL position

variance-explained of 44.2%. Dominant inheritance mode had almost the same values as genetics free model. Genetics free model for this QTL with recessive and additive modes confirmed that this QTL expressed dominant action at the locus(Table 5).

5. Molecular marker combinations for SCN

Multiple regression analysis using various molecular marker combinations was used to explain the combined effects of QTL loci and an estimate of the total variation for SCN response. Two pairs of flanking markers; E02[3] and W03, pK418C and pK418E$_1$, and one unlinked RAPD marker, G10[1] were used. G10[1] showed an F = 12.74, P = 0.0006, and R^2 = 14.2%(Table 4). Twenty-six sets of marker combinations are presented in Table 6. Thirteen marker combinations were significant at 99.9% confidence level. Five marker combinations were significant at 99.0% confidence level, and 4 marker combinations were significant at 95.0% confidence level. Marker combination set 21, which was composed of 4 markers, E02[3], G10[1], W03, and pK418C, explained the highest amount of phenotypic variation by SCN(35.2%)(Table 6).

In this study, we identified one dominant and one recessive genes for SCN race 5 and explained portion of the phenotypic variation

Table 6. Molecular marker combinations for multiple regression analysis for resistance to soybean cyst nematode race 5 in $F_{2:3}$ progenies of 'Essex' × PI 437654 cross

Set	E02[3]	G10[1]	W03	pK418C	pK418E[1]	F	Pr > F	R^2
1	+	+		−	−	6.47	0.002**	0.145
2	+	−	+	−	−	2.38	0.099	0.060
3	+	−	−	+	−	4.91	0.011*	0.173
4	+	−	−	−	+	7.44	0.001***	0.171
5	−	+	+	−	−	6.65	0.002**	0.151
6	−	+	−	+	−	12.68	0.0001***	0.351
7	−	+	−	−	+	14.25	0.0001***	0.282
8	−	−	+	+	−	4.41	0.018*	0.158
9	−	−	+	−	+	7.35	0.001***	0.171
10	−	−	−	+	+	1.84	0.171	0.073
11	+	+	+	−	−	4.42	0.006**	0.152
12	+	+	−	+	−	8.30	0.0002***	0.351
13	+	+	−	−	+	9.93	0.0001***	0.283
14	+	−	+	+	−	3.25	0.030*	0.175
15	+	−	+	−	+	4.85	0.004**	0.172
16	+	−	−	+	+	2.82	0.049*	0.158
17	−	+	+	+	−	8.28	0.0002***	0.351
18	−	+	+	−	+	9.31	0.0001***	0.285
19	−	+	−	+	+	7.66	0.0003***	0.338
20	−	−	+	+	+	2.51	0.071	0.143
21	+	+	+	+	−	6.10	0.0005***	0.352
22	+	+	+	−	+	6.89	0.0001***	0.285
23	+	+	−	+	+	5.62	0.001***	0.338
24	+	−	+	+	+	2.11	0.096	0.161
25	−	+	+	+	+	5.62	0.001***	0.338
26	+	+	+	+	+	4.41	0.002**	0.339

* : Significant at 95% confidence level
** : Significant at 99% confidence level
*** : Significant at 99.9% confidence level
+ : Marker was included, − : Marker was not included

in F_2 segregating population of Essex × PI 437654. According to Myers & Anand[15], there might be 2 dominant and 2 recessive genes for resistance to SCN race 5. Anand & Rao-Arelli[2] have suggested that at least one gene for race 5 resistance was different in Peking, PI 90763, and PI 404166. Riggs & Schmitt[18] have suggested that more than 10 genes might be needed for resistance to SCN. Our results necessitate identification of the remaining QTLs for resistance to SCN race 5 in order to explain larger portion of phenotypic variation in soybean. The further localization of QTLs and examination of interaction between QTLs will accelerate the exploitation of resistance to SCN.

摘 要

콩 品種 Essex와 PI 437654間 交雜 후 F_2 由來 F_3 系統들을 材料로 하여 作成된 RAPD 遺傳子 地圖上에 cyst 線蟲 race 5에 대한 저항성 QTLs 分析을 實施한 바 結果를 要約하면 다음과 같다.

1. 回歸分析 결과 26개의 marker들(22 RAPD, 4 RFLP)에서 cyst 線蟲 race 5 저항성 반응에 대한 有意性이 認定되었다.
2. MAPMAKER /QTL 分析 결과 2개의 저항성 QTL들이 探索되었는데, 이 QTL들은 2개의 linkage groups(LGC-20와 Group 2)에 位置하였다.
3. 探索된 2개의 QTL들 중 1개는 優性遺傳, 그리고 나머지 하나는 劣性遺傳樣相을 나타내었다.
4. 콩 cyst 線蟲 race 5의 저항성에 대한 有意性이 認定되는 5개의 marker들간 相互作用을 알아보기 위한 多重回歸分析 결과 총 26개의 조합들 중 4개의 marker들($E02^3$, $G10^1$, W03, pK418C)로 구성된 조합에서 가장 높은 表現的 변이의 값(35.2%)을 나타내었다.

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