

## MARKER-ASSISTED SELECTION FOR DISEASE RESISTANCE IN COMMON BEAN

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Marker-assisted selection can provide an effective and efficient breeding tool for detecting, tracking, retaining, combining, and pyramiding disease resistance genes (for reviews see Kelly and Miklas, 1998 and 1999). For common bean, PCR-based RAPD and SCAR markers linked with more than 20 disease resistance genes have been obtained to date (Table 1).

Table 1. Resistance genes in common bean for which linked markers have been identified.

<u>Gene</u>	<u>Disease</u>	<u>Gene</u>	<u>Disease</u>	<u>Gene</u>	<u>Disease</u>
<i>Co-1</i>	Anthracnose	<i>I*</i>	BCMV	<i>Ur-3</i>	Rust
<i>Co-2</i>	“	<i>bc-I<sup>2</sup>*</i>	“	<i>Ur-4*</i>	“
<i>Co-4</i>	“	<i>bc-3</i>	“	<i>Ur-5*</i>	“
<i>Co-4<sup>2</sup>*</i>	“	<i>Phg-2</i>	Angular leaf spot	<i>Ur-6</i>	“
<i>Co-5</i>	“	<i>Mp-1</i>	Macrophomina	<i>Ur-7</i>	“
<i>Co-6</i>	“	<i>Mp-2</i>	“	<i>Ur-9</i>	“
		<i>bgm-1*</i>	BGYMV	<i>Ur-11</i>	“
				Ouro Negro	“

\*genes for which MAS has been applied in our program.

Note: an in depth list of SCAR markers linked with resistance genes and QTL in common bean is available on the web at: <http://www.usda.prosser.wsu.edu/Scartable3.htm>

The utility of many of these linkages for marker-assisted selection of the resistance gene or QTL, however, has not been demonstrated outside of the original mapping population. There are various reasons why some of the linked markers may not be useful or have restricted utility, including: i) the linkage is not tight enough, or the linkage intensity may vary widely across different genetic backgrounds due to recombination suppression, ii) the gene is not expressed in certain genetic backgrounds (for example *bc-I<sup>2</sup>* is not expressed in a recessive *i*-gene background that lacks *bc-u*), iii) the marker is difficult to assay in certain genetic backgrounds or using different PCR protocols and equipment, which may even be true for certain SCAR markers, iv) the gene is easier to screen for using the pathogen, v) the gene may have nominal effect and not be worthwhile retaining in a breeding program, and vi) the resistance-linked markers are present in susceptible lines or susceptible-linked markers are present in resistant lines, which can occur in a gene-pool or race within gene-pool specific pattern. For instance, the A14 RAPD marker linked with *Ur-4* was found to be present in all Andean germplasm lines tested, whether they were resistant or susceptible (Miklas et al., 1993). Conversely, A14 marker was absent in all Middle American germplasm lines lacking the *Ur-4* gene; therefore, use of this RAPD marker for indirect selection is restricted to the Middle American gene pool.

Markers linked with quantitative trait loci conditioning resistance to ashy stem blight, bean golden yellow mosaic virus (BGYMV), common bacterial blight (CBB), and web blight was reviewed recently by Kelly and Miklas, (1999). Since then, additional QTL conditioning resistance to CBB (Tar'an et al., 2001), white mold (Miklas et al., 2001; Park et al., 2001; Kolkman and Kelly, 2001), fusarium root rot (Schneider et al., 2001; Chowdury et al., 2002), fusarium wilt (Fall et al., 2001),

and halo blight (Ariyaratne et al., 1999) have been tagged. SCARs are available for MAS of four CBB, one white mold (Miklas et al., 2001), and one BGYMV QTL (Miklas et al., 2000). Unequivocal evidence for effective MAS of these QTL, however, has only been demonstrated for the CBB resistance QTL linked with the SU91, BC420, and SAP6 SCAR markers (Jung et al., 1999; Miklas et al., 1999 and 2000; Park et al., 1999; Yu et al., 2000; Fourie and Herselman, 2002; Mutlu et al., 2002). Some specific applications of MAS for disease resistance in bean are mentioned below.

Bean rust is a hyper-variable pathogen that can rapidly overcome newly deployed resistance genes. PI 181996 was found to be resistant to 89 rust races. The resistance was conditioned by the *Ur-11* gene. This gene was quickly deployed into most common bean market types by Stavelly et al. (1997). *Ur-11* is epistatic to less effective resistance genes like *Ur-4* and *Ur-5*. Linked markers are useful for retaining these defeated genes in the presence of a broadly effective gene like *Ur-11*. The A14 marker was used to select those *Ur-11* lines which retained the *Ur-4* gene (Stavelly et al., 1994). The *Ur-4* + *Ur-11* combination was later found to hold up against a newly identified race in Honduras, whereas *Ur-11* by itself was susceptible.

