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Development and Application of Molecular Markers to Breed Common Bean (*Phaseolus vulgaris* L.) for Resistance to Common Bacterial Blight (CBB) – Current Status and Future Directions

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1. Introduction

Common bean (*Phaseolus vulgaris* L.) is grown and consumed principally in developing countries in Latin America, Africa, and Asia. It is a major source of dietary protein that complements carbohydrate-rich sources such as rice, maize, and cassava. It is also a rich source of dietary fibres, minerals and certain vitamins (Gepts et al.2008). Common bacterial blight (CBB), incited by *Xanthomonas axonopodis* pv. *Phaseoli* (Smith) Dye (*Xap*), is one of the most destructive bacterial diseases of common bean. CBB is a seed-transmitted disease that is a major yield-limiting factor of common bean production worldwide. CBB can reduce seed quality through staining and browning, which render the bean seed unacceptable for the food processing industry (Yu et al., 2000a). Currently, CBB outbreaks are managed through the use of expensive pathogen free seeds reproduced in certain locations (Scott & Micheals, 1992) and seed treatment with antibiotics such as Streptomycin or via foliar spraying with copper-based compounds (e.g. Kocide™) that are not only costly, but partially effective, and have serious long-term consequences on human and animal health (Forbes & Bretag, 1991; Fininsa, 2003). The exploitation of natural resistance to CBB is the only effective and environmentally sound approach to control this disease in bean production. Sources of genetic resistance to CBB have been identified in common bean and its related species, tepary bean (*P. aculifolius*) and runner bean (*P. coccineus*), but most of them are inherited as quantitative trait loci (QTL) and vary in their levels of genetic effects and their expressions are influenced by environmental conditions (Kelly et al.2003; Miklas et al. 2006). However, dominant gene controlling CBB resistance in common bean was also reported (Zapata et al. 2010). Conventional breeding for resistance to CBB is further aggravated by the pathogen variability, linkage of resistance with undesirable traits (Liu et al. 2008), and different genes conditioning resistance in different plant organs, including leaves, pods, and seeds (Jung et al. 1997; Liu et al., 2009; Aggour et al., 1989; Mutlu et al., 2008; López et al., 2006; Mkandawire et al., 2004; Zapata, 1997). The advent of DNA-based molecular marker (MM) technology has provided an efficient selection tool to breeders in plant breeding (Tanksley et al. 1989). Molecular marker can be defined as a gene or a piece

of DNA that is located on a chromosome and can be used as a point of reference (Weber & Wricke, 1994). Since DNA-based MM is phenotypic and environmental neutral, and can be accurately and automatically analyzed with little quantity of DNA (nanogram) in a laboratory at any time for any plant tissue, they can reduce the breeding cost and improve the selection efficiency. Significant progresses on the development and application of MMs to breed bean for CBB resistance have been made in recent years. In this chapter, we will review the current status on the development and application of MMs for CBB breeding in common bean and discuss the future prospects of research in this area.

2. Genetic resistance sources of the CBB resistance gene(s)/or QTL

CBB resistance bean plants can somehow decrease or stop the movement of the *Xap* pathogen through vascular tissues, which can abate the accumulation of bacterial population in leaves or barricade internal seed infection (Goodwin et al., 1995; Aggour & Coyne,1989). Genetic resistance to CBB is relatively low in common bean in comparison with its related species, scarlet runner bean and tepary bean (Singh & Munoz, 1999; Coyne and Schuster, 1973; Yoshii et al., 1978; Mohan, 1982).

Although the genetics of CBB resistance in *P. coccineus* are largely unknown, low to moderate levels of resistance from *P. coccineus* were transferred to common bean (Table 1; Freytag et al., 1982; Park & Dhanvantari, 1987; Miklas et al., 1994; Miklas et al., 1999; Singh & Munoz, 1999). The CBB resistance source for the bean line XR-235-1-1 released by Freytag et al., (1982) was from scarlet runner (*P. coccineus*) bean line PI273667 (Ethiopia). Two minor CBB resistance QTL, derived from PI273667, were identified in the XR-235-1-1 bean line and together they explained about 27% of the phenotypic variation for CBB resistance (Yu et al., 1998). Four CBB resistance lines, C1, C2, C3 and C4 were developed by inter-specific crosses between Common bean and *P. coccineus* (Park and Dhanvantari, 1987). The CBB resistance source for the multiple disease resistance line TARS VCI-4B released by Miklas et al. (1994) was from two *P. coccineus* PI lines, PI311950 and PI311977 (Mexico). Miklas et al. (1999) released four CBB resistant bean lines, ICB-3, ICB-6, ICB-8 and ICB-10, which derived their CBB resistance either from *P. coccineus* or from Great Northern varieties. Singh and Munoz (1999) screened 166 promising germplasm accessions of four *Phaseolous* species including 55 Scarlet Runner beans and found that moderate resistance present in *P. coccineus*. Except the two CBB resistance QTL in XR-235-1-1 was mapped to linkage group A (chromosome 7) and F (chromosome 8) (Yu et al., 1998), the rest of the CBB resistance genes / or QTL from *P. coccineus* are unknown.

Because tepary bean has the highest level of resistance to CBB (Arnaud-Santana et al., 1993; Coyne and Schuster, 1983; Mohan, 1982; Schuster et al., 1983; Singh and Munoz, 1999; Zapata et al., 1985), efforts have been made successfully to transfer the genetic factors controlling CBB resistance from tepary bean into common bean through inter-specific hybridizations (Thomas and Waines, 1984; McElroy, 1985; Parker, 1985; Scott & Michaels, 1992; Singh & Munoz, 1999). The XAN lines, XAN-159, XAN-160 and XAN-161 were developed at CIAT (Centro International de Agricultura Tropical) through inter-specific crosses between *P. vugaris* and *P. acutifolius* accession PI319443 (Thomas and Waines, 1984; Jung et al., 1997). PI319443 showed quantitative inheritance, predominately additive effects, and partial dominance for CBB resistance (McElory, 1985). Based on the CBB resistance data, McElory (1985) hypothesized that one prominent major gene and two other minor genes controlling CBB resistance in PI319443 (McElory, 1985), and the XAN-159, derived its CBB

resistance from PI319443, is likely to obtain most of the genes. Parker (1985) transferred CBB resistance from tepary bean into *P. vulgaris* by hybridizing PI440795 (*P. acutifolius*) to 'ICA Pijao' (*P. vulgaris*) and crossing the F_1 progeny to 'Ex Rico 23' (*P. vulgaris*). CBB resistance in both OAC-88-1 line and OAC- Rex Cultivar are originated from PI 440795 (Scott & Michaels, 1992; Michaels et al., 2006). The VAX lines, VAX1, VAX2, VAX3, VAX4, VAX5 and VAX6, were developed from inter-specific cross between the common bean cultivar ICA Pijao and the tepary bean accession G40001 in 1989 by embryo rescue (Mejia-Jimenez et al., 1994; Singh & Munoz, 1999). By far, tepary bean PI319443, PI44079, and G40001 (Table 1) are sources of the major CBB resistance gene(s) /or QTL that have been incorporated into common bean breeding programs (Yu et al., 2000a; Yu et al., 2004; Liu et al., 2008; Bai et al., 1997; Tar'an et al., 2001; Jung et al., 1997; Miklas et al., 2000; Singh & Munoz, 1999; Pedraza et al., 1997).

