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Genetic analysis of resistance to bean leaf crumple virus identifies a candidate *LRR-RLK* gene

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SUMMARY

Bean leaf crumple virus (BLCrV) is a novel begomovirus (family Geminiviridae, genus *Begomovirus*) infecting common bean (*Phaseolus vulgaris* L.), threatening bean production in Latin America. Genetic resistance is required to ensure yield stability and reduce the use of insecticides, yet the available resistance sources are limited. In this study, three common bean populations containing a total of 558 genotypes were evaluated in different yield and BLCrV resistance trials under natural infection in the field. A genome-wide association study identified the locus *BLC7.1* on chromosome Pv07 at 3.31 Mbp, explaining 8 to 16% of the phenotypic variation for BLCrV resistance. In comparison, whole-genome regression models explained 51 to 78% of the variation and identified the same region on Pv07 to confer resistance. The most significantly associated markers were located within the gene model Phvul.007G040400, which encodes a leucine-rich repeat receptor-like kinase subfamily III member and is likely to be involved in the innate immune response against the virus. The allelic diversity within this gene revealed five different haplotype groups, one of which was significantly associated with BLCrV resistance. As the same genome region was previously reported to be associated with resistance against other geminiviruses affecting common bean, our study highlights the role of previous breeding efforts for virus resistance in the accumulation of positive alleles against newly emerging viruses. In addition, we provide novel diagnostic single-nucleotide polymorphism markers for marker-assisted selection to exploit *BLC7.1* for breeding against geminivirus diseases in one of the most important food crops worldwide.

Keywords: bean leaf crumple virus (BLCrV), *Begomovirus*, common bean (*Phaseolus vulgaris* L.), disease resistance, genomic prediction, leucine-rich repeat receptor-like kinase (LRR-RLK), marker-assisted selection (MAS).

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is affected by a broad diversity of begomoviruses (family Geminiviridae, genus *Begomovirus*) in the American tropical and subtropical regions (Blair & Morales, 2008; Meziadi et al., 2017). These viruses often occur in disease complexes, and their genetic material can go through events of mutation, recombination and reassortment, favoring the emergence of new pathogenic variants (Lefeuvre & Moriones, 2015; Lima et al., 2017). Bean leaf crumple virus (BLCrV) is a recently characterized bipartite begomovirus infecting common bean in Colombia (Beebe, 2012; Carvajal-Yepes et al., 2017; Morales et al., 2002). The virus is transmitted by the whitefly vector *Bemisia tabaci* and can cause significant yield losses in susceptible cultivars (Morales, 2010). Disease symptoms include mosaic patterns and crumpled leaves, as well as deformation of pods (Morales, 2011). The genome sequence of BLCrV was grouped to other begomoviruses causing leaf-curling symptoms in tomato (*Solanum lycopersicum* L.) and cucurbit hosts, sharing up to 85% nucleotide identity (Carvajal-Yepes et al., 2017). More distantly related are the bean viruses causing mosaic

symptoms such as bean dwarf mosaic virus (BDMV), bean golden mosaic virus (BGMV) and bean golden yellow mosaic virus (BGYMV), which also occur in the Americas (Carvajal-Yepes et al., 2017; Morales et al., 2002).

The potential of rapid and widespread dissemination of begomoviruses as well as the heavy yield losses they can cause have been observed for BGMV and BGYMV in Latin America and the Caribbean (Morales, 2010). Many of the begomoviruses are persistent, i.e., they can replicate in the whitefly and therefore cause long-term infection (Czosnek et al., 2017; Levy & Tzfira, 2010). Control of the virus requires the use of insecticides to restrict whitefly advancement. However, proper pest management is difficult because resistance to insecticides may evolve rapidly (Dângelo et al., 2018). This situation can be aggravated by current climate change scenarios, as whitefly populations are able to reach higher altitudes and move towards temperate regions, expanding their range of infestation (Aregbesola et al., 2019; Jones, 2016; Navas-Castillo et al., 2011; Sharma, 2014). Therefore, genetic resistance is an important and sustainable solution to avoid yield losses caused by BLCrV in common bean. However, in contrast to many other viruses, resistance sources for BLCrV are rare (Beebe, 2012; Meziadi et al., 2017).

Plant defense against pathogen infections employs a two-layer system to restrict disease development (Calil & Fontes, 2017; Paudel & Sanfaçon, 2018). First, basal innate immunity is deployed by pattern recognition receptors (PRRs) that recognize pathogen-associated molecular patterns (PAMPs) in the plasma membrane, inducing a cascade of signaling pathways that activate PAMP-triggered immunity (PTI). This mechanism involves transcriptional reprogramming and biosynthesis of specific defense molecules (Gouveia et al., 2017). This strategy was initially associated with bacterial and fungal infection but recent evidence suggests that the concept of PTI can be extended to plant–virus interactions (Teixeira et al., 2019). In response, some adapted pathogens deliver virulence effectors into the host cell cytoplasm, preventing the activation of PTI. Plant cells have evolved intracellular immune receptors called R (resistance) proteins, often containing nucleotide-binding leucine-rich repeats (NB-LRRs) that recognize the virulence effectors and activate effector-triggered immunity (ETI) as a second layer of defense against pathogens. ETI is often associated with a hypersensitive reaction (HR), which is considered a resistance response against several pathogens and causes rapid cell death and the formation of visible necrotic lesions on infected tissue (Paudel & Sanfaçon, 2018).

