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Mapping Genes for Specific and Adult Plant Resistance to Rust and Abaxial Leaf Pubescence and their Genetic Relationships Using Randomly Amplified Polymorphic DNA (RAPD) Markers in Common Bean

Geunhwa Jung[1], Dermot P. Coyne,[2] James Bokosi,[3] and James R. Steadman[4]
University of Nebraska, Lincoln, NE 68583

James Nienhuis
Department of Horticulture, University of Wisconsin, Madison, WI 53706

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Abstract. **Dry bean (*Phaseolus vulgaris* L.) production is limited by bean rust [*Uromyces appendiculatus* (*Pers.*) Unger var. *appendiculatus*]. An effective control strategy for this disease is to breed cultivars with durable resistance. Information on the inheritance, genetic relationships, and mapping of genes with molecular markers for specific resistance (SR), adult plant resistance (APR), and abaxial leaf pubescence (ALP) is needed to pyramid the desired genes for durable resistance. ALP was found to be associated previously with APR in Andean germplasm. The objective here was to identify and map RAPD markers for the genes controlling SR, APR, and ALP and to examine their relationships. Five rust pathotypes were inoculated on the unifoliate leaves of 68 recombinant inbred (RI) lines derived from 'PC-50' (presence of SR, APR, and ALP) x XAN-159 (absence of SR, APR, and ALP). SR was determined by a single major gene (*Ur*-9) to the five rust pathotypes with no detection of recombinants. The fourth trifoliolate leaves were inoculated with one pathotype (A88T1-4b). A single major gene *Ur*-12 controlled APR to that pathotype. The *Ur*-9 gene (SR) was independent of and epistatic to the *Ur*-12 gene (APR). Because of the low number of APR lines in the RI population resulting from the elimination of RI lines with SR, an F_2 population was developed from a cross of two homozygous RI lines selected for unifoliate susceptibility to pathotype A88T1-4b and for resistance and susceptibility of the fourth trifoliolate leaves to tag RAPD markers linked to the *Ur*-12 gene (APR). The single major gene *Pu*-a determinated ALP and was not linked to *Ur*-9 (SR) and *Ur*-12 (ALP). The gene *Ur*-9 (SR) was linked to RAPD marker J13-1100 at 5 cM and was not assigned to any linkage group or other markers. The gene *Pu*-a (ALP) was mapped at 20.2 cM from I16.500 and 3.9 cM from marker G3.1150 in linkage group 3. The *Ur*-12 gene (APR) was mapped at 34.6 cM from marker O13.1350 in linkage group 4b. This is the first report of mapping a gene for APR in common bean.**

Common bean (*Phaseolus vulgaris*) is an important dietary protein source in developing countries, a cash crop, and a health food in the United States. However, dry bean yields are limited by the bean rust fungal pathogen (*Uromyces appendiculatus*) in many production regions (Stavely and Pastor-Corrales, 1989). More than 200 races and pathotypes have been identified worldwide (Stavely et al., 1989). This high pathogenic variability is attributed to genetic recombination occurring in the sexual stage, although mutations also may play a role (Ballantyne, 1978). An environmentally safe, yet effective, control strategy for this disease is breeding for resistance. Pyramiding monogenic resistance genes may increase the stability of resistance to this highly variable pathogen as illustrated by the resistance to 63 of 65 U.S. races conferred by three genes (*Ur*-3, *Ur*-4, and *Ur*-5). However, this procedure can be expensive and time-consuming because of epistatic interactions among rust resistance genes that necessitate progeny testing. Therefore, RAPD markers tightly linked to genes for rust resistance could be useful for pyramiding several different rust resistant genes into susceptible cultivars, as suggested by Miklas et al. (1993).

RAPD markers linked to the rust resistance genes *Ur*-3 found in 'Aurora', Ecuador 299, Mexico 235, 51051, and 'Nep-2'; *Ur*-3[2] found in PI 181996; *Ur*-4 found in 'Early Gallatin'; and *Ur*-5, a gene block in Mexico 309, have been identified in common bean (Kelly, 1995). All RAPD markers were linked in coupling-phase to the respective resistance gene. RAPD molecular maps of common bean using RI lines from intra-Andean and Middle American crosses were developed by Jung et al. (1996, 1997).