Miklas et al. 2003, however, revealed that the major CBB resistance gene(s)/or QTL present in the great northern cultivars GN# 1 and GN#1 Selection 27 (GN#1Sel 27) was actually derived from common bean cultivar, Montana No. 5 that was released as a cultivar in 1947 from a selection out of the common great northern landrace (Sutton & Coyne, 2007). CBB resistance gene(s) / or QTL were also identified from the Mesoamerica bean line BAC 6 (Jung et al., 1996). A dominant gene conferring resistance to CBB was recently found in the small white bean line PR0313-58 (Zapata et al. 2010). Four CBB resistance QTLs were also reported in the Middle American breeding line BAT93 (Nodari et al., 1993b). Table 1 summarized the major CBB resistance sources commonly used by common bean breeders.

3. Genomic mapping of bean with MMs

A genetic map is a graphic representation of the arrangement of a gene or a MM on a chromosome, which can be used to locate and identify the gene or group of genes that determines a particular inherited trait. If a linkage between a MM and a trait is established, the MM can be used to carry out indirect selection of the trait in a plant breeding program. The first comprehensive genetic map of bean was developed in 1992 with a seed and flower color marker (P), nine seed protein, nine isozyme and 224 restriction fragment length polymorphism (RFLP) marker loci. Eleven linkage groups (LGs), corresponding to the *P. vulgaris* L. (n = 11), were established in the backcross population derived from the cross between the Mesoamerican breeding line XR-235-1-1 and the Andean cultivar, Calima. The 11 linkage groups covered 960 centiMorgans (cM) of the bean genome (Vallejos et al., 1992). The second bean genetic map was constructed in an F_2 population from the cross of BAT93/Jalo EEP 558 (BJ) with 152 MMs, and 143 of the 152 MMs were assigned to 15 LGs, which covered 827 cM of the bean genome (Nodari et al., 1993a). Adam-Blondon et al. (1994) developed the third bean genetic map with 51 RFLP, 100 random amplified polymorphic DNA (RAPD), and 2 sequence characterized amplified region (SCAR) loci in a backcross population derived from the Ms8EO2 x Corel cross. Twelve LGs were formed in this map that covered 567.5 cM of the bean genome.

Because there are different breeding objectives in bean, which include crop quality, disease and pest resistance, tolerance to abiotic stresses, domestication syndrome, etc. (Gepts et al., 1998, 1999), no single mapping population would segregate for all the economic traits of interest. Thus, genes for these traits have been mapped to different segregating populations. In addition, each map was constructed with most of markers that are not common among them. Therefore, there is a need to compare among these maps to determine the linkage

Origin	cv. or lines	Gene/QTL number	MM	Explained phenotypic variation (R^2)	Reference
Resistance source from common bean (*P. vulgaris*)					
Montana No. 5	GN#1		SAP6	35%	Miklas et al. 2003
	GN#1Sel 27				
BAC6		3	BC409	12%(first trifoliate leaves); 13%(later-developed trifoliate leaves); 17%(pods)	Jung et al. 1996, 1999
			K19		
BAT 93		4		75% (all QTLs)	Nodari et al. 1993b
Belneb RR-1		4	W10	44% (first trifoliate leaves); 41% (pods)	Ariyarathne et al., 1999
	PR0313-58	1	SAP6	55%	Zapata et al. 2010
Resistance source from tepary bean (*P. acutifollus*)					
PI 440795	OAC-88-1	2	R7313	81% (all QTLs)	Bai et al. 1996
	OAC-Rex	3	PV-ccct-001	42.2%(One QTL)	Tar'an et al. 2001
PI 319443	XAN 159	4	BC420 SU91	28% (leaves); 11% (pods); 18% (seeds) 17%	Jung et al. 1997 Mutlu et al. 2005a
	HR 45	2	BC420	25-52%	Liu et al. 2008
	HR 67	2	BC420	70%	Yu et al. 2004
G40001	VAX 1				Singh & Munoz, 1999
	VAX 2				
	VAX 3				
	VAX 4				
	VAX 5				
	VAX 6				
Resistance source from scarlet runner bean (*P. coocineus*)					
PI 273667	XR235-1-1	2		27%	Yu et al. 1998
PI 311950 PI 311977	TARS VCI-4B				Miklas et al. 1994
PI 165421	C1				Park & Dhanvantari, 1987
	C2				
	C3				
	C4				

Table 1. The genetic origins of the major CBB resistance gene(s) /or QTL of common bean (*P. vulgaris*) derived from the *Phaseolus* species. The major QTL and the associated MM present in the genotypes are also shown if they are available.

relations for the different genes and MMs located on the different maps. A deliberate effort was therefore initiated to make a consensus map by determining the segregation in a core mapping population of a few markers selected from each linkage group (LG) on the different maps (Freyre et al., 1998; Kelly et al., 2003). The recombinant inbred line (RIL) population of the cross between the Middle American genotype BAT93 and Andean genotype Jalo EEP558 (BJ) was chosen to develop the integrated core map because it displayed high level of polymorphisms for RFLP and other MMs (Nodari et al., 1992; McClean et al., 2002), and segregated for multiple host–microorganism interactions (Nodari et al., 1993b; Geffroy et al., 1999). Two or more markers from the Florida map (Vallejos et al., 1992), the Paris map (Adam-Blondon et al., 1994), the Davis map (Nodari et al., 1993a) and the Nebraska–Wisconsin maps (Jung et al., 1996, 1997–1999) were mapped in the BJ population. This approach allowed researchers to correlate and align the different LGs of these maps and to establish the collinear relationships of the different LGs among the maps with more than two markers being mapped per LG (Freyre et al., 1998; Gepts, 1999). Although no detailed correlation (i.e., over short distances of the order of 10–15 cM or less) among the LGs of the different maps was provided, the information developed thus far allows at least a rough comparison of the location for genes or QTL placed on independent maps (Kelly et al., 2003). The total map length of the consensus or core map is approximately 1200 cM (Gepts, 1999) and the average relationship between genetic and physical distances is approximately 500 kb/cM, which was verified around the *Phs* (phaseolin) locus on LG B7 (Llaca and Gepts, 1996). The core map is composed of some 550 markers, including RFLP, RAPD, SCAR, allozyme, and seed protein markers; when considering markers from the other, correlated maps, at least 1000 markers have been mapped which would average one marker per 1–2 cM (Freyre et al., 1998; Gepts, 1999; Kelly et al., 2003, Miklas et al., 2006).

