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ORIGINAL PAPER

QTL underlying the resistance to soybean aphid (*Aphis glycines* Matsumura) through isoflavone-mediated antibiosis in soybean cultivar 'Zhongdou 27'

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Abstract Soybean aphid (*Aphis glycines* Matsumura) results in severe yield loss of soybean in many soybean-growing countries of the world. A few loci have been previously identified to be associated with the aphid resistance in soybean. However, none of them was via isoflavone-mediated antibiosis process. The aim of the present study was to conduct genetic analysis of aphid resistance and to identify quantitative trait loci (QTL) underlying aphid resistance in a Chinese soybean cultivar with high isoflavone content. One hundred and thirty $F_{5:6}$ derived recombinant inbred lines from the 'Zhongdou 27' × 'Jiunong 20' cross were used. Two QTL were directly associated with resistance to aphid as measured by aphid damage index. qRa_1, close to Satt470 on soybean linkage group (LG) A2 (chromosome 8), was consistently detected for 3- and 4-week ratings and explained a large portion of phenotypic variations ranging from 25 to 35%. qRa_2, close to Satt144 of LG F (chromosome 13), was detected for 3- and 4-week ratings and could explain 7 and 11% of the phenotypic variation, respectively. These two QTL were highly associated with high isoflavone content and both positive alleles were derived from 'Zhongdou 27', a cultivar with higher isoflavone content. The results revealed that higher individual or total isoflavones contents in soybean lines could protect soybean against aphid attack. These two QTL detected jointly provide potential for marker-assisted selection to improve the resistance of soybean cultivars to aphid along with the increase of isoflavone content.

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Introduction

Soybean aphid (*Aphis glycines* Matsumura) is one of the most damaging pests of soybean (Sun et al. 2000), causing a considerable yield loss through plant damage during feeding, including leaf distortion, stunting and desiccation (Wu et al. 2004; Hartman et al. 2001), or transmitting various viruses such as soybean dwarf virus, soybean mosaic virus, potato virus, alfalfa mosaic virus and tobacco ring spot virus (Clark and Perry 2002; Davis et al. 2005). The soybean aphid originated in Asia, and then has spread to USA, Canada, Australia and New Zealand in the recent years (Fletcher and Desborough 2000; Hartman et al. 2001). Yield losses of soybean attributed to the aphid were reported to be greater than 50% in Minnesota of USA (Ostlie 2002) and up to 52% in China (Wang et al. 1994). Currently, the major soybean cultivars in China, especially in Northeastern China, are all susceptible to the soybean aphid, and producers depend on insecticides or natural enemies to control aphid. However, most natural enemies can only control soybean aphids at a low density. Meanwhile, chemical control may require scouting, kill beneficial insects, and cause environmental pollution (Zhang et al. 2010). Compared to biological and chemical controls, host resistance is more effective, economical and environmentally sound for pest control. Hence, selecting endogenously resistant cultivars and germplasm may be the effective method to control soybean aphid.

Traditionally, plant improvement had relied on the phenotypic selection of populations from crosses between

commercial cultivars or experimental lines (Stuber et al. 1992). However, like most harvest traits, aphid resistance evaluation is complicated by environmental conditions including temperature, moisture and light, which influenced the life cycle of aphid (Wu et al. 2004). Consequently, selection for soybean cultivars with high and stable resistance against aphid requires evaluation in multiple environments over several years, which is expensive, time consuming and labor intensive. Molecular marker offers a faster and more accurate approach to breeding, because selection could be based on genotype rather than solely on phenotype. The use of molecular markers for indirect selection of important agronomic traits, or marker-assisted selection (MAS) could improve the efficiency of traditional plant breeding. Cregan et al. (1999) and Song et al. (2004) developed an integrated genetic linkage map of soybean containing 1,849 markers, including 1,015 SSR markers in one or more of five different populations, and aligned the molecular linkage groups (LGs) into a consensus map of 20 LGs that correspond to the 20 pairs of soybean chromosomes (Zou et al. 2003;Choi et al. 2007). This information has greatly facilitated MAS in soybean breeding.