Adult plant resistance (APR) or partial resistance was identified by Shaik (1985) and Mmbaga and Steadman (1992) in the greenhouse and the field, and is characterized by reduced uredinium size on fourth or higher trifoliolate leaves compared to the unifoliolate leaf reaction. This resistance is hypostatic to specific resistance (SR) (Bokosi, 1996) and thus can not be easily combined with SR. Screening can only be done with carefully controlled inoculations with appropriate spore concentrations in greenhouse or field tests on adult plants. Greenhouse inoculations may be too severe to identify APR related to slow-rusting or physical barriers to infection while field tests need to have broad rust virulence represented. Screening techniques using linked molecular markers, however, may allow APR to bean rust to be identified at any stage of plant development and so would be useful for gene pyramiding.

Dense, abaxial leaf pubescence (ALP) on the third or higher trifoliolate leaves in beans had been reported to be associated with race-nonspecific adult plant resistance (Steadman and Shaik, 1988;

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[1]Current address: Dept. of Horticulture, Univ. of Wisconsin, Madison, WI 53706.
[2]Department of Horticulture.
[3]Former PhD graduate student, Dept. of Horticulture.
[4]Department of Plant Pathology.

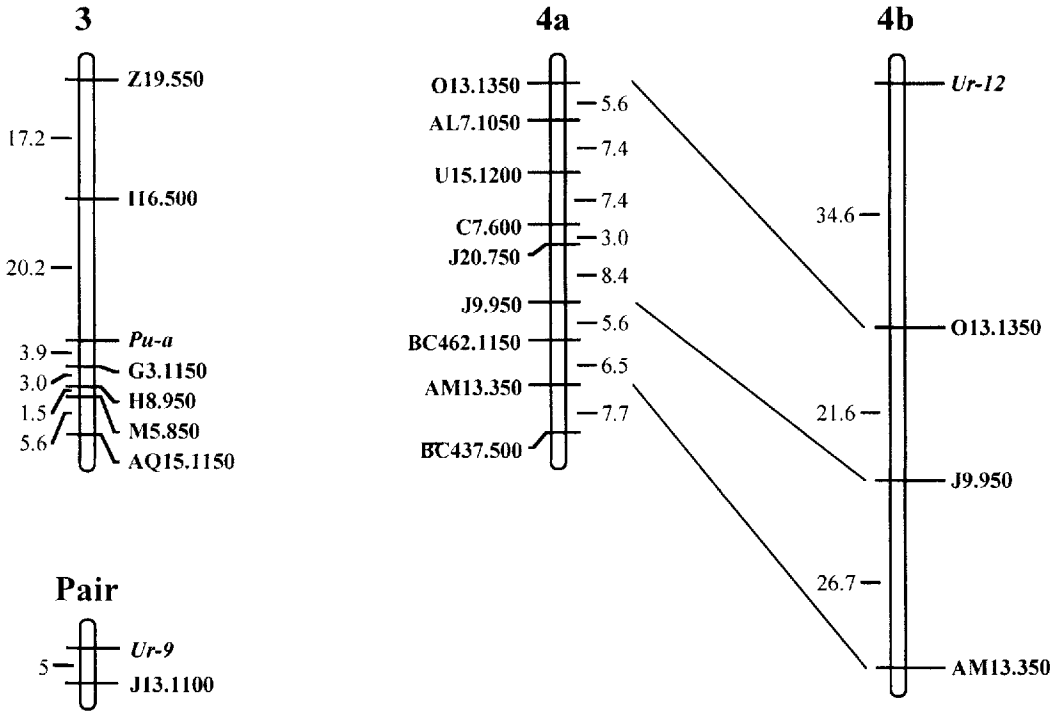

Fig. 1. Partial genetic linkage maps showing linkage groups 3 and 4 and a pair with the location of loci (in italic) for abaxial leaf pubescence (*Pu*-a) and for adult plant resistance (*Ur*-12) and specific resistance (*Ur*-9) to *Uromyces appendiculatus*, respectively. The genetic linkage group 4 constructed in a recombinant inbred population (4a) (Jung et al., 1997) and an F_2 population (4b) were compared. Shared markers with the same molecular weight between two linkage groups are connected with lines.