Another development in the genetic mapping of the bean genome includes the development of simple sequence repeat (SSR) or microsatellite markers (Yu et al., 1999, 2000b; Blair et al., 2003; Melotto et al., 2005; Hanai et al., 2007). The first preliminary study of the existence of microsatellite markers in the bean genome was conducted by Yu et al. (1999, 2000b), who were able to map 15 of the SSR markers onto the core linkage map. One hundred fifty SSR markers derived from either gene-coding or anonymous genomic-sequences were developed by Blair et al. (2003) and 100 of them were mapped to an F_9 RIL population of the DOR364 x G19833 cross and the BJ core mapping population (Freyre et al., 1998). These loci are integrated with the 15 microsatellite markers previously mapped to seven chromosomes on the BJ population (Yu et al., 2000b). Therefore these studies bring to a total of 115 microsatellite loci on the common bean genetic map, and provides coverage for every chromosome in the genome with from five to 20 SSR markers each (Blair et al., 2003). Two hundreds forty-three SSR sequences were identified from 5,255 bean expressed sequence tag (EST) sequences (Melotto et al., 2005) and 471 SSRs were found from 714 genomic sequences in a microsatellite-enriched genomic library (Hanai et al., 2007). These SSR markers will be invaluable for fine mapping the bean genome and tagging genes of economic interest.

Single nucleotide polymorphisms (SNPs) discovery and marker development is the current focus of several genetic mapping efforts. SNPs are by far the most common form of DNA polymorphism in a genome (Hyten et al., 2010). It was estimated that common bean genome has one SNP per 88 bp (Gaitán-Solís et al., 2008). With an estimated bean

genome size of 588 Mb (Liu et al., 2010), over 6 millions of SNPs would present in the entire bean genome. Three hundreds eighteen SNPs were detected from 5,255 bean EST sequences (Melotto et al., 2005). With EST sequence information from two bean genotypes, the Mesoamerican cultivar Negro Jamapa 81 and the Andean cultivar G19833 (Ramirez et al., 2005) , over 1,800 SNPs and indels were detected (McConnell et al., 2010). PCR primers were designed to amplify the BAT93 and Jalo EEP558 fragments and 534 useful fragments with an average size of 500bp were sequenced. Of the 534 gene fragment, 395 (74%) were polymorphic between BAT93 and Jalo EEP558. Three hundreds of the 395 gene fragments (most of them have at least one SNP) were mapped on the BJ core map population (McConnell et al., 2010). The genetic map (LOD 2.0) composed of the 300 gene-based markers, 103 core map markers and 24 other markers covered 1545.5 cM of the bean genome (McConnell et al., 2010). Using a modified deep sequencing of reduced representation library (RRL) (Van Tassell et al., 2008) approach, named as Muli-tier RRL, Hyten et al (2010) discovered 3487 SNPs from the parental lines, BAT 93 and Jalo EEP 558, which was used to develop the bean core map RIL population (Freyre et al., 1998). Because of the abundance of SNPs present in the bean genome, it is expected that SNPs will soon become the most commonly used MMs in bean mapping and breeding programs. Because the SNPs discovered so far are mainly from bean genotypes between, rather than within, the bean gene pools, one limitation of using these SNPs in common bean breeding programs, however, is the lack of polymorphisms (about 10%) of these SNPs between parental lines within each of the bean market classess (Shi et al., 2011b). Therefore, discovering SNPs within gene pool is necessary for routine application of SNPs in bean improvement.

Bacterial artificial chromosome (BAC) libraries are important resources for physical mapping and the development of molecular markers (Yu, 2011). The Development of SSR markers from BAC-end sequences is very cost-effective and offers genome-wide coverage as all repeat types are systematically sampled in the randomly selected BACs (Cho et al., 2004). BAC libraries were constructed for 10 common bean genotypes (Table 2). The first common bean BAC library was developed by Vanhouten & MacKenzie (1999) with the Sprite snap bean-derived genotype for physical mapping of the nuclear fertility restorer *Fr* locus. In 2006, four BAC libraries, three for common bean genotypes BAT93, G21245, G02771, and one for lima bean, cv Henderson (*P. lunatus*), were developed to study the evolution of the arcelin–phytohemagglutinin–a-amylase inhibitor (APA) multigene family (Kami et al., 2006) The four BAC libraries have a range of 9-20 fold genome coverage that should make them useful genetic resources for studying common bean and lima bean. The BAT 93 BAC library has been used successfully for cytogenetic studies of bean chromosomes (Fonseca, et al. 2010; Pedrosa-Harand et al. 2009). BAC libraries were also developed for common bean genotypes G19833 (Schlueter et al., 2008), G12949 (Galasso et al., 2009), HR45 (Liu et al., 2010), G02333 (Melotto et al., 2003), HR67 and OAC-Rex (Gepts et al., 2008).

The G19833 BAC library was used for BAC end sequence analysis to develop BAC derived SSR markers and for physical mapping of the common bean genome (Fonseca, et al., 2010; Córdoba et al., 2010). Liu et al. (2010) used the HR45 BAC library to physically map the major CBB resistance QTL of common bean to the end of chromosome 6. Currently, the OAC-Rex BAC library is being used to sequence the whole genome of the CBB resistance cultivar, OAC-Rex (Pauls, et al. personal communication) and whole genomic sequencing of G19833 is also under way (McClean et al. personal

communication). However, the potential utilization of the bean BAC libraries for MM development has not been fully explored in bean.

P. vulgaris L. (Common bean)	Genotype	Vector	R-site[a]	No. of clones	A.I-size[b] kb	Cov[c].	Ref.
	Sprite	pECSBC4	RcoRI	33,792	100	5.3	Vanhouten, & MacKenzie.1999
	Bat 93	pIndigoBac5	HindIII	110,592	125	21.7	Kami et al. 2006
	G21245	pIndigoBac5	HindIII	55296	105	9.2	Kami et al. 2006
	G02771	pIndigoBac5	HindIII	55296	139	12.1	Kami et al. 2006
	G12949	pIndigoBac5	HindIII	30720	135	6.5	Pedrosa-Harand et al. 2009
	G19833	pIndigoBac 536	HindIII	55296	145	12.6	Schlueter et al. 2008
	HR45	pIndigoBac5	HindIII	33,024	107	5.5	Liu et al. 2010
	HR67	BIBAC2	BamHI	22,560	300	10.6	Gepts et al. 2008
	OAC-Rex	BIBAC2	BamHI	31,776	150	7.5	Gepts et al. 2008
	G02333	pBeloBac11	HindIII	24,960	125	4.9	Galasso, et al. 2009

Table 2. BAC libraries developed for common bean genotypes. [a] R-site is restriction cutting site for cloning; [b] A.I.-size is the average insert size of the BAC library in kilo bases; [c] Cov is the times of genome equivalent coverage.

