Genetic Mapping and QTL Analysis in Common Bean

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Abstract

Common bean (*Phaseolus vulgaris* L.) is the most important legume for direct human consumption and a well-studied crop species in terms of genetics, genomics and breeding. Genome maps are important tools that are an integral part of genetic resource conservation and breeding programmes. Several maps have been developed or are being developed in common bean. Different types of molecular markers such as RFLP, AFLP, SSR, CAPS, RGA and EST have been developed and mapped onto the 11 common bean chromosomes. Markers have been used extensively for identification and mapping of genes and QTL for many biologically and agriculturally important traits, including disease resistance genes, photoperiod sensitivity, growth habit, pod size, seed weight, pigmentation, phenology and abiotic stress tolerance, and occasionally for germplasm screening, fingerprinting and marker-assisted breeding. MAS has been employed mainly for improving simply inherited traits and not much for improving complex traits. The utility of MAS in common bean breeding has been restricted largely due to inaccurate estimation of main QTL, epistatic and QTL × environmental interaction effects. GWAS has also proved to be a powerful tool for investigating complex traits and developing new markers for breeding. The huge amount of sequence information available for common bean via whole-genome sequencing projects facilitates in the next years the development of a rapid and

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cost-effective generation of high-density functional maps, which could also lead to the direct gene tagging for QTL mapping of important agronomic traits, improving the efficiency of common bean breeding programmes via MAS.

Keywords
Phaseolus vulgaris · Quantitative trait locus · Epistasis · Diseases
Abiotic stress · Agronomic traits · Genome-wide association

4.1 Introduction

Continuous progress made in the last two decades on phenotypic and DNA marker analyses has provided a set of useful tools both for genetic research and for plant breeding. They have also led to the construction of genetic linkage maps for most of the crop species, particularly for legume species such as common bean (*Phaseolus vulgaris* L.), where genes involved in valuable traits have been located. Currently, selectable DNA marker development entails the main goal for most public research institutions and private companies working on plant breeding. Mapping DNA markers does allow not only for an efficient genotype selection but also for the detection of quantitative trait loci (QTL) for interesting traits. QTL analysis combines linkage analysis and molecular and statistical genetics, providing consistent information about the chromosome regions contributing to the variance and the inheritance pattern of such traits. This basic genetic information can be used by plant breeders to accelerate introgression of desirable traits and to manage environmental interactions.

The genome sequence of the common bean has already been published from the results of two sequencing consortia, which have focused their works on 'G19833' and 'BAT93' genotypes representing the Andean (Schmutz et al. 2014) and Mesoamerican (Vlasova et al. 2016) gene pools, respectively. In addition, a Canadian consortium has also sequenced a third genotype ('OAC-Rex9') and the genome information is also available (http://www.beangenomics.ca/research/projects/view/draft-genome-sequence-for-common-bean-i-p-vulgaris-i/). Hopefully, the integration of linkage maps, QTL and genomic tools will be essential not only for the development of more accurate tools useful for genomics-assisted breeding, but also for the map-based cloning approaches devoted to the isolation of genes controlling important traits.

In this review, genetic mapping approaches performed in common bean are summarized, from the more classical ones to the more recent maps based on molecular and genomic data. Similarly, the more relevant contributions of QTL analysis are also reviewed despite the elevated number of quality reports recently published on this topic. Particular attention has been paid to epistatic and environmental interactions as the single-locus QTL only reveal part of the genetic determinants underlying phenotypic variance. Finally, a section is devoted to mapping results from genome-wide association study (GWAS) as it provides an alternative to linkage mapping for the dissection of complex traits.

4.2 The Beginnings of Genetic Mapping in Common Bean

The first genetic analysis of common bean was conducted by Gregor Mendel in the mid-nineteenth century (Mendel 1866). It was performed in a progeny from *P. vulgaris* and *Pseudomys nanus* (= *P. vulgaris*, bush type) and

was aimed to corroborate results on the inheritance of growth habit, pod colour and shape that Mendel had obtained in pea (*Pisum sativum* L.). Later, Shaw and Norton (1918) used intraspecific crosses and determined that pigmentation and pigmentation patterns of the seed coat were controlled by multiple independent factors. The first report of a linkage in common bean was performed by Tjebbes and Kooiman (1921), who reported the so-called non-constant mottling, which is due to the tight repulsion linkage between the *B* gene (now *C*, a colour factor) and the *S* gene (now *M*, a cis-acting factor of the *C* locus). A few years later, Sax (1923) began to identify the multiple components that determine the inheritance of pattern and colour of the seed. Indeed, metabolic control of seed colour in common bean was one of the first QTL to be identified. Differences for seed weight were associated with one or both factors that determine the pattern and colour of the seed. Later, an association between seed weight and phaseolin protein type was observed on the linkage group (LG) 07 (Johnson et al. 1996). Vallejos and Chase (1991) reported a linkage between isozyme loci *Adh-1* and *Got2* and seed size; Weeden and Liang (1985) also observed an association between isozyme loci *EST-2* and white flower. The second half of the twentieth century provided much more evidence for genetic linkage affecting a wide variety of traits. Among others, the *I* allele, which confers resistance to all known strains of the bean common mosaic virus (BCMV) and four related potyviruses, was found to be linked to seed coat (Temple and Morales 1986; Kyle and Dickson 1988) and hilum-region-darkening allele *B* (Park and Tu 1986). A genetic linkage of coloured seed coat to resistance to *Pythium* and/or *Rhizoctonia* root rots was also reported (Dickson and Petzoldt 1986). Likewise, genetic linkage was described between maturity and indeterminate growth habit by Valladares-Sánchez et al. (1979), among genes for rust resistance by Stavely (1984) and between arcelin and lectin genes by Osborn et al. (1986). Since then, tagging of many other traits with molecular markers has been reported.

Lamprecht (1961) published the first genetic linkage map for common bean, which consisted mainly of morphological markers distributed over eight LGs. This linkage map was rudimentary, with many loci that could only be reliably evaluated in a limited number of populations or which were subject to epistasis. Lamprecht's map was extended with additional isozymes, seed proteins and induced mutations (Bassett 1988; Gepts 1988; Koenig et al. 1990; Vallejos and Chase 1991). These classical maps showed a reduced genomic coverage and scarce usefulness for marker-assisted selection (MAS), but they provided a point of reference for subsequently developed DNA-based linkage maps.

A problem encountered in establishing the classical maps was the use of different gene symbols for the same gene by different researchers (Bassett 1991). A subcommittee of the *Phaseolus* Genetics Committee addressed this lack of coordination among geneticists and formulated guidelines for gene designation and nomenclature (Myers and Bassett 1993; Bassett and Myers 1999). As a result, an updated list of genes for *P. vulgaris* was published (Bassett 2004). Another problem was that many previously described mutants could not be tested due to the lack of a seed source. To solve this drawback, in 1987, the *Phaseolus* Genetics Committee (Gepts 1988) advocated for a repository of genetic stocks that M Bassett established and which is currently maintained by the USDA-ARS National Plant Germplasm System (NPGS, Pullman, WA, USA).

4.3 Development of DNA-Based Linkage Maps in Common Bean

The progress of genetic linkage mapping in common bean is closely related to the development of different generations of molecular markers. Random DNA markers such as Restriction Fragments Length Polymorphism (RFLP), Random Amplification Polymorphic DNA (RAPD), Amplified Fragments Length Polymorphism (AFLP) or Simple Sequence

Repeat (SSR) have been the basis for most of the common bean genetic maps currently published. However, the increasing availability of high-throughput molecular marker technology and the reduction of costs of marker development and genotyping technologies have provided a wealth of sequence information. Thus, the incessant evolution in genomic research is driving a trend away from random DNA markers towards those called functional markers; whereas the former are derived at random from polymorphic sites in the genome, functional markers are specifically developed from the transcribed genomic regions (Andersen and Lubberstedt 2003). Thereby, throughout this section, common bean genetic maps are described as first- or second-generation genetic maps according to the type of molecular marker used for linkage mapping (i.e. random DNA or functional markers, respectively).

4.3.1 First-Generation Genetic Maps

The first DNA-based genetic maps of common bean were mainly based on RFLP markers (Vallejos et al. 1992; Nodari et al. 1993a). Divergent parents were chosen for both maps in order to maximize polymorphism at the nucleotide level, as well as the phenotypic variation. The mapping population used by Vallejos et al. (1992) consisted of a backcross progeny from the 'XR-235-1-1' (Mesoamerican) × 'Calima' (Andean) cross, whereas Nodari et al. (1993a) used a F_2 population derived from the 'BAT 93' (Mesoamerican) × 'Jalo EEP558' (Andean) cross. The map developed by Vallejos et al. (1992) included the pigmentation gene *P*, 224 RFLP, nine seed proteins and nine isozyme markers, which were sorted into 11 LGs covering 960 cM of the bean genome. This map was later expanded to 980 cM by adding seven additional markers (Vallejos 1994). Subsequently, Vallejos et al. (2001) increased the number of markers up to 294; however, the map coverage was reduced up to 900 cM as a bigger stringency was used for placement of markers on the map. The map developed by Nodari et al. (1993a) was constructed using 108 RFLPs (from *Pst*I and *Eco*RI-*Bam*HI genomic libraries), seven isozymes, seven RAPDs and 18 marker loci corresponding to known genes that were selected after hybridization, as well as three phenotypic traits. These markers were distributed into 15 LGs spanning 827 cM of the genome. Gepts et al. (1993) rapidly improved this map, which finally included 204 markers grouped into 13 LGs covering 1060 cM. The following genetic map published was developed by Adam-Blondon et al. (1994) from a BC_1 population derived from the 'Ms8EO2' × 'Corel' cross, which in addition to 51 RFLPs included 100 RAPDs, two sequence-characterized amplified regions (SCARs) and four morphological markers, covering 567.5 cM of the common bean genome. Furthermore, Adam-Blondon et al. (1994) carried out the first effort to align LGs with the map published by Vallejos et al. (1992), as 19 of the 51 RFLP markers were shared, which established a preliminary correspondence between both maps.

In successive years, the initial F_2 'BAT 93' × 'Jalo EEP558' mapping population was advanced to a Recombinant Inbred Line (RIL) one in order to perform the core linkage map of common bean (Freyre et al. 1998; Hanai et al. 2010); additionally, many RIL populations were developed and used for genetic mapping studies. Koinange et al. (1996) used a RIL population from the 'Midas' (Andean cultivar) × 'G12873' (Mesoamerican wild bean) cross to create a map composed of 77 RFLP and 5 isozyme markers in order to identify the major alleles and QTL that differentiate the wild from cultivated beans. Furthermore, RIL populations from intra-gene pool crosses were also used for linkage mapping. Jung et al. (1996) used a RIL population obtained from the cross between two Mesoamerican genotypes, 'BAC 6' × 'HT 7719', and mapped 75 RAPD markers distributed into 9 LGs covering 545 cM. Likewise, a RIL population obtained between two Andean genotypes, 'PC-50' × 'XAN 159', was used by Jung et al. (1997) to map 168 RAPD markers distributed into 10 LGs covering 426 cM, to study the common bacterial blight disease

resistance. Subsequently, more than twenty-five RIL mapping populations have been developed to map individual or multiple traits, most of them created from inter-gene pool crosses, which include divergent parents showing high genetic polymorphism (Broughton et al. 2003; Kelly et al. 2003). A complete description of the main mapping populations used for linkage map construction purpose is provided in Table 4.1.

Whereas RAPD and AFLP markers were used for saturating previous RFLP maps and to create new genetic maps from additional populations (Miklas et al. 1996, 1998, 2000; Ariyarathne et al. 1999; Tar'an et al. 2001, 2002; Vallejos et al. 2001; Johnson and Gepts 2002), RFLP markers were also useful to anchor different genetic maps. The 'BAT93' × 'Jalo EEP558' RIL population as well as RFLP and RAPD markers of three previous maps (Vallejos et al. 1992; Nodari et al. 1993a; Adam-Blondon et al. 1994; Vallejos 1994) were used to create the core linkage map (Freyre et al. 1998). This map comprised a total of 563 markers, including 120 RFLPs and 430 RAPDs, in addition to a few isozyme and phenotypic marker loci, which were grouped into 11 LGs spanning 1226 cM (Freyre et al. 1998). Later, Vallejos et al. (2001) integrated three linkage maps based on three RIL mapping populations obtained from the 'XR-235-1-1' (Mesoamerican) × 'Calima' (Andean), 'Jamapa' (Mesoamerican) × 'Calima' (Andean) and 'Eagle' (Andean) × 'Puebla 152' (Mesoamerican) crosses that allowed for the placement of 230 RFLPs and 464 RAPDs on the map.

Nevertheless, as with other species, the development of single-locus PCR-based markers such as SSRs or SCARs brought about incomparable progress for common bean genetic mapping research; these quickly replaced RFLPs as the markers of choice for comparing and integrating genetic maps. Among their advantages, SSR markers are multiallelic, codominant, highly polymorphic and have an abundant distribution in plant genomes (Kalia et al. 2011). Yu et al. (2000) published the first successful assignment of 15 SSRs to a framework map based on RAPD and RFLP markers. Subsequently, Blair et al. (2003) developed a total of 150 SSRs: 81 were anonymous genomic or non-coding SSRs and 69 were developed from expressed sequence tag (EST) databases. In this study, 100 SSRs were integrated in a base map developed from the 'DOR364' (Mesoamerican) × 'G19833' (Andean) population in order to anchor two existing linkage maps. The base map comprised a total of 246 loci (78 SSR, 48 RFLP, 102 RAPD and 18 AFLP markers) spanning 1720 cM, with an average distance between SSR loci of 19.5 cM. Thereby, the linkage map developed by Blair et al. (2003) could be classified as the first second-generation genetic map of common bean.