Molecular markers within or tightly linked to different resistance genes can be used to efficiently transfer qualitative resistance to susceptible varieties in a breeding program (Meziadi et al., 2017; Singh & Schwartz, 2010). Many resistance loci that protect against virus infections are reported for common bean. For example, one dominant and one recessive resistance gene in common bean have been described for BGYMV (Osorno et al., 2007). Another resistance locus (*bcm-1*) was mapped close to a known recessive resistance locus against bean common mosaic virus (BCMV), a potyvirus (Blair et al., 2007). Virus resistance can also have a quantitative behavior controlled by several loci, such as the response to beet curly top virus (BCTV), a curtovirus (Larsen et al., 2010), and the analog response to BDMV (Miklas et al., 2009). In such a case, genomic prediction, considering many marker effects throughout the whole genome, could be more useful to elucidate the genetic control of virus resistance.

The main objective of this study was the identification of BLCrV resistance loci to enable marker-assisted selection (MAS) in common bean. Specifically, we aimed at (i) evaluating a diverse panel of common bean genotypes for BLCrV resistance under natural disease pressure in the field, (ii) investigating the suitability of this panel for resistance gene identification through population structure and linkage disequilibrium (LD) analyses, (iii) characterizing the genetic architecture of the resistance to BLCrV following a complementary approach that combined association mapping and genomic prediction and (iv) providing haplotype-specific single-nucleotide polymorphism (SNP) markers to implement MAS for BLCrV resistance breeding.

RESULTS

Yield and bean leaf crumple virus resistance trials

A common bean population (hereafter referred to as AxM population) was evaluated in three yield trials carried out in Palmira, Colombia in 2013, 2014 and 2015 (Pal13, Pal14 and Pal15). The AxM population was derived from biparental crosses between Andean and Mesoamerican breeding lines and comprised 190 F4-derived recombinant inbred lines (RILs) and its ten parental lines (Figure 1a, green). Flowering time (DF) of the AxM population was not different between Pal13, Pal14 and Pal15 (Figure 1b). However, the number of days to physiological maturity (DPM) differed between the rainfed trials (Pal13 and Pal14) and the irrigated trial (Pal15), with an average of 65.4, 63.7 and 69.7 DPM, respectively (Figure 1b). As expected, lower yields per hectare (YdHa) were observed in the rainfed trials, with an average of 640 and 628 kg/ha for Pal13 and Pal14, respectively, and 1310 kg/ha for the irrigated trial Pal15. Despite these differences, the correlations between DF, DPM and YdHa, as well as their broad-sense heritabilities, were stable, with correlation values ranging from 0.53 to 0.79 and heritabilities ranging from 0.74 to 0.95 (Figure 1c). The trial Pal14 was strongly affected by a whitefly infestation followed by BLCrV infection during the reproductive stage of the crop. This caused a moderate reduction in DPM and YdHa for some genotypes in the population (Figure 1b).