Cytological analysis of the common bean chromosomes has long been hampered by the small size and overall similarity of its (2n=22) chromosomes. Unambiguous identification of each bean chromosome was not possible until the double fluorescent in situ hybridization (FISH) with 45S and 5S rRNA probes, followed by 4'-6-diamidino-2-phenylindole (DAPI) counterstaining techniques were developed (Moscone et al., 1999). The first common bean mitotic chromosome nomenclature was proposed by Moscone et al. (1999). Chromosomes were characterized with respect to size, morphology, heterochromatin content and distribution of rDNA genes by fluorescent in situ hybridization (FISH) and assigned numbers from 1 to 11, based on size from largest to smallest, using the European cultivar 'Wax' as a reference (Pedrosa et al. 2003). Because the numbering of linkage groups according to Freyre et al. (1998), B1 to B11, has been widely used by the bean community, it was agreed during the Phaseomics III meeting in 2004 that chromosomes should be reassigned numbers based on the linkage group nomenclature (Pedorsa et al., 2008). The integration of the common bean LGs and the chromosome map was a breakthrough in common bean genetics. The standard nomenclature can be found in the BIC website (http://www.css.msu.edu/bic/Genetics.cfm). In two recent studies, a cytogenetic map of common bean was built by FISH of BACs selected with markers mapping to the 11 linkage groups, plus 2 plasmids for 5S and 45S ribosomal DNA and one bacteriophage (Pedrosa et al., 2009; Fonseca et al., 2010). It was found that about 50% of the bean genome is heterochromatic and that genes and repetitive sequences on the bean chromosomes are intermingled in the euchromation and heterochromatin.

4. Molecular tagging of CBB resistance gene(s) /or QTL and application of MMs in bean breeding

The positioning of MMs linked to CBB on the bean genetic maps has revolutionized our understanding of resistance to CBB (Kelly et al., 2003). Nodari et al. (1993) used 150 restriction fragment length polymorphism (RFLP) markers and 70 F_2-derived F_3 families of the BAT 93 X Jalo EEP558 cross to map the genetic factors controlling CBB resistance present in BAT 93. They identified 4 putative QTLs located on 4 different linkage groups. These four QTLs explained 75% of the phenotypic variation for CBB resistance. Random amplified polymorphic DNA (RAPD) markers were used by Jung et al. (1996) to tag the QTL in a recombinant inbred line (RIL) population derived from the cross between BAC 6 x HT 7719. The CBB resistance of this RIL population was from the BAC 6 Mesoamerican bean line. RAPD marker BC409.1250 was found to associate with a major QTL conditioning CBB resistance to the *Xap* EK-11 strain in first trifoliate leaves (account for 12% of the phenotypic variation), later-developed trifoliate leaves (13% of the phenotypic variation), and pods (17% of the phenotypic variation). Another QTL conditioning CBB resistance to the *Xap* Epif-IV strain in the later-developed trifoliate leaves (10% of the phenotypic variation) was found to link with RAPD marker U16.600-H11.650. Other QTL were also detected but they were of less significance (account for less than 10% of the phenotypic variation). MMs associated with QTL of less effect are usually not very effective in MAS. The two genomic regions that enclose the CBB resistance QTL were confirmed later with 3 additional mapping populations and 1 additional *Xap* strain (Jung et al., 1999). It is interesting that the BC 409.1250 RAPD marker was significantly associated with CBB resistance to all 3 *Xap* strains (EK-11, Epif-IV, and DR-7) in all the 4 populations {BAC 6 x HT 7719 (BH), Venezuela x BAC6 (BV), BeIneb RR-1 x A55 (BA), and PC50 x BAC6 (PB)}. The BC 409.1250 marker, however, was not linked to any of the other markers used in their experiments (Jung et al., 1996, 1999).

The other genomic region tagged by U16.600-H11.650 in the BH population (Jung et al., 1996) was also confirmed in 3 of the 4 populations (BH, BA, and PB). Two RAPD markers, K19.450 and AG15.660, were mapped to the same location across the 4 populations (Jung et al., 1999). The BC409.1250 RAPD marker was converted into a sequence characterised amplified region (SCAR) marker (Paran & Michelmore, 1993) for reliable PCR amplification in MAS of bean breeding programs (Jung et al., 1999; Ariyarathne et al., 1999). The two genomic regions tagged, respectively, by BC409.1250 and K19.450 (Jung et al.,1996, 1999) was later mapped to the same linkage group (LG) B10 by Ariyarathne et al. (1999). Another RAPD marker, W10.550, located in the same LG was also identified, which is tightly associated with the CBB resistance QTL in first trifoliate leaf and pod. This QTL explained up to 44% and 41% of the phenotypic variation in first trifoliate leaf and pod, respectively (Ariyarathne et al., 1999). These results provideed strong evidence that more than one QTL are located on the bean linakge group 10 (Chromosome 10). The SCAR marker, derived from the BC409.1250 RAPD marker, was named as BAC 6 SCAR marker, which is one of the commonly used MM in MAS of bean for CBB resistance (http://www.css.msu.edu/bic/PDF/SCAR_Markers_2010.pdf).

By using selective genotyping and bulked segregant analysis (Michelmore et al., 1991), Miklas et al. (1996) detected two genomic regions affecting CBB resistance with an $F_{5:7}$ RIL population derived from the Dorado x XAN 176 cross. One genomic region defined by RAPD marker AP6.820 on LG PR14 explained 60%, 10% and 30 of the phenotypic

variation in greenhouse-leaf, field-leaf and greenhouse-pod reactions, respectively. The AP6.680 RAPD marker was mapped to the end of LG 10 by Miklas et al. (2000a) and converted to the SCAR marker SAP6.680 (Miklas et al., 2003) which was positioned between the W10.550 and the BAC 6.1250 markers on LG10 in the BAC 6/HT 7719 population (Ariyaranthe et al., 1999). So there is a good chance that both the SAP 6.680 and the BAC 6.1250 markers are actually linked to the same QTL (Miklas, personal communication). The other genomic region on LG PR7 or B7 defined by A19.600 explained 12% and 40% of the phenotypic variation in greenhouse-leaf and field-leaf reaction. The occurrence of common genomic region affecting CBB resistance in different tissues and under different environmental conditions indicate that the expression of CBB resistance may be conditioned by more than one genetic factors clustered together or by one genetic factor with pleiotropic effect.