4.3.2 Second-Generation Genetic Maps

In the past few years, common bean genome and EST sequencing programmes have generated large amounts of sequence data, which led to the acceleration in the identification of functional markers. Nowadays, approximately 168,500 common bean sequences have been deposited in the GenBank nucleotide database (http://www.ncbi.nlm.nih.gov/nuccore/; July 2016), and the vast majority of them are EST sequences (~129,000). These have resulted in new research opportunities, such as data mining approaches for searching repetitive motifs (e.g. SSR), single nucleotide polymorphism (SNP) and insertion/deletion (InDel). A large-scale sequence analysis was carried out by Ramírez et al. (2005), who examined over 21,000 EST sequences derived from different cDNA libraries of the Mesoamerican ('Negro Jamapa') and the Andean ('G19833') gene pools. This analysis allowed for the identification of 529 SNPs in 214 kb of contigs, giving one SNP every 387 bp. More recently, data mining approaches have led to the detection of a huge number of polymorphisms from both coding and non-coding regions. Thus, for example, Zou et al. (2014) used 36 common bean genotypes to construct DNA libraries for next-generation sequencing (NGS). By analysing 76 million

Table 4.1 Main common bean populations used for common bean linkage mapping studies

Mapping population	Gene pool	Markers mapped	Reference
XC[a] (68 BC_1)[b]	MA[c]	294 loci (224 RFLPs, 9 seed proteins, 9 isozymes, the gene *P*), 11 LGs, 960 cM	Vallejos et al. (1992, 2001), Vallejos (1994),
MsCo (128 BC_1)	MA	157 loci (51 RFLPs, 100 RAPDs, 2 SCARs, 4 morphological markers), 11 LGs, 567.5 cM	Adam-Blondon et al. (1994)
MiG12 (65 RIL)	AM	82 loci (77 RFLPs, 5 isozymes), 15 LGs, 1,111 cM	Koinange et al. (1996)
DX (79 RIL)	MA	155 loci (147 RAPDs, 2 SCARs, 1 ISSR, and the *R, V, Asp* and two rust resistance genes), 11 LGs, 930 cM	Miklas et al. (1996, 1998, 2000)
BH (128 RIL)	MM	75 RAPDs, 8 LGs, 545 cM	Jung et al. (1996)
PXA (70 RIL)	AA	168 RAPDs, 10 LGs, 426 cM	Jung et al. (1997), Park et al. (2001)
BA (78 RIL)	MM	174 loci (172 RAPDs, 2 SCARs), 11 LGs, 755 cM	Ariyarathne et al. (1999)
EP (75 RIL)	AM	361 RAPDs, 11 LGs, 825 cM	Vallejos et al. (2001)
JaCa (76 RIL)	MA	243 loci (155 RAPDs, 88 RFLPs), 11 LGs, 950 cM	Vallejos et al. (2001)
S95 (142 RIL)	MA	115 loci (49 AFLPs, 43 RFLPs, 11 SSRs, 9 RAPDs, 1 SCAR, 2 morphological markers), 12 LGs, 1,717 cM	Tar'an et al. (2001, 2002)
CDRKY (150 RIL)	AM	192 AFLPs, 15 LGs, 862 cM	Johnson and Gepts (2002)
WOSp (110 F_5)	AM	105 loci (99 RAPDs, 3 SSRs, 3 SCARs), 8 LGs, 641 cM	Beattie et al. (2003)
BG21 (94 RIL)	MM	115 loci (26 SSRs, 89 RAPDs), 8 LGs, 611.2 cM	Frei et al. (2005)
G23G19 (84 RIL)	MA	149 loci (79 SSRs, 57 RAPDs, 11 SCARs, and 1 biochemical and 1 morphological markers), 11 LGs, 1,175 cM.	Ochoa et al. (2006)
IG24 (157 $BC_2F_{3:5}$)	AM	84 loci (80 SSRs, 1 SCAR, 3 morphological markers), 11 LGs, 869,5 cM	Blair et al. (2006)
JulCa (103 F_2)	MA	103 loci (21 RAPDs, 82 AFLPs), 12 LGs, 1,983.6 cM	Yaish et al. (2006)
G19AND (75 RIL)	AA	167 loci (64 SSRs, 11 RAPDs, 91 AFLPs, 1 phenotypic trait), 11 LGs, 1,105 cM	Cichy et al. (2009a)
G14G48 (110 RIL)	MM	114 loci (68 SSRs, 46 RAPDs), 11 LGs, 915.4 cM	Blair et al. (2010)
DB (113 RIL)	MM	291 loci (22 AFLPs, 98 RAPDs, 153 SSRs, 18 ESTs), 11 LGs, 1,788 cM	Blair et al. (2012), Galeano et al. (2011, 2012)
XCo (104 RIL)	AM	349 loci (175 AFLPs, 115 SSRs, 30 SCARs, 12 RAPDs, 13 proteins, 4 genes), 11 LGs, 1,042 cM	Pérez-Vega et al. (2010), Casañas et al. (2013), Trabanco et al. (2014)

(continued)

Table 4.1 (continued)

Mapping population	Gene pool	Markers mapped	Reference
IACAL (380 RIL)	MA	292 SSRs, 11 LGs, 2,058 cM	Campos et al. (2011), Oblessuc et al. (2012, 2013, 2014)
P1037 (185 RIL)	AA	229 loci (86 AFLPs, 98 SSRs, 42 SNPs, 2 SCARs and *P* locus), 11 LGs, 858.4 cM	Yuste-Lisbona et al. (2012, 2014a, b), González et al. (2015)
BJ (70 F_2, 70 RIL)	MA	428 loci (300 gene-based, 103 core and 24 other markers), 11 LGs, 1,545.5 cM	Nodari et al. (1993a), Gepts et al. (1993), Freyre et al. (1998), Gepts (1999), Yu et al. (2000), Hougaard et al. (2008), Hanai et al. (2010), McConnell et al. (2010)
DG (87 RIL)	MA	534 gene-based markers, 11 LGs, 2,400 cM.	Blair et al. (2003), Córdoba et al. (2010a, b); Galeano et al. (2011, 2012)
StRe (267 F_2, 85 RIL)	MA	7,276 SSRs and SNPs, 11 LGs	Schmutz et al. (2014)
SEA5CAL (125 RIL)	MA	2,122 SNPs, 11 LGs, 1,351 cM	Mukeshimana et al. (2014)

[a]Mapping population acronyms: BA = Belneb-RR-1 × A55; BH = BAC6 × HT7719; BJ = BAT93 × JaloEEP558; BG21 = BAT881 × G21212; BA = Belneb-RR-1 × A55; CDRKY = CDRK × Yolano; DB = DOR364 × BAT477; DG19 = DOR364 × G19833; DX = DOR364 × XAN176; EP = Eagle × Puebla152; G14G48 = G14519 × G4825; G19AND = G19833 × AND696; G23G19 = G2333 × G19839; IACAL = IAC-UNA × CAL 143; IG24 = ICACerinza × G24404; JaCa = Jamapa × Calima; JuCa = Jules × Canela; MiG12 = Midas × G12873; MsCo = Ms8EO2 × Corel; P1037 = PMB0225 × PHA1037; PX = PC50 × XAN159; SEACAL = SEA5 × CAL96; StRe = Stampede × Red Hawk; S95 = OACSeaforth × OAC95; Xco = Xana × Cornell49242; XC = XR-235-1-1 × Calima; WOSp = WO3391 × OAC Speedvale

[b]RIL = Recombinant Inbred Line; BC_1 = backcross first generation

[c]M = Mesoamerican; A = Andean

sequence reads generated by the Illumina's HiSeq 2000 Sequencing System, they identified a total of 43,698 putative SNPs and 1267 putative InDels and located 24,907 SNPs and 692 InDels in 8835 and 637 genes, respectively. Likewise, Müller et al. (2014) analysed the ends of 52,270 Bacterial Artificial Chromosome (BAC) libraries from the Mesoamerican breeding line 'BAT93' and identified a total of 3789 SSR loci with a distribution of one SSR per 8.36 kbp. Meanwhile, Wu et al. (2014), using a RNA-seq approach for the discovery of drought-responsive genes, identified a total of 10,482 SSR and 4099 SNP loci in transcripts of the 'Long 22-0579' (Mesoamerican) and 'Naihua' (Andean) common bean cultivars. However, despite the gigantic number of SSR and SNP polymorphisms identified to date, most of them have remained untapped as a source of functional markers and need future validation for practical use in common bean breeding and research.

Common bean linkage maps have been progressively incorporating functional markers. For instance, 108 markers based on genes known to be involved in the nodulation process in model legumes (Galeano et al. 2012) were evaluated by Ramaekers et al. (2013) in the RIL population generated from the cross between the Mesoamerican 'G2333' and the Andean 'G19839' genotypes. This mapping population has been previously used in several genetic studies (Ochoa et al. 2006; Checa and Blair 2008; Caldas and Blair 2009); thus, the existing genetic map was improved through the mapping of 42 out of 108 nodulation gene-based markers. The final linkage map consisted of a total of 207 markers (57 RAPDs, 106 SSRs, 42 SNPs, one SCAR and one isozyme) grouped into 11 LGs with a total map length of 1601 cM. Using this improved genetic map, Ramaekers et al. (2013) performed QTL analysis for the symbiotic nitrogen fixation capacity, and candidate genes were tentatively identified among the nodulation markers.

Nowadays, the RIL populations derived from the 'BAT93' × 'Jalo EEP558' and 'DOR364' × 'G19833' inter-gene pool crosses are considered as core mapping populations since both populations have been widely used for genetic mapping studies and QTL identification (Freyre et al. 1998; McClean et al. 2002, 2010; Blair et al. 2003, 2009a; Liao et al. 2004; Beebe et al. 2006; Hougaard et al. 2008; Caldas and Blair 2009; López-Marín et al. 2009; Hanai et al. 2010; McConnell et al. 2010; Galeano et al. 2011, 2012). Markers with putative gene functions have also been included in the extension of both core linkage maps. For the RIL population 'BAT93' × 'Jalo EEP558', EST libraries from anthracnose-infected common bean leaves (Melotto et al. 2005) were screened for microsatellites by Hanai et al. (2010), yielding a set of 140 EST-SSR markers. In addition, Resistance Gene Analogs (RGAs)-based markers were also developed. The merging of the data of the 285 new loci (50 EST-SSR, 32 RGA and 203 AFLP markers) mapped by Hanai et al. (2010) with the data of 143 markers previously mapped by Freyre et al. (1998) resulted in a map which comprised 413 loci. These loci were placed across 11 LGs and spanned a genetic distance of 1259 cM with an average distance between neighbouring loci of 3.0 cM. Likewise, the previous genetic map of the RIL population 'DOR364' × 'G19833' was updated by Galeano et al. (2012), who developed a total of 313 intron-based EST-SNP markers. Thus, the final genetic map consisted of 534 marker loci distributed into 11 LGs with a full map length of 2400 cM.

In addition, in order to map individual or multiple traits, new mapping populations have been developed in the last few years. Thus, a RIL population derived from an inter-gene pool cross between 'Xana' (Andean) and 'Cornell 49242' (Mesoamerican) was used to develop a genetic map including 349 markers (175 AFLPs, 115 SSRs, 30 SCARs, 12 RAPDs, 13 loci codifying for seed proteins and four genes) distributed into 11 LGs, with a total length of 1042 cM (Pérez-Vega et al. 2010; Casañas et al. 2013; Trabanco et al. 2014). Likewise, the 'PMB0225' × 'PHA1037' Andean intra-gene pool RIL population has been used to study the inheritance of different agronomic and resistance traits (Yuste-Lisbona et al. 2012, 2014a, b; González et al. 2015). The last version of this genetic map consisted of 229 loci (86 AFLPs, 98 SSRs, 42 SNPs, 2 SCARs and the *P* locus), which were distributed into 11 LGs and spanned 858.4 cM (González et al. 2015). Moreover, the 'IAC-UNA' (Mesoamerican) × 'CAL 143' (Andean) RIL population has been used to detect loci controlling growth habit and disease resistance (Campos et al. 2011; Oblessuc et al. 2012, 2013, 2014). The updated version of this map had 292 SSR markers distributed into 11 LGs spanning a total map length of 2058 cM (Oblessuc et al. 2014). Furthermore, in order to assign markers to chromosomes and construct the LGs, SSR markers were located in the *P. vulgaris* chromosomes using the native Phytozome's BLAST and default algorithm parameters (http://www.phytozome.net/). As a result, the Oblessuc et al. (2014) map was more consistent with the genome sequence, and some markers mapped to different chromosomes in relation to the previous analysis (Campos et al. 2011; Oblessuc et al. 2012, 2013).

The International Center for Tropical Agriculture (CIAT), as part of the Harvest Plus challenge programme on Biofortification, has developed different mapping populations in order to improve the iron and zinc concentration in both gene pools (Blair et al. 2009a, 2010, 2011; Cichy et al. 2009a, b). In addition to the 'DOR364' × 'G19833' inter-gene pool RIL population, a genetic map of the 'G19833' × 'AND696' Andean intra-gene pool RIL population was developed by Cichy et al. (2009a). This linkage map consisted of a total of 167 markers (64 SSRs, 11 RAPDs, 91 AFLPs and 1 phenotypic trait) with 11 LGs and a total length of 1105 cM. Likewise, another Andean genetic map was created with the RIL population derived from the 'G21242' × 'G21078' cross. The genetic map was created using a total of 74 SSRs so as to anchor the map to previously published reference maps and 42 RAPDs, which were distributed into 11 LGs and spanned 726 cM (Blair et al. 2011). Furthermore, the 'G14519' 'G4825' Mesoamerican RIL population was used to examine the inheritance of seed iron and zinc concentrations (Blair et al. 2010). The

genetic map for the 'G14519' × 'G4825' population was constructed with a total of 68 SSRs and 46 RAPDs grouped into 11 LGs spanning 915.4 cM of the common bean genome.

Additionally, a new linkage map was developed by Galeano et al. (2011) using the 'DOR364' × 'BAT477' Mesoamerican intra-gene pool population. This map was constructed by evaluating a total of 2706 molecular markers (including SSR, SNP and gene-based markers) and consisted of 291 loci distributed into 11 LGs with a total map length of 1788 cM. In order to create a consensus map for fine mapping and synteny analysis in common bean, the 'DOR364' × 'BAT477' map was merged with the previously existing linkage maps of both 'BAT93' × 'JALO EEP558' and 'DOR364' 'BAT477' core populations. Thereby, the consensus map consisted of a total of 1,060 markers distributed into 11 LGs and a total map length of 2041 cM with an average distance between adjacent loci of 1.9 cM (Galeano et al. 2012). The common bean consensus map includes a higher number of loci than most single cross maps, thus increasing the number of potentially useful markers across divergent genetic backgrounds and providing broader genome coverage.

Functional genetic maps based on genes involved in physiological processes potentially underlying important agronomic traits allow for the identification of candidate genes through translational genomics. Thereby, Kwak et al. (2008) identified common bean homologues of 12 *Arabidopsis thaliana* genes related to floral transition and flowering pathways. Seven out of 12 genes could be mapped using the 'BAT93' 'JaloEEP558' and 'Midas' × 'G12873' RIL populations. Thus, three *Terminal Flower 1* homologues (*PvTFL1x*, *PvTFL1y* and *PvTFL1z*) were mapped. *PvTFL1y* co-segregated with the phenotypic locus for determinacy growth habit (*fin*) on LG01, whereas *PvTFL1z* mapped near or at a second determinacy locus on LG07 (Kolkman and Kelly 2003). In addition, a *Zeitlupe* homologue mapped close to a QTL for flowering time on LG09 (Kwak et al. 2008). These results support the role of functional maps including genes of known function as an important component of the candidate gene approach. However, further studies are needed to confirm the role of these homologues as potential candidate genes.