The aphid resistance in crop plants is often qualitative (controlled by one or two genes) rather than quantitative (controlled by many genes or quantitative trait loci) (Klingler et al. 2005). In soybean, the aphid resistance in Dowling and Jackson was both controlled by a single dominant gene (Hill et al. 2006a, b). The gene in Dowling was named as Rag1 (Hill et al. 2006a). Later, Rag1 and the resistance gene (Rag) in Jackson were both mapped to the same genomic region on chromosome 7 (LG M, Li et al. 2007a), which seemed to be allelic. Similarly, resistance in PI 243540 was controlled by a single dominant gene (Kang et al. 2008), named as Rag2 and was mapped on chromosome 13 (LG F) (Mian et al. 2008). The aphid resistance in PI 567541B and PI 567598B has been determined to be controlled by two recessive genes (Mensah et al. 2008). These two genes in PI 567541B were anchored on chromosomes 7 (LG M) and 13 (LG F), respectively (Zhang et al. 2009). The gene on chromosome 7 (LG M) was mapped to the same genomic region as *Rag*1 and was later designated as *rag*1_provisional (Zhang et al. 2010). The gene on chromosome 13 (LG F) was located distantly from *Rag*2 and was later designated as *Rag*4 (Zhang et al. 2009). One quantitative trait loci (QTL) was identified in an interval between Sat_339 and Satt414 on chromosome 16 (LG J) in PI 567543C (Zhang et al. 2010).

Quantitative trait loci associated with aphid resistance in modern Chinese soybean cultivars has not been reported by 2010. The objective of the present study was to identify QTL associated with aphid resistance using the recombinant inbred lines (RILs) of 'Zhongdou 27' × 'Jiunong 20' in both greenhouse and field conditions using SSR markers.

Materials and methods

Mapping population and the evaluation of aphid resistance

The mapping population contained 130 $F_{5:9}$ RILs that were advanced by single-seed-descent from the cross between 'Zhongdou 27' (resistance to aphid, developed by the Chinese Academy of Agriculture, Beijing, China) and 'Jiunong 20' (susceptible to aphid, developed by Jinlin Academy of Agriculture, Jilin, China).

Field and greenhouse tests were conducted for aphid resistance evaluation. One hundred and thirty RILs were grown together with the parents at the experimental station of Northeast Agricultural University, Harbin, China (45°N, fine-mesic chernozen soil). Seeds were planted on May 7 of 2009, with rows 3 m long, 0.75 m apart and a space of 6 cm between plants. Two row plots were used in the experiment with border rows in a complete randomized design with three replicates. The experiment was covered by a plastic-net cabinet to control aphid and other insect infestations. The greenhouse experiment was maintained at temperature of 26°C by day, 15°C by night, and sodium vapor lights were used to supplement light intensity during the day (14 h). Each plant in field and greenhouse was inoculated with two wingless aphids at the V1 stage. The aphid inoculated in the field and greenhouse trial was a single clone, which was collected from the naturally infested field in experimental station of Northeast Agricultural University in 2008 and has been maintained in the greenhouse ever since. The aphid biotype used in this study is the same as the Ohio biotype (Kim et al. 2008) (data not shown).

Aphid resistance was visually rated for each plant three and four weeks after inoculation using a scale of 0–4 developed by Mensah et al. (2005, 2008). The damage index (DI) for each line was calculated by the following formula: DI = (scale value × no. of plants in the category)/(4 × total no. of plants) × 100 (Mensah et al. 2005). The DI ranges between 0 (for no infestation) and 100 (for the most severe damage).

Molecular linkage map

The map including 99 SSR markers on 20 LGs had been previously constructed (Zeng et al. 2009), which encompassed about 2,019.77 cM with mean distance of 20.41 cM between markers. In this study, QTL analysis, associated with aphid resistance, was based on this map.