Jung et al. (1997) identified four genomic regions present in XAN 159, a bean line was developed by inter-specific crossing between tapary bean PI319443 and common bean, conditioning CBB resistance at first trifoliate leaves, pods and seeds. One genomic region on their LG 5 defined by BC420.900 RAPD marker was found to contain a major QTL conferring CBB resistance to *Xap* strain DR-7 and EK-11 in leaf (accounted for 28% of the phenotypic variation), pod (11%) and seed (18%). The CBB resistance in XAN159 was successfully transferred into common bean by Park & Dhanvantari (1994) and Park et al. (2007) to develop the HR45 and HR67 bean germplasm, which are highly resistant to CBB in both field and greenhouse test (Yu et al., 2000a; Liu et al., 2008). The major QTL of XAN159 origin linked with BC420.900 RAPD marker was confirmed by Yu et al. (2000a) in a RIL population derived from the HR 67 x W1744d cross. This QTL explained 62% of the phenotypic variation in CBB reaction tested in the greenhouse environment. The BC420.900 RAPD marker was also converted into a SCAR marker, which is one of the most commonly used markers for MAS in breeding bean for CBB resistance (Yu et al., 2000a; Liu et al., 2008; Kelly et al., 2003; Miklas et al., 2006; Fourie and Herselman 2002, Park and Yu 2004; Ibarra-Perez and Kelly 2005; Liu et al. 2005; Mutlu et al.2005b). An SSR marker in the bean nitrate reductase gene was also found to have tight linkage with the BC 420.900 marker and CBB resistance in a RIL population derived from the HR67 x OAC-95-4 cross (Yu et al., 2004). The same RIL population was also used by Liu et al. (2008) to develop several sequence tagged site (STS) markers that are tightly linked to this major QTL. They also confirmed that the BC420.900 QTL is located on LG B6 (chromosome 6) rather than LG B7 reported by Yu et al. (2004). One negative aspect of this QTL is its linkage with the *V* gene, which conditions purple flower color and dark seed colors (Jung et al., 1997; Miklas et al., 2006; Kelly et al., 2003; Liu et al., 2008). This linkage drag is unfavourable for breeding CBB resistance pinto, cranberry, and red kidney beans If XAN159, HR67 or HR45 were used as the CBB resistance source (Liu et al., 2008; Miklas et al., 2006). Bai et al. (1997) detected tight association between the RAPD marker BC73.700 and a major CBB resistance QTL in OAC-88-1, a breeding line developed by the inter-specific cross between tapary bean PI440795 x common bean 'ICA Pijao' (Scott & Michaels, 1992). This QTL could explain 45% of the phenotypic variation and believed to be located on LG8 (Chromosome 8) (Kelly et al., 2003) because of its linkage with the SU91.700 SCAR marker that is tightly linked to a major QTL in XAN159, which derived its CBB resistance from tapary bean PI 319443 (Pedraza et al., 1997; Kelly et al., 2003; Liu et al., 2009). The BC73.700 RAPD marker was also converted into a SCAR marker for efficient use in MAS (Beattie et al., 1998). The exact map location of the SU91 is

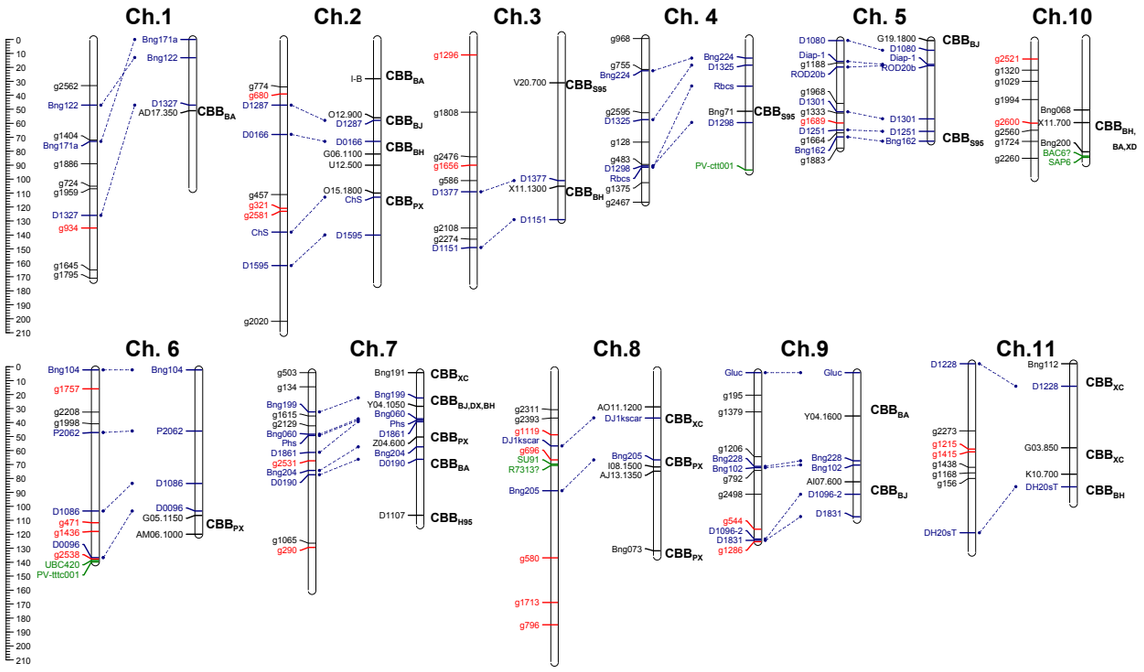

Fig. 1. The chromosome distribution of the identified CBB resistance gene (s) /or QTLs and the associated molecular markers, resemble the maps presented by Shi et al. (2011b). For each linkage group, the map on the left is reproduced from McClean (2007) map {(http://www.comparative-legumes.org/); McConnell et al. 2010}, the map on the right is reproduced from the bean core map {(http://www.comparative-legumes.org/); Freyre et al. 1998}, adopted from Miklas et al (2006) . Both maps are integrated by shared markers except for linkage group B10 (Shi et al. 2011b). In McClean (2007) map, only molecular markers used in association study (in black, Shi et al. 2011b), shared markers (in blue, Freyre et al. 1998), and the Breeder-friendly SCAR and SSR markers (in green) are shown. The markers in red were found significantly ($P \leq 0.05$) associated with CBB resistance (Shi et al. 2011b). In Freyre (1998) map, loci placed on the left side of each chromosome were shared markers in blue and molecular markers closest to previous identified CBB-QTLs. To the right of each linkage group are previously identified CBB-QTLs in different populations (Miklas et al. 2006). Symbols in subscript represent the source population of the QTL: BA Belneb-RR-1/A55, BJ BAT93/JaloEEP558, BH BAC6/HT7719, DX DOR364/XAN176, H95 HR67/OAC95, PX PC50/XAN159, S95 Seaforth/OAC95 and XC XR-235-1-1/Calima. Marker UBC420, PV-ttc001, PV-ctt001, SU91, SPA6 and QTL locations are approximate because most were not directly mapped in the BAT93/JaloEEP558 population. The total distance of each linkage group is expressed in cM (Kosambi mapping function).

not determined so far. Recent studies, however, found that SU91.700 SCAR marker appears to have two similar PCR fragments co-migrating on agrose gel and shows distorted segregation in some mapping populations (Xie et al., unpublished results). Interestingly, the two QTL associated with BC73.700 and SU91.700 were derived from tapary bean PI 440795 and PI 319433, respectively. This suggests that the two QTL may be the same or of the same origion. Tar'an et al. (2001) identified three QTL from OAC-95-4, which is a sibling breeding line of the OAC-88-1 and the experimental name of the OAC-Rex cultivar (Michaels et al., 2006). One