Mmbaga and Steadman, 1992; Mmbaga et al., 1996). However, some nearly-glabrous lines exhibited APR (Bokosi, 1996; Mmbaga et al, 1996). Thus both APR and ALP may contribute to durable rust resistance and will need to be selected in breeding programs.

The objectives of this study were to 1) identify RAPD markers linked to genes controlling SR, APR, and ALP and 2) investigate the genetic relationships between the genes APR and ALP, and SR and APR, in a recombinant inbred (RI) mapping population derived from 'PC-50' (presence of SR, APR, ALP) x XAN-159 (in absence of SR, APR, and ALP) in common bean.

Materials and Methods

Rust cultures. Seven different single uredinium rust pathotypes or races were used. A88T1-4b and A88T1-20a pathotypes were isolated from Tanzania. D82C1-1 and D82VC-74fh pathotypes were from the Dominican Republic (DR). PR911-19a was from Puerto Rico and U.S. races 49 and 50 were from Nebraska and North Dakota, respectively.

Mapping populations. Mapping populations of 68 F_6 recombinant inbred (RI) and 64 RI F_4BC_2 'PC-50' x XAN-159 lines from the cross 'PC-50' x XAN-159 were developed using the single seed descent breeding method (Arnaud-Santana et al., 1994). A partial genetic linkage map using RAPD markers was constructed in the F_7 RI derived from the above F_6 RI population (Jung et al., 1997). 'PC-50' has abaxial leaf pubescence (ALP), dense, long (1 to 2 mm), straight leaf hairs on third trifoliolate and above leaves and is resistant (*Ur*-9) (Finke et al., 1986) to *U. appendiculatus* pathotypes A88T1-4b, PR911-19a, D82VC-74fh, and races 49 and 50 but is susceptible to pathotypes D82C1-1 and A88T1-20a. It was derived from a single plant selection made by F. Saladin (unpublished, Ministry of Agriculture, DR) in the landrace Pompadour Checa in the Dominican Republic (DR). XAN-159 [McElroy (1985), Centro International de Agricultura Tropical (CIAT), Colombia] has an abaxial leaf surface that is nearly-glabrous and is susceptible to all the above pathotypes having no SR or APR.

A low number of RI lines segregating only for APR out of the 68 F_7 RI lines was due to the epistatic interaction of the SR on the APR (Bokosi, 1996). Thus, an F_2 segregating population of 86 individuals was developed from the cross between two homozygous RI lines selected for their unifoliolate leaf susceptibility to rust pathotype A88T1-4b and for resistance and susceptibility of their fourth trifoliolate leaves to this rust pathotype.

Phenotypic evaluation. In Fall 1994 the 68 RI F_7 lines and their parents were planted in a randomized complete block design with two replications (two plants per replicate) in a greenhouse in Lincoln, Nebr. The abaxial leaf surface of a unifoliolate leaf or fourth trifoliolate leaf of each plant was inoculated separately with $\approx 2 \times 10^4$ urediniospores of each selected rust pathotype/mL suspension of water with 40 $\mu L \cdot L^{-1}$ of Tween-20 using the multiple inoculation technique (Stavely, 1983). Inoculated plants were incubated in a mist chamber at 20 to 22 °C for 16 h. Plants were then removed from the chamber, transferred to greenhouse benches, and maintained at 21 to 25/20 to 23 °C day/night temperatures and a 12- to 14-h photoperiod. Rust reactions were recorded 14 d after inoculation using the rating scale reported by Stavely et al. (1983). Resistant reactions were a hypersensitive response (necrosis, no sporulation) and uredinia <300 µm; susceptible reactions were uredinia >300 µm. Also, abaxial leaf pubescence on the fourth trifoliolate leaf was recorded as present or absent.

Eighty-six F_2 progenies and parental F_7 RI lines were planted on 20 Sept. 1996, in a randomized complete-block design with two groups (two plants per pot per group) in a greenhouse in Lincoln. The unifoliolate and fourth trifoliolate leaves of each plant were inoculated with rust pathotype A88T1-4b. Inoculation procedures and disease ratings were performed as described above.