Statistical analysis

Quantitative trait loci were identified by single-factor analysis of variance (PROC. GLM. SAS) as described by

Primomo et al. (2005), based on individual environment value. Locus main effects were considered as linear models if they were significant at $P \leq 0.01$. Significant loci on the same LG were tested by two-factor analysis of variance without interactions. If both loci were significant at $P \leq 0.05$ in the two-factor model, they would be both considered as linear models. Otherwise, the locus with the larger individual R^2 value was chosen to represent the effect of the putative QTL on the LG. If two loci remained significant in a two-factor model, both loci were considered as linear models.

Results

Resistance evaluation of aphid

Cultivars 'Zhongdou 27' and 'Jiunong 20' significantly differed in the aphid resistance in both field and greenhouse conditions. The resistant and susceptible parents, as well RILs were clearly separated based on damage index in field condition at Harbin in 2009. The frequency distribution of the field resistance to aphid displayed a continuous distribution (Table 1; Fig. 1), ranging from a low value of 11.73% to a high value of 75.54% for 3-week rating, and from 22.85 to 90.60% for 4-week rating, respectively. The frequency distribution of greenhouse resistance to aphid also showed a continuous distribution (ranged from a low value of 14.28% to a high value of 71.88% for 3-week rating, and from 17.59 to 87.62% for 4-week rating, Table 1; Fig. 2). Both skewness and kurtosis values of aphid resistance in field or greenhouse condition were less than 0.5 in both 3- and 4-week ratings, suggesting that the segregation of this trait fit a normal distribution model. Broad-sense heritability for 130 RILs across years or environments were listed in Table 1, most of them showed relatively high heritability.

QTL associated with aphid resistance

The aphid damage indexes were utilized for mapping QTL controlling resistance to aphid. Two QTL were detected and located on LG A2 (chromosome 8) and F (chromosome 13), respectively (Table 2). The 'Zhongdou 27' allele conferred aphid resistance at both loci. qRa_1, associated with Satt470 of LG A2, was consistently detected for 3- and 4-week ratings and explained a large portion of phenotypic variations ranging from 25 to 35%. qRa_2, associated with Satt144 of LG F, was associated with 3- and 4-week rating and explained a moderate portion of phenotypic variation (explained from 7 to 11% of the phenotypic variations).

Discussion

In comparison with biological and chemical controls, host resistance is the most desirable, effective, and economical means of pest control. Host resistance to insects has three types including tolerance, antibiosis and antixenosis (Painter 1951). Tolerance is expressed as the ability of plant to withstand or recover from the insect damage. Antibiosis resistance affects the insect biology and causes reduced insect abundance. Antixenosis resistance affects the insect behavior and is expressed as the non-preference of the insect for certain plants. The field aphid resistance in this study was evaluated with choice test that could not determine the resistant types of 'Zhongdou 27'. Therefore, both choice test and no choice test were adopted for aphid resistant assay in the greenhouse and the resistant type of 'Zhongdou 27' was determined as antibiosis (data not shown).

Table 1 Aphid damage index (DI) in the greenhouse in 2009 for the parents, 'Zhongdou 27' and 'Jiunong 20', and 130 $F_{5:9}$-derived lines of the mapping populations

Trait	Parents[a]		RILS population						
	Zhongdou 27	Jiunong 20	Mean	Range	SE[b]	CV[c]	Skewness	Kurtosis	H^2 (%)[d]
Field cage									
3-week rating	35a	63b	39.4	11.73–75.54	11.68	29.64	0.5657	0.5384	84.77
4-week rating	36.8a	75b	52.5	22.85–90.60	14.63	27.86	0.1105	0.3944	87.25
Greenhouse									
3-week rating	31.5a	52.3b	41.43	14.28–71.88	12.85	31.01	−0.3963	0.3348	83.25
4-week rating	37.5a	59.4b	46.14	17.59–87.62	16.08	34.85	−0.2688	0.2213	92.36