Major QTL	Chromo-some Location	Linked MMs	Primer sequences	Size (bp)	Reference
PV-cttt001	4 or 5 ?	PV-cttt001 SSR	F- gagggtgtttcactattgtccctgc R- ttcatggatggtggaggaacag	152	Tar'an et al. 2001 Kelli et al. Unpublished
BC 420	6	BC420 SCAR	F- gcagggttcgaagacacaxtgg R- gcagggttcgcccaataacg	900	Yu et al. 2000a
		PV-tttc001 SSR	F- tttacgcaccgcagcaccac R- tggactcatagaggcgcagaaag	161	Yu et al. 2004
		STS 183	F- cctatgtacttcttgagggagac R- agaagcccagggacttggat	142	Liu et al. 2008
		STS 333	F- cataagatgaatggttcttgac R- ccatttggtgagattcactt	274	Liu et al. 2008
		GTM 1	F- ccacctgccacatagacctt R- tctcgagaagggcagaggta	459	Yu et al. Unpublished
		GTM 2	F- cgagactcgtgtgctctctg R- acgaaggttgattcccagtg	519	Yu et al. Unpublished
SU 91	8	SU91 SCAR	F- ccacatcggttaacatgagt R- ccacatcggtgtcaacgtga	700	Pedraza et al. 1997
		GTM 3	F- atggtggagacgagatgacc R- tccgacattgaaaccagttg	425	Yu et al. Unpublished
		GTM 4	F- ggcgacggcttctttgac R- tccaaagaccaaagggtgag	464	Yu et al. Unpublished
R 7313	8	R7313 SCAR	F- attgttatcgtcgacacg R-aatatttctgatcacacgag	700	Bai et al. 1997 Beattie et al. 1998
SPA6	10	SPA6 SCAR	F-gtcacgtctccttaatagta R-gtcacgtctcaataggcaaa	820	Miklas et al. 2003
BAC6	10	BAC6 SCAR	F- taggcggcggcgcacgttttg R-taggcggcggaagtggcggtg	1250	Jung et al. 1999

Table 3. Commonly used MMs associated with the major CBB resistance gene(s) /or QTL and their primer sequences

major QTL associated with the SSR marker *PHVPVPK-1* was mapped on LG G5 (corresponding to B5 or chromosome 5), which explained over 42% of the phenotypic variation. The SSR marker *PHVPVPK-1* linked to this major CBB resistance locus in the study of Tar'an et al. (2001) is equivalent to the PV-ctt001 reported by Yu et al. (2000b) that was positioned on LG B4 (chromosome 4) of the 'BAT93' – 'Jalo EEP558' core map (Yu et al.

2000b). The mapping position discrepancy may be because the marker is located at the end of a linkage group in both maps, a position known to be difficult to map accurately, especially in a small population. Differences in the genetic background between the two mapping populations may also contribute to the discrepancy in marker location (Tar'an et al., 2001). Recent studies, however, indicate again that PV-ctt001 is likely located on B4 (chromosome 4) rather than B5 (chromosome 5) and the QTL associated with PV-ctt001 is not significant in the absence of BC 420 (Kelli et al., unpublished results). The other major QTL was positioned on LG G2 (corresponding to B4 or chromosome 4), which was tagged by*BNG71Dra*I and explained 36% of the phenotypic variation for CBB resistance.
The SCAR markers, BC420, SU91 and SAP6 linked with the 3 major QTL of *P. acutifolius* (BC420 and SU91) and *P.vulgaris* (SAP6) origins on B6, B8, and B10, respectively, have been used for MAS of CBB resistance (Table 3, Mutlu et al., 2005b; Yu et al., 2000a) and to validate QTL presence in resistant lines selected by phenotypic selection (see review by Miklas et al., 2006, Fouie & Herselman, 2002). Because of the differences in molecular weights among the 3 SCAR markers, they can be multiplexed in one PCR reaction (Miklas et al., 2000) to speed up MAS for combined resistance to CBB (Miklas et al., 2006). The SU91 SCAR marker associated QTL tentatively positioned on LG 8 or chromosome 8 has been used the most often for MAS (Miklas et al 2005, Mutlu et al 2005b, Hou et al. 2010, Navabi et al., unpublished results; Yu et al unpublished results). Epistatic interactions, however, between BC420 and SU91 CBB resistance QTL were detected (Vandemark et al., 2008), which would complicate the use of MMs in MAS. Epistatic interactions between other CBB resistance QTL were also reported in several other studies (Jung et al.1997; Tar'an et al., 1998; O'Boyle et al., 2007,).

5. Cloning CBB resistance gene(s) / or QTL

Current technical progress in the area of molecular biology and genomics have made the cloning of QTL [i.e. the identification of the DNA sequences (coding or non-coding) responsible for QTL] possible. To date, most plant QTL have been cloned using a positional cloning approach following identification in experimental crosses. In some cases, an association between sequence variation at a candidate gene and a phenotype has been established by analysing existing genetic accessions (Salvi & Tuberosa, 2005). A literature survey shows that although about 150 research papers reporting original QTL data are published yearly (average of 2000–2004, considering Arabidopsis, soybean, rice, sorghum, maize, barley and wheat), only a handful of studies have reported the cloning of QTL (Salvi & Tuberosa, 2005). In common bean, there is only one report on efforts to clone the major CBB resistance QTL by Yu et al. (2010). Two approaches were used by Yu's group to clone the major CBB resistance QTL associated with the BC 420 SCAR and the SU 91 SCAR markers present in the CBB resistance line HR45. Namely, map-based position cloning and candidate gene approaches. With the first approach, they first mapped the BC420 QTL to chromosome 6 at a distal region (Figure 3A, Yu et al., 2000a, Liu et al., 2008,). A BAC library was then developed to physically map the QTL to a genomic region of about 750 kb with 6 BAC clones (Figure 3 B, Liu et al., 2010). One BAC clone, 4k7, containing the BC420 marker was sequenced. The sequenced 4k7 BAC was assembled into a 90kb single contig for functional annotation. Since the BAC was selected by marker BC420, the entire sequence of BC420 was fully recovered from this BAC sequence. Twenty-one genes were *ab initio* predicted

by FGENESH using Medicago gene model, including 11 from sense chain and 10 from anti-sense chain (Figure 3C). Although no homology to any previously identified common bean genes was found, six of the putative genes were supported by common bean ESTs and three of them were supported by runner bean ESTs. The expression of 6 putative genes with supported bean ESTs was assessed and verified by real time RT-PCR. For each putative gene, one or two primer pairs were designed and tested in the contrasting NILs (Near Isogenic Lines) (Vandemark et al. 2008). Fifty-seven percent (8 of 14) of the primer pairs were polymorphic. Seven of them are dominant markers present in the NILs harboring the BC420-QTL, but one is a co-dominant marker. Based on the simple repetitive elements found in the BAC sequences, seven SSR markers were designed and tested in the contrasting NILs. Three of them turned out to be polymorphic, including two dominant and one co-dominant markers. Overall, eleven new markers have been developed in association with CBB resistance in HR45. Another BAC clone harbouring the SU91 SCAR marker was also identified from the HR45 BAC library and sequenced. A single contig of 58kb was assembled. Sixteen genes were also predicted by FGENESH using Medicago gene model (Figure 2).