Bulked segregation analysis. Total genomic DNA was prepared from lyophilized fully expanded trifoliolate leaves of the 68 RI lines and the two parental lines and also prepared from fresh fully expanded trifoliolate leaves of the 86 F_2 plants mentioned above using the method previously described by Skroch and Nienhuis (1995). The polymerase chain reaction was used to generate RAPD (Williams et al., 1990) genetic markers for RI population in an air thermalcycler (model 1605; Idaho Technology, Idaho Falls) in thin-walled glass capillary tubes as described by Skroch and Nienhuis (1995) and for F_2 plants in 96-well plates in an MJ PTC 100 thermocycler (MJ Research, Watertown, Mass.) as described by Johns et al. (1997).

Bulked segregant analysis (BSA) (Michelmore et al., 1991)

was performed to identify RAPD markers tightly linked to target loci. Two DNA bulks for specific rust resistance in the F_7 RI lines were formed as follows. One bulk consisted of equal amounts of DNA from 8 F_7 individuals that carried the *Ur*-9 allele, and the other bulk was formed from the same number of F_7 individuals that were susceptible. The two DNA bulks, representing a contrasting F_7 phenotype pair, were screened with 600 single 10-mer primers of arbitrary sequence (Operon Technologies, Alamedia, Calif.). Random amplified fragments observed only in the resistant bulk were classified in the entire segregating population.

About 600 primers were tested against two separate DNA bulks formed from seven APR resistant and seven susceptible F_2 plants, respectively, as based on rust reactions of fourth trifoliolate leaves compared to unifoliolate leaves described above. Each segregating marker was named using its approximate size in base pairs, combined with the primer name as described by Jung et al. (1996).

BSA was not used to find markers for ALP (*Pu*-a) because the monogenic inheritance of the trait so linkage analysis could be performed between this trait and the markers identified previously (Jung et al., 1997).

Linkage analysis. The linkage analysis using 179 RAPD markers already mapped (Jung et al., 1997) plus an additional 100 RAPD markers and the gene (*Pu*-a) for ALP was performed using MAPMAKER Macintosh version 2.0 software (Lander et al., 1987). A LOD (log of the likelihood of the odds ratio that two genes are linked versus unlinked) score of 3.0 was used as a linkage threshold with 0.3 as the maximum recombination fraction for linkage groups in the RI population. An LOD score of 2.0 was used as a linkage threshold with 0.3 as the maximum recombination fraction for linkage groups in the F_2 population. An LOD score of 2.0 was used instead of 3 because of the loose linkage between *Ur*-12 and markers (Fig. 1, 4b). Map distances (centiMorgan, cM) were estimated using recombination fractions and Kosambi's mapping function (Kosambi, 1944) for ordered marker loci.

Results and Discussion

Inheritance of SR, APR, and ALP. For the cross 'PC-50' x XAN-159, segregation for reaction to rust pathotypes A88T1-4b, D82VC-74fh, PR911-19a, race 49, and race 50 gave a good-fit to a 1:1 ratio of resistant to susceptible lines with no recombinants for resistance and susceptibility, indicating that SR to the pathotypes was determined by a single major gene (*Ur*-9) (Table 1). All lines in the RI population were susceptible to rust pathotypes A88T1-20a and D82C1-1. 'PC-50', a bean of Andean origin with red mottled seed, most probably possesses a single gene or tightly linked block of single genes inherited as a single factor that confers specific resistance to the five different *U. appendiculatus* rust cultures. This is consistent with other rust resistances studied to date and found to be generally under monogenic control (Steadman et al., 1995). The lines from the cross F_4BC_2 'PC-50' x XAN-159 gave a good-fit to a 7:1 segregation ratio ($\chi^2 = 0.14$, $P = 0.71$) for specific resistance (57) to susceptibility (7) confirming that a single major gene controlled specific resistance (Bokosi, 1996).

Recombinant inbred lines with SR plus APR segregated in a 3:1 ratio due to an epistatic effect of SR on APR (Table 1). Bokosi (1996) observed a good-fit to a segregation ratio of 2:1:1 for SR to APR to S instead of an expected 1:1:1:1 for number of lines with SR plus APR to SR only to APR only and to all susceptible (no APR and no SR). It is postulated that a single major gene (*Ur*-12) controlled APR to pathotype A88T1-4b. APR was not expressed in the presence of SR. A good-fit to a 1:1 ratio of APR (12 lines) to S (16 lines) on the fourth trifoliolate leaves was detected despite the low number of available APR lines because of the epistatic interaction of SR on the expression of APR (Bokosi, 1996) (Table 1).