[a] Mean followed by different letters within the same row are significantly different at $P < 0.05$

[b] Standard deviation

[c] Coefficient of variation

[d] Broad sense heritability

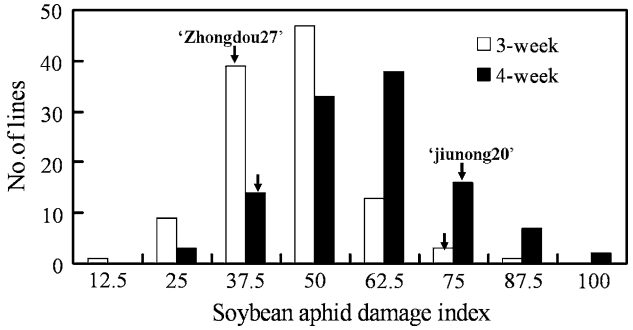

Fig. 1 Frequency distribution of damage index (DI) in greenhouse condition among the 130 $F_{5:9}$ lines derived from a cross between cultivar 'Zhongdou 27' × 'Jiunong 20'

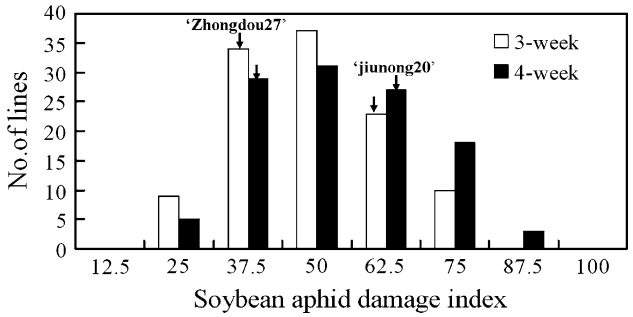

Fig. 2 Frequency distribution of damage index (DI) in field condition among the 130 $F_{5:9}$ lines derived from a cross between cultivar 'Zhongdou 27' × 'Jiunong 20'

'Zhongdou 27' was proved to have high individual and total isoflavones contents (daidzein 1,865.38 μg g^{-1}, genistein 1,614.08 μg g^{-1}, glycitein 311.52 μg g^{-1} and total isoflavones 3,791.09 μg g^{-1}) and 'Jiunong 20' was proved to have lower isoflavones contents (daidzein 844.02 μg g^{-1}, genistein 1,046.41 μg g^{-1}, glycitein, 193.17 μg g^{-1} and total isoflavones 2,061.87 μg g^{-1}) in our previous report (Zeng et al. 2009). The aphid resistance of 'Zhongdou 27' might be due to the presence of high individual or total isoflavones contents. In order to verify the relationship between aphid resistance and isoflavone content in soybean leaves, we analyzed the individual and total isoflavones contents in the leaves of four soybean genotypes injured or non-injured by soybean aphid. Both before and after aphid damage, the glycitein content in soybean leaves did not show a significant difference among the four genotypes tested (Table 3). Leaves from resistant accessions PI 567598B and 'Zhongdou 27' (higher contents of daidzein and genistein) damaged by soybean aphid had higher daidzein and genistein contents than those without aphid attack, while the susceptible accession 'Jiunong 20' and Williams 82 produced less daidzein and genistein than the other two genotypes even after being damaged by soybean aphid, and showed no difference in individual or total isoflavones contents between damaged and non-damaged leaves. Former reports had proved that the increased production of isoflavonoid was correlated with the increased *Nezara viridula* resistance (Piubelli et al. 2003). Mensah et al. (2005) reported that PI 567598B was resistant to soybean aphid via antibiosis. Graham and Graham (1991) as well as Graham et al. (1990) have proposed that pre-existing pools of daidzin and malonyldaidzin, glucosides of the isoflavonoid daidzein, and of genistin and malonal-genistin, glucosides of genistein, might contribute to the resistance to infection in soybean seeds and seedling tissues or leaves. Activation of the general defence response in soybean leaves resulted in the production of isoflavone that was the products of multiple branched pathways, of which only one branch lead to the synthesis of the phytoalexins or the glyceollins (Morris et al. 1991). These phytoalexins were primarily fungitoxic, but the phytoalexins of legumes could be toxic to insects (Hart et al. 1983). Furthermore, some of isoflavones themselves had antibiotic effects on insects. Genistein and daidzin were found to be