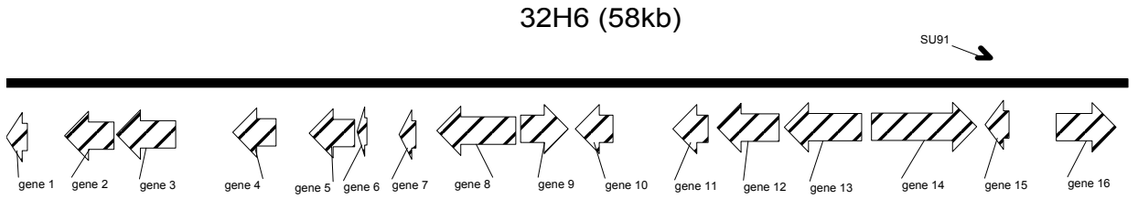

Fig. 2. Annotated genes that are present in the SU91 BAC clone.

In parallel, cDNA-amplified fragment length polymorphism (AFLP) technique was used to identify the candidate genes (CG) that are differentially expressed in the leaves of HR45 sampled at different time-periods after inoculation (Shi et al., 2011a). Selective amplifications with 34 primer combinations allowed the visualization of 2,448 transcript-derived fragments (TDFs) in infected leaves; 10.6% of them were differentially expressed. Seventy-seven differentially expressed TDFs (DE-TDFs) were cloned and sequenced. 50.6% (39 of 77) of the DE-TDFs representing modulated bean transcripts were not previously reported in any EST database then. The expression patterns of 10 representative DE-TDFs were further confirmed by real-time RT-PCR. BLAST analysis suggested that 40% (31 of 77) of the DE-TDFs were homologous to the genes related to metabolism, photosynthesis, and cellular transport, whereas 28% (22 of 77) of the DE-TDFs showed homology to the genes involved in defence response, response to stimulus, enzyme regulation, and transcription regulation. Thus, the 22 pathogenesis-related DE-TDFs were selected as functional candidate genes (FCGs) in association with CBB resistance. Meanwhile, six of the FCGs were *in silico* mapped to the distal region of the chromosome 6 (the genomic region of the CBB resistance QTL linked to BC420 in HR45) and were chosen as positional candidate genes (PCGs) for comparative mapping. Comparing the CGs found from map-based cloning to the CGs derived from cDNA-AFLP, none of them is overlapped. This indicates that gene expression studies may characterize the downstream transcriptional cascade of the QTL. The PCGs could be the genes for CBB resistance, whereas the FCGs genes that map to other locations may be involved in the molecular responses related to the QTL (Shi et al., 2011a).

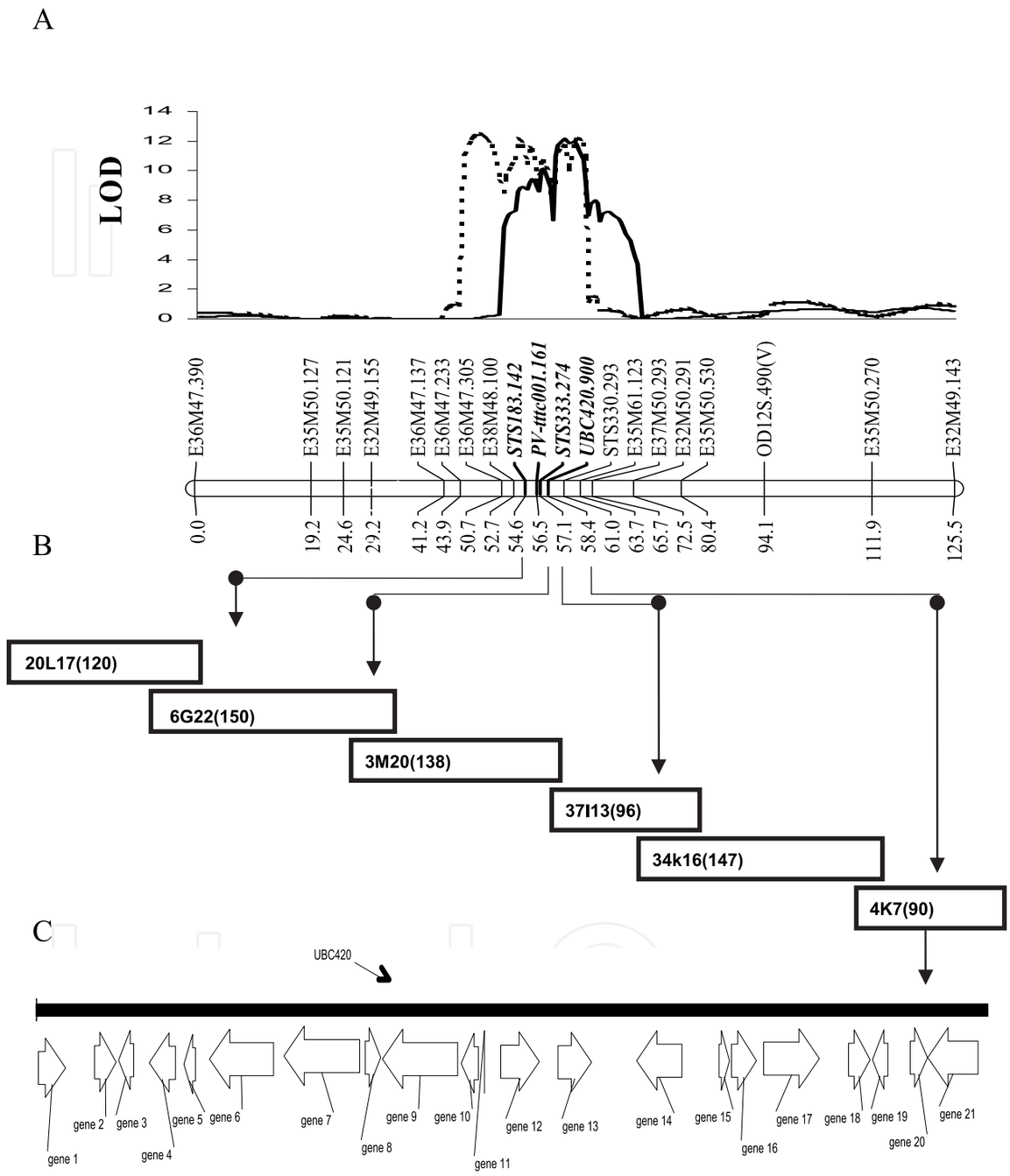

Fig. 3. Genetic, physical and sequence map of the BC420 QTL. A) A genetic map showing the map location of the BC420 QTL in the HR 67 X OAC-95-4 recombinant inbred line population (Liu et al. 2008);B) A physical map showing the contig that contains the BC420 SCAR marker (Liu et al. 2010); C) Annotated genes present in the BAC clone 4k7 (Yu et al. unpublished).