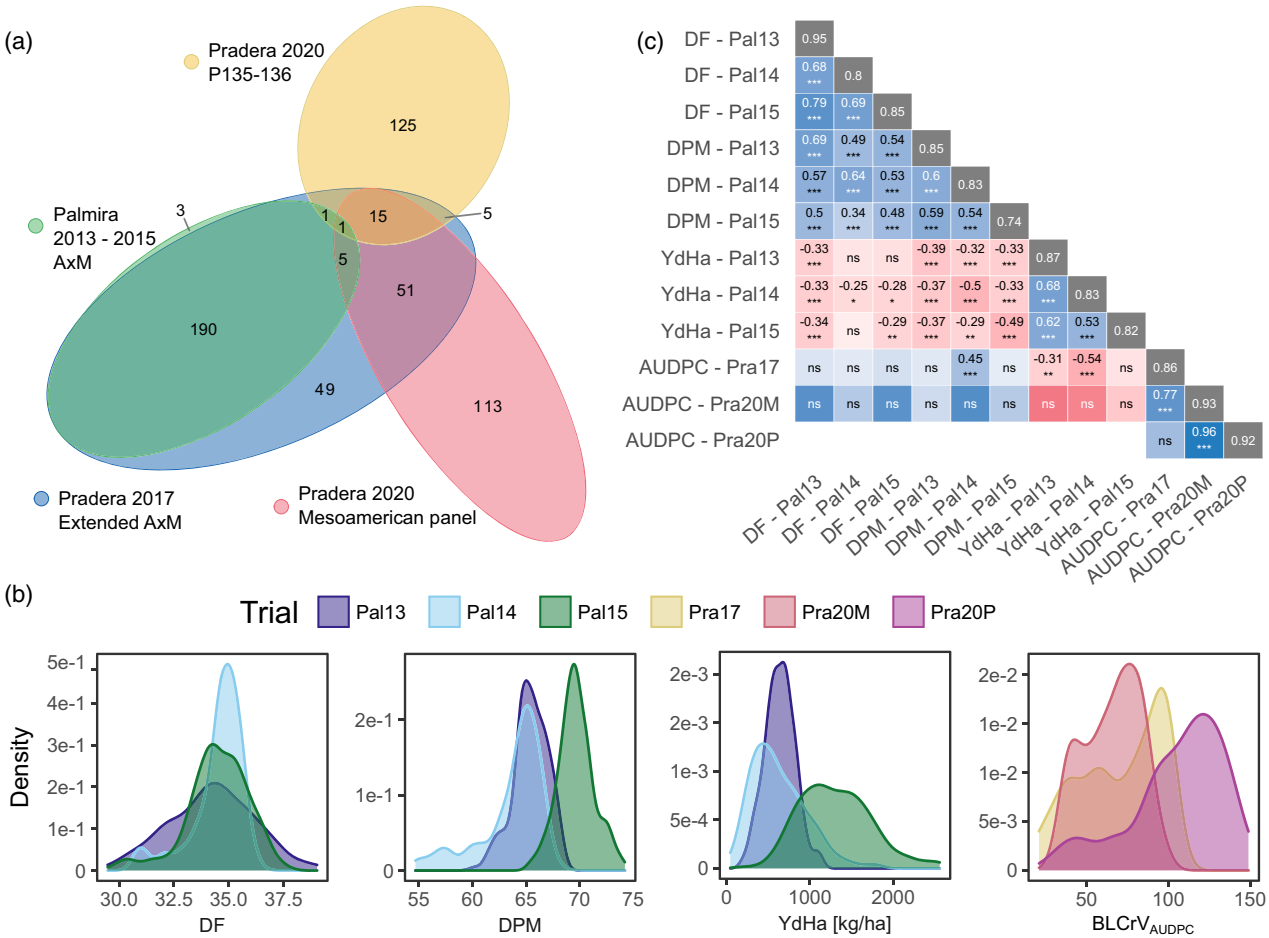

Figure 1. Composition of the plant material and its phenotypic evaluation for resistance to bean leaf crumple virus (BLCrV) in six field trials. (a) The plant material included the AxM population (green) tested in three yield trials in Palmira (Pal13, Pal14 and Pal15) and the extended AxM population (blue) tested in a BLCrV resistance trial in Pradera in 2017 (Pra17); the Mesoamerican panel (red) and P135-136 (yellow) were tested in separate BLCrV resistance trials in Pradera in 2020 (Pra20M and Pra20P, respectively). The Venn diagram shows the number of unique and common lines for each location and trial. (b) Phenotypic variability represented as density curves for the traits days to flowering (DF), days to physiological maturity (DPM) and seed yield (YdHa) evaluated in Palmira (Pal13, Pal14 and Pal15) and BLCrV resistance (BLCrV$_{AUDPC}$) evaluated in Pradera (Pra17, Pra20M and Pra20P). (c) Pearson correlation coefficients between best linear unbiased estimators from each trait and trial. The broad sense heritabilities are indicated within the main diagonal with gray background. ***$P < 0.0001$; **$P < 0.001$; *$P < 0.01$; ns, not significant.

The effect of BLCrV infections was further evaluated in Pradera, Colombia, using the AxM population extended with 120 breeding lines of Andean and Mesoamerican origin in 2017 (Pra17, Figure 1a, blue), a panel of 185 Mesoamerican breeding lines (Pra20M, Figure 1a, red) and 147 lines including pre-breeding material (termed P135-136, Pra20P, Figure 1a, yellow) in 2020. These three disease trials Pra17, Pra20M and Pra20P revealed that the phenotypic variability for BLCrV resistance (BLCrV$_{AUDPC}$, Figure 1b) was of genetic origin, as indicated by its high heritability (0.86 to 0.93, Figure 1c). The correlations between BLCrV$_{AUDPC}$ in Pra17 and DPM and YdHa in Pal14 were 0.45 and −0.54, respectively. These correlations were higher when compared to the other trials in Palmira. The correlations between the BLCrV trials in Pradera were high: 0.96 between Pra20P and Pra20M and 0.77 between Pra17 and Pra20M.

Genotyping, linkage disequilibrium and population structure analysis

The complete set of 558 common bean lines was genotyped using either genotyping-by-sequencing (GBS) or targeted amplicon sequencing offered by the DArTag pipeline. A joint genotypic matrix constructed with all available GBS data contained 14 588 SNP markers for 433 individuals. Using this matrix, the genome-wide LD decay rate was 240 kbp at 0.16 r^2_V. Similarly, the chromosome-wise LD decay rate ranged between 181 kbp at 0.166 r^2_V (chromosome Pv07) and 352 kbp at 0.204 r^2_V (chromosome Pv09) (Figure S1). In addition, an individual genotypic matrix was obtained for the extended AxM population and the Mesoamerican panel using GBS data, containing 317 and 185 lines and 14 868 and 14 765 SNP markers,

respectively. Similarly, a genotypic matrix for the 147 lines of P135-136 yielded 1861 SNP markers. Based on these data, the population structure analysis from a neighbor-joining (NJ) tree and principal components analysis (PCA) showed two large clusters of lines that can be identified as the Andean (containing accessions such as G19833, CAL143 and AFR298/ICA Quimbaya) and the Mesoamerican (containing accessions such as G4495/Porrillo Sintético, MD23-24 and Mexico54) gene pools (Figure 2). The individuals found in the middle with longer matching distances from either cluster can be identified as those with higher levels of admixture between both gene pools, i.e., those lines that were obtained from direct crosses between Andean and Mesoamerican parents, such as the 190 lines of the AxM population. The first and second principal components of the combined matrix accounted for 34.28% of variance (Figure 2), while the same value for each population ranged between 16.40 and 33.11% (Figure S2). These results indicate the presence of a strong population structure in the combined matrix as well as in the independent population-specific matrices. In that sense, the population structure effect is measurable and can be accounted for to reduce its negative impacts on the search for significant SNP–trait associations.

Genome-wide association study and candidate gene identification

A genome-wide association study (GWAS) using the available GBS and DArTag genotypic data and the phenotypic data from the trials that were affected by the disease