Table 2 Summary of QTL for soybean aphid resistance detected in the mapping populations from the cross of 'Zhongdou 27' × 'Jiunong 20 using the composite interval mapping method

Trait	QTL	LG[a]	Marker	Environment	*P*	R^{2a}	Allelic mean ± SEM[b]	
							'Zhongdou 27'	'Jiunong 20'
3-week rating	qRa_1	A2	Satt470	Field cage	<0.0001	25.63	20.61 ± 1.24	67.32 ± 1.24
				Greenhouse	<0.0001	33.53	23.56 ± 1.36	65.76 ± 1.41
4-week rating				Field cage	<0.0001	29.87	27.89 ± 2.14	73.57 ± 2.21
				Greenhouse	<0.0001	35.76	25.56 ± 1.89	69.89 ± 1.90
3-week rating	qRa_2	F	Satt144	Field cage	<0.0001	10.43	19.98 ± 1.10	68.98 ± 1.26
				Greenhouse	<0.0001	7.05	22.67 ± 1.62	63.34 ± 1.57
4-week rating				Field cage	<0.0001	9.03	20.67 ± 1.14	67.98 ± 1.14
				Greenhouse	<0.0001	11.01	23.45 ± 1.92	75.08 ± 1.93

[a] R^2 is *R*-square or the proportion of the phenotypic data explained by the marker locus

[b] SEM: mean + $SD/\sqrt{N}$; where *N* was the number of each of allele

Table 3 Individual or total isoflavones contents in leaves of four soybean genotypes injured or non-injured by soybean aphid

Genotype	Daidzin concentration (μg g^{-1}) (mean ± SEM)		Glycitein concentration (μg g^{-1}) (mean ± SEM)		Genistein concentration (μg g^{-1}) (mean ± SEM)		Total isoflavones concentration (μg g^{-1}) (mean ± SEM)	
	Injured	Non-injured	Injured	Non-injured	Injured	Non-injured	Injured	Non-injured
Zhongdou 27	1,259.5 ± 161.7aA[a]	880.9 ± 26.6aB	123.2 ± 2.8abA	127.3 ± 6.3aA	1,625.3 ± 91.6aA	1,190.5 ± 86.2abB	3,008.0 ± 185.9aA	2,198.7 ± 109.7aB
PI567598B	1,083.3 ± 57.7abA	678.1 ± 50.2abB	122.6 ± 2.9abA	121 ± 10.9abA	1,366.7 ± 76.6bA	1,158.8 ± 68.3abAB	2,572.7 ± 135.7bA	1,958.0 ± 81.7abB
Jiunong 20	461.3 ± 47.2dA	486.4 ± 11.9cA	115.9 ± 6.4abA	124.9 ± 9.7abA	631.5 ± 58.0dA	741.8 ± 115.0cA	1,208.7 ± 93.6cA	1,353.1 ± 115.3dA
Williams 82	688.0 ± 32.6cA	606.3 ± 23.2cdA	99.1 ± 9.1bA	108.7 ± 10.3bA	941.2 ± 34.7cA	951.2 ± 51.0bcA	1,728.4 ± 63.9dA	1,748.0 ± 43.3cA

[a] Means followed by the same lowercase letter in the column and capital letter in the row did not differ by Tukey's test ($P < 0.05$)

toxic to larvae of mosquito (Rao et al. 1990). Genistein and daidzin were also known to cause higher mortality, lower initial larval and pupal weight, less growth and elongated larval cycle to *N. viridula*, *Trichoplusia ni* and *Anticarsia gemmatalis*, which were important pests of soybean in Brazil (Sharma and Norris 1991; Hoffmann-Campo et al. 2001; Piubelli et al. 2003, 2005).

