International Journal of Academic and Applied Research (IJAAR)
ISSN: 2643-9603
Vol. 3 Issue 11, November – 2019, Pages: 32-42

Review On: Marker Assisted Selection In Common Bean Breeding For Disease Resistance

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Abstract : *Common bean (Phaseolus vulgaris L) is the most important food legume consumed worldwide (Miklas et al., 2006) and an important source of human dietary protein, calories, vitamins and minerals necessary for a healthy community. Molecular marker-assisted selection, often simply referred to as marker-assisted selection involves selection of plants carrying genomic regions that are involved in the expression of traits of interest through molecular markers. With the development and availability of an array of molecular markers and dense molecular genetic maps in crop plants, MAS has become possible for traits both governed by major genes as well as quantitative trait loci. The potential benefits of using markers linked to genes of interest in breeding programme, thus moving from phenotype based towards genotype-based selection, have been obvious for many decades.*

Keywords: Marker Assisted Selection, Quantitative Trait Loci, Molecular Marker, Genotype-based selection.

Introduction

Common bean (*Phaseolus vulgaris* L) is the most important food legume consumed worldwide (Miklas *et al.,* 2006) and an important source of human dietary protein, calories, vitamins and minerals necessary for a healthy community. It has a great impact on food security of people in developing countries (Miklas *et al.,* 2006). The world largest producers of common bean are India, Brazil, Myanmar and Mexico (FAOSTAT, 2014). In Africa large producers are East African countries where Tanzania is the leading producer contributing 4.9 % of the production (FAOSTAT, 2015). However, production of common bean in various parts of the world is faced with a number of major biotic and abiotic constraints. Biotic stresses include those which are caused by fungi, bacteria, viruses and insect pests. The abiotic bean production constraints include macro nutrients such as nitrogen and phosphorus, micronutrients deficiency; such as excessive rain/flooding, drought, heat and cold stress factors, each of which causes yield loss significantly (Beebe *et al*., 2012).

Plant breeding is the art and science of changing the traits of plants in order to produce desired characteristics and it can be accomplished through many different techniques ranging from simply selecting plants with desirable characteristics for propagation, to methods that make use of knowledge of genetics and chromosomes, to more complex molecular techniques. Conventional plant breeding is primarily based on phenotypic selection of superior individuals among segregating progenies resulting from hybridization. Although significant strides have been made in crop improvement through phenotypic selections for agronomical important traits, considerable difficulties are often encountered during this process, primarily due to genotype – environment interactions. Besides, testing procedures may be many times difficult, unreliable or expensive due to the nature of the target traits (e.g. abiotic stresses) or the target environment (Babu *et al.,* 2004).

A new variety in conventional breeding could take 8 to 10 years to develop. Breeders are very interested in new technologies to speed up this process or make it more efficient. The development of molecular markers was therefore greeted with great enthusiasm as it was seen as a major breakthrough promising to overcome this key limitation. With the advent of DNA-based genetic markers, it became possible to identify large numbers of markers dispersed throughout the genetic material of any species of interest and use the markers to detect associations with traits of interest (John R and Andrea S 2007). Thus allowing marker assisted selection (MAS) finally to become a reality. Molecular marker-assisted selection, often simply referred to as marker-assisted selection (MAS) involves selection of plants carrying genomic regions that are involved in the expression of traits of interest through molecular markers. With the development and availability of an

array of molecular markers and dense molecular genetic maps in crop plants, MAS has become possible for traits both governed by major genes as well as quantitative trait loci (QTLs). The potential benefits of using markers linked to genes of interest in breeding programme, thus moving from phenotype based towards genotype-based selection, have been obvious for many decades. By now a stage has been reached, where genomics research is focusing on generating functional markers that can help identifying genes that underlie certain traits, thus facilitating their exploitation in crop improvement programs. The mapping of genes controlling agronomic traits coupled with the widespread availability of easy to use simple sequence repeat (SSR) markers and quick DNA extraction methods has provided breeders with an excellent opportunity to apply marker assisted selection (MAS) methods in varies of crops (David 2007).

Objective

- To review the Application of Molecular markers assisted selection for common bean diseases

Literature Review

Origin, distribution and botany of common bean

The common bean (*Phaseolus vulgaris L*.) originated from wild growing vines and is diversified in the Andes and the highlands of Middle America (Gichangi *et al.,* 2012). It was domesticated in two region distributed from Mesoamerican gene pool and the Andean gene pool (Gichangi *et al.,* 2012). The domestication of common bean has changed the phenology, morphology and the form of the plant. The modification is visible also on the seed size, growth habit, maturity and seed retention (Beebe *et al.,* 2014). Therefore, the dissimilarity among the cultivated and wild common bean is due to the seed size, pod size and the presence of edible parts such as the dry seed and green immature pod (Oshone *et al.,* 2014).

Phaseolus vulgaris L. is the scientific name of common bean. It's within the legume family with a taxonomic hierarchy namely as older is Fabales, family is fabacea, Genus is Phaseolus L., and the species is Phaseolus vulgaris L. The genus Phaseolus is diverse with around 80 wild and cultivated species, but it remains the most commonly cultivated species (Porch, 2013). Common bean is a multipurpose diploid (2n=2x=22) self-pollinated crop and the most widely grown pulse in eastern and central Africa (Gichangi *et al.,* 2012).