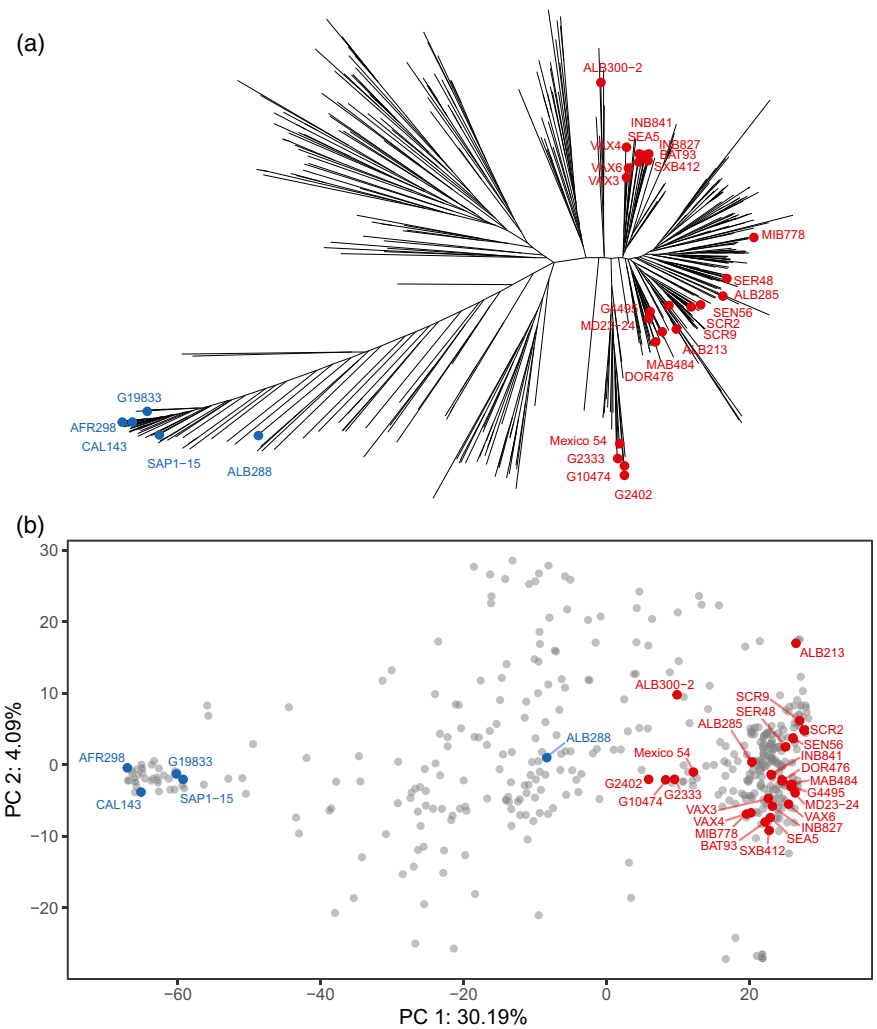

Figure 2. Overall population structure of the evaluated populations (the extended AxM population, the Mesoamerican panel and P135-136) based on genotypic data. The accessions for which whole-genome resequencing data were available and evaluated in this study are marked with labeled tags and helped identifying the Andean (leftmost, blue group) and Mesoamerican (rightmost, red group) gene pool clusters in this population. (a) Unrooted neighbor-joining tree; the length of the lines in the tree shows the simple matching distance. (b) Principal component analysis; the location of each genotype is represented by a point in the two-dimensional space defined by the eigenvectors of the first and second principal components.

(Pal14, Pra17, Pra20M and Pra20P) revealed a single region located on chromosome Pv07 that was associated with BLCrV resistance (Figure 3). The most significantly associated markers were Pv07_3313494_G/A in Pra17 and Pv07_3313706_C/T in Pra20M, explaining 8.22 and 9.91% of the observed variance, respectively (Figure 4c,d; Table S3). This region was surrounded by the most significantly associated markers identified for YdHa in the disease-affected trial Pal14: Pv07_3044767_G/A and Pv07_3644749_G/T, which independently explained 11.41 and 16.08% of the observed variance, respectively (Figure 4a,b; Table S3). Despite the absence of significant associations in Pra20P, differences between the alleles of the adjacent SNP marker Pv07_3359118_A/G were observed in this panel (Figure 4e). No significant associations were found for other traits in Pal14, despite the significant phenotypic correlation between DPM, YdHa and BLCrV resistance in Pra17 (Figure S3). Likewise, no significant associations were identified on Pv07 for other yield trials that were not affected by the disease (Pal13 and Pal15). These results indicate that