We previously reported that four QTL, QDZF-1, QGTA2-1, QGTF-1 and QTIF-1 underlay daidzein and genistein components of isoflavone in soybean seeds of 'Zhongdou 27' × 'Jiunong 20' (Zeng et al. 2009). The aphid resistance QTL qRa_1, which was mapped on linkage group (LG) A2 and closely linked to Satt470. The QGTA2-1 was associated with GT and has also been mapped to the same genomic region (http://www.soybase.org). Meanwhile, the cultivars PI 567598B and 'Zhongdou 27' with higher genistein contents are aphid resistance. Therefore, qRa_1 and GTA2-1 on chromosome 8 (LG A2) may be the same locus, which should be considered to contribute for both aphid resistance and high genistein content. QDZF_1 associated with DZ, QGTF_1 associated with GT, QTIF_1 associated with TI, were all linked with SSR marker Satt144 in LG F. The aphid resistant QTL qRa_2 was also closely linked to Satt144. The results revealed that higher individual DZ/GT or total isoflavones contents in soybean lines could protect soybean against aphid attack and the resistance to aphid from 'Zhongdou 27' seemly belonged to antibiosis category.

In this study, a major QTL qRa_1 explained a large portion of phenotypic variations ranging from 25 to 35%, while qRa_2 explain 7 and 11% of the phenotypic variation. The QTL qRa_1 and qRa_2 detected here contributed only less than 50% of the resistant variations. QTL with small contributions have also been observed in *Glycine max* (Jiang et al. 2010), *Triticum aestivum* (Wang et al. 2009), corn (Li et al. 2007b), *Oryza sativa* (Li et al. 2006). Therefore, more undetected QTL that underlie the resistance to aphid might exist. To detect more QTL resistant to soybean aphids we need more mapping populations, more markers (such as SSR and SNP) and more soybean aphid biotypes. Furthermore, QTL with relatively small effects ($R^2 < 0.10$) are difficult to identify because the risk of detecting false-positives is high in the small mapping populations (<200 individuals) used in many mapping studies (Boerma and Walker 2005). Therefore, more attention should be paid to the stable QTL detected in different environments and with pleiotropic effects in future study.

Several loci (QTL) had been found to underlie aphid resistance in the different regions of the LG F (Mian et al. 2008; Zhang et al. 2009). In this study, QTL qRa_2, located in LG F, was found to be strongly associated with aphid resistance on a region of LG F different from the published ones. Both QTL qRa_1 and qRa_2 from the

present work were new loci underlying aphid resistance in soybean and was consistently detected for 3- and 4-week ratings in field and greenhouse. The two QTL were also highly associated with high isoflavone content. Therefore, the two QTL detected jointly provide potential for MAS to improve the resistance of cultivar to aphid along with the increase of isoflavone content.

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References

Boerma HR, Walker DR (2005) Discovery and utilization of QTLs for insect resistance in soybean. Genetica 123:181–189

Choi IY, Hyten DL, Matukumalli LK, Song Q, Chaky JM, Quigley CV, Chase K, Gorden Lark K, Reiter RS, Yoon MS, Wang EY, Yi SI, Yong ND, Shoemaker RC, van Tassell CP, Specht JE, Cregan PB (2007) A soybean transcript map: gene distribution, haplotype and single nucleotide polymorphism analysis. Genetics 176:685–696

Clark AJ, Perry KL (2002) Transmissibility of field isolates of soybean viruses by *Aphis glycines*. Plant Dis 86:1219–1222

Cregan PB, Jarvik T, Bush AL, Shoemaker RC, Lark KG, Kahler AL, Kaya N, VanToai TT, Lohnes DG, Chung J, Specht JE (1999) An integrated genetic linkage map of the soybean genome. Crop Sci 39:1464–1490