Cultivation of common bean in Africa though widespread is mainly concentrated in East and Central African region (Katungi, *et al.,* 2010). Kenya is the principal producer of common bean in terms of area cultivated, followed by Uganda and Tanzania (Katungi, *et al.,* 2010). Though, Uganda occupies the first place in terms of production, then Kenya followed by Tanzania (Balcha and Tigabu, 2015). The climatic of common bean ranges from temperate to sub-tropical with defined wet and dry seasons. Production of common bean is high in areas where precipitation is moderate rather than in dry areas with excessive rainfall (Beebe *et al.,* 2014). Common bean is cultivated twice a year in eastern and central Africa and sowing season start from March to April and from September to October, but in Ethiopia the long season is June to August (Katungi, *et al.,* 2010). Beans are grown in various cropping system.

Marker Assisted Selection (MAS)

The development of DNA (or molecular) markers has irreversibly changed the disciplines of plant genetics and plant breeding. While there are several applications of DNA markers in breeding, the most promising for cultivar development is "marker assisted selection". MAS refer to the use of DNA markers that are tightly-linked to target loci as a substitute for or to assist phenotypic screening. By determining the allele of a DNA marker, plants that possess particular genes or quantitative trait loci (QTLs) may be identified based on their genotype rather than their phenotype. Five main considerations for the use of DNA markers in MAS (Mohler and Singrun, 2004) are;

- **Reliability:** Molecular markers should co-segregate or tightly linked to traits of interest, preferably less than 5 cm genetic distance. The use of flanking markers or intragenic markers will greatly increase the reliability of the markers to predict phenotype.
- **DNA quantity and quality:** Some marker techniques require large amounts and high quality DNA, which may sometimes be difficult to obtain in practice and this, adds to the cost of the procedures.
- **Technical procedure:** Molecular markers should have high reproducibility across laboratories and transferability between researchers. The level of simplicity and time required for the technique are critical

considerations. High-throughput simple and quick methods are highly desirable.

- **Level of polymorphism:** Ideally, the marker should be highly polymorphic in breeding material and it should be co-dominant for differentiation of homozygous and heterozygous individuals in segregating progenies.
- **Cost:** Molecular markers should be user-friendly, cheap and easy to use for efficient screening of large populations. The marker assay must be cost-effective in order for MAS to be feasible.

Molecular Breeding for Disease Resistance

In the scope of using molecular breeding for improving disease resistance, there has been considerable effort focused on the identification of markers linked with major genes and mapping quantitative trait loci (QTLs) for disease resistance. With molecular markers for R genes, direct selection with these markers for disease resistance can be implemented in the breeding programs. Many hundreds of R genes have been mapped across important crops, including rice (Bryan, *et al.,* 2000), wheat (Saintenac, *et al.,* 2013) and maize (Webb, *et al.,* 2002). Work in *Arabidopsis thaliana* has identified many genes and provided a strong fundamental foundation in molecular plant-pathogen interactions. However, given the economic importance of these diseases and the overall effort invested in genetic markers, there are relatively few examples of large-scale implementation of MAS for disease resistance in applied breeding programs. As noted by (Miedaner & Korzun 2012) in relation to wheat and barley breeding, the lack of markers applied in commercial breeding for disease resistance could be due to having few diagnostic markers, the prevalence of QTL background effects, and overall economic constraints from a low return of investment for implementing markers.

Furthermore, few monogenic resistances are durable, and only a few QTLs with high effects have been successfully transferred into elite breeding material, limiting the practical use of MAS for R genes (Miedaner & Korzun 2012). Following mapping and implementation of markers for multiple R genes, pyramiding of selected genes is possible (Pradhan, *et al.,* 2015). However, the practical implementation of MAS for stacking multiple disease resistance genes adds an additional layer of complexity to applied breeding programs, as the population size needed for maintaining and fixing multiple genes quickly outgrows the reasonable available resources for MAS. For example, in a simple F2 population, an expected 25% of the lines would be fixed for the favorable allele at any given locus and only 1.5% of the plants would be fixed for all favorable alleles at three different loci. It is possible with a reasonable population size of a few hundred to identify plants through MAS with a three-gene pyramid.

However, this must be put in the context of breeding programs in which breeders want to have many hundreds of lines for advancement to yield testing. It would take a population size of 10,000 with MAS to identify 150 lines carrying a three resistance gene pyramid that could be advanced for yield testing, which is still far too few for making progress for yield. Although the probability in the F2 increases to 42% if just maintaining the favorable allele combination (in a heterozygous or homozygous condition), further rounds of marker testing are needed in subsequent generations. Backcross introgression is another option for combining multiple R genes into an elite background.

Angular Leaf Spot

Resistance genes against *Phaeoisariopsis griseola* the causal agent of ALS are controlled by major genes, that are either dominant or recessive, acting singly or duplicated and which may interact in an additive manner with or without epistasis (Mahuku *et al.,* 2003). Diverse sources of resistance to angular leaf spot in bean genotypes have been reported (Beebe *et al.,* 1991). Examples of resistant cultivars include A 75, A 140, A 152, A 175, A 229, BAT 76, BAT 431, BAT 1432, BAT 1458 and G5686, MAR 1, MAR 2 (Ferreira *et al.,* 2000). In reference (Ragagnin *et al.,* 2005) found the ALS resistance in AND 277 to race 63:23 to be conferred by a single dominant gene (*Pgh-1*). Cornell 49-242 has *Pgh-2* which confers resistance to *P. griseola* pathotype 31:17 (Nietsche *et al.,* 2000) while (Mahuku *et al.,* 2004) found that resistance to ALS in Mexico 54 is due to a single dominant gene that confers resistance to pathotype 63:63 and G06727 has resistance to *P. griseola* pathotype 63:59.