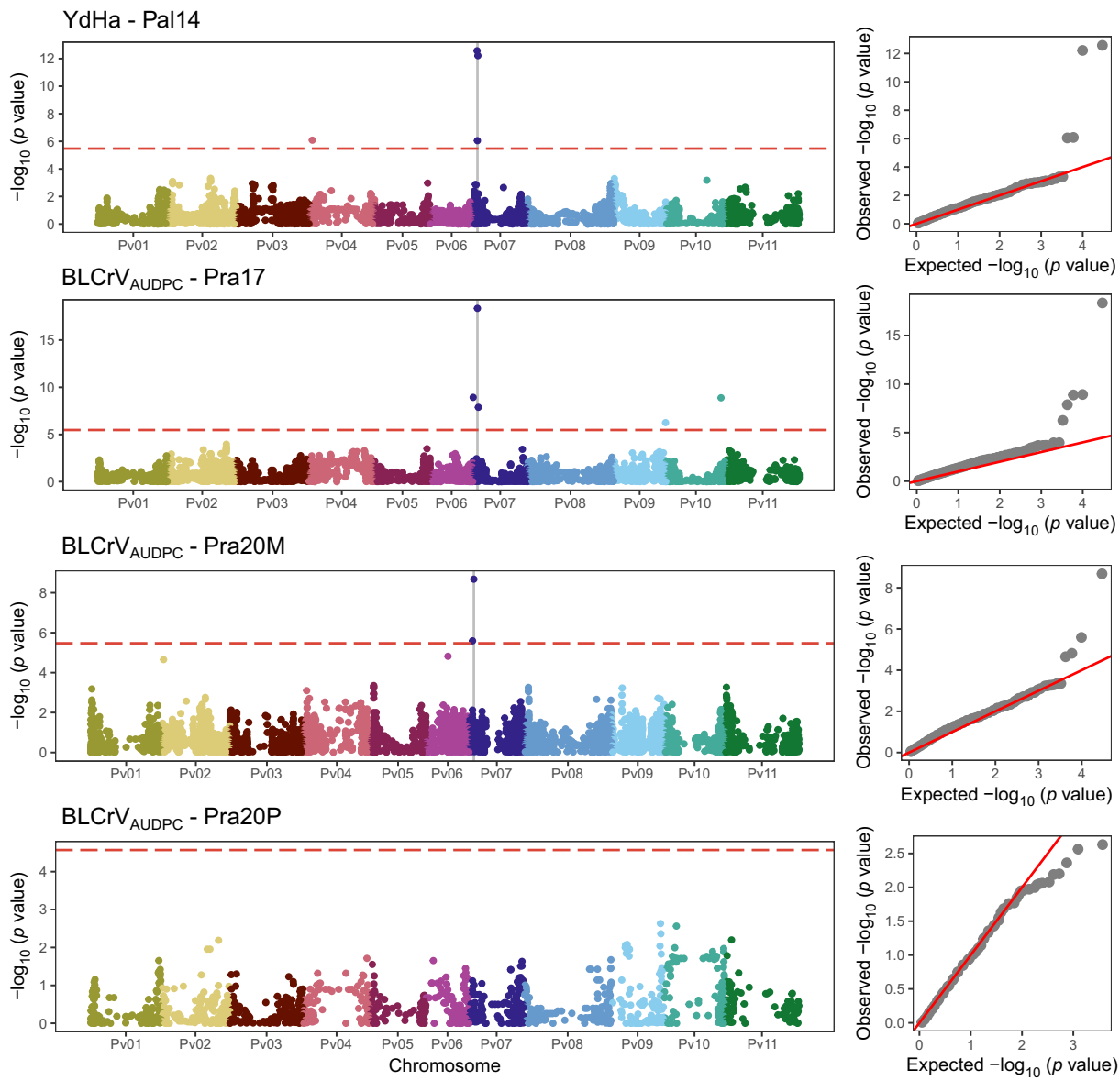

Figure 3. Results of the genome-wide association study for resistance to bean leaf crumple virus (BLCrV) in an infected yield trial in Palmira in 2014 (YdHa – Pal14) and in BLCrV resistance trials in Pradera in 2017 and 2020 (BLCrV$_{AUDPC}$ – Pra17, Pra20M and Pra20P). The results are presented as individual Manhattan plots showing the significance of the SNP markers (colored dots) and their physical location on each of the 11 chromosomes of the reference genome of the accession G19833 (v2.1). The horizontal red-dashed lines represent the Bonferroni-corrected threshold, which was calculated with a genome-wide type I error rate $\alpha = 0.05$ ($P < 2.028 \times 10^{-6}$). The corresponding quantile-quantile plots to the right compare the deviation between the observed and the expected significance of the SNP markers from a theoretical χ^2 distribution.

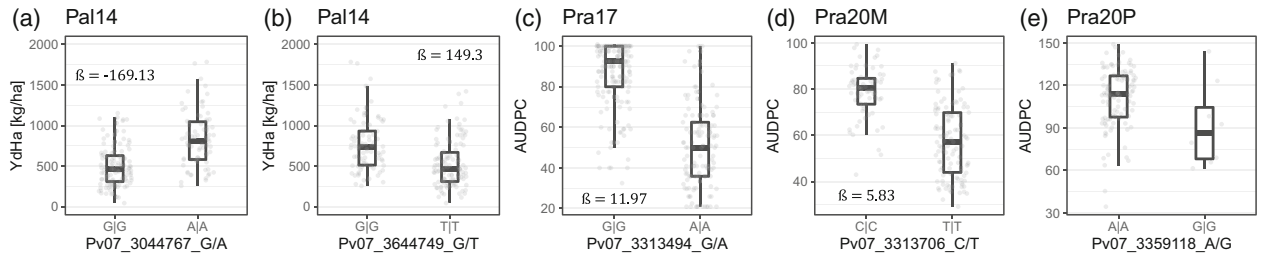

Figure 4. Distribution of phenotypic values between the two allelic states of the most significantly associated markers. (a) Pv07_3044767_G/A and (b) Pv07_3644749_G/T for the yield trial (YdHa) in Palmira in 2014 (Pal14), (c) Pv07_3313494_G/A for the resistance trial ($BLCrV_{AUDPC}$) of the extended AxM population in Pradera in 2017 (Pra17) and (d) Pv07_3313706_C/T for $BLCrV_{AUDPC}$ in the Mesoamerican panel tested in Pradera in 2020 (Pra20M). The plot under (e) displays the same distribution for P135-136 tested in Pradera in 2020 (Pra20P) on the neighboring marker Pv07_3359118_A/G. The approximate effect size for each significantly associated marker is displayed as the β value.

there is a single region of interest (ROI) associated with the resistance to BLCrV, located on Pv07 between 3.04 and 3.65 Mbp (Figure 5a).

The ROI associated with BLCrV resistance contained 81 annotated gene models in the reference genome. The significantly associated markers identified in Pal14, Pv07_3044767_G/A and Pv07_3644749_G/T, fall in the coding region of the gene models Phvul.007G037400, encoding a glycosyltransferase 14 family member, and Phvul.007G045200, encoding a protein with unknown function, respectively (Table S3). On the other hand, the significantly associated markers identified for BLCrV resistance, Pv07_3313494_G/A and Pv07_3313706_C/T, fall in the coding region of Phvul.007G040400, which encodes a leucine-rich repeat receptor-like kinase (LRR-RLK) family protein (Figure 5a,b). Despite the physical proximity of the markers identified in Pal14, Pra17 and Pra20M (up to 600 kbp), their LD pattern indicates that they are genetically distant (Figure S4). Considering that Pv07_3313494_G/A and Pv07_3313706_C/T were identified in two separate populations and in two different trials, and that both are in the same exonic region, Phvul.007G040400 was selected as a candidate gene that is likely to be involved in the resistance to BLCrV. The protein motif structure of the gene model contains a signal peptide in the N-terminal region followed by three leucine repeat motifs, a transmembrane domain and a kinase domain at the C-terminal end. The genetic diversity within this gene, revealed by whole-genome resequencing (WGS) data (69 sequence variants), was used to analyze its variability in a separate collection of 699 accessions that included breeding lines and landraces of common bean. The results indicate the presence of at least five different haplotype groups for this gene (H1–H5) (Figure 5c; Figure S5). Due to the lower SNP marker density yielded by GBS, it was not possible to identify haplotype membership in the evaluated populations of this study. However, using phenotypic data for 29 of those 699 accessions (Figure 2), it was possible to identify significant differences for the predicted BLCrV resistance between these five haplotypes. Haplotype H1, which was defined by the marker Pv07_3313706_C/T, is present mainly in lines of the Mesoamerican gene pool and represents the most resistant group compared to other haplotypes in this gene (Figures 2 and 5c,d).