Gene(s)	Races (No.)	
	R	S
<i>Ur-4</i>	22	57
<i>Ur-11</i>	89	1
<i>Ur-4</i> + <i>Ur-11</i>	90	0

Similarly, the hypostatic *I* gene is retained in the presence of the *bc-3* gene by MAS for the SW13 SCAR (Melloto et al., 1996; Miklas et al., 2002). This combination of a dominant and a recessive gene, likely possessing different resistance mechanisms, should provide more durable resistance to bean common mosaic virus.

Linked markers can also be used to quickly deploy a resistance gene into an adapted background. The R2 codominant RAPD marker identified by Urrea et al. (1996), and later converted to a SCAR by CIAT (S. Beebe), was used to backcross the *bgm-1* recessive resistance gene into snap bean (Stavelly et al., 1997), with the pole bean cultivar Genuine a direct result of this effort. The marker is widely used by CIAT for MAS in early generations (F<sub>1</sub> gamete) because of the recessive inheritance of *bgm-1*, and because the disease can be difficult to screen for in field and greenhouse environments (S. Beebe, personal communication).

Backcross scheme for introgressing *bgm-1* into snap bean via MAS:

Generation	Cross
F <sub>1</sub>	A 429 ( <i>bgm-1</i> ) x snap bean cultivar ( <i>Bgm-1</i> )
BC <sub>1</sub>	F <sub>1</sub> ( <i>Bgm-1</i> // <i>bgm-1</i> ) x snap bean cultivar
BC <sub>2</sub> *	1/2 BC <sub>1</sub> F <sub>1</sub> ( <i>Bgm-1</i> // <i>bgm-1</i> ) x snap bean cultivar
BC <sub>3</sub> *	1/2 BC <sub>2</sub> F <sub>1</sub> ( <i>Bgm-1</i> // <i>bgm-1</i> ) x snap bean cultivar
BC <sub>3</sub> F <sub>1</sub> *	1/2 BC <sub>3</sub> F <sub>1</sub> ( <i>Bgm-1</i> // <i>bgm-1</i> ) is selfed
BC <sub>3</sub> F <sub>2</sub>	25% R ( <i>bgm-1</i> // <i>bgm-1</i> ) and 75% S ( <i>Bgm-1</i> // <i>Bgm-1</i> or <i>bgm-1</i> ) as expected

\* denotes generation where MAS was used.

A similar MAS-backcrossing scheme was used to rapidly introgress the *Co-4<sup>2</sup>* resistance gene into pinto bean to combat the emerging anthracnose disease problem in North Dakota (Miklas and Kelly, 2002). *Co-4<sup>2</sup>* is the most effective anthracnose resistance gene

characterized to date (Balardin and Kelly, 1998).

Use of linked markers for indirect selection of quantitative resistance traits, is more difficult because QTL generally have minor cumulative effects, and are greatly influenced by environment and genetic background. For these reasons most studies have focused on identifying markers linked with “major-effect” QTL because they offer the best opportunity for MAS. For example the SCAR markers linked with the major-effect QTL for CBB resistance have been observed to singly explain from 20 to 80% of the variability for disease resistance in segregating populations.

The utility of a SCAR marker for MAS of a major-effect QTL for BGYMV resistance was partially validated in a set of advanced lines with resistance derived from a similar source; however, direct use of SW13 for MAS of the resistance has not yet been demonstrated (Singh et al., 2000). The SCAR marker (*Phs*) linked with white mold resistance, detects a major-effect QTL that is expressed in both greenhouse (38%) and field (26%) environments, but successful MAS for the QTL has not yet been reported (Miklas et al., 2001). The RAPD marker linked with fusarium wilt resistance has not been converted to a SCAR yet, but the QTL should be amenable to MAS because it explains 63% of the variation for disease reaction in the original mapping population (Fall et al., 2001).

Once the limitations of a marker for the purpose of indirect selection have been determined, and its effective use for MAS outside the original mapping population validated, it can become an invaluable tool in disease resistance breeding as highlighted above. Integration of the resistance genes and QTL into the core map should be conducted, but if unsuccessful should not circumvent publication of important findings. Future QTL mapping studies should attempt to use larger segregating populations to enable better resolution of minor-effect QTL and better characterization of gene clusters. Once an important QTL is found, the region should be saturated with markers using phenotype- and map-based bulked-segregant analysis in an effort to obtain tightly linked flanking markers. Further fine-mapping, using BACs for development of contigs, may eventually lead to identification and perhaps cloning of the gene responsible for the QTL. As more resistance-linked markers are found, characterized and mapped, the power of MAS for developing more durable and multiple disease resistant cultivars will increase substantially in dry and snap bean breeding programs across the world.

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