Resistance to specific isolates of *P. griseola* has been reported to be simply inherited and molecular markers have been identified for some of these resistance genes (Ferreira *et al.,* 2000, and Miklas 2005). Sources of resistance reported from Africa include GLP 24, GLP X-92, GLP - 806 and GLP 77 (Ferreira *et al.,* 2000). Resistance to various diseases is monogenically determined, but cases of duplicate, complementary and other interactions have been

reported (Singh and Munoz 1996). The breed for ALS resistance, molecular markers linked to angular leaf spot resistance genes have been identified in beans. Example of successfully used Markers in common bean disease resistance: SCAR markers for selecting for genes for resistance to ALS include SH13 for *phg-1* gene in linkage group *6* (Queiroz *et al.*, 2004) and SNO2 for phg-2 gene in linkage group 8 (Miklas *et al.*, 2002). Others include, SAA19 (Queiroz *et al.*, 2004), SBA16 (Queiroz *et al.*, 2004) and SMO2 (Queiroz *et al.*, 2004) which is ouro Negro dominant gene.

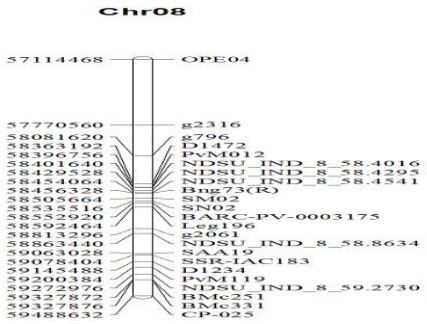

Figure 1 Partial map of chromosome Pv08 of common bean showing twenty-three markers used to screen the Ugandan breeding parents (NABE12C, Mexico 54, RWR719 and G2333) for an alternative to the SN02 marker (Schmutz et al., 2014).

Common bacterial blight

The control of common bacterial blight (CBB) disease caused by *Xanthomonas axonopodis pv phaseoli (Xap)* is challenging due to its complexity and seed borne nature (Singh and Munoz 1996). The number of genes involved in resistance to *Xap* range from one to several genes with varying degrees of action and interactions (Zapata *et al.*, 2010). Breeding for CBB resistance is complicated pathogen genetic diversity and coevolution (Allen and Lenne 1998) different genes conditioning resistance in leaves, pods and seeds (Crous *et al.*, 2006) and linkage of resistance with undesirable traits (Crous *et al.*, 2006). Resistance of CBB is quantitatively and qualitatively controlled depending on the source of germplasm with pod and leaf resistance being controlled by different genes (Chataika *et al.*, 2010). Quantitative inheritance was observed after making original interspecific crosses between resistant *P. acutifolius* 'tepary 4' and susceptible *P. Vulgaris* (Singh and Munoz 1996). Sources of resistance to *Xap* in common bean have been reported (Miklas 2005). CIAT lines VAX 3, VAX 4, VAX 6, and XAN 159 have also been reported to have good level of resistance to common bacterial blight (Singh and Munoz 1996). Albeit, genetic studies have shown that resistance to CBB is quantitatively inherited, it involves a few major genes (Kelly and Miklas 1998).

The identification of QTL influencing resistance to CBB combined with phenotypic data implying the involvement of few genes, suggests that MAS may be useful in combining resistance sources to CBB in common bean. To date, SCAR markers used in selecting resistance to CBB are dominant and are scored as presence or absence of a single band on an agarose gel. SCAR markers available in screening are SU91, BC420, SAP 6, BAC 6, R7313 and R4865. SU91 is linked to a QTL for CBB resistance in bean in the linkage group B8 (Yu *et al.*, 2000). BC420 is linked to a QTL for CBB resistance on bean linkage group B6. SAP 6 is for a major QTL in the linkage group B10 (Miklas *et al.*, 2000), BAC 6 for a major QTL in linkage group B10 (Jung *et al.*, 1996) R7313 for a major QTL in linkage group B8 (Bai *et al.*, 1997) and R4865 for another major QTL (Bai *et al.*, 1997). Thus, molecular markers allow distinct QTLs to be screened and consequently provide an opportunity to pyramid multiple QTL for CBB resistance into a single genotype.

Anthracnose

Two new sources of anthracnose resistance within the Andean gene pool were identified in germplasm from Brazil (Gonçalves-Vidigal *et al.*, 2009). The two independent genes were identified as *Co-12* in Jalo Vermelho and *Co-13* in Jalo Listras Pretas and represent unique resistance patterns. These are significant findings as the multi allelic *Co-1* locus with five alleles was the only resistance sources previously known in Andean germplasm. This is particularly important given the recent breakdown of the *Co-12* gene by race 105 in Manitoba. The rapid evolution of this new race underscores the need to monitor the pathogenic variability in different production areas. The availability of new resistance genes of Andean origin offers breeders more choices for pyramiding genes with the more common Middle American resistance sources.