There was a good-fit to a 3:1 ratio of numbers of plants with APR (59) and susceptible plants (27) to rust pathotype A88T1-4b on the fourth trifoliolate in 86 F_2 plants of the cross between two RI lines derived from 'PC-50' x XAN-159 (Table 2). This indicated that APR in this cross was controlled by a single dominant gene and confirms the single gene hypothesis established on the basis of segregation in the RI lines. All unifoliolate leaves of these F_2 plants were susceptible to pathotype A88T1-4b. Although only one pathotype was used for APR testing, Mmbaga and Steadman (1992) found that 'PC-50' expressed APR against seven pathotypes of rust. However, subsequently Sandlin et al. (1995) found that some pathotypes can overcome APR. Although APR is effective against many pathotypes it is not race nonspecific.

A good-fit to a ratio of 1:1 for number of lines with pubescent to glabrous abaxial leaf surfaces was detected in 68 RI lines of the cross F_7 'PC-50' x XAN-159 (Table 1). It was hypothesized that abaxial leaf pubescence (ALP) was inherited as a single gene (*Pu*-a) in this cross. This single gene hypothesis was confirmed in the F_4BC_2 'PC- 50' x XAN-159 where a good-fit to 7:1 plants with

Table 1. Segregation ratios[z] for specific rust resistance (SR) on unifoliolate leaves, and also for adult plant resistance (APR) and susceptibility (S) and abaxial leaf pubescence (ALP) on fourth trifoliolate leaves in 68 recombinant inbred bean lines of the cross 'PC-50' (presence of SR, APR, ALP) x XAN-159 (absence of SR, APR, and ALP).

Trait	Observed no. of plants		Expected ratio	χ^2	P
SR[y]	SR	S			
	40	28	1:1	2.11	0.18
APR[x]	APR	S			
	12	16	1:1	0.57	0.57
SR and APR	SR + APR[w]	S			
	52	16	3:1	0.07	0.89
ALP	ALP	Glabrous (G)			
	40	28	1:1	2.11	0.18

[z]Data source: Bokosi, 1996.

[y]Phenotypic data using five *Uromyces appendiculatus* pathotypes (A88T1-4b, PR911-19a, D82VC-74fh, race 49, and race 50) showed the same segregation pattern. No recombination was detected.

[x]APR was exhibited by a smaller uredinium size (<300 µm) on the fourth trifoliolate compared to unifoliolate leaves when inoculated with pathotype A88T1-4b (Tanzania) and was recorded on RI lines uniform for susceptibility on unifoliolate leaves (absence of SR).

[w]SR + APR = plants with both resistances.

[v]χ^2 test for paired segregation of SR and ALP indicated independence of the genes *Ur*-9 and *Pu*-a ($\chi^2 = 4.471$, df3, $P \leq 0.22$) with 16 plants SR and ALP, 24 SR and G, 12 S and ALP, and 16 S and G.

Table 2. Segregation for adult plant resistance (APR) to rust in F_2 plants derived from a cross between two homozygous RI lines APR x susceptible selected from F_7 RI lines of the cross 'PC-50' x XAN-159 with both lines susceptible in unifoliolate leaves.

	Observed number of plants				
Trait	APR	Susceptible	Expected ratio	χ^2	P
APR[z]	59	27	3:1	2.84	0.21

[z]APR was recorded on F_2 plants uniform for susceptibility on unifoliolate leaves (no specific resistance) and exhibited by a smaller uredinium size (<300 μm) on fourth trifoliolate leaves compared to unifoliolate leaves when inoculated with *Uromyces appendiculatus* pathotype A88T1-4b (Tanzania).

pubescent leaves (58) and glabrous (6) was observed ($\chi^2 = 0.57$, P 0.45).

Mapping RAPD markers linked to SR, APR, and ALP. More than 100 RAPD markers collected later were added to the 179 RAPD markers already mapped in the RI lines of the cross 'PC-50' x XAN-159 by Jung et al. (1997). However, linkage was not detected between a gene (*Ur*-9) for SR and any of the mapped markers. Therefore, BSA method was used to tag markers linked to the *Ur*-9 gene. The RAPD marker J13.1100 detected using BSA method was linked in coupling phase at 5.0 cM from the *Ur*-9 gene for resistance (Fig. 1). Even though 18 out of the 600 primers screened amplified polymorphic bands between the two bulks, all except one turned out to be false positives.