Genomic predictions

A large amount of the phenotypic variance was not explained by the significantly associated markers identified by GWAS. Therefore, a series of genomic prediction models were tested for their ability to identify complementary additive effects across the genome for BLCrV resistance. These models yielded prediction abilities ranging from 0.55 (YdHa – Pal14) to 0.77 ($BLCrV_{AUDPC}$ – Pra17) (Figure 6a). The proportion of genetic variance explained by the markers, indicated as the genomic heritability, reached values between 0.51 (YdHa – Pal14) and 0.78 ($BLCrV_{AUDPC}$ – Pra20M). The difference between the estimated broad-sense heritability and the genomic heritability defines the proportion of missing heritability, and in the present study ranged between 0.12 ($BLCrV_{AUDPC}$ – Pra17) and 0.32 (YdHa – Pal14) (Figure 6a). No significant differences were observed between the prediction models evaluated, including the model that used the most significantly associated markers as fixed effects (quantitative trait locus [QTL] + Bayesian ridge regression [BRR]). In line with the GWAS results, the estimated marker effects of the prediction model indicate the presence of a locus with a major effect on Pv07 (Figure 6b).

DISCUSSION

The AxM population was initially developed to combine drought tolerance, good agronomic performance, and commercial grain quality from the Mesoamerican and the Andean common bean gene pool. However, the yield trial Pal14 was strongly affected by a whitefly infestation carrying a novel bipartite begomovirus infecting common bean, named BLCrV (Carvajal-Yepes et al., 2017). This virus caused a shortened pod filling stage in susceptible genotypes and a moderate reduction in seed yield compared to the rainfed trial Pal13. This virus is genetically related to other begomoviruses