6. Summary and future prospects

Significant progresses have been made in introducing CBB resistance gene(s)/or QTL into common bean from its related species, in dissecting genetic factors underlying CBB resistance, and in mapping CBB resistance QTL on bean LG or chromosomes. Molecular markers linked to several major CBB resistance QTL have been developed and used in marker assisted breeding of common bean for CBB resistance. However, all of the QTL mapping studies reviewed so far have been based on the analysis of populations derived from bi-parental crosses that segregated for trait(s) of interest. To date, at least 24 different CBB resistance QTL have been positioned across all eleven LG or chromosomes of common bean (Figure 1, Kelly, et al., 2003; Miklas et al., 2006; Shi et al., 2011b). Because these QTL were mapped in a number of different bi-parental populations, the MMs associated with each of the major QTL only segregate in one or a few of the mapping populations (Shi et al., 2011b). As a result, co-alignment of the QTL associated MMs from the different studies could not be done accurately and several questions remain unanswered. For example, what is the map location of SU91 QTL relative to the R7313 QTL on LG 8 or Chromosome 8? Are they the same or different? What is the map location of SPA6 QTL relative to BAC6 QTL on LG10 or chromosome 10? Are the same or different? Thus markers linked to these QTLs are not immediately available for use in other bean breeding programs. Validation of QTL effects in other genetic backgrounds is necessary prior to widespread application of the QTL linked markers for MAS (Shi et al. 2011b).

Alternatively, association mapping is a new QTL mapping approach that can be done in natural populations, cultivars released over years, and/or the breeding materials within a breeding program (Oraguzie et al., 2007). These types of populations or a subset of them may represent a smaller set of the available genetic diversity within a breeding program. Collections of these breeding lines may provide great potential for applied association mapping experiments because they are routinely evaluated in the breeding programs and regional trials to assess their local adaptation or response to biotic and/or abiotic stresses (Oraguzie et al., 2007).

Association mapping is increasingly being used to identify marker-QTL linkage associations using plant materials routinely developed in breeding programs. Compared with conventional QTL mapping approach, association mapping using breeding populations should be a more practical strategy for cultivar development, considering that markers linked to a major QTL can immediately be applied for MAS, once new QTL are identified. For instance, in soybean (*Glycine max* L. Merr.) two markers, Satt114 and Satt239, were found to be associated with iron deficiency chlorosis loci using advance breeding lines (Wang et al., 2008). In rice (*Oryza sativa* L.), microsatellite markers associated with yield and its components were identified in a variety trial, and many of them were located in regions where QTL had previously been identified (Agrama et al., 2007). Association mapping studies have also been used to investigate the genetic diversity within crop species. High levels of LD (Linkage Disequilibrium) (pairwise LD: 56%; average $r^2 = 0.1$) was found in common bean (Monica et al., 2009). Much higher LD was observed in domesticated populations (pairwise LD: 57.3%; average $r^2 = 0.18$) compared to wild populations (pairwise LD: 31.5%; average $r^2 = 0.08$) (Monica et al., 2009). In the presence of high LD, lower marker density is required for a target region with greater potential for detecting markers strongly

associated with the target gene polymorphism, even if distant physically (Shi et al., 2011b). Thus, whole-genome-scan association study is feasible for bean domestic populations (Monica et al., 2009). In association mapping, where unlike conventional QTL mapping, populations of un-structurally related individuals are employed, it is important to consider population structure and kinship among individuals, because false associations may be detected due to the confounding effects of population admixture (Oraguzie et al., 2007).This may indeed be the case for populations sampled from large collections, breeding materials, or from released cultivars. Therefore, it is necessary to apply appropriate statistical methods that account for population structure and kinship among individuals. A Mixed Linear Model (MLM) approach has been developed to account for multiple levels of relatedness simultaneously as determined by kinship estimates based on a set of random genetic markers (Yu et al., 2006). This model has been proven useful in genome-wide association studies to control the biases that may be caused by population structure and relatedness in other species e.g., maize (*Zea mays* L.) (Yu et al., 2006), rice (Wang et al., 2008). Another issue for association mapping is reliability, an issue of particular concern when the goal is to discover marker/trait associations that have broad application. Shi et al. (2011b) conducted the first association mapping study in common bean for CBB resistance. Using CBB resistance data collected in a CBB field nursery from 395 of the 469 dry bean lines of different market classes representing plant materials routinely developed in a bean breeding program in Ontario, Canada and 132 SNPs evenly distributed across the bean genome, significant associations between CBB resistance data, collected at 14 and 21 days after inoculation, and 14 MMs were detected. Among the 14 MMs, previously identified BC420 and SU91 SCARS were confirmed for their association with CBB resistance (Shi et al., 2011b). The rest of the markers were SNPs, which were co-localized with or close to the CBB-QTLs identified previously in bi-parental QTL mapping studies. Given the abundance of SNPs exits in the common bean genome, the possibility of automation for the analysis of SNPs, and the efficiency of association mapping approach, it is expected that the use of association mapping with SNPs will be the method of choice for mapping CBB resistance gene(s) /or QTL in the future.

Despite of the progresses that have been made for understanding CBB resistance in common bean, there are still a number of challenges lying ahead, such as 1) what is the molecular mechanisms underlying the epistatic interactions between CBB resistance QTL, such as the BC 420 and SU91 QTL? 2) The epistatic interaction between CBB resistance gene(s)/or QTL would make the application of MMs to assist in breeding CBB resistance bean and the cloning of the QTL more complex, and 3) the recalcitrant nature of common bean to transformation make the validation of CGs coding for CBB resistance more difficult to accomplish. So it is an urgent task for bean researchers to develop novel approaches, such as virus induced gene silencing (VIGS) (Zhang et al., 2010) for validating the functions of CGs in common bean.

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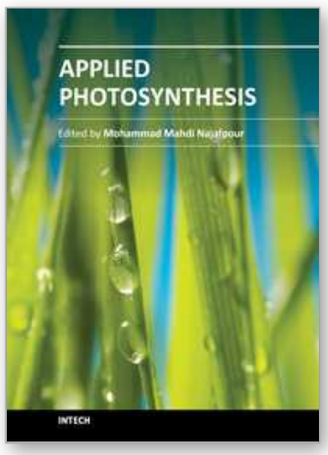

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Photosynthesis is one of the most important reactions on Earth, and it is a scientific field that is intrinsically interdisciplinary, with many research groups examining it. This book is aimed at providing applied aspects of photosynthesis. Different research groups have collected their valuable results from the study of this interesting process. In this book, there are two sections: Fundamental and Applied aspects. All sections have been written by experts in their fields. The book chapters present different and new subjects, from photosynthetic inhibitors, to interaction between flowering initiation and photosynthesis.

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