Because of the low number of RI lines segregating for APR due to the epistatic effect of SR, an F_2 population consisting of 86 plants was developed from the cross of two RI lines segregating for APR but uniform susceptibility in the unifoliolate leaves and was used in order to tag a gene *Ur*-12 for APR. After screening 600 RAPD primers against the two bulks 36 of them were found to be polymorphic. However, the RAPD marker J9.950 was initially detected to be loosely linked to the gene *Ur*-12 using bulked segregant analysis. Since the same RAPD marker 79.950 also mapped to linkage group 4 previously constructed using the RI population (Jung et al., 1997) there was no need to screen more primers to find a more tightly linked marker using BSA. Therefore, nine markers mapped in linkage group 4 were tested between the two RI parents that were used to develop the F_2 population. Fortunately only two markers, O13.1350 and AM13.350, located at each end of the linkage group 4a, were tested and found to be polymorphic between two RI lines used for the construction of the F_2 population. Then a linkage analysis was performed using the gene (*Ur*-12) for APR and three RAPD markers in the F_2 population. The RAPD marker J9.950 was mapped to a location in linkage group 4b flanked on one side at 21.6 cM from marker O13.1350 and on the other side at 26.7 cM from marker AM13.350 in the same linkage group as shown in the RI population (Fig. 1). The *Ur*-12 gene for APR was linked at 34.6 cM from O13.1350 in the linkage group 4b. Further research is needed to find a RAPD marker with tighter linkage to the *Ur*- 12 gene for use in gene pyramiding to provide for more durable resistance to rust. This is the first report of the mapping of the gene for APR in common bean.

The *Pu*-a gene determined the presence of dense, long (1 to 2 mm), straight leaf hairs (pubescent) on the abaxial trifoliolate leaf surfaces of 'PC-50' or the absence of hairs (nearly-glabrous) on all leaf surfaces of XAN-159. The *Pu*-a gene was mapped to a location in linkage group 3 (Jung et al., 1997) flanked on one side at 20.2 cM from marker I16.500 and on the other side at 3.9 cM from marker G3.1150 (Fig. 1). The *Pu*-a gene was not linked with the *Ur*-12 gene.

Implications for breeding. Pyramiding different genes for rust resistance should provide more durable protection to the highly variable rust pathogen. For example, pyramiding genes *Ur*-3, *Ur*-4, and *Ur*-5 in one genotype would confer resistance to 63 of the 65 bean rust races that are currently in the USDA collection (Steadman et al., 1995) and could only be overcome by two races, 58 and 67. Additionally, the incorporation of different types of independently inherited resistance genes *Ur*-9 and *Ur*-12 in a line should be considered in developing more durable resistance. It is not known if these two genes are independent of the other designated rust resistant genes (Kelly et al., 1996). 'PC-50' was identified here as an additional source of SR and a new source of APR and could be used to complement the known rust resistant genes in common bean. Markers tightly linked to APR are still needed, however, to facilitate incorporation of this resistance into cultivars along with SR to obtain more durable resistance.

RAPD markers for major rust resistance genes such as *Ur*-3, *Ur*-3[2] (now tentatively named *Ur*-11 by J.R. Stavely, USDA, Beltsville, Md.), *Ur*-4, and *Ur*-5, have already been identified (Kelly, 1995). These RAPD markers may be utilized in marker-assisted selection (MAS) for monogenic resistance to different rust races in common bean. MAS should enhance selection efficiency and reduce the time-consuming procedures involved in extensive progeny testing needed to identify and incorporate epistatic genes as was found here between SR and APR. RAPD markers that flank the gene of interest should also facilitate selection for rust resistance (Young and Kelly, 1996). The tagged RAPD markers for the above different rust resistance genes should also be mapped in an integrated common bean molecular map to examine the relationships among these genes.

Efforts should be continued to gain understanding of the basis of and the inheritance of APR in other bean germplasm, the contributions of ALP as an avoidance mechanism to rust infections, and the epistatic interactions of different genes for SR on other SR and APR genes.

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