Davis JA, Radcliffe EB, Ragsdale DW (2005) Soybean aphid, *Aphis glycines* Matsumura, a new vector of *potato virus Y* in potato. Am J Potato Res 82:197–201

Fletcher MJ, Desborough P (2000) The soybean aphid, *Aphis glycines*, present in Australia. http://www.agric.nsw.gov.au/hort/ascu/insects/aglycin.htm

Graham MY, Graham TL (1991) Rapid accumulation of anionic peroxidases and phenolic polymers in soybean cotyledon tissues following treatment with *Phytophthora megasperma f. sp. Glycinea* wall glucan. Plant Physiol 97:1445–1455

Graham TL, Kim JE, Graham MY (1990) Role of constitutive isoflavone conjugates in the accumulation of glyceollin in soybean infected with *Phytopthora megasperma*. Mol Plant Microbe Interact 3:157–166

Hart SV, Kogan M, Paxton JD (1983) Effect of soybean phytoalexins on the herbivorous insects, Mexican bean beetle and soybean looper. J Chem Ecol 9:657–672

Hartman GL, Domier LL, Wax LM, Helm CG, Onstad DW, Shaw JT. Solter LF, Voegtlin DJ, D'Arcy CJ, Gray ME, SteVey KL, Isard SA, Orwick PL (2001) Occurrence and distribution of *Aphis glycines* on soybeans in Illinois in 2000 and its potential control. http://www.plantmanagementnetwork.org/pub/php/brief/aphisglycines/

Hill CB, Li Y, Hartman GL (2006a) A single dominant gene for resistance to the soybean aphid in the soybean cultivar Dowling. Crop Sci 46:1601–1605

Hill CB, Li Y, Hartman GL (2006b) Soybean aphid resistance in soybean Jackson is controlled by a single dominant gene. Crop Sci 46:1606–1608

Hoffmann-Campo CB, Harborne JB, McCaffery AR (2001) Pre-ingestive and post-ingestive effects of soya bean extracts and rutin on *Trichoplusia ni* growth. Entomol Exp Appl 98:181–194

Jiang ZF, Han YP, Teng WL, Zhang ZC, Sun DS, Li YH, Li WB (2010) Identification of QTL underlying the filling rate of protein at different developmental stages of soybean seed. Euphytica 175:227–236

Kang ST, Rouf Mian MA, Hammond RB (2008) Soybean aphid resistance in PI 243540 is controlled by a single dominant gene. Crop Sci 48:1744–1748

Kim KS, Hill CB, Hartman GL, Rouf Mian MA, Diers BW (2008) Discovery of soybean aphid biotypes. Crop Sci 48:923–928

Klingler J, Creasy R, Gao L, Nair RM, Calix AS, Jacob HS, Edwards OR, Singh KB (2005) Aphid resistance in *Medicago truncatula* involves antixenosis and phloem-specific, inducible antibiosis, and maps to a single locus flanked by NBS-LRR resistance gene analogs. Plant Physiol 137:1445–1455

Li SB, Zhang ZH, Hu Y, Li CY, Jiang X, Mao T, Li YS, Zhu YG (2006) Genetic dissection of developmental behavior of crop growth rate and its relationships with yield and yield related traits in rice. Plant Sci 170(5):911–917

Li Y, Hill CB, Carlson SR, Diers BW, Hartman GL (2007a) Soybean aphid resistance genes in the soybean cultivars Dowling and Jackson map to linkage group M. Mol Breed 19:25–34

Li YL, Niu SZ, Dong YB, Cui DQ, Wang YZ, Liu YY, Wei MG (2007b) Identification of trait-improving quantitative trait loci for grain yield components from a dent corn inbred line in an advanced backcross BC2F2 population and comparison with its F2:3 population in popcorn. Theor Appl Genet 115:129–140

Mensah C, DiFonzo C, Nelson RL, Wang D (2005) Resistance to soybean aphid in early maturing soybean germplasm. Crop Sci 45:2228–2233