Moreover, functional maps are useful for synteny studies among different species. Sequence data from legumes are available in the Legume Information System (LIS: http://phavu.comparative-legumes.org/gb2/gbrowse/Pv1.0/; Dash et al. 2015) which is focussed on legume comparative analysis. Thus, in order to investigate the syntenic relationship between *P. vulgaris*, *A. thaliana*, *Medicago truncatula* and *Lotus japonicus*, a gene-based map was developed by McConnell et al. (2010) using the 'BAT93' × 'JaloEEP558' RIL population. The map included a total of 420 loci (304 gene-based markers, 103 core markers and 13 colour gene markers), which were sorted into 11 LGs and spanned 1545.5 cM. The genetic map information and the marker sequences were used as a query in a 'tblastx' analysis with the genome sequence of each of the species. The results showed that while only short blocks of synteny were observed with *A. thaliana*, large-scale macrosyntenic blocks were observed with *M. truncatula* and *L. japonicus*. These syntenic relationships are in accordance with the results previously obtained by Hougaard et al. (2008), who carried out the first attempt at estimating the extent of synteny and collinearity among these species based on 104 legume anchor-marker loci representing single-copy genes. Similarly, this gene-based map was used by McClean et al. (2010) to understand syntenic relationship between common bean and soybean. Genetically positioned transcript loci of common bean were mapped in relation to the soybean 1.01 genome assembly (http://soybase.org/gbrowse/cgi-bin/gbrowse/gmax1.01/). In nearly every case, each common bean locus mapped into two positions in soybean, a result consistent with the duplicate polyploidy history of soybean. Furthermore, by this genetic/physical synteny approach, McClean et al. (2010) were also able to electronically position ~15,000 common bean sequences (primarily EST contigs and EST singletons) onto the common bean map using the shared syntenic

blocks as reference points. Therefore, this extensive gene-based map significantly expands the genomic resources available for common bean and provides a framework for comparative genetics and genomics of legumes.

Currently, the genomes of the Andean 'G19833' (http://www.phytozome.net/commonbean.php/; Schmutz et al. 2014) and the Mesoamerican 'BAT93' are available (http://denovo.cnag.cat/genomes/bean; Vlasova et al. 2016), while the genome of the Andean 'OAC-Rex' is underway (Canadian team, http://www.beangenomics.ca/research/projects/view/draft-genome-sequence-for-common-bean-i-p-vulgaris-i/). In this way, a large number of specific disease resistance genes have been identified and located in the genome, constituting a valuable material to design new functional molecular markers in common bean (Meziadi et al. 2015). Hence, the huge amount of sequence information available for common bean via whole-genome sequencing projects facilitates the development of an almost unlimited number of genetic markers suitable for high-throughput genotyping and easily transferable across different mapping populations. The PhaseolusGenes database was developed as part of the BeanCAP project (http://www.beancap.org/; http://phaseolusgenes.bioinformatics.ucdavis.edu/), including phenotypic, genotypic and molecular marker data collected from publications and projects throughout the world and a genome browser in order to place markers on assembled common bean and soybean genomes. The BeanCAP project has also carried out the design of BeadChips that are being used to genotype bean populations (Song et al. 2015). Two of these BeadChips (BARCBEAN6K_1 with 5,232 SNP markers and BARCBEAN6K_2 with 5,514 SNP markers) have been used by Schmutz et al. (2014) in the 'G19833' genome sequencing. The resulting assembled sequence was organized into 11 chromosomes by integration with a map of 7015 SNP markers, typed on 267 F_2 lines from the 'Stampede' (Mesoamerican) × 'Red Hawk' (Andean) cross, and a similar set of SNP and 261 SSR markers, typed on 88 F_5-RIL population derived from the same cross. Thus, the final genetic map contained 7276 SSR and SNP markers arranged in 11 LGs. Another BeadChip (BARCBEAN6K_3 with 5389 SNP markers) has been recently used by Mukeshimana et al. (2014). A total of 2122 SNP markers were mapped in the 'SEA5' (Mesoamerican) × 'CAL96' (Andean) RIL population. The genetic map spanned 1351 cM and covered all 11 LGs with an average distance of 0.64 cM between markers. The Mukeshimana et al. (2014) results showed that SNP marker order and location in the 'SEA5' × 'CAL96' map generally agreed with order and chromosome assignment in the 'Stampede' × 'Red Hawk' common bean map. Therefore, such high-throughput genotyping approaches allow for the rapid and cost-effective generation of high-density functional maps, which could also lead to the direct gene tagging for QTL mapping of important agronomic traits, improving the efficiency of common bean breeding programmes via MAS.

4.4 Molecular Mapping of Simple and Complex Traits

QTL mapping has become very popular in bean genetics and breeding research, where QTL have been identified for numerous agronomical and biological important complex traits. This section will summarize the most important genes and QTL, which have been described and mapped during the past decades in common bean.

4.4.1 Genes and QTL Involved in Biotic Stress Resistance

Identification of genetic markers associated with disease resistance in common bean started in 1970s with the pioneering work of Coyne and his co-workers who identified an association between the common bacterial blight (CBB) resistance and late flowering (Coyne et al. 1973). Since then, numerous genetic markers have been

used to map major genes and QTL conferring resistance to common bean diseases. An overview of the main resistance genes and QTL as well as their location on the common bean linkage map is summarized in Table 4.2.

4.4.1.1 Virus Diseases

Resistance to different pathogroups of the potyviruses such as bean common mosaic virus (BCMV) and bean common mosaic necrosis virus (BCMNV) is conferred by four different recessive loci: *bc-1*, *bc-2*, *bc-3* and *bc-*

Table 4.2 Details of QTL mapping studies performed for the mapping of major genes and QTL for different biotic stress resistance in common bean

Disease[a]	Gene/QTL name[b]	LG[c]	Mapping population[d]	Reference
BCMV/BCMNV	*I*	2	BA	Ariyarathne et al. (1999)
	bc-1^{2}, *bc-u*	3	DG, OS	Strausbaugh et al. (1999)
	bc-3	6	CIAT breeding lines	Johnson et al. (1997)
BGYMV	*bgm-1*	3	DS	Blair et al. (2007)
	BGMV	4, 7	DX	Miklas et al. (2000)
ANT	*Co-1, Co-w, Co-x, Co-1*$^{65-X}$, *Co1*$^{73-X}$, *SDC*^{23}*-1, PDC*^{1545}*-1, PAUDPC*^{1545}*-1*	1	BJ, XCo, P1037	Geffroy et al. (2008), Campa et al. (2014), González et al. (2015)
	Co-u, CoPv02c^{3-X}, *CoPv02c*$^{7-X}$, *CoPv02c*$^{19-X}$, *CoPv02C*$^{449-X}$	2	BJ, XCo	Geffroy et al. (2008), Campa et al. (2014)
	Co-13, Co-17, PAUDPC^{1545}*-3, LDC*^{23}*-3*	3	JMex, JCo, SM, P1037	Gonçalves-Vidigal et al. (2009), Trabanco et al. (2015), González et al. (2015)
	Co-3, Co-10, Co-9, Co-y, Co-z, Co-15, Co-3c^{3-X}, *Co-3c*$^{7-X}$, *Co-3c*$^{19-X}$, *Co-3c*$^{449-X}$, *Co3c*$^{453-X}$, *SDC*$^{23-}$*4, SAUDPC*$^{23-}$*4, LDC*^{23}*-4.1, LAUDPC*^{23}*-4*	4	RuOu, Aou, BJ, CoCo, XCo, P1037	Geffroy et al. (2000), Gonçalves-Vidigal et al. (2013), Campa et al. (2014), González et al. (2015), Sousa et al. (2015)
	SDC^{1545}*-5, SAUDPC*^{1545}*-5, PDC*^{1545}*-5, PAUDPC*^{1545}*-5, LDC*^{1545}*-5, LAUDPC*$^{1545-5}$	5	P1037	González et al. (2015)
	Co-5, Co-6, LDC^{1545}*-7, LAUDPC*^{1545}*-7*	7	TM, ABMi, P1037	Campa et al. (2007, 2009), González et al. (2015)
	Co-4, SDC^{1545}*-8, SAUDPC*^{1545}*-8, LDC*^{1545}*-8, LAUDPC*^{1545}*-8*	8	SM, P1037	Trabanco et al. (2015), González et al. (2015)
	CoPv09c^{453-C}, *LDC*^{23}*-9, LAUDPC*^{23}*-9*	9	XCo, P1037	Campa et al. (2014), González et al. (2015)
	Co-2, Co-2^{6-C}, *Co-2*$^{39-C}$, *Co-2*$^{38-C}$, *and Co-2*$^{357-C}$	11	MsCo, XCo	Adam-Blondon et al. (1994), Campa et al. (2014)
FRR	$P7_{1550}$, $P7_{700}$, $P10_{1600}$, $G6_{1100}$, $D3_{600}$, $I18_{1800}$, $I18_{1700}$, $AG2_{800}$, $G17_{900}$, $G3_{800}$, $G3_{2000}$, $P9_{1550}$, $Y11_{600}$, $O12_{800}$, $S8_{500}$, $V12_{1100}$	1, 2, 3, 4, 5, 6, 7	MF	Schneider et al. (2001)
	$UBC2I8_{1200}$/$UBC503_{640}$, $UBC503_{640}$/$UBC211_{1000}$	6	ACNY	Chowdhury et al. (2002)
	$AL20_{850}$/$G8_{1400}$, $O12_{800}$/$AL20_{850}$, $S19_{1000}$/$S19_{1100}$, $G17_{900}$/$AL20_{350}$, $AL20_{700}$/$G6_{2000}$, $AJ4_{350}$/$X3_{3054}$, $AN19_{1300}$/$H4_{1200}$	1, 5, 7, 9, 8	CNSL	Román-Avilés and Kelly (2005)
	FRR3.1 ^{km}	3	K32MLB, K20MLB	Kamfwa et al. (2013)
	ARR2.1, ARR4.1, ARR6.1, FRR3.1,FRR7.1	1, 3, 4, 6, 7		Hagerty et al. (2015)

(continued)

Table 4.2 (continued)

Disease[a]	Gene/QTL name[b]	LG[c]	Mapping population[d]	Reference
WM	*WM1.1, WM7.1*	1, 7	AG	Miklas et al. (2001)
	WM2.1, *WM4.1*, *WM5.1*, *WM8.1*	2, 4, 5, 8	PX	Park et al. (2001)
	WM2.2, WM7.2	2, 7	BuN	Kolkman and Kelly (2003)
	WM2.3, WM5.2, WM7.2, WM8.4	2, 5, 7, 8	IBR	Ender and Kelly (2005)
	WM1.2, *WM2.4, WM8.2, WM8.3, WM9.1*	1, 2b, 8, 9	GCO	Maxwell et al. (2007)
	WM2.2, WM4.2, WM5.3, WM5.4, WM6.1, WM7.3, *WM8.4*	2, 4, 5, 6, 7	R31	Soule et al. (2011)
	WM2.2, WM8.3	2, 8	BV	Soule et al. (2011)
	WM3.3, WM7.5, WM9.2, WM11.1	3, 7, 9, 11	TPI95, TPI50	Mkwaila et al. (2011)
	WM1.3, WM3.2, WM6.2, WM7.4, WM8.5, WM11.12	1, 3, 6, 7, 8, 11	XCo	Pérez-Vega et al. (2012)
Rust	*Ur-9*	1	PC	Miklas et al. (2002)
	Pu-a	3	PX	Jung et al. (1998)
	Ur-5, Ur-Dorado108, *Ur-ON* (*Ur-14*)	4	DX, OUB, OUM	Miklas et al. (2000), Souza et al. (2011)
	Ur-4	6	BJ	Miklas et al. (2002)
	Ur-12	7	PX	Jung et al. (1998)
	Ur-13, *Crg*	8	KB, Sierra mutagenized seed	Kalavacharla et al. (2000), Mienie et al. (2005)
	Ur-3, Ur-11, Ur-Dorado53, Ur-6, Ur-7, Ur-BAC6	11	P07Be, P32Be, DX, BH, BA	Stavely (1998), Miklas et al. (2000, 2002)
CBB	*D1, D2, D5, D9*	1,5,7,9	BJ	Nodari et al. (1993b)
	CBB-1LL, CBB-2LL, CBB-2S, CBB-2P, CBB-2FL	1, 2, 3, 4, 5, 6	BH	Jung et al. (1996)
	CBLEAF, CBPOD	1, 2, 9, 10	BA	Ariyarathne et al. (1999)
	FT-1, FT-2, LDT-2, Pod-1, Pod-2, Seed-1, Seed-2	1, 4, 5, 9	PX	Jung et al. (1997)
	Bng40, Bng139	7, 8	XC	Yu et al. (1998)
	CBB-GH-leaf, CBB-GH-pod, CBB-field	7, 10	DX	Miklas et al. (2000)
	SU91, SAP6, Xa11.4^{OV1,OV3}	8, 10, 11	OV1, OV3	Viteri et al. (2015)
HB	*Pse1, Pse2, Pse3, Pse4, Pse6*	2, 4, 10	CWU, ZCW, BA	Miklas et al. (2009, 2011, 2014)
	Stem, 96LFA, 98LFA, 96BBS, 98BBS	1, 2, 3, 4, 5, 6, 7, 8, 9, 11	BA	Jung et al. (2003)
	Rpsar-1, Rpsar-2	8, 11	BJ	Fourie et al. (2004)
	HB83, HB16	2, 3, 4, 5, 9, 10	BA	Ariyarathne et al. (1999)
	Psp4^{812XC}, *Psp6.1*^{812XC}, *Psp6.1*^{684XC}, *Psp6.2*^{684XC}	4, 6	XCo	Trabanco et al. (2014)

(continued)

Table 4.2 (continued)