The identification of additional resistance specificities in anthracnose resistant genotypes through classical bi-parental population analysis and afterwards allelism tests is time-consuming. GWAS are complementary to bi-parental analysis and can identify traits of economic importance in the crops. GWAS take advantage of natural variation in the population accumulated during historic recombination. GWAS provided comprehensive

insights to identify complex traits in both model and non-model plants. The availability of bean genome sequence (Schmutz *et al.*, 2014) and SNP markers in BARCBean6K_3 BeadChip (Hyten *et al.*, 2010; Song *et al.*, 2015) has given fresh impetus to the bean scientific community to map R genes. The SNP chip has resulted in the fine mapping of many resistance sources including: *Co-x* (Richard *et al.*, 2014), *Co-1* (Zuiderveen *et al.*, 2016) *Co-12* (Vazin, 2015) and the *Co-42* (Oblessuc *et al.*, 2015) and the discovery of new genomic regions and candidate genes associated with anthracnose resistance (Gonzalez *et al.*, 2015). The *Co-x* gene was fine mapped to Pv01, independent of the *Co-1* locus, and to a syntenic region, located at one end of soybean (*Glycine max*) chromosome 18 that carries *Rhg1*, a major gene conditioning resistance to soybean cyst nematode (Richard *et al*., 2014). Fine mapping of the *Co-4* (COK-4) locus to Pv08 revealed 18 copies of the COK-4 gene in a 325kbp segment of that chromosome (Oblessuc *et al.*, 2015).

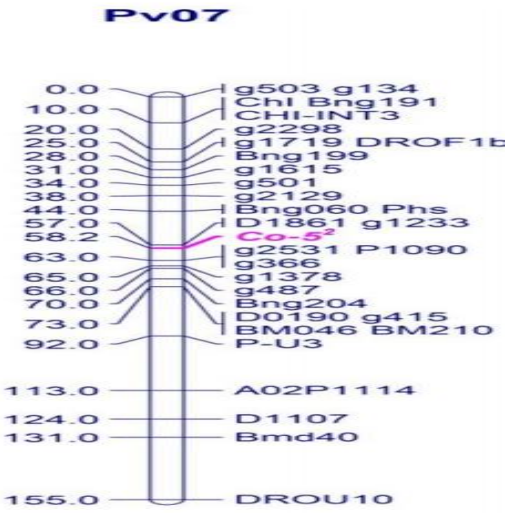

Figure 2 Genetic distance and location of the *Co-52* allele, which confers resistance to common bean anthracnose, and the g1233 molecular marker within linkage group Pv07 of *Phaseolus vulgaris* L. using the population obtained from the Mexico 222 × MSU 7-1 cross. The map was constructed with Map Chart (Voorrips 2002).

Bean common mosaic virus and bean common mosaic necrosis virus

Genetic resistance to both poty viruses is conditioned by a series of independent multi-allelic loci in common bean is affected by four different loci: *bc-1*, *bc-2*, *bc-3* and *bc-u* (Drijfhout, 1987). Resistance controlled by alleles at these loci is inherited as recessive characters (Naderpour *et al.*, 2008). In addition to the recessive *bc* genes, the dominant *I* gene in *P. vulgaris* confers resistance to BCMV and other poty viruses through a hypersensitive response (Naderpour *et al.*, 2008) and has also been the focus of positional gene cloning activities (Vallejos *et al.*, 2000). The *I* gene located on B2 (Kelly *et al.*, 2003), is independent of recessive resistance conditioned by three different *bc* genes. The *bc-3* gene is located on B6 (Miklas, *et al.*, 2000), whereas the *bc-1*2 allele was mapped to B3 (Miklas, *et al.*, 2000). The non-specific *bc-u* allele, needed for expression of *bc-2*2 resistance, also resides on B3 based on the loose linkage with the *bc-1* locus (Strausbaugh *et al.*, 2005).

The independence of the BCMV resistance genes provides opportunities to use gene pyramiding as a strategy in breeding for durable resistance. Bean breeders recognize that the combination of the dominant *I* gene with recessive *bc* resistance genes offers durability over single gene resistance to BCMV and BCMNV, since the two types of genes have distinctly different mechanisms of resistance (Kelly 1997). The dominant *I* gene is defeated by all necrotic strains, whereas the three most effective recessive genes (*bc-1, bc-2 and bc-3*) act constitutively by restricting virus movement within the plant, probably through the virus movement proteins. The action of the dominant '*I'* gene is masked by the recessive *bc-3* gene, so as efforts to incorporate the *bc-3* gene into new germplasm proceed, the risk of losing the *I* gene in improved germplasm increases, since direct selection for the *I* gene is not possible. Linked markers offer the only realistic opportunity to maintain and continue to utilize the '*I'* gene as a pyramided resistance gene in future bean cultivars. A marker tightly linked to the '*I'* gene (Melotto *et al.,* 1996) has been demonstrated in many laboratories to be effective across a wide range of germplasm from both gene pools. Breeders have used markers linked to the *I* gene to develop enhanced germplasm with the *I +bc-3* gene combination. In addition, (Johnson *et al.*, 1997) developed SCAR markers from the OC11350/420 (ROC11) and OC20460 RAPD markers linked to the *bc-3* gene to improve their utilization.