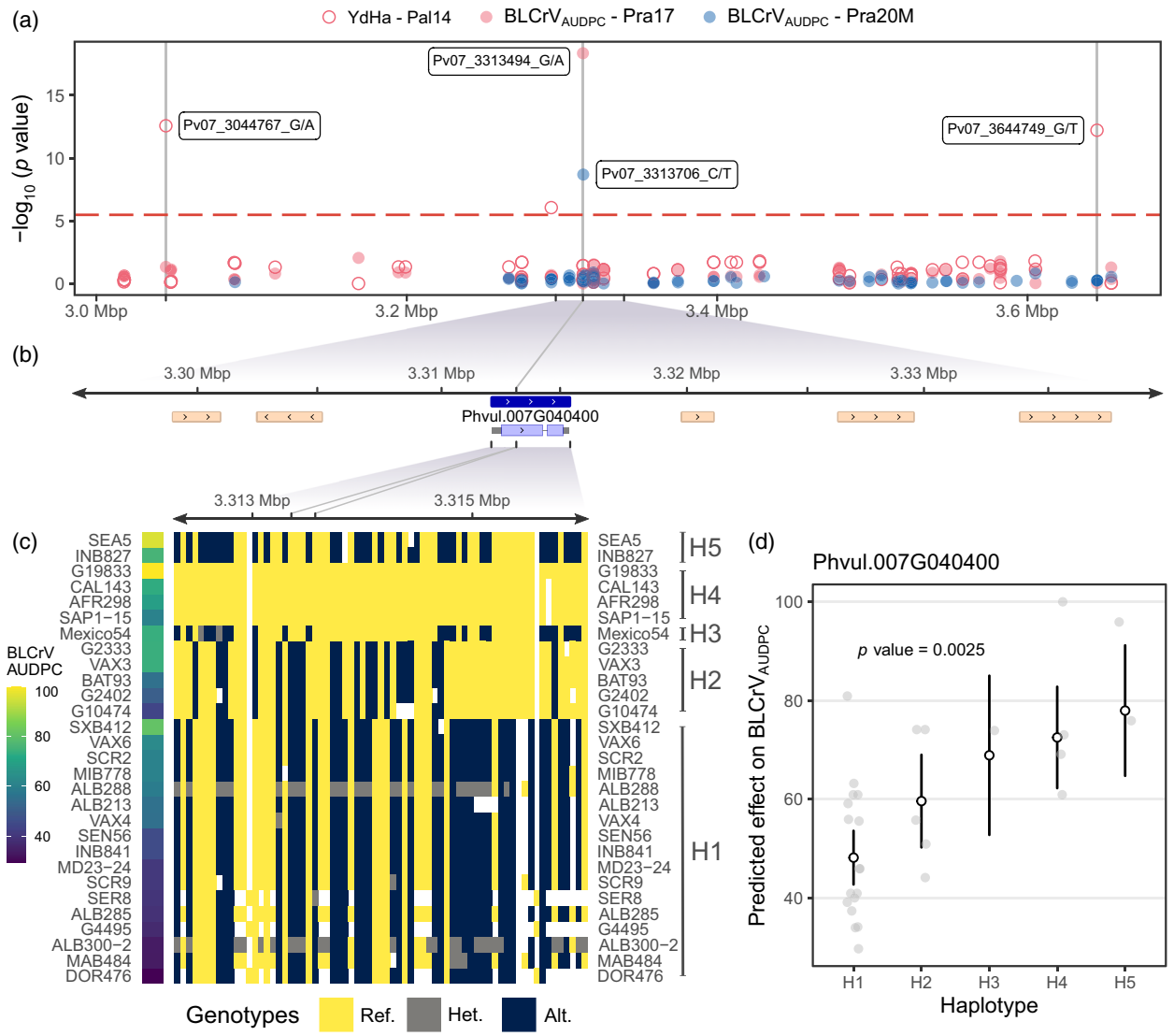

Figure 5. Genetic dissection of the region of interest (ROI) associated with resistance to bean leaf crumple virus (BLCrV) on chromosome Pv07, between 3.04 and 3.65 Mbp in the reference genome of G19833. (a) Marker–trait associations in the ROI for seed yield in Palmira in 2014 (YdHa – Pal14) and for BLCrV resistance in Pradera in 2017 (BLCrV$_{AUDPC}$ – Pra17) and 2020 (BLCrV$_{AUDPC}$ – Pra20M). The most significantly associated SNP markers are indicated with vertical gray lines. The horizontal red-dashed line represents the Bonferroni-corrected threshold, which was calculated with a genome-wide type I error rate $\alpha = 0.05$ ($P < 2.028 \times 10^{-6}$). (b) The SNP marker Pv07_3313494_G/A falls within the gene model Phvul.007G040400, which encodes a leucine-rich repeat receptor-like kinase. This gene model stands out as a candidate gene involved in resistance to BLCrV. (c) Five different haplotypes (H1–H5) were found in the coding region of Phvul.007G040400 using 69 SNP markers identified from whole-genome resequencing (WGS) data in a combined panel of 699 accessions. The tiles represent the observed genotypes for 29 accessions that were evaluated in the BLCrV trials in Pradera. The leftmost tile column represents BLCrV$_{AUDPC}$ for each of these lines. (d) Predicted effect on BLCrV$_{AUDPC}$ for each one of the identified haplotypes. The mid-points and their corresponding error bars indicate the best linear unbiased predictors and standard errors for each haplotype. The jittered points indicate the observed response for each of the 29 lines with WGS data available. The *P*-value was obtained from a likelihood ratio test for the significance of the haplotype effect in the model.

causing leaf curling symptoms, such as bean calico mosaic virus (BCaMV), which causes significant losses in northwestern Mexico (Morales, 2006). Even though BLCrV can cause yield losses in susceptible cultivars (Morales, 2010), the economic impact that the disease can cause has yet to be studied. Using a broad collection of breeding germplasm and two complementary approaches for gene mapping (GWAS and genomic prediction), we identified a single locus associated with BLCrV resistance at the proximal arm of Pv07. This locus is hereafter named *BLC7.1*, following the guidelines proposed by Miklas and Porch (2010). The underlying molecular markers are simple to use for MAS applications. This is of particular importance, considering that genetic resistance appears as one of the best and most cost-efficient control strategies for the management of viral diseases (Meziadi et al., 2017; Rojas et al., 2018).