Disease[a]	Gene/QTL name[b]	LG[c]	Mapping population[d]	Reference
	SAUDPC^{3}-2, PLAUDPC^{3}-2, PDC-^{3}2, PDC^{4}-2, PDC^{5}-2, PAUDPC^{3}-2, PAUDPC^{4}-2, SDC^{7}-6	2, 6	P1037	González et al. (2016)
ALS	*Phg-1*	1	AOu	Gonçalves-Vidigal et al. (2011)
	Phg-2	8	BJ	Miklas et al. (2006)
	Phg_{G5686A}, Phg_{G5686B}, Phg_{G5686C}, $Phg_{G10909A}$, $Phg_{G10909B}$	4, 8, 9	G56Sp, G10Sp	Mahuku et al. (2009, 2011)
	Phg-ON (Phg-3)	4	RuOu, Aou	Gonçalves-Vidigal et al. (2013)
	ALS	4, 10	DG19	López et al. (2003)
	ALS2.1^{UC}, ALS3.1^{UC}, ALS4.1^{GS,UC} ALS4.2^{GS,UC}, ALS5.1^{UC}, ALS5.2^{UC}, ALS10.1^{DG,UC}	2, 3, 4, 5, 10	UC, GS	Oblessuc et al. (2012)

[a]*ALS* = angular leaf spot; *ANT* = anthracnose; *BGYMV* = bean golden yellow mosaic virus; *BCMV* = bean common mosaic virus; *BCMNV* = bean common mosaic necrosis virus; *CBB* = common bacterial blight; *FRR* = *Fusarium* root rot; *HB* = halo blight; *WM* = white mould

[b]*Co* = anthracnose; *Ur* = rust; *Pse* and *Rpsar* = halo blight; *Phg* and *ALS* = angular leaf spot; *I* and *bc* = BCMV/BCMNV resistance loci

[c]LG = linkage group

[d]Mapping population acronyms: ABMi = AB136 × Michelite; ACNY = A.C. Compass × NY2114-12; AG = A55 × G122; AOu = AND277 × Ouro Negro; BA = Belneb-RR-1 × A55; BH = BAC6 × HT7719; BJ = BAT93 × JaloEEP558; BV = Benton × VA19; BuN = Bunsi × Newport; CoCo = Corinthiano × Cornell 49-242; CNSL = C97407 × Negro San Luís; CWU = Canadian Wonder × UI-3; DG19 = DOR364 × G19833; DS = DOR476 × SEL1309; DX = DOR 364 × XAN 176; EP = Eagle × Puebla 152; EEP = Eagle*2 × Puebla 152; EPH = Eagle*2 × Hystyle; GCO = G122 × CO72548; G10Sp = G10909 × Sprite; G56Sp = G5686 × Sprite; H95 = HR67 × OAC95; IBR = ICA Bunsi × Raven; JCo = JLP × Cornell 49242; JMex = JLP × Mexico 222; JuCa = Jules × Canela; KB = Kranskop × Bonus; K32MLB = K132 × MLB-49-89A; K20MLB = K20 × MLB-49-89A; MF = Montcalm × FR266; MsCo = Ms8EO2 × Corel; OS = Olathe × Sierra; OUB = Ouro Negro × Belmidak RR-3; OUM = Ouro Negro × Mexico309; OUS = Ouro Negro × US Pinto 111; OV1 = Othello × VAX 1; OV3 = Othello × VAX 3; P07Be = P94207 × Beltsville; P32Be = P94232 × Beltsville; P1037 = PMB0225 × PHA1037; PC = PC50 × Chichara-83-109; PX = PC50 × XAN159; R31 = Raven × I9365-31; RNSL = Red Hawk × Negro San Luís; RuOu = Ruda × Ouro Negro; S95 = OACSeaforth × OAC95; SM = SEL1308 × MDRK; TPI95 = Tacana × PI318695; TPI50 = Tacana × PI313850; UC = IAC-UNA × CAL143; XC = XR-235-1-1 × Calima; XCo = Xana × Cornell49242; ZCW = ZAA12 × Canadian Wonder

u (Drijfhout 1978). In addition, the dominant *I* gene confers immune resistance to all strains of BCMV through a hypersensitive response (Ariyarathne et al. 1999). As regards the begomovirus such as bean golden yellow mosaic virus (BGYMV), a Mesoamerican source of partial resistance, is conditioned by the recessive *bgm-1* gene (Blair et al. 2007). The Andean-derived recessive *bgm-2* gene (Velez et al. 1998) and dominant *Bgp-1* gene, which confers resistance to pod deformation and requires the presence of *bgm-1* for complete expression (Molina Castañeda and Beaver 1998), have also been reported. Furthermore, two independent QTL have been identified for BCMV resistance, explaining 60% of the phenotypic variation (Miklas et al. 2000). One of these QTL was located on LG07 close to the *Asp* and *Phs* loci, together with other QTL or major genes conditioning resistance to CBB, white mould, anthracnose and stem blight (Nodari et al. 1993a; Miklas et al. 2000, 2001).

4.4.1.2 Fungal Pathogens

With respect to fungal diseases, over 40 genes have been described as conferring resistance to anthracnose (labelled as *Co-*), caused by the fungus *Colletotrichum lindemuthianum*. Anthracnose resistance is related to the presence of closely linked race-specific loci, which comprise different single, duplicate or complementary dominant genes, except for the recessive *co-8* (Ferreira et al. 2013; Campa et al. 2014). Moreover, a major QTL located on LG04 explained 70% of resistance to race 45 co-localized with the genes *Co-9*, *Co-y* and *Co-z* at the end of LG04 (Geffroy et al. 2000). Clusters of nucleotide-binding site–leucine-rich

repeat (NBS-LRR) genes have been identified on this region of the LG04, as well as on LG11, which co-localize with previously mapped *Co-3* and *Co-2* genes, respectively (Schmutz et al. 2014). Likewise, 17 out of 26 main-effect QTL and 20 epistatic interactions were detected by González et al. (2015) harbouring NBS-LRR genes.

Several studies demonstrated that resistance to Fusarium root rot (FRR, fungus: *Fusarium* species) is controlled by several genes located at different loci. Over 30 QTL for FRR resistance (many minor in effect) have been reported in RIL populations derived from several resistance sources (Table 4.2). Most of the QTL detected by Schneider et al. (2001) were located on LGs 02 and 03, close to a region where defence response genes, *Pgip* and *ChS*, and pathogenesis-related protein genes, *PvPR-1* and *PvPR-2*, have been positioned. Additionally, Román-Avilés and Kelly (2005) identified two QTL on LGs 02 and 05, the former located near the QTL previously detected by Schneider et al. (2001) on LG02. Likewise, Kamfwa et al. (2013) mapped the *FRR3.1*^{KM} close to the *PvPR-1* gene on LG03.

The *Sclerotinia sclerotiorum* fungus is the causal agent of white mould (WM). Genetic resistance to WM is quantitatively inherited with low–to-moderate heritability (Park et al. 2001). Single major and numerous weak QTL for WM resistance have been identified. Among them, Miklas et al. (2001) reported a single major-effect QTL located on LG07 that accounted for 38% of the total phenotypic variation and was closely linked to the *Phs* locus. Association among WM physiological resistance and disease avoidance traits was also investigated by Miklas et al. (2013), whose results showed 13 WM resistant QTL associated with disease avoidance traits.

Resistance to bean rust, caused by the *Uromyces appendiculatus* fungus, is mainly controlled by major single dominant genes (named as *Ur-1* to *Ur-14*). Clustering is observed for rust resistance and other disease resistance genes. Thus, *Co-1* and *Ur-9* genes co-localize on LG01 and *Co-3/Co-9*, *Co-10*, *Ur-5* and *Ur-Dorado-108* co-localize on LG04 (Miklas et al. 2006), while *Ur-3*, *Ur-11* and *Ur-Dorado53* map close to *Co-2* on LG11 (Miklas et al. 2002). Similarly, *Ur-13* is located on LG08 near the *Phg-2* gene for resistance to angular leaf spot (Garzon et al. 2014).

4.4.1.3 Bacterial Diseases

Studies on CBB genetics, disease caused by *Xanthomonas axonopodis* pv. *phaseoli*, reported quantitative inheritance with largely additive effects. Thus, more than twenty minor and major QTL for CBB resistance have been identified across all 11 LGs (Nodari et al. 1993b; Jung et al. 1996, 1997; Yu et al. 1998; Ariyarathne et al. 1999; Miklas et al. 2000; Viteri et al. 2015). Although specific genes associated with resistance to CBB have not been identified, genomic regions which are likely to contain genes for resistance to this disease have been found. Thus, for example, Miklas et al. (2003) detected one genomic region on LG10 associated with the RAPD marker *AP6*^{820} that explained up 60% of the phenotypic variance for CBB resistance.

Both qualitative and quantitative responses to halo blight (HB), caused by *Pseudomonas syringae* pv. *phaseolicola* (*Psp*), have been described (Ariyarathne et al. 1999; Fourie et al. 2004; Miklas et al. 2009, 2011, 2014; Trabanco et al. 2014; González et al. 2016). Five dominant (*Pse-1*, *Pse-2*, *Pse-3*, *Pse-4* and *Pse-6*) and one recessive (*pse-5*) genes were identified among the set of differential cultivars by means of complementary tests (Ferreira et al. 2013; Miklas et al. 2014). Furthermore, two independent genes that confer *AvrRpm1*-specific resistance (*Rpsar-1* and *Rpsar-2*) were located near genes that confer resistance to the *C. lindemuthianum* fungus (Fourie et al. 2004). Quantitative response has also been observed; thus, González et al. (2016) detected 76 main-effect QTL that explained up to 41% of the phenotypic variation for HB resistance, although they also identified 101 epistatic QTL, which suggest that epistasis plays an important role in the genetic control of this trait. Additionally, Trabanco et al. (2014) searched for candidate genes associated with HB resistance and identified 16 candidate genes in the physical positions in which the QTL *Psp6.1*^{812XC}, *Psp6.1*^{684XC} and *Psp6.2*^{684XC} were mapped.

These candidate genes carried sequences homologous to the resistance genes *RPM1*, *FLS2*, *RPG1*/*RPG1*-B and *Pto*, all of which confer resistance to *P. syringae* in different species.

Resistance to the angular leaf spot (ALS) (caused by the *Pseudocercospora griseola* Sacc. fungus) is mediated by several independent genes, which possess one or more alleles conferring resistance to several races of the fungus (Miklas et al. 2006; Mahuku et al. 2009, 2011; Gonçalves-Vidigal et al. 2011, 2013). Quantitative resistance has also been reported (López et al. 2003; Oblessuc et al. 2012). López et al. (2003) found a cluster of RGA on LG10 associated with one major QTL for resistance to different ALS isolates, explaining from 47 to 64% of the phenotypic variance depending on the isolate used. Moreover, seven QTL on five LGs were detected by Oblessuc et al. (2012). Among these, $ALS10.1^{DG,UC}$ on LG10 presented major effects, explaining between 16 and 22% of the phenotypic variance for ALS resistance. The QTL $ALS4.1^{GS,UC}$ was fine-mapped with two closely linked SNP markers (Marker50 and 4M437) to a region on LG04 containing 36 candidate genes (Keller et al. 2015).

4.4.2 Genes and QTL Involved in Abiotic Stress Resistance

To date, several studies have reported QTL that may play a role in mitigating the negative effects of abiotic stresses in common bean (Table 4.3). The importance of abiotic stress is unquestionable, especially in low-input agricultural systems of underdeveloped countries, where conventional breeding may be insufficient because of the fact that global climate change increases the frequency and severity of abiotic constraints. It is important to unravel molecular mechanisms in response to abiotic stress in common bean, which would help accelerate genetic improvement through MAS.

4.4.2.1 Drought Tolerance

Drought is the most important abiotic stress that limits crop productivity worldwide (Lauer et al. 2012). Although some drought-responsive genes have been reported in common bean (Blair et al. 2016), breeding for drought is complex due to the number of traits involved, quantitative inheritance and environmental influence (Mir et al. 2012).

In the absence of an effective linkage map, Schneider et al. (1997) studied the genetics of the response to drought across a broad range of environments in the 'Sierra' × 'AC1028' and 'Sierra' × 'Lef-2RB' populations using RAPD markers and multiple regression analyses. Nine markers were reported for drought resistance, although they were located on non-anchored LGs. A RIL population from the 'SEA 5' × 'MD 23-24' cross was evaluated under drought and irrigated conditions in two seasons, and common and specific QTL for drought were identified (Beebe et al. 2007). The most significant result was that in no case were one locus' alleles specifically adapted to the contrary environments (i.e., one allele to drought conditions and the other allele to favourable conditions). This implies that yield under drought and yield under well-watered conditions are not mutually exclusive and can be combined. Five QTL were detected by composite interval mapping for seed yield under drought irrigated conditions over 3 years in an intra-gene pool RIL population derived from 'BAT477' × 'DOR364' cross (Blair et al. 2012). Positive alleles for the QTL came from each parent, indicating that both contributed to yield in the drought treatment. The same mapping population was analysed with a mixed model methodology to dissect QTL of root traits associated with contrasting water availability (Asfaw and Blair 2012), and nine QTL were mapped for drought stress tolerance on six of the 11 LGs. Mukeshimana et al. (2014) used an inter-gene pool RIL population derived from the 'SEA5' × 'CAL96' cross for the identification of QTL for performance under drought stress. A mapping population from the

Table 4.3 Details of QTL mapping studies performed for the mapping of major genes and QTL for different abiotic stress tolerance in common bean