Rust

Two new races of rust have been recently reported in Michigan and North Dakota. The new races have reoccurred in Michigan since 2007 and in North Dakota since 2008. Preliminary results are showing that both races are similar, but not identical (Markell *et a.,l* 2008). Resistance to

both races is conditioned by the *Ur-5*, *Ur-11*, and CNC genes. A new source of resistance was mapped to LG 4 near the *Ur-5* and Ur-Dorado loci in black bean populations derived from Tacana (Wright *et al.*, 2009). Several new cultivars with different combinations of rust resistance genes have been released (Pastor-Corrales *et al.*, 2007). Salient among these are six unique great northern bean germplasm lines named BelDakMi-RMR-8, to 13. These are the first great northern beans that combine four genes for rust resistance and two genes for resistance to the two bean common mosaic poty viruses. These beans combine two Andean (*Ur-4 and Ur-6*) and two Middle American (*Ur-3 and Ur-11*) rust resistance genes (Pastor-Corrales *et al.*, 2007).

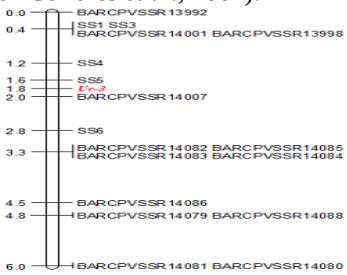

Figure 3 Genetic map of common bean linkage group Pv11 containing *Ur-3* locus and simple sequence repeats (SSRs) and single nucleotide polymorphism (SNPs) markers.

Application of MAS in common bean for Resistance Breeding

Marker assisted selection has been used in common bean for bacterial, virus and fungal disease resistance (Kelly *et al.*, 2003). For common bean bacterial blight resistance which is caused by *Xanthomonas axonopodis pv phaseoli (Xap)* several SCAR marker have been found linked to resistance QTL from different source: UBC420 linked to the QTL on chromosome 1 with resistance alleles from XAN 159; SU91 Linked to the QTL on chromosome 3 from the same source; SAP6 linked to the QTL on chromosome 8 with resistance from great northern Nebraska No.1 sel. 27 (Yu *et al.*, 2000a). These markers have been used in practical breeding to pyramid CBB resistance.

SW13 is linked to the '*I'* gene for resistance to bean common mosaic virus (BCMV) and has proved very reliable in different genetic background (Melotto *et al.*, 1996). SCAR marker SBD5.1300 tightly linked to *bc-1*^{2}, which confers resistance to specific strains of BCMV and bean common mosaic necrosis virus (BCMNV). However, its resistance is masked by *bc-2*^{2} and bc-3 (Miklas *et al.*, 2000b). Therefore, the marker should be useful in MAS breeding. A recessive gene, *bgm*-1, confers bean gold mosaic virus resistance. Its tightly linked marker SR2 is also close to *bc-1* (Blair *et al.*, 2007).

Breeding for anthracnose (caused by *Colletotrichum lindemuthianum*) resistance from different source using MAS to combine different genes (*co-1* to *co-10*) conferring resistance to various predominant races based on geographic regions is practical and realistic (Balardin and Kelly 1998). SAS13 is linked to *co-4*^{2} gene which has the broadest resistance to fungal races (Melotto and Kelly 2001). Pyramiding different resistance genes or QTL with different disease resistance is very common. Integration of UBC420 linked QTL for CBB resistance, SW13 linked I gene for BCMV resistance, and SAS13 linked to *co-4*^{2} gene for anthracnose resistance to breed bean varieties with multiple disease resistance in several market classes including navy, black, pinto, red kidney and cranberry bean is under way (Park and Yu 2004).

In white mold resistance breeding, marker assisted backcrossing successfully transferred a B_7 QTL from G122 and a B_8 QTL from Ny6020-4 into susceptible pinto bean (Miklas and Bosak 2006c). Ender et al 2007 applied markers linked QTL for resistance to white mold from Bunsi to enhance the selection of resistance in breeding.

Table 1 Examples of MAS application in common bean for disease resistance breeding

s.no.	Traits	Resistance QTL/genes	Markers for MAS	Reference
1	CBB	QTL	BC420, SU91, SAP6	Yu *et al.*, 2000b
2	BCMV	*I*	SW13	Melotto *et al.*, 1996

3	BCMNV	*bc*-1^2, *bc*-2^2 and *bc*-3	SBD5.1300, RCO11	Miklas *et al.*, 2000b
4	BGMV	*bgm*-1	SR2	Blair *et al.*, 2007
5	ANT	*Co*-4	SAS13	Melotto & Kelly 2001
6	WM	QTL	AFLP	Ender *et al.*, 2007

*CBB=common bacterial blight, BCMV= bean common mosaic virus, BCMNV= bean common mosaic necrosis virus, BGMV= bean gold mosaic virus, ANT= anthracnose and WM= white mold

Marker assisted selection using LGC KASP platform

Marker-assisted selection (MAS) is the principal molecular breeding approach by which a phenotype such as disease resistance is predicted based on a molecular marker. To demonstrate the usefulness of this SNP platform for MAS, three markers were genotyped including two SNPs in the eIF4E gene that were previously reported to be associated with the *bc- 3* resistance gene to Bean Common Mosaic Virus (BCMV)/Bean Common Mosaic Necrotic Virus (BCMNV) (Hart and Griffiths 2013; Naderpour *et al.*, 2010). Markers *bc-3* and *bc-3a* query the same SNP (C227A), hence, marking the same samples with the resistance associated allele A: A, Bc-3b is based on a different SNP (T194A) in the same eIF4E gene. The resistant allele is detected in lines known to harbor BCMV resistance, like the BRB and RCB lines, as well as in lines not previously known to have the *bc-3* resistance gene like MCM1015 and RWV1129 for which phenotypic information is not available. Available data for BCMV/BCMNV resistance correlate well with the data from all three markers, which shows that these markers are suitable for MAS. Most African varieties display the susceptible alleles (C: C/T: T). The genotyping using these specific markers demonstrates the usefulness of this SNP platform for MAS. This allows breeders to use SNP markers in an easy and cost-effective way without having a molecular analysis infrastructure.