Mensah C, DiFonzo C, Wang D (2008) Inheritance of soybean aphid resistance in PI 567541B and PI 567598B. Crop Sci 48:1759–1763

Mian R, Kang ST, Beil SE, Hammond RB (2008) Genetic linkage mapping of the soybean aphid resistance gene in PI 243540. Theor Appl Genet 117:955–962

Morris PF, Savard ME, Ward EWB (1991) Identification and accumulation of isoflavonoids and isoflavone glucosides in soybean leaves and hypocotyls in resistance responses to *phytophthora megasperma* f.sp. *glycinea*. Physiol Mol Plant Pathol 39:229–244

Ostlie K (2002) Managing soybean aphid, University of Minnesota Extension Service, St Paul. http://www.soybeans.umn.edu/crop/insects/aphid/aphid_publication_managingsba.htm

Painter RH (1951) Insect resistance in crop plants. Macmillan, New York

Piubelli GC, Hoffmann-Campo CB, de Arruda IC, Franchini JC, Lara FM (2003) Flavonoid increase in soybean as a response to *Nezara viridula* injury and its effect on insect-feeding preference. J Chem Ecol 29:1223–1233

Piubelli GC, Hoffmann-Campo CB, Moscardi F, Miyakubo SH, de Oliveira MC (2005) Are chemical compounds important for soybean resistance to *Anticarsia gemmatalis*? J Chem Ecol 31:1509–1524

Primomo VS, Poysa V, Ablett GR, Jackson CJ, Gijzen M, Rajcan I (2005) Mapping QTL for individual and total isoflavone content in soybean seeds. Crop Sci 45:2454–2464

Rao KV, Chattopadhyay SK, Chandrasekhara Reddy G (1990) Flavonoids with mosquito larval toxicity. J Agric Food Chem 38:1427–1430

Sharma HC, Norris DM (1991) Chemical basis of resistance in soy bean to cabbage looper, *Trichoplusia ni*. J Sci Food Agric 55:353–364

Song QJ, Marek LF, Shoemaker RC, Lark KG, Concibido VC, Delannay X, Specht JE, Cregan PB (2004) A new integrated

genetic linkage map of the soybean. Theor Appl Genet 109:122–128

Stuber CW, Lincoln SE, Wolff DW, Helentjaris T, Lander ES (1992) Identification of genetic factors contributing to heterosis in a hybrid from two elite maize inbred lines using molecular markers. Genetics 132:832–839

Sun B, Liang SB, Zhao WX (2000) Outbreak of the soybean aphid in Suihua prefecture in 1998 and its control strategies. Soybean Bull 8:5

Wang XB, Fang CH, Zheng XP, Lin ZZ, Zhang LR, Wang HD (1994) A study on the damage and economic threshold of the soybean aphid at the seedling stage. Plant Prot 20:12–13

Wang RX, Hai L, Zhang XY, You GX, Yan CS, Xiao SH (2009) QTL mapping for grain filling rate and yield related traits in RILs of the Chinese winter wheat population Heshangmai 9 Yu8679. Theor Appl Genet 118:313–325

Wu Z, Schenk-Hamlin D, Zhan W, Ragsdale DW, Heimpel GE (2004) The soybean aphid in China: a historical review. Ann Entomol Soc Am 97:209–218

Zeng G, Li D, Han Y, Teng W, Wang J, Qiu L, Li W (2009) Identification of QTL underlying isoflavone contents in soybean seeds among multiple environments. Theor Appl Genet 118:1455–1463

Zhang G, Gu C, Wang D (2009) Molecular mapping of soybean aphid resistance genes in PI 567541B. Theor Appl Genet 118:473–482

Zhang G, Gu C, Wang D (2010) A novel locus for soybean aphid resistance. Theor Appl Genet 120:1183–1191

Zou JJ, Singh RJ, Lee J, Xu SJ, Cregan PB, Hymowitz T (2003) Assignment of molecular linkage groups to the soybean chromosomes by primary trisomics. Theor Appl Genet 107:745–750