Stress[a]	Gene/QTL name[b]	LG[c]	Population[d]	Reference
Drought	$OA08_{780}$, $OA04_{560}$, $OX11_{680}$, $OZO8_{750}$, $OXI8_{980}$	unknown	SL, SAC	Schneider et al. (1997)
	Yld4.1, *Yld6.1*, *Yld8.1*, *Yld8.2*, *Yld10.1*	4, 6, 8, 10	DB	Blair et al. (2012)
	Cbm3.1, Ppi3.1, Hri3.1, *Stc5.1, Stc6.1*, *Scr6.1, Yld8.1*, *Sbr9.1*	3, 5, 6, 8, 9,	DB	Asfaw et al. (2012)
	$PHI1.1^{SC}$, $NP3.1^{SC}$, $SW3.1^{SC}$, $SW7.2^{SC}$, $SY9.2^{SC}$,	1, 3, 7, 9	SC	Mukeshimana et al. (2014)
	$SY1.1^{BR}$, $SY2.1^{BR}$	1, 2	BR	Trapp et al. (2015)
Zn Deficiency	*Znd*	unknown	MT	Singh and Westermann (2002)
	QTL1-Zn	4	BG	Guzmán-Maldonado et al. (2003)
	BM154/BM184	9	VA	Gelin et al. (2007)
	Zn-ICPa3, Zn-ICPa7, Zn-ICPa11	3, 7, 11	DG	Blair et al. (2009a)
	SeedZn	1, 5, 6, 11	AG	Cichy et al. (2009a)
	QZnPoAA2.1, QZnPoAA3.1, QZnPoAA6.1, QZnDaAA8.1, QZnPaAA6.1, QZnPaAA8.2,	2, 3, 6, 8,	G14G48	Blair et al. (2010)
	Zn-AAS2c, Zn-AAS7c, Zn-AAS8c	2, 7, 8	G42G78	Blair et al. (2011)
	Zn_cont3.1, Zn_cont5.1, Zn_cont5.2, Zn_cont7.1	3, 5, 7	CCCG	Blair and Izquierdo (2012)
Fe Deficiency	*QTL1-Fe*, *QTL2-Fe*	2, 3	BG	Guzmán-Maldonado et al. (2003)
	Fe-ICPa4, *Fe-ICPa6*, *Fe-ICPa7*, *Fe-ICPa8.1*, *Fe-ICPa8.2*, *Fe-ICPa11.1*	4, 6, 7, 8, 11	DG	Blair et al. (2009a)
	SeedFe	1, 5, 6, 8, 9, 11	AG	Cichy et al. (2009a)
	QFeDaAA4.1, QFePaAA6.1, QFePoAA6.1, QFePaAA7.1,	4, 6, 7	G14G48	Blair et al. (2010)
	Fe-AAS2a, Fe-AAS6b, Fe-AAS6c	2, 6	G42G78	Blair et al. (2011)
	Fe7.1, Fe_cont8.1	7, 8	CCCG	Blair and Izquierdo (2012)
P Efficiency Al Toxicity	*Pup4.1*, *Pup10.1*	4, 10	DG	Yan et al. (2004)
	Pup3.1, Pup4.1, Pup7.1, Pup9.1, Pup10.1, Pup11.1	3, 4, 7, 9, 10, 11	DG	Liao et al. (2004)
	Pup4.1, Pup10.1	4, 10	DG	Beebe et al. (2006)
	LPAdvNoF.1, LPAdvNoF.2	2, 9	G23G19	Ochoa et al. (2006)
	PupLP, PueLP, PupLP	7, 8, 11	AG	Cichy et al. (2009b)
	Tsp2.1, Npc6.1, Npc7.1, Npc10.1, Tsp2.1, Tsp2.1 Tsp11.1	2, 6, 7, 10, 11	G23G19	Blair et al. (2009b)
	Srl2.1, Nrt3.1, Nrt5.1, Ard6.1, Srl7.1, Ard7.1, Trl9.1, Nrt9.3, Nrt11.1, Rdw11.1, Trl11.1, Trl11.2	2, 3, 5, 6, 7, 9, 11	DG	López-Marín et al. (2009)

(continued)

Table 4.3 (continued)

Stress[a]	Gene/QTL name[b]	LG[c]	Population[d]	Reference
SNF	*D1, D3.1, D3.2, D7*	1, 3, 7	BJ	Nodari et al. (1993b)
	D1 N-, D1 N + , D3.1 N-, D3.2 N-, D3.3 N + , D4 N + , D7 N-, D7 N+	1, 3 4, 7	BJ	Tsai et al. (1998)
	NN_A	2, 3, 5, 6, 7, 9, 11	BJ	Souza et al. (2000)
	NN_B	3, 5, 7, 10	BJ	Souza et al. (2000)
	%NROOT, %NPLANT_1, % NPLANT_2, NROOT_1, NROOT_2, NPLANT_1, NPLANT_2	1, 3, 4, 10	G23G19	Ramaekers et al. (2013)

[a]Zn = zinc; Fe = iron; P = phosphorus; Al = aluminium; SNF = symbiotic nitrogen fixation
[b]*Yld*, *SY*: yield; *Cbm*: canopy biomass; *Ppi*: pod portioning index; *Sbr*: stem biomass reduction; *Hri*: harvest index; *Stc*: stem TNC; *Scr*: SPAD chlorophyll metre reading; *NP*: number of pods per plant; *SW*: seed weight; *PHI*: pod harvest index; *ICP*: mineral concentration with inductively coupled plasma-optical emission spectrometry; *AA*: mineral concentration with atomic absorption method (trials Popayán, Darién and Palmira); *AAS*: mineral concentration with absorption spectroscopy method; *Pup*: phosphorus uptake; *LPAdvNoF:* adventitious root traits under low-phosphorus availability in the field; Pue: P use efficiency under low-phosphorus availability; PupLP: phosphorus uptake under low-phosphorus availability; *Npc*: Net P content; *Tsp*: total seed phosphorus; *Ard*: average root diameter; *Nrt*: average number of root tips; *Srl*: specific root length; *Rdw:* root dry weight; *Trl*: total root length; *D*: number of *Rhizobium* nodules QTL; NN_A and NN_B: number of *Rhizobium* nodules trials A and B; *%NROOT, %NPLANT, NROOT, NPLANT*: %N and total N content of root and total plant
[c]LG = linkage group
[d]Mapping population acronyms: AG = AND696 × G19833; BJ = BAT93 × Jalo EEP558; BG = Bayo Baranda × G-22837; BR = Buster × Roza; CCCG = Cerinza × (Cerinza × (Cerinza × G10022; DB = DOR364 × BAT477; DG = DOR364 × G19833; G14G48 = G14519 × G4825; G23G19 = G2333 × G19839; G42G78 = G21242 × G21078; MT = Matterhorn × T-39; SL = Sierra × Lef-2RB; SAC = Sierra × AC1028, SC = SEA5 × CAL96; SMD = SEA 5 × MD 23-24; VA = Voyager × Albion

'Buster' × 'Roza' cross was tested for yield under multiple stresses (intermittent drought, compaction and low fertility) across several location-years, resulting in the detection of two major QTL (located on LGs 01 and 02), which explained up to 37% of the phenotypic variance for seed yield (Trapp et al. 2015).

4.4.2.2 Tolerance to Zinc and Iron Deficiency

Zinc (Zn) and iron (Fe) deficiency is one of the most widespread crop micronutrient deficiencies and is capable of causing severe yield reductions. The inheritance of Fe and Zn concentration in common bean seeds has been suggested to be quantitative in most studies (Guzmán-Maldonado et al. 2003; Blair et al. 2009a, 2010; Cichy et al. 2009a), even while a few initial reports suggested that a single dominant gene (named with the symbol *Znd*) was involved in the tolerance to Zn deficiency (Singh and Westermann 2002). An interesting feature of several studies (Cichy et al. 2009a; Blair et al. 2009a, 2010, 2011) was that a number of QTL for Fe and Zn co-localized or overlapped, suggesting a possibly pleiotropic locus effect for mineral uptake. This provides further support for the suggestion that the same genetic and molecular mechanisms are controlling both Zn and Fe mobilization, uptake, distribution and accumulation in the plant (Clemens et al. 2002).

Using AFLP markers in the 'Bayo Baranda' 'G-22837' cross, Guzmán-Maldonado et al. (2003) found a locus that accounted for 15% of the phenotypic variation associated with Zn content. Gelin et al. (2007) identified a locus on LG09 that accounted for 18% of the seed Zn accumulation. An inter-gene pool RIL population

of the cross 'DOR364' × 'G19833' was used to scan for Fe and Zn accumulation loci (Blair et al. 2009a). QTL clustered on the upper half of LG11, explaining up to 48% of phenotypic variance. QTL for Fe and Zn content also co-localized on LGs 01, 06 and 11 in a RIL population developed from a 'AND696' × 'G19833' cross (Cichy et al. 2009a). A new QTL for Fe and Zn concentrations was mapped on LG06 in the inter-gene pool RIL population from 'G14519'× 'G4825' cross (Blair et al. 2010). Other QTL for both mineral concentrations were found on LGs 02, 03, 04, 07 and 08, which were also mostly novel compared to loci found in previous studies. In addition, the evaluation of a $BC_2F_{3:5}$ introgression line population derived from genotype 'G10022' backcrossed into 'Cerinza' allowed for the identification of four QTL associated with Fe and Zn content on LGs 03, 05 and 07 (Blair and Izquierdo 2012).

4.4.2.3 Phosphorus Use Efficiency

Among the edaphic stresses, phosphorus (P) deficiency is the primary constraint to common bean production in the tropics and subtropics, limiting seed yield to 60% of the bean-producing areas of Latin America and Africa (Wortmann et al. 1998). Root hair length, adventitious rooting and basal root growth angle in low-P soils were shown to be under the control of QTL (Miguel 2004).

The RIL population derived from the 'DOR364' (P inefficient) × 'G19833' (P efficient) cross has been widely used to study the morphological, physiological and genetic mechanisms underlying P efficiency. Yan et al. (2004) detected an association between root hair growth, acid exudation and P uptake, as well as two QTL for P uptake on LGs 04 and 10, which were closely linked to three QTL for root-exudation. The same mapping population was used to detect QTL associated with root gravitropism and their influence in the acquisition of P (Liao et al. 2004). QTL for P uptake were closely linked to QTL for shallow basal root length on LGs 04, 07 and 11. Beebe et al. (2006) confirmed in the same population that P acquisition was associated with basal root development and specific root length. A RIL population derived from the 'G2333' × 'G19839' cross was used to identify a total of 19 QTL for adventitious root traits (Ochoa et al. 2006). Two QTL for the number of adventitious roots under low P were mapped on LGs 02 and 09 and explained 61% of total phenotypic variation. In low-P conditions, two P-uptake QTL on LGs 07 and 11, and one P use efficiency QTL on LG08 were identified in the 'AND696' × 'G19833' Andean mapping population (Cichy et al. 2009b). A total of six QTL, three under each high and medium P soil, were mapped on LGs 06, 07 and 11 and on LGs 02, 07 and 10, respectively, in the mapping population from 'G2333' × 'G19839' cross (Blair et al. 2009b). The QTL on LG11 co-localized with a QTL for the number of adventitious roots (Ochoa et al. 2006), suggesting that this trait may have led to increased P uptake.

Several candidate genes induced by low P have been isolated in various plant species including legumes. In common bean, a total of 3,165 ESTs belonging to P-starved root cDNA library were reported by Ramírez et al. (2005) and a limited number (575) was registered in the NCBI database (http://www.ncbi.nlm.nih.gov/dbEST). An in silico approach for the identification of genes involved in the adaptation of common bean and other legumes to P-deficiency has also been reported (Graham et al. 2006). Over 240 putative P starvation-responsive genes were identified in a cDNA library (Tian et al. 2007). Full-length cDNAs for three genes, representing *PvIDS4*-like, *PvPS2* and *PvPT1,* were cloned and characterized. The open reading frames contained a SPX domain, a putative phosphatase and a P transporter, respectively. It is also worth mentioning that Hernández et al. (2007) have completed a transcript profiling of bean plants grown under P-deficient and P-sufficient conditions and showed 126 genes with a significant differential expression, of which 62% were induced in P-deficient roots. Finally, variations in the microRNA 399-mediated *PvHO2* regulation within the *PvPHR1* transcription factor were found in two contrasting genotypes (P-tolerant 'BAT477' and

P-sensitive 'DOR364') (Valdés-López et al. 2008; Ramírez et al. 2013).

4.4.2.4 Tolerance to Aluminium Toxicity

Other edaphic constraints include toxicities of aluminium (Al) associated with acid soil together with low calcium (Ca) availability (Rao 2001). Genetic variation exists for Al tolerance among common bean genotypes (Rangel et al. 2005). The most frequently measured effect of Al excess is inhibition of root elongation (Rangel et al. 2005). While root traits in the presence of Al are controlled by many genes in common bean, QTL for root morphological traits identified under the stress of Al were located on six genomic regions of the 'DOR364' × 'G19833' RIL population (López-Marín et al. 2009). A total of 12 QTL were involved in specific mechanisms of Al resistance, two of them in the same genomic regions, where QTL for the length of shallow basal roots and P acquisition efficiency were identified by Liao et al. (2004).

Rangel et al. (2010) hypothesized that the expression of a citrate transporter and the enhanced synthesis of citrate are crucial for sustained Al resistance in common bean. Eticha et al. (2010) corroborated these results, showing that the Al-induced expression of a citrate transporter gene family *MATE* (multidrug and toxin extrusion family protein) in root apices is a prerequisite for citrate exudation and Al resistance in common bean. In addition, Al-induced inhibition of root elongation was positively correlated with the expression of an *ACCO* (1-aminocyclopropane-1-carboxylic acid oxidase) gene in the root apex (Eticha et al. 2010). The expression of *MATE* and *ACCO* genes has been used as a sensitive indicator of Al impact on the root apex in common bean (Yang et al. 2011).

4.4.2.5 Symbiotic Nitrogen Fixation (SNF)

Enhancing the natural capacity for biological nitrogen (N) fixation has proved to help overcome the loss of soil fertility (Hungria and Vargas 2000). Studies on symbiotic nitrogen fixation (SNF), nitrogenase activity and nodulation-related traits (Nodari et al. 1993b; Tsai et al. 1998; Souza et al. 2000; Ramaekers et al. 2013; Kamfwa et al. 2015b) suggest that these traits have a complex inheritance with the involvement of multiple genes. Most of these studies focused on QTL analyses of nodulation traits and shoot dry weight under N-fixing conditions.

The first QTL study on nodule number (NN) trait in common bean was performed by Nodari et al. (1993b), who reported four genomic regions for NN in a RIL population derived from the 'BAT93' × 'Jalo EEP558' cross; all QTL together accounted for 50% of the phenotypic variation. One of these four genomic regions influenced both NN resistance and CBB resistance. Tsai et al. (1998) screened the same RIL population, but at two different soil N levels. They confirmed previous findings by Nodari et al. (1993b) and reported three QTL for NN in high N levels, one QTL associated with *CH18* (chitinase) and the other two QTL with *CHS* (chalcone synthase) and *PAL-1* (phenylalanine ammonia lyase). Souza et al. (2000) identified also in the same RIL population seven QTL under low N conditions and five under high N conditions, accounting for 34 and 28% of the total phenotypic variation, respectively. In accordance with Nodari et al. (1993b), Souza et al. (2000) indicated that NN resistance and CBB resistance in common bean have overlapping QTL on LGs 02, 03, 07 and 11. Ramaekers et al. (2013) conducted a QTL analysis of SNF and related traits under greenhouse and field conditions in a RIL population derived from the 'G2333' × 'G19839' cross, which resulted in two QTL for per cent N fixed in greenhouse located on LGs 01 and 04.