Summary and Conclusion

Molecular marker-assisted selection, often simply referred to as marker-assisted selection (MAS) involves selection of plants carrying genomic regions that are involved in the expression of traits of interest through molecular markers. Breeders are very interested in new technologies to speed up this process or make it more efficient. The development of molecular markers was therefore greeted with great enthusiasm as it was seen as a major breakthrough promising to overcome this key limitation.

In the scope of using molecular breeding for improving disease resistance, there has been considerable effort focused on the identification of markers linked with major genes and mapping quantitative trait loci (QTLs) for disease resistance. With molecular markers for R genes, direct selection with these markers for disease resistance can be implemented in the breeding programs. Marker assisted selection has been used in common bean for bacterial, virus and fungal disease resistance (Kelly *et al.* 2003). For common bean bacterial blight resistance which is caused by *Xanthomonas axonopodis pv phaseoli (Xap)* several SCAR marker have been found linked to resistance QTL from different source: UBC420 linked to the QTL on chromosome 1 with resistance alleles from XAN 159; SU91 Linked to the QTL on chromosome 3 from the same source; SAP6 linked to the QTL on chromosome 8 with resistance from great northern Nebraska No.1 sel. 27 (Yu *et al.* 2000a).

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From the above points we can conclude that applications of MAS are successful for disease resistance breeding as well as for trait identification (which is desirable trait) in common bean. It is quite clear that the application of MAS for breeders is more important because it facilitates the breeding program, especially, when we use for disease resistance breeding. There are several factors that affect the successfully using of MAS in disease resistance breeding, those, will be the type of marker used, the type of gene to be transferred and the conditions that characterize a suitable molecular marker. The availability of bean genome sequence (Schmutz *et al.*, 2014) and SNP markers in BARCBean6K_3 BeadChip (Hyten *et al.*, 2010; Song *et al.*, 2015) has given fresh impetus to the bean scientific community to map R genes.

Future Prospects

The high cost of MAS will continue to be a major obstacle for its adoption for some crop species and plant breeding in developing countries in the near future. Specific MAS strategies may need to be tailored to specific crops, traits and available budgets. New marker technology can potentially reduce the cost of MAS considerably. If the effectiveness of the new methods is validated and the equipment can be easily obtained, this should allow MAS to become more widely applicable for crop breeding programmes. Breeders in the future should to develop new markers technology with potentially reduced cost because even if there are many markers developed before there are many new race disease will be develop in the future. The continues development of marker technologies and improved genetic understanding of complex trait, relations among traits and between target trait and environments will make MAS breeding more broadly useful and efficient, as well as cost- effective. Linked markers offer the only realistic opportunity to maintain and continue to utilize the '*I'* gene as a pyramided resistance gene in future bean cultivars should be continued.

Acknowledgment

First of all, I would like to thank for ALLAH who give us Life, Patience and Peace. I would like to acknowledge my Advisor Mrs. Zeleke W. for his valuable comments and my instructor Mrs. Techale B. by giving this current topic which is very important for understanding the topics. Lastly I would like to thank Mr. Dagnaw A. for his support in doing my current topic.

Reference

Allen, D.J. and Lenne, J.M. Diseases as constraints to production of legumes in agriculture. In Pathology of Food and Pasture Legumes. Allen, D. J. and Lenne, J.M. (Eds.). CAB International, Wallingford, UK. 1998; p1-61.

Bai, Y., Michaels, T.E., & Pauls, K.P. Identification of RAPD markers linked to common bacterial blight resistance genes in *Phaseolus vulgaris* L. Genome, 1997; 40: 544–551.

Balardin RS, Kelly JD 1998 interaction among race of Colletotrichum linemuthiamum and diversity in *Phaseolus vulgaris* L. journal of the American society for horticultural science 123, 1038-1047

Beebe, S., Rao, I., Mukankusi, C. and Buruchara, R. (2012). *Improving Resources Use Efficiency and Reducing Risk of Common Bean Production in Africa, Latin America, and Caribbean, International Center for Tropical Agriculture* (CIAT). Cali, 18pp.

Beebe, S.E. and Pastor-Corrales, M.A. 1991. Breeding for disease resistance. In: van Schoonhoven, A. and Voysest, O. (Eds.). Common beans: Research for crop improvement. C.A.B. Intl., Wallingford, UK and CIAT, Cali, Colombia. 1991, 561-617pp.

Bilgi, V.N., C.A.Bradley, S. Ali, S.D. Khot, and J.B. Rasmussen. 2007. Reaction of dry bean genotypes to root rot caused by Fusarium graminearum. Phytopathology 2007; 97:S10.

Blair MW, Rodriguez LM, Pedraza F, Morales F and Beebe S. (2007) Genetic mapping of the bean golden mosaic germini virus resistance gene Bgm-1 and linkage with poty virus resistance in common bean (Phaseolus vulgaris L.) theoretical and applied genetics 114, 261-271

Bradley, C.A., and J.L. Luecke. 2004. 2002 dry bean grower survey of pest problems and pesticide use in Minnesota and North Dakota. Ext. Rep. 1265. North Dakota State Univ. Fargo.

Chataika, B.Y.E., J. M. Bokosi, M. B. Kwapata, R. M. Chirwa, V. M. Mwale,P. Mnyenyembe, J. R. Myers, Performance of parental genotypes and inheritance of Angular Leaf Spot (*Phaeosariopsis griseola*) resistance in the common bean (*Phaseolus vulgaris*). African Journal of Biotechnology 2010; 9(28): 4398-4406.

CIAT. (2001). Solutions that cross-frontiers. [http://www.ciat.cgiar.org/beans] site visited on 27/04/2006

Cramer, R.A., P.F. Byrne, M.A. Brick, L. Panella, E. Wickliffe, and H.F. Schwartz. 2003. Characterization of Fusarium oxysporum isolates from common bean and sugar beet using pathogenicity assays and random-amplified polymorphic DNA markers. J. Phytopathology 2003; 151:352-306.

Crous PW, Slippers B, Wingfield MJ, Rheeder J, Marasas WFO. Phylogenetic lineages in the Botryosphaeriaceae. Studies in Mycology 2006; 55: 235–253.

Drijfhout, E. and Blok, M.J. Inheritance to *Xanthomonas axonopodis pv. phaseoli* in tepary bean (*Phaseolus acutifolius*). Euphytica 1987; 36: 803-808.

Drijfhout, E. Genetic interaction between *Phaseolus vulgaris* and BCMV with implication for strain identification and breeding for resistance. Centre for Agricultural Publication and Documentation, Wageningen. 1978; 33 – 50pp.

Ferreira, C.F., Borém, A., Caravalho, G.A., Neitsche, S., Paula, T. J., De Barros, E.G. and Moreira. M. A. Inheritance of angular leaf spot resistance in common bean and identification of a RAPD marker linked to a resistance gene. Crop Science 2000; 40:1130-1133.

Food and Agricultural Organization of the United Nations (2015). *State of diversity of major and minor crops*. The Second Report on the state of the world's 322pp.

Food and Agricultural Organization of the United Nations (FAOSTAT). (May 2014). Dry Bean Statistical Database. [http://faostat3.fao.org] site visited on 17/6/2016.

Garcia-Espinosa, R. Breeding for horizontal resistance in bean: an example from Mexico. Biotechnology and Development Monitor 1997; 33:5

Gonçalves-Vidigal, M.C., P.S. Vidigal Filho, A.F. Medeiros, and M.A. Pastor-Corrales. 2009. Common bean landrace Jalo Listras Pretas is the source of a new Andean anthracnose resistance gene. Crop Science 2009; 49:133-138.

Johnson, W.C., Guzman, P., Mandala, D., Mkandawire, A. B. C., Temple, C., Gilbertson, R. L. and Gepts, P. Molecular tagging of the bc-3 gene for introgression into Andean common bean. Crop Science 1997; 37:248–254

Kelly, J. D. A review of varietal response to bean common mosaic potyvirus in *Phaseolus vulgaris*. Plant Varieties and Seeds 1997; 10: 1- 6.

Kelly, J. D. and Miklas, P. N. The role of RAPD markers in breeding for disease resistance in common bean. Kluwer Academic Publishers. Molecular Breeding 1998; 4:1-11.

Kelly, J. D., Gepts, P., Miklas, P. N., and Coyne, D. P. Tagging and mapping of genes and QTL and molecular marker-assisted selection for traits of economic importance in bean and cowpea. Field Crops Research *2003;* 82: 135–154.

Kelly, J.D., Hosfield, G. L., Varner, G. V., Uebersax, M. A. and J. Taylor, J. 1999. Registration of 'Matterhorn' great northern bean. Crop Science 1999; 39: 589–590.

Mahuku, G. S., M.A. Henriquez, J. Munoz, R.A. Buruchara, Molecular markers dispute the existence of the Afro-Andean Group of the bean angular leaf spot pathogen, *Phaeoisariopsis griseola*, Phytopathology 2002; 92 580–589.

Mahuku, G.S., Jara, C., Cajiao, C. and Beebe, S. Sources of resistance to angular leaf spot (*Phaeoisariopsis griseola*) in common bean core collection, wild *Phaseolus vulgaris* and secondary gene pool. Euphytica 2003; 130:303-313.

Markell, S.M., M.A. Pastor-Corrales, J.G. Jordahl, R.S. Lampa, F.B. Mathew, J.M. Osorno, and R.S. Goswami. Virulence of Uromyces appendiculatus to the resistance gene Ur-3 identified in North Dakota in 2008. Annual Report of the Bean Improvement Cooperative 2009; 52: 82-83.

Melotto, M., L. Afanador, and J.D. Kelly. Development of a SCAR marker linked to the I gene in common bean. Genome 1996; 39:1216-1219.

Melotto M and Kelly JD (2001) fine mapping of the co-4 locus of common bean reveal a resistance gene candidate, COK-4 that encodes for protein kinase. *Theoretical and applied genetics* 103, 508-517.

Miklas, P. N. List of DNA SCAR markers linked with disease resistance traits in bean. [http://www.usda.prosser.wsu.edu/miklas/Scartable3.pdf] site visited on 13/04/2012. (2005).