DNA sequence comparison of markers closely linked to these QTL allowed for the detection of some potential candidate genes. One of these genes encodes an auxin-responsive transcription factor and explained differences in N accumulation between climbing and bush beans. Alternatively, an AP2/ERF-domain-containing transcription factor underlies the QTL for the total amount of symbiotic N fixed in the field

(Ramaekers et al. 2013). Recently, Kamfwa et al. (2015b) detected 11 significant SNPs (five on LG03 and six on LG09) for nitrogen derived from atmosphere (Ndfa) in the shoot at flowering and for Ndfa in the seed in an Andean diversity panel of 259 common bean genotypes. Two genes *Phvul.007G050500* and *Phvul.009G136200* that code for leucine-rich repeat receptor-like protein kinases were identified as candidate genes for Ndfa.

4.4.3 Genes and QTL for Agronomic and Quality Traits

Agronomic and quality-related traits are almost always quantitative traits in plant species. Table 4.4 includes many of the research studies that have been carried out to identify QTL for agronomic and quality traits in common bean.

4.4.3.1 Plant Height and Traits Related to Vegetative Growth

Determinacy is controlled by the *FIN* gene that is located on LG01, where the dominant allele causes an indeterminate growth habit (Koinange et al. 1996). *PvTFL1y* is homologous to the *Arabidopsis TFL1* (Kwak et al. 2008) and is responsible for determinacy in common bean (Repinski et al. 2012; González et al. 2016). The *TOR* gene controls twining and correlates with *FIN,* suggesting that either *FIN* had a pleiotropic effect on twining or *TOR* was tightly linked to *FIN* (Koinange et al. 1996). Likewise, Koinange et al. (1996) reported pleiotropic effects or tight linkage of *FIN* on plant height, number of days to flowering and maturity, number of pods and harvest index. Another gene for growth habit was located at the end of LG11 and was significantly associated with QTL for days to flowering and maturity (Tar'an et al. 2002). Growth habit

Table 4.4 Details of QTL mapping studies performed for the mapping of major genes and QTL for agronomical and quality traits in common bean

Trait	Gene/QTL name[a]	LG[b]	Population[c]	Reference
Plant height	*FIN, TOR*	1	MG	Koinange et al. (1996)
	GH	11	S95	Tar'an et al. (2002)
	HABIT	4	A55G	Kolkman and Kelly (2003)
	PH, TB, TN, Ag	1, 2, 9, 11	S95	Tar'an et al. (2002)
	BP	7	A55G	Kolkman and Kelly (2003)
	Angle, Height	3, 4, 5	WO	Beattie et al. (2003)
	ph1.1, ph6.1, ph6.2, pw6.1, pw6.2, pw7.1, ph7.1	1, 6, 7	CCCG	Blair et al. (2006)
	Plh1-2, Plh1-1, Plh1-3, Plh1-4, Plh2-1, Plh2-3, Plh2-2, Int2, Int3, Int4, Int2, Int3, Int1, Cab1-1, Cab1-4, Cab1-2, Cab1-1, Cab1-3, Cab1-5, Cab1-5, Cab2-1, Cab2-1, Brn1	3, 4, 5, 7, 8, 10, 11	G23G19	Checa and Blair (2008)
	Ph1.1 ^{AG}, *Ph1.2* ^{AG}, *Ph1.3* ^{AG}, *Ph7.1* ^{AG}	1, 7	A55G	Chavarro and Blair (2010)
Days to flowering	*PPD, HR*	1	MG, RM, RR	Koinange et al. (1996), Gu et al. (1998), Kwak et al. (20080
	Tip	unmapped	GC, FR	White et al. (1996)
	PvTFl1y, PvTFL1z, PvGI, PvZTL, PvFLD	1, 7, 9, 11	BJ, MG	Kwak et al. (2008)
	DF1.1, DF1.2, PD	1	MG	Koinange et al. (1996)
	DF11	11	S95	Tar'an et al. (2002)
	df1.1, df2.1, df6.1, df6.2, df9.1, df9.2, df11.1	1, 2, 6, 9, 11	CCCG	Blair et al. (2006)
	Df1.1, Df1.2, Df1.3, Df2.1, Df2.2, Df2.3, Df3.1, Df3.2, Df7.1	1, 2, 3, 7	A55G	Chavarro and Blair (2010)
	DF1, DF2, DF8, DE1, DE2, DE6.1, DE6.2	1, 2, 6, 8	Xco	Pérez-Vega et al. (2010)
	Df4.1, Df4.2, Df5.1, Df5.2, Df5.3, Df6.1, Df7.1, Df11.1	4, 4, 5, 5, 5, 6, 7, 11	DB	Blair et al. (2012)

(continued)

Table 4.4 (continued)

Trait	Gene/QTL name[a]	LG[b]	Population[c]	Reference
Seed size	*SW*	1, 3, 4, 7	BJ	Nodari (1992)
	SW	1, 7, 7, 11	MG	Koinange et al. (1996)
	SW, SL, SH	2, 3, 4, 5, 6, 7, 8, 11	PX	Park et al. (2000)
	QTL1-SM, QTL2-SM, QTL3-SM, QTL4-SM, QTL5-SM	1, 2, 3, 4, 5	BG	Guzmán-Maldonado et al. (2003)
	sw2.1, sw2.2, sw3.1, sw6.1, sw7.1, sw8.1, sw8.2, sw9.1, sw10.1, sw11.1	2, 3, 6, 7, 8, 9, 10, 11	CCCG	Blair et al. (2006)
	Swf3.1, Swf4.1, Swf11.1	3, 4, 11	DG	Beebe et al. (2006)
	SW6, SW8.1, SW8.2, SL2, SL3, SL6, SL8, SL10, SH6, SH8, WI3, WI6, WI7	2, 3, 6, 7, 8, 10	Xco	Pérez-Vega et al. (2010)
	SW1^{PP}, SW6^{PP}, SW9.1^{PP}, SW9.2^{PP}, SL1.1^{PP}, SL1.2^{PP}, SL2.1^{PP}, SL6^{PP}, SL7^{PP}, SL10^{PP}, SWI2^{PP}, SWI7^{PP}, SWI9^{PP}, ST2^{PP}, ST9^{PP}	1, 2, 6, 7, 9, 10	P1037	Yuste-Lisbona et al. (2014a)
Pod size	*PL*	1, 2, 7	MG	Koinange et al. (1996)
	Podlength, podheight, podwidth	2, 6, 8, 10	MO	Davis et al. (2006)
	PH, PL, PWT	1, 3, 4, 5, 6, 7, 9, 10	RO	Hagerty (2013)
	PL1^{PP}, PL4^{PP}, PL11^{PP}, PWI1^{PP}, PWI4^{PP}, PT4.1^{PP}, PSI1.1^{PP}, PSI1.2^{PP}, PSI4^{PP}, PBL1.1^{PP}, PBL1.2^{PP}, PBL1.3^{PP}, PBL4^{PP}	1, 4, 11	P1037	Yuste-Lisbona et al. (2014b)
Yield	*NP*	1, 8, 4	MG	Koinange et al. (1996)
	PPP, SPP, SY	2, 5, 9, 11	S95	Tar'an et al. (2002)
	SYD, HI	5, 6, 7, 11,	CDRKY	Johnson and Gepts (2002)
	PP, Y	2, 3, 5	WO	Beattie et al. (2003)
	pp7.2, pp9.2, pp11.3, sp6.1, sp7.1, sp7.2, yld2.1, yld3.1, yld3.2, yld4.1, yld4.2, yld4.3, yld4.4, yld9.1, yld9.2	2, 3, 4, 6, 7, 9, 11	CCCG	Blair et al. (2006)
	yield2004, yield2005, yield2006	3, 5, 10, 11	J115	Wright and Kelly (2011)
	nppr4.1, nppr5.1, npp4.1, npp10.1, yld3.1, yld3.2, yld4.1, yld10.1	3, 4, 5, 10	G23G19	Checa and Blair (2012)
	PHI1.1^{SC}, SY3.3^{SC}, SY9.1^{SC}, SY9.2^{SC}, NP3.1^{SC}, NP8.1^{SC}	1, 3, 8, 9	SC	Mukeshimana et al. (2014)
Colour	*P, Asp*	7	MG	Koinange et al. (1996)
	Gy, C, R, J, G, B, Rk	2, 4, 8, 10	W593	Bassett et al. (2002)
	Ana, Bip, C, G, V, Gy, Z, T	3, 4, 6, 8, 9, 10	BJ	McClean et al. (2002)
	Prp	8	BJ	Kelly and Vallejo (2004)
	QTL1-TA, QTL2-TA, QTL3-TA, QTL4-TA	2, 3, 4	BG	Guzmán-Maldonado et al. (2003)
	Cst1a, Cit1a, Ctt1a, Cst1b, Cst2b, Cit1b, Ctt1b, Ctt2b, Ctt3b, Cst1c, Cit1c, Ctt1c	3, 6, 7, 8, 9, 10	BJ	Caldas and Blair (2009)
	Color2005, Color2006, Color2007	1, 3, 5, 8, 11	J115	Wright and Kelly (2011)
	PSC3^{PP}, PSC4^{PP}, PSC7.1^{PP}, PSC7.2^{PP}, PSC9^{PP}, SSC4^{PP}, SSC7^{PP}, SSC8.2^{PP}, SSC9^{PP}, PC2^{PP}, PC6^{PP}, PC7.1^{PP}, PC8^{PP}	2, 3, 4, 6, 7, 8, 9	P1037	Yuste-Lisbona et al. (2014a, 2014b)

(continued)

Table 4.4 (continued)

Trait	Gene/QTL name[a]	LG[b]	Population[c]	Reference
Quality	*St*	2	MG	Koinange et al. (1996)
	PvIND	2	BJ, MG	Gioia et al. (2012)
	PvSHP1	6	BJ, MG	Nanni et al. (2011)
	APP, SPLT	8, not assigned	MCDRK, MCELRK	Posa-Macalincang et al. (2002)
	podstring	6	MO	Davis et al. (2006)
	WA3, WA4, CP3, CP7	3, 4, 7	Xco	Pérez-Vega et al. (2010)
	Texture2005, Texture2006, Visual appearance2005, Visual appearance2006, Washed-drainedweight2006	3, 5, 6, 8, 10, 11	J115	Wright and Kelly (2011)
	SpA, SpB, SpE, SpI, SpJ, Phs, SpF, SpG, SpK, SpL, SpM, SpC, SpD	3, 4, 5, 7	Xco	Campa et al. (2011)
	ct1.3, ct1.1, ct9.2	1, 9	CL	Vasconcelos et al. (2012)
	PST, PFB	1, 4	RO	Hagerty (2013)
	A5xc, AA9xc, As1xc, As7xc, Ca1xc, Ca7xc, Ca9xc, DF6xc, DF7xc, Mg7xc, Pt5xc, Pt7xc, S1xc, S2xc, S4xc, S9.1xc, S9.2xc, UA5xc, UA7xc	5, 9, 1, 7, 1, 7, 9, 6, 7, 7, 5, 7, 1, 2, 4, 9, 9, 5, 7	Xco	Casañas et al. (2013)
	Anthavg, L10, b11, L11, color11, W_uptake, Soak_Anth, text11, hc11, text10, wdc10, hc10, wdc11, a11, b11, L10	4, 5, 7, 11	BS	Cichy et al. (2014)

[a]*PH*, *H*, *ph*, *Plh* = plant height; *TN* = total nodes; *TB*, *Brn* = total branches; *Ag*, *A* = branch angle; *BP* = branching pattern; *pw* = plant width; *Int* = internode length; *Cab* = climbing ability; *DF*, *df*: days to flowering; *PD*: photoperiod-induced delay influencing; *DE*: days to end of flowering; *SW*, *sw*, *Swf*: seed weight; *SL*: seed length; *SH*: seed height; *SM*: seed mass; *WI*, *SWI*: seed width; *ST* = seed thickness; *PL*: pod length; *PH*: pod height; *PWT*: pod wall thickness; *PT*: pod thickness; *PSI*: pod size; *PBL*: pod beak length; *NP*, *PPP*, *PP*, *pp*, *npp*: number of pods per plant; *SPP*, *sp*: seeds per plant; *Y*, *SYD*, *yld*, *SY*: seed yield; *HI*: harvest index; *npr*: number of pods per raceme; *PHI*: pod harvest index; *TA*: tannins; *Cst*: condensed soluble tannins; *Cit*: condensed insoluble tannins; *Ctt*: condensed total tannins; *PSC*: primary seed colour; *SSC*: secondary seed colour; *PC*: pod colour; *WA* = water absorption; *CP*: coat proportion; *ct*: cooking time; *PBF*: pod fibre; *PST*: strings; *A*: amylose; *AA*: apparent amylose; *As*: Ashes; *Ca*: calcium; *DF*: dietary fibre; *Mg*: magnesium; *Pt*: protein; *S*: starch; *UA*: uronic acids; *W uptake*: water uptake; *Soak-Anth*: anthocyanin concentration of the soak water after 12 h; *Anthavg*: anthocyanins; *wdc*: washed drained weight coefficient; *text*: texture; *HC*: hydration coefficient; *L*: lightness; *b*: blue/yellow; *a*: red/green

[b]LG = linkage group

[c]Mapping population acronyms: A55G = A 55 × G 122; BJ, BG = Bayo Baranda × G-22837; BAT93 x JaloEEP558; BS = Black Magic × Shiny Crow; CCCG = Cerinza × Cerinza × (Cerinza × G24404); DB = DOR364 × BAT477; CDRKY = California Dark Red Kidney × Yolano; CL = CNFM7875 × Laranja; DG = DOR364 × G19833; FR = Flor de Mayo × Rojo 70; GC = Gordo × de Celaya; G23G19 = G2333 × G19839; J115 = Jaguar × 115 M; MG = Midas × G12873; MO = Minuette × OSU5630; MCDRK = Montcalm × California Dark Red Kidney 82; MCELRK = Montcalm × California Early Light Red Kidney; S95 = OACSeaforth × OAC95; P1037 = PMB0225 × PHA1037; PX, PC50 × XAN159; RM = Redkloud × MAM; RO = RR6950 × OSU5446; RR = Redkloud Rojo; SC = SEA5 × CAL96; W593 = Wagenar × BC3 5-593; Xco = Xana × Cornell49242; WO = WO3391 × OAC Speedvale

(locus *habit*) mapped on LG04 (Kolkman and Kelly 2003). Tar'an et al. (2002) mapped one QTL for total branches on LG02 and one QTL for branch angle on LG11. QTL were detected on LG07 for branching pattern (Kolkman and Kelly 2003). Beattie et al. (2003) mapped two QTL for plant height on LGs 03 and 04 and three QTL for branch angle on LGs 03 and 05. QTL for plant height were found at the *ATA5* locus on LG01, at the *V* locus on LG06 and at the *Phs* locus on LG07 (Blair et al. 2006). QTL were found for plant height, climbing ability, internode length and branch number on LGs 03, 04, 08 and 11 (Checa and Blair 2008). Chavarro and Blair (2010) discovered a cluster of QTL for different plant height traits on LG01.