Miklas, P.N., R. Delorme, W.C. Johnson, and P. Gepts. 1999. Dry bean G122 contributes a major QTL for white mold resistance in the straw test. Annu. Rpt.Bean Improvement Coop. 42:43–44.

Miklas, P.N., Smith, J.R., Riley, R., Grafton, K.F., Singh, S.P., Jung, G. and Coyne, D.P. Marker-assisted breeding for pyramided resistance to common bacterial blight in common bean. Annual Report of the Bean Improvement Cooperative 2000; 43: 39-40.

Miklas, P. N., Pastor-Corrales, M. A, Jung, G., Coyne, D. P., Kelly, J. D., McClean, P. E., and Gepts, P. Comprehensive linkage map of bean rust resistance genes. Bean Improvement Cooperative 2002*;* 45: 125-129.

Miklas, P.N., Kelly, J.D., Beebe, S.E. and Blair, M.W. 2006. Common bean breeding for resistance against biotic and abiotic stresses: from classical to MAS breeding. *Euphytica* 147(1-2):105-131.

Miklas PN, Bosak KM (2006c) marker assisted backcrossing of QTL for resistance to sclerotinia white mold in pinto bean. The annual report of the bean improvement cooperative 49, 67-68

Mohan, S.T. (1982). Evaluation of *Phaseolus coccineus* Lam. Germplasm for resistance to common bacterial blight of bean. Turrialba 1982*;* 32: 489–490.

Naderpour, M., Søgaard Lund, O., Larsen, R. and Johansen, E. Potyviral resistance derived from cultivars of *Phaseolus vulgaris* carrying *bc-3* co-segregates with homozygotic presence of a mutated *eIF4E* allele. 2008

Ngulu, F. S. Final Report on Pathogenic Variation in *Phaeoisariopsis griseola* in Tanzania. Selian Agricultural Research Institute, Arusha, Tanzania.1999;13pp

Nietsche, S., A. Borém, G. A. Carvalho, R. C. Rocha, T. J. Paula, E. G. de Barros, M. A. Moreira, RAPD and SCAR markers linked to a gene conferring resistance to angular leaf spot in common bean, Phytopathology 2000; 148: 117-121

Park, S.O., Coyne, D.P. and Jung, G. Gene estimation, associations of traits, and confirmation of QTL for common bacterial blight resistance in common bean. Annual Report of the Bean Improvement Cooperative 1998; 41:145-146.

Park SJ, and Yu K, (2004) Molecular marker assisted selection techniques for gene pyramiding of multiple disease resistance in common bean: a plant breeder prospective. Annual report of the bean improvement cooperative 47, 73-74.

Pastor-Corrales, M.A., A. Sartorato, M.M. Liebenberg, M.J. del Peloso, P.A. ArraesPereira, J. Nunes-Junior, and H. Dinis-Campo. 2007. Evaluation of common bean cultivars from the United States for their reaction to soybean rust under field conditions in Brazil and South Africa. Annual Report of the Bean Improvement Cooperative 2007; 50: 123-124.

Queiroz V.T., C.S. Sousa, M.R. Costa, D.A. Sanglad, K.M.A. Arruda, T.L.P.O. Souza, V.A. Regagnin, E.G. Barros, M.A. Moreira. Development of SCAR markers linked to common bean anthracnose resistance genes *Co-4* and *Co-6*. Annual Report of the Bean Improvement Cooperative 2004; 47:249-250

Ragagnin, V., Sanglard, D., de Souza, T. L., Costa, M., Moreira, M. and Barros, E. A new inoculation procedure to evaluate angular leaf spot disease in bean plants (*Phaseolus vulgaris* L.) for breeding purposes. Bean Improvement Cooperative 2005; 48: 90-91.

Santos-Filho HP, Ferraz S and Sediyama CS (1976) Isolamento e esporulac ˜ ¸ao ‘‘in vitro’’ de Isariopsis griseola Sacc. Experientiae 7: 175–193.

Sartorato, A. Resistance of Andean and Mesoamerican common bean genotypes to *Phaeoisariopsis griseola.* Annual Report of the Bean Improvement Cooperative 2005; 48:88-89.

Singh, S.P. and Munoz, C.G. Resistance to common bacterial blight among Phaseolus species and common bean improvement. Crop Science 1996; 39: 80–89.

Strausbaugh C.A., Overturt K., Koehn A.C. Pathogenicity and real-time PCR detection of Fusarium spp. in wheat and barley roots. Canadian Journal of Plant Pathology 2005; 27: 430–438.

Vallejos, C. E., Malandro, J. J., Sheehy, K. and Zimmermann, M. J. Detection and cloning of expressed sequences linked to a target gene. Theoretical and Applied Genetics 2000; 101: 1109–1113.

Vennette, J.R., and Lamey, H.A. Dry Edible Bean Diseases. NDSU Extension Service Publication 1998;576pp

Wright, E.M., Awale, H.E., M.A. Pastor-Corrales, and J. D. Kelly. Persistence of a new race of the common bean rust pathogen in Michigan. Annual Report of the Bean Improvement Cooperative 2009; 52: 84-85.

Yu, K., Park, S.J. and Poysa, V. Marker-assisted selection of common beans for resistance to common bacterial blight: efficacy and economics. Plant Breeding 2000; 119:411-415

Zapata, M., Beaver, J.S. and Porch, T.G. Dominant gene for common bean resistance to common bacterial blight caused by Xanthomonas axonopodis pv. phaseoli. Euphytica 2010; 179:373-382