4.4.3.2 Flowering Date and Photoperiod Response

Flowering time is a key issue contributing to the adaptation and range of expansion of the short-day common bean (Vadez et al. 2012; Weller and Ortega 2015). Two loci, *PPD* and *HR,* are known to affect photoperiod response in common bean. *PPD* has been mapped within

5 cM of the *FIN* locus on LG01 (Koinange et al. 1996; Kwak et al. 2008), where recessive alleles confer reduced photoperiod response and early flowering under long days. This region is syntenic with the region in soybean containing the *E3/PHYA3* gene (McClean et al. 2010) and, as expected, contains the bean *E3* orthologue, suggesting this as an attractive candidate for the *PPD* locus. The second locus *HR* is less well-defined but is positioned towards the other end of the same linkage group (Gu et al. 1998), a region containing homologues of *ELF3* and the *FTa/c* cluster. Mapping *HR* was difficult because homozygous *ppd* is epistatic over *HR*, and thus, genotypes, *ppdHR* and *ppdhr*, give the same insensitive response to photoperiod. In addition, the expression of *HR* is influenced by the environment, producing an overlap of intermediate and highly sensitive genotypes. A third locus identified as a QTL on LG9 is located near the bean orthologue of *ZEITLUPE*, an important gene for circadian clock regulation in *Arabidopsis* (Tar'an et al. 2002; Kwak et al. 2008). QTL controlling flowering time and other flowering-related traits have now been identified in common bean (Koinange et al. 1996; Tar'an et al. 2002, Chavarro and Blair 2010; Pérez-Vega et al. 2010). Blair et al. (2006) detected two QTL on LG09, explaining 13 and 22% of the phenotypic variation, while Chavarro and Blair (2010) found a cluster of QTL for flowering time on LG01 close to *FIN* genomic region. Clusters of QTL for days to flowering were also found by Pérez-Vega et al. (2010) and González et al. (2016) on LG01 (close to the *FIN* gene) and LG02 (close to the *I* gene), as well as Blair et al. (2012) on LGs 04, 05, 06, 07 and 11.

4.4.3.3 Seed and Pod Size

The Danish plant scientist Wilhelm Johannsen (1911) concluded that a genetic effect could influence seed size in self-fertilizing beans, detecting segregation for seed size in a progeny from a large × small seed size population. A few years later, seed size was described as a polygenic trait in common bean (Sax 1923) with at least 10 genes involved in its genetic control (Motto et al. 1978). Quantitative inheritance of seed size has been reported by Vallejos and Chase (1991). QTL for seed size were mapped on LGs 01, 03, 04, 07 and 11 (Nodari 1992; Koinange et al. 1996). Park et al. (2000) found QTL for seed size traits on LGs 02, 03, 04, 05, 06, 07, 08 and 11. QTL on LG07 span the *PHS* locus that codes for phaseolin seed protein (Nodari 1992; Koinange et al. 1996; Park et al. 2000). Guzmán-Maldonado et al. (2003) reported five QTL for seed weight, explaining 42% of the phenotypic variation, while Blair et al. (2006) identified 10 QTL across eight LGs, explaining from 4 to 17% of the phenotypic variation, which agrees with the previous studies (Koinange et al. 1996; Park et al. 2000; Tar'an et al. 2002). Seed size QTL mapped near the upper end on LGs 02 and 06; the lower end on LGs 03, 07, 08 and 10; and near the centre on LGs 06 and 08 (Park et al. 2000; Blair et al. 2006; Pérez-Vega et al. 2010). Yuste-Lisbona et al. (2014a) detected QTL for seed weight on LGs 01, 02, 06, 07, 09 and 10 that were consistent with QTL mapped by Park et al. (2000) and Pérez-Vega et al. (2010).

Four genes (*Ea*, *Eb*, *Ia* and *Ib*) control pod cross-sectional shape although the exact genetics is uncertain (Leakey 1988). QTL for pod size have been reported by Koinange et al. (1996) on LGs 01, 02 and 07. QTL for pod length and height clustered together on LGs 01 and 03 (Hagerty 2013). Yuste-Lisbona et al. (2014b) detected 17 QTL for pod size traits, which were distributed throughout most of the LGs except for LG02. Most of the QTL affecting pod size clustered on LGs 01 and 04, which indicates that these genomic regions may contain linked genes or a gene with pleiotropic effects governing these traits.

4.4.3.4 Pod and Seed Yield

Yield is a quantitative trait influenced by many genes and is primarily conditioned by three components: number of pods per plant, number of seeds per pod and 100 seed weight (Adams 1967). Koinange et al. (1996) identified QTL for the number of pods per plant on LGs 01 (associated with *FIN*), 04, 08 and 11. Tar'an et al. (2002) mapped three QTL for seed yield on LGs 05, 09 and 11, explaining 28% of the total

phenotypic variation, one QTL for the number of pods per plant on LG02 and one QTL for seeds per pod on LG05. Six QTL for seed yield and four QTL for harvest index were detected on LGs 05, 06, 07 and 11 (Johnson and Gepts 2002). Beattie et al. (2003) reported three yield QTL on LGs 03 and 05 and three QTL for the number of pods per plant on LGs 02, 03 and 05. Blair et al. (2006) reported nine QTL for seed yield on LGs 02, 03, 04 and 09, accounting from 9 to 21% of the phenotypic variation, and three QTL for the number of pods per plant, explaining up to 64% of the phenotypic variation. Likewise, they reported three QTL for the number of seeds per plant located on LGs 06, 07, 09 and 11, which explain from 15 to 29% of phenotypic variation. Wright and Kelly (2011) reported seven QTL for seed yield on LGs 03, 05, 10 and 11. Checa and Blair (2012) identified four QTL for yield on LGs 03, 04 and 10; two QTL for the number of pods per raceme on LGs 04 and 05; and two QTL for the number of pods per plant on LGs 04 and 10. Mukeshimana et al. (2014) reported QTL for seed yield on LGs 03 and 09. Three QTL for the number of pods per plant and pod harvest index mapped on LGs 01, 03 and 08. In spite of the different procedures used, several studies found QTL associated with the number of pods per plant and seed yield on LG03 (Beattie et al. 2003; Blair et al. 2006; Wright and Kelly 2011; Checa and Blair 2012). Recently, Qi (2015) characterized the *A. thaliana* homologue of *BnMicEmUp*/*AT1G74730* gene in common bean (*Phvul.009G190100*), which encodes a cbZIP transcription factor that could affect seed yield.

4.4.3.5 Seed and Pod Colour

The genetic control of the different patterns and colours of bean seeds has been studied by Beninger et al. (2000). The *P* gene determines the presence or absence of flavonoids in the seed coat, and the specific colour depends on the epistatic interactions of the alleles at the other genes (Erdmann et al. 2002). The *Asp* gene controls the shine of the seed coat. Both genes are located on LG07 (Koinange et al. 1996). In *P_* individuals, a multiallelic serie at *V* gene controls flower colour, with the genotypes *V_* (purple) > v^{lae}_ (pink) > *vv* (white) (Beninger et al. 1999, 2000). Alleles at other genes (*Gy*, *C*, *R*, *J*, *G*, *B* and *Rk*) interact with *V* and with each other to determine the many colours found in the seed coat (Bassett et al. 2002). *Ana*, *Ane*, *Bip*, *L*, *T* and *Z* are genes of the pattern and colour of the seed (McClean et al. 2002). The *J* locus for seed coat shininess is located on LG10 (Freyre et al. 1998; Galeano et al. 2011). The colour modifying *B* gene is linked to the *I* gene for BCMV resistance on LG02 (Nodari et al. 1993b). The seed coat colour genes *C*, *G*, *V* and *Gy* have been mapped on LGs 08, 04, 06 and 08, respectively (McClean et al. 2002). The seed pattern of *Z* and *T* genes was located on LGs 03 and 09, and the *Bip* and *L* loci on LG10. Wax bean pod colour is controlled by a single recessive gene (*y*), but may be affected by a second gene (*arg*) and perhaps other modifiers (Currence 1931). *P* and *V* genes control solid purple colouring or purple stripes depending on the allele at the [*C Prp*] complex locus (Bassett 1996; Bassett et al. 2005). The *Prp* (purple pod) locus was located on LG 08 (Kelly and Vallejo 2004).

The pigments responsible for variations in seed colour are flavonoids, principally flavonol glycosides, anthocyanins and condensed tannins (Beninger et al. 1999). Four QTL for tannin content were detected (Guzmán-Maldonado et al. 2003), explaining 42% of the phenotypic variation. Caldas and Blair (2009) found twelve QTL for tannin content, explaining from 10 to 64% of the phenotypic variation. Yuste-Lisbona et al. (2014a) showed that seed colour is controlled by a QTL located near the *P* locus, explaining from 27 to 42% of the phenotypic variation. In addition, QTL for pod colour were found on LGs 06 and 08, which may correspond to the *Prp* and *V* genes, and a major QTL on LG07, where the locus *P* was previously identified (Erdmann et al. 2002; Koinange et al. 1996; Vallejos et al. 1992; Yuste-Lisbona et al. 2014a). A QTL analysis revealed that the region near the *Asp* gene (seed coat shininess) on LG07 contained 141 genes, the best gene candidate for *Asp* being a FAE1/Type III polyketide synthase-like protein

that acts as a fatty acid elongase (Cichy et al. 2014).

4.4.3.6 Other Quality Traits

Despite the genetic complexity of most common bean quality traits, many studies have recently reported interesting molecular and functional results about this topic. Koinange et al. (1996) found that the lack of pod suture fibres was controlled by a major gene (*St* locus) on LG02. Gioia et al. (2012) amplified a gene homologous to *INDESHICENT* (*IND*, a factor required for silique shattering in *Arabidopsis*) that mapped next to the *St* locus. The homologue *PvSHP1* (*SHATTERPROOF-1* in *Arabidopsis*) was mapped on LG06, linked to seed colour gene *V* (Nanni et al. 2011) and close to a QTL for pod string, explaining 26% of the phenotypic variation (Davis et al. 2006). Hagerty (2013) detected a pod suture string QTL on LG01 and a pod fibre QTL on LG04 clustered to QTL for pod length, height and thickness. QTL for seed weight and length have been mapped on LG08 (Park et al. 2000; McClean et al. 2002).

In the last two decades, several markers and QTL have been associated with organoleptic quality traits of different nature, from water absorption and coat proportion (Pérez-Vega et al. 2010) to cooking time (Vasconcelos et al. 2012). It is interesting to note that major QTL for colour retention mapped in overlapping positions to four flavonoid biosynthesis genes (two of which code for chalcone synthase proteins), while other minor effect QTL co-localized with anthocyanin-related genes (Wright and Kelly 2011). In addition, five QTL associated with content of ash, calcium, dietary fibre, magnesium and uronic acid were mapped on LG07, close to *P* locus and QTL for content of tannins and seed coat proportion (Caldas and Blair 2009; Pérez-Vega et al. 2010). Two QTL for content of ash and calcium were detected close to the *FIN* gene (LG01), and one QTL for protein content was located on LG07 close to the *Phs* cluster (Campa et al. 2011).

4.5 Epistatic and Environmental Interactions Among QTL

The goal of QTL mapping is to identify the genes/regions responsible for generating differences between individuals within a polymorphic population. Such phenotypic variation in the population can be divided into three components: (i) the contribution of QTL main effects, (ii) the role of QTL × QTL interactions or epistatic effects and (iii) the influence of QTL × environmental interaction effects. Inaccurate estimation of these effects further reduces the power and precision of QTL detection. Nonetheless, most of the QTL reports on common bean have not taken into account the identification of both epistatic and environmental effects.

Epistasis is considered an integral part of the genetic architecture of quantitative traits (Parvez et al. 2007), and in autogamous plants, it is expected to have significant effects on traits controlled by several genes/QTL, as pointed out by Holland (2001). Therefore, not only can epistasis be considered the major barrier to inferring the genetic basis of a given trait, but it also hampers the efficiency of breeding programmes. A direct implication of epistasis is that the fitness of individual alleles could be affected (increase or decrease) when they are found together in a given genotype (Holland 2007). If alleles involved in positive epistatic interactions are not transferred together to the cultivar that is being developed, improvement will be unsuccessful due to the presence of epistatic effects (Lark et al. 1995). Thus, any attempt to use QTL for improved plant performance and adaptation to different environmental conditions should take into account such epistatic effects, involving selection methods which tend to accumulate favourable allele combinations in the same genotype. Hence, the identification of QTL and the elucidation of their genetic control (main and epistatic effects) are essential for the development of efficient MAS programmes aimed at improving breeding efficiency.

The presence of epistasis can greatly obscure the mapping between genotype and phenotype. The effects of QTL may be masked by interactions with other loci, which can make mapping difficult (Phillips 2008). According to Asins (2002), the lack of information about QTL × QTL interaction could be explained by the plant material employed in the experiments. Epistatic interactions can hardly be detected in F_2 or BC populations. The reason is that in F_2 generations, even if large populations are used, there are insufficient individuals with two-locus double homozygotes, whereas in BC, every generation reduces the number of gene combinations while increasing genes from the recurrent genotype. Thus, the appropriate segregant populations would be RIL or doubled haploid (DH) populations, as additive and epistatic interactions effects can be detected but not dominant or over-dominant effects.

Johnson and Gepts (2002) found a reduced average fitness in the progeny of an inter-gene pool RIL population 'California Dark Red Kidney' × 'Yolano' that could be attributed to a break-up of co-adapted gene complexes or low viability of preferred epistatic relationships. They found that digenic epistatic interactions clearly played an important role for the number of days to maturity, average daily biomass, seed yield accumulation and harvest index. Both independently acting and digenic epistatic QTL of similar magnitude were identified. A total of 22 epistatic interactions were detected for the four traits evaluated. Each of the interactions accounted on average for 10% of the variation in the traits. In addition, eight interactions included a locus that also had a significant effect as independently acting QTL. Hence, the results obtained by Johnson and Gepts (2002) showed that, in addition to independent QTL action, epistatic QTL interactions play an important role in the cross-analysis.

The importance of epistatic QTL in the genetic control of pod size and colour traits has been recently revealed by Yuste-Lisbona et al. (2014b), who used an Andean intra-gene pool RIL population from a cross between a cultivated common bean ('PMB0225') and an exotic 'nuña' bean ('PHA1037'). A common feature of the epistatic interactions detected for pod-related traits is that most of them occur between QTL with main additive effects, but QTL that showed only epistatic effects were also detected. Thus, 12 out of 18 epistatic QTL identified were previously detected as main-effect QTL. Interestingly, 6 out of the 12 epistatic interactions detected were identified for pod colour, whose interactions explained 13.3% of the phenotypic variance observed, indicating the significant role of epistasis in the genetic control of this trait. This complex genetic inheritance is in accordance with the results obtained by McClean et al. (2002), who reported the existence of many genes that exhibit epistatic interactions that define the many colours observed within the species. Likewise, the role of epistatic effects in the genetic control of popping ability and others seed quality traits has also been studied using the same RIL population (Yuste-Lisbona et al. 2012, 2014a). Overall, the results showed that digenic epistatic interactions clearly play a significant role in the genetic control of these traits in the Andean common bean intra-gene pool.

A qualitative digenic model of inheritance, discerning an interaction between two QTL conditioning disease resistance in plants, was reported by Vandemark et al. (2008). Two QTL based on the closest markers such as *BC420* and *SU91* are of particular interest to breeding programmes focused on enhancing resistance to CBB in common bean, which is caused by *Xanthomonas axonopodis* pv. *phaseoli* (Xap). Results mainly showed that the expression of *BC420* was epistatically suppressed by a homozygous recessive *su91/su91* genotype and the highest level of disease resistance was conferred by genotypes with at least a single resistance allele at both QTL (*BC420/-*; *SU91/-).* The observed recessive epistatic interaction between the two QTL suggests that *SU91* is essential for the expression of an effective resistance mechanism. Moreover, this finding emphasized the need for breeders to correctly identify plants that are homozygous for both *SU91* and *BC420* loci, since breeding materials that are not fixed for *BC420* may produce moderately resistant

progeny in subsequent generations, while plants that are not fixed for *SU91* may produce susceptible progeny.

Recently, new insights into the role of epistasis in anthracnose resistance were provided by González et al. (2015). A total of 39 epistatic QTL (21 for resistance to race 23 and 18 for resistance to race 1545) involved in 20 epistatic interactions (eleven and nine interactions for resistance to races 23 and 1545, respectively) were identified in an Andean intra-gene pool RIL population. Depending on the race and organ tested, the total phenotypic variation explained by epistatic interactions ranged from 3 to 15%. Most of the epistatic interactions detected were due to loci without detectable QTL additive main effects, which showed the importance of the epistatic effects in genetic resistance to anthracnose.

What is more, in addition to epistatic effects, QTL × environmental interaction effects similarly complicate the use of MAS as genetic variance at one QTL may be sufficiently large in one environment but not in another. For instance, Jung et al. (2003) clearly showed the discrepancies among different environments regarding the locations and effects of QTL for bacterial brown spot resistance in the same mapping population. Thus, mapping QTL under natural infection in the field and artificial inoculation in growth chamber in two years revealed the existence of four QTL on LGs 02, 03, 04 and 09 in 1996, whereas two QTL on LGs 02 and 08 were detected in 1998. Only the genomic region on LG02 was significantly associated with bacterial brown spot resistance over both years (see Table 4.2). Similarly, depending on the experimental conditions, different QTL were identified by Beattie et al. (2003). Of the 21 QTL identified for plant architecture and yield traits, only 10 QTL (48%) were detected across all environments and, in most cases, these were the QTL with the largest influence on a given trait.

Asfaw et al. (2012) identified QTL for traits related to photosynthate mobilization across different drought stress and non-stress environments. The results showed that when using composite interval mapping for each individual environment, many QTL were detected, but these tend to be site-specific. However, when using a multienvironmental approach, only a small number of stable QTL and a high QTL × environmental interaction effects were identified. In addition, Asfaw and Blair (2012) detected root length QTL with significant QTL × environmental interaction effects under drought stress *versus* non-stress conditions. Interestingly, the QTL × environmental interaction effects were not attributed to the contrasting effects of the parental alleles between non-stress and stress environment, rather they were attributed to the differential expression of paternal alleles in different environments.

Long-day and short-day natural photoperiod conditions have been used by Yuste-Lisbona et al. (2012, 2014a, b) in order to extend the knowledge of the QTL × environmental interaction effects involved in common bean seed and pod quality traits. Among main-effect QTL detected by Yuste-Lisbona et al. (2014b), 11 QTL only exhibited significant genetic main effects, while 6 showed both significant genetic main effects and environmental interaction effects. As regards the epistatic interaction effects, only 2 out of 12 digenic interactions had environmental interaction effects. For seed shape and weight, as well as seed coat colour, 12 out of 32 main-effect QTL detected showed environmental interaction effects. Furthermore, only 6 out of 26 epistatic interactions identified had environmental interaction effects (Yuste-Lisbona et al. 2014a). Likewise, Yuste-Lisbona et al. (2012) showed that popping ability of 'nuña' bean is controlled by several QTL, which only have individual additive effects or may also be involved in epistatic or environmental interactions. Overall, even though most of the QTL detected were consistent over environment, some of them were subject to environmental modification. Despite this, QTL with differential effect on long-day and short-day environments were not found for seed and pod quality traits.

Finally, QTL × environmental interaction effects have also been reported for common bean resistance to angular leaf spot by Oblessuc et al. (2012). They revealed the existence of seven

QTL with variable magnitudes of phenotypic effects depending on the environments. One major QTL (*ALS10.1*) is highlighted with stable effect across environments. In addition, two QTL with minor effects (*ALS5.2* and *ALS4.2*) showed an interesting QTL × environmental interaction. *ALS5.2* revealed a greater resistance effect under greenhouse conditions, but only a small effect in the field experiments, whereas *ALS4.2* presented an opposite interaction with a greater resistance effect only under field conditions but not in the greenhouse. Hence, it is necessary to perform trials in different environmental conditions in order to draw conclusions about the genetic architecture of quantitative traits. However, a few multienvironmental QTL analyses have been carried out in common bean.

4.6 Genome-Wide Association Study (GWAS) Mapping

The association mapping (AM) exploits historical recombination events and has become a powerful alternative to linkage mapping for the dissection of complex trait variation at the sequence level (Zhu et al. 2008). There are two kinds of AM approaches: (i) candidate gene (CG) association mapping, which relates polymorphisms in selected candidate genes that have putative roles in controlling phenotypic variation for specific traits, and (ii) genome-wide association study (GWAS) mapping, also named genome scan, which surveys genetic variation in the whole genome to find associations with various complex traits (Risch and Merikangas 1996). The former implies good understanding of the biochemistry and genetics of the trait, while the latter requires a large number of well-distributed molecular markers and a broader reference population for the identification of numerous causative genetic polymorphisms with previously unappreciated biological function.

Advances in common bean genomics such as the sequenced genome (Schmutz et al. 2014) and the application of high-throughput and efficient genotyping platforms (Hyten et al. 2010; Goretti et al. 2014; Gujaria-Verma et al. 2016) have created the opportunity to conduct GWAS to dissect the genetic architecture of several complex traits in common bean. Moreover, common bean has been recognized as a valuable target for GWAS because of its extensive genetic diversity (Blair et al. 2009a). An extra advantage of the GWAS design for common bean is the homozygous nature of most varieties, which makes it possible to employ a 'genotype or sequence once and phenotype many times over' strategy, whereby once the lines are genomically characterized, the genetic data can be reused many times over across different phenotypes and environments. Additionally, in AM, unlike conventional QTL mapping, it is important to consider population structure and kinship among individuals, since false associations may be detected due to the confounding effects of population admixture (Oraguzie et al. 2007). Therefore, the divided population structure for common bean has made it necessary to consider the Andean and Mesoamerican gene pools as separate subgroups for AM. Several statistical methods have been proposed to account for population structure and familial relatedness, structured association (Falush et al. 2003), genomic control (Devlin and Roeder 1999), mixed model approach (Yu et al. 2006) and principal component approach (Price et al. 2006).

Several examples can be found in the literature in the 2010s to identify significant associations between agronomical and resistant traits and common polymorphisms in or near genes. GWAS not only identified previously reported QTL, but also resulted in narrower genomic regions than the regions reported as containing these QTL (see, e.g. Kamfwa et al. 2015b; Perseguini et al. 2015, 2016). Moreover, the results have also provided unprecedented views into the contribution of common variants to complex traits and new valuable markers for breeding that can now be used in common bean in future programmes. However, the time of GWAS is actually the beginning of a new age: one characterized by many new regions of the genome worthy of pursuit as candidate genes to explore. Advances in GWAS methodology and continued improvements in different genetic and genomic

techniques would eventually make it possible to realize the potential offered by AM in identifying as many as possible of new genes underlying complex traits (Korte and Farlow 2013).

The application of GWAS for common bean was originally assessed in Shi et al. (2011). In this work, 395 dry bean lines of different market classes were genotyped with 132 SNPs and evaluated for association with CBB resistance. Twelve SNP markers co-localized with or close to previously identified CBB-QTL, and eight new resistance loci were identified. Later on, a panel including 93 genotypes, mainly of Andean origin, was genotyped with 110 SNPs and 24 SSRs, and several flowering and pod features were characterized (Galeano et al. 2012). From GWAS, four putative proteins (i.e. acyl-acp thioesterase, auxin response factor 2, transcription factor bhlh96-like and oxygen-evolving enhancer protein chloroplastic-like protein) were found to be associated with several traits. A whole-genome sequencing approach was conducted for a 280-member panel of modern Mesoamerican cultivars (34,799 SNPs) in order to understand the genetic architecture of days to flower, days to maturity, growth habit, canopy height, lodging and seed weight. About 30 candidate genes were detected by GWAS; among them, most of the components of cytokinin biosynthesis pathways, multiple-component phosphorelay regulatory systemand genes relative to the *Arabidopsis* flowering pathway were identified (Schmutz et al. 2014; Moghaddam et al. 2016). Similar agronomical traits were subjected to GWAS analysis by Nemli et al. (2014) in 66 common bean genotypes of different geographic regions. In addition, an Andean diversity panel of 237 genotypes of common bean was conducted to gain insight into the genetic architecture of phenology, biomass, yield components and seed yield traits (Kamfwa et al. 2015a). Interestingly, the *phyA* gene, which codes for phytochrome, was identified as a candidate gene involved in the genetic control of these traits. In addition, significant SNPs for seed yield were also identified on LGs 03 and 09, co-localizing with QTL for yield from the previous studies on LG09 (Tar'an et al. 2002; Blair et al. 2006; Wright and Kelly 2011; Checa and Blair 2012; Mukeshimana et al. 2014). These QTL were stable and expressed in diverse genetic backgrounds, which makes them useful tools for MAS breeding of yield in common bean. Taken together, GWAS results may provide markers and genes that are useful for common bean genetics, trait selection, breeding applications and genetic dissection of novel traits to widely characterize common bean germplasm diversity. Furthermore, the markers detected will be interesting for future association studies, wherein marker–trait associations are compared. However, further elucidation of gene function has not yet been achieved, but it must be acquired by further functional and experimental analysis.

4.7 Perspectives and Future Direction

Since the early 1990s, numerous studies have identified molecular markers linked to QTL involved in the inheritance of agronomically important traits in common bean (Kelly et al. 2003). QTL mapping approaches have proved to be enormously useful to identify loci of large effect and dissect the genetic basis of fairly simple traits. Following the discovery of promising loci and identification of molecular markers, MAS has been used to transfer single genes in adapted cultivars (Yu et al. 2000; Kelly et al. 2003; Faleiro et al. 2004) and to develop multiple-introgression lines with improved resistance (Mutlu et al. 2005a, b; Miklas et al. 2006). However, most QTL mapping studies used small population size and low marker density, which allows only for an approximate mapping of the chromosomal region. Therefore, identification of reliable QTL is a preliminary step in developing a MAS programme for genetic improvement. So as to transfer QTL in selective breeding or to identify functional genes, the identified major QTL should be fine-mapped to a higher level of resolution and verified or

validated in additional genetic backgrounds and environments by developing advanced segregating populations with large number of recombinants in the region of interest. However, it is difficult to fine map several minor QTL associated with highly complex traits, such as drought tolerance and yield. Different factors may contribute to such failure or to an unexpected result in MAS: the magnitude of inconsistency of minor QTL, most QTL effects will have limited transferability across populations, epistatic and genotype-by-environment interactions, and population sizes of 500–1000 are needed for mapping QTL in order to eliminate the effects of sampling error (Bernardo 2008).

A new generation of genetic mapping populations must be designed with the aim to overcome many of the limitations of biparental QTL mapping and association mapping. A widely adopted strategy to estimate the position and effect of a mapped QTL more accurately is to create a new experimental population by crossing nearly isogenic lines (NILs) that differ only in the allelic constitution at the short chromosome segment harbouring the QTL (QTL-NILs) (Yamashita et al. 2014). In such populations, because of the absence of other segregating QTL, the target QTL becomes the only genetic source of variation, and the phenotypic means of the QTL genotypic classes can be statistically differentiated and genotypes recognized. Other populations combine the controlled crosses of QTL mapping with multiple parents and multiple generations of intermating. In this sense, MAGIC (multiparent advanced generation intercross) (Huang et al. 2015) and NAM (nested association mapping) populations (Buckler et al. 2009) would be an ideal resource to generate high-density maps using germplasm of direct relevance to the breeders. These designs require trade-offs among the amount of genetic variation sampled, the resolution of genetic mapping, the confounding effects of population substructure and the effort required to generate the mapping population. Another alternative genetic mapping strategy with higher resolution includes association analysis (Myles et al. 2009).

The advent of fast-evolving DNA sequencing technology has given a new direction in the field of common bean genomics by enabling sequencing of whole genome, extracting precious genomic information and resequencing in quick time and under manageable cost. Reduction of cost for sequencing leads to the development next-next- or third-generation sequencing technologies such as single-molecule real-time (SMRT) sequencing capable of generating longer sequence read (Thudi et al. 2012). A combination of GWAS and next-generation-mapping populations will improve the ability to connect phenotypes and genotypes, while genomic selection could take advantage of all these data for rapid selection and implementation in common breeding programmes. In addition to this, the next frontier in mapping and identification of candidate genes involved in complex traits is high-throughput phenotyping. Due to the development of NGS technologies, genomic resources are rapidly accumulating, but phenotypic data collected in a global context remain scarce. Automated platforms must be developed for phenotyping in growth chambers and controlled environments to provide new technologies for high-throughput phenotyping. The combination of these approaches and the promise of improved and cheaper genomic technologies will provide an opportunity to apply our understanding of the past to the future of common bean improvement.

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