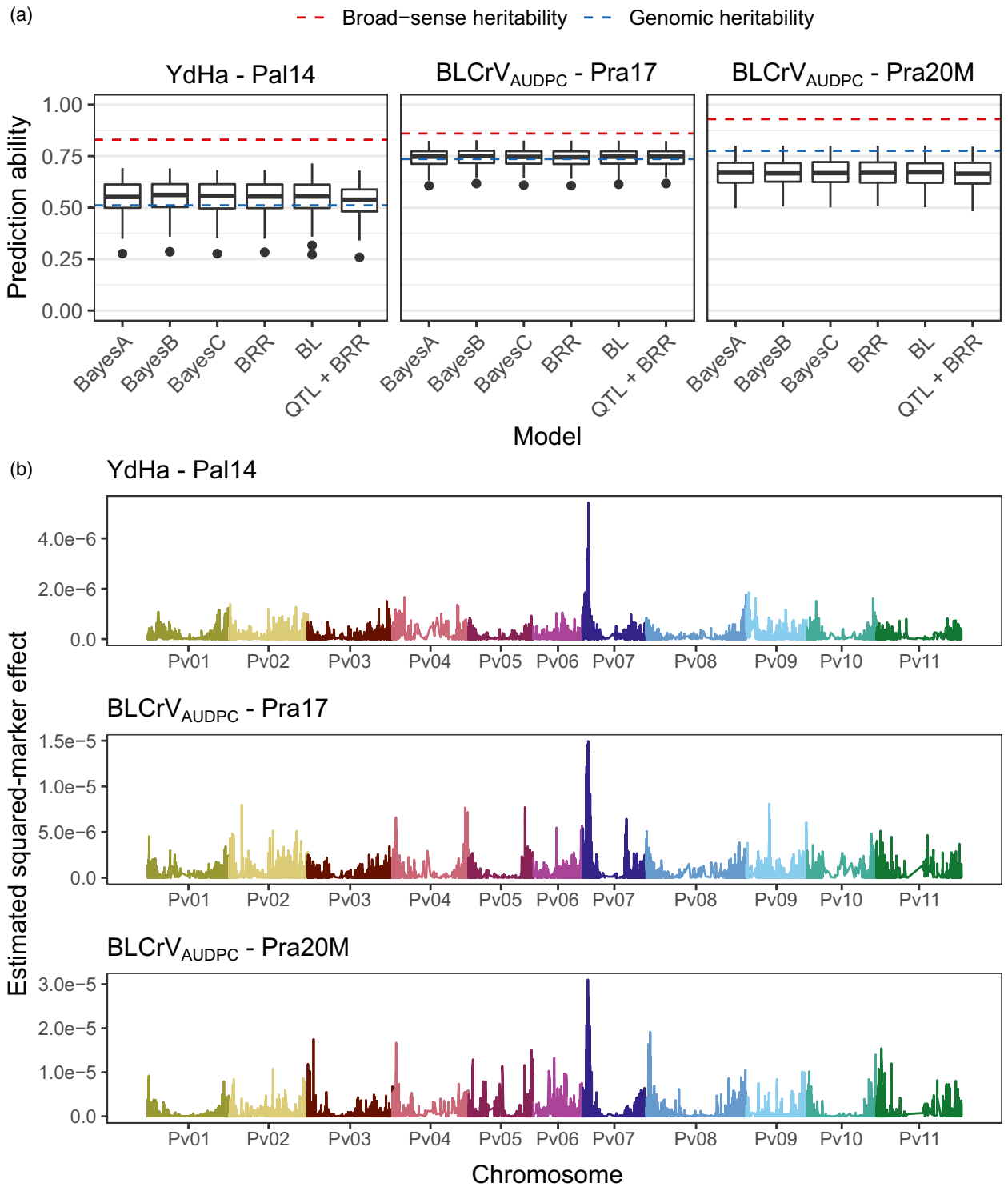

Figure 6. Genomic prediction for the resistance to bean leaf crumple virus (BLCrV) in an infected yield trial in Palmira in 2014 (YdHa – Pal14) and in BLCrV trials in Pradera in 2017 and 2020 (BLCrV$_{AUDPC}$ – Pra17 and Pra20M). (a) Prediction abilities for the additive models Bayes A, B and C, Bayesian ridge regression (BRR), Bayes Lasso (BL) and BRR including significant SNP markers as a fixed effect term (QTL + BRR). The boxplots show the distribution of correlations between predicted and observed values of the validation subsets (30%) in a cross-validation process repeated 100 times. The red and blue dashed lines represent the estimated broad-sense and genomic heritabilities, respectively. (b) Estimated squared-marker effects from the BRR model are given for each trial along their physical position in the reference genome.

A candidate *LRR-RLK* gene for BLCrV resistance

The locus *BLC7.1* is in a genome region of fast LD decay. The markers that delimitate this locus were identified independently in two separate common bean populations evaluated in different years, and both are in the exonic regions of the gene model Phvul.007G040400, which encodes a member of the LRR-RLK protein superfamily (Restrepo-Montoya et al., 2020). The predicted protein is orthologous to six legume RLKs, including Glyma.10G261600 and Glyma.20G129200 of *Glycine max* (L.) Merr. (Restrepo-Montoya et al., 2021), both belonging to subfamily III of the LRR-RLK superfamily (Zhou et al., 2016). The closest Arabidopsis homolog is the receptor dead kinase 1 (RDK1; AT4G34220) (69.7% sequence similarity), which functions as a positive regulator in plant responses to abscisic acid (Kumar et al., 2017; Paul & Srinivasan, 2020) and is also involved in the response against geminivirus infection (Ascencio-Ibáñez et al., 2008).

In the cell, LRR-RLKs work as co-receptors of PRRs that recognize PAMPs in the plasma membrane, inducing a signaling cascade that activates PTI (Gouveia et al., 2017). The most abundant co-receptors are members of subfamilies II and III of the LRR-RLK superfamily (Xi et al., 2019). However, the best-characterized co-receptors are members of subfamily II, which can be involved in antiviral and fungal/bacterial-related PTI in plants (Gouveia et al., 2017; Hosseini et al., 2020; Sakamoto et al., 2012; Teixeira et al., 2021). The most notable example is the antiviral signaling cascade mediated by nuclear shuttle protein-interacting kinase 1 (NIK1), which was reported to be activated upon perception of begomovirus infection in Arabidopsis, triggering a signaling cascade that culminates in suppression of host and viral mRNA translation (Brustolini et al., 2015; Zorzatto et al., 2015). The current knowledge about subfamily III is more limited, but some reports indicate that a few members may be involved in pollen development, secondary cell wall formation and root and cambium development processes (Xi et al., 2019). Despite that, the two subfamilies are the most abundant members of the LRR-RLK superfamily, and both share a high degree of structural similarity in their domain composition (Liu et al., 2017; Xi et al., 2019). The gene model Phvul.007G040400, most significantly associated with BLCrV resistance in our study, encodes a subfamily III member of the LRR-RLK superfamily and represents a candidate gene involved in the immune response against BLCrV.

Importance of *BLC7.1* for other geminiviruses and fungal pathogens

Common bean breeding for resistance against geminivirus diseases in the tropics has focused primarily on BGMV and BGYMV, which affect crop production in South and Central America, respectively (Beebe, 2012). The underlying genetic basis controlling the resistance to BGYMV can also work against other begomoviruses (Blair & Morales, 2008). Recently, different genetic analyses led to the identification of a plant-specific transcription factor NAM/ATAF1,2/CUC2 (NAC) (Puranik et al., 2012) on chromosome Pv03, linked to the resistance locus *bgm-1* against BGYMV (Soler-Garzón et al., 2021). Those analyses allowed the further characterization of *BGY7.1*, a QTL on Pv07 at 2724611–3525083 bp, which had been reported previously (Miklas et al., 1996) and corresponds to the same ROI as identified in this study for *BLC7.1*. This region overlaps also with the dominant *Bct-1* locus that confers resistance to BCTV (Larsen & Miklas, 2004). In addition, *Bct-1* co-segregates with a major QTL conferring quantitative resistance to BDMV (Miklas et al., 2009). These reports indicate the co-location of a QTL in the proximal end of Pv07 with minor effects against BGYMV and major effects against BDMV and BCTV. Further, it suggests that the breeding effort against BGYMV contributed to the accumulation of other loci with major effects against other geminiviruses in common bean.

Previous studies demonstrated that the defense response against BDMV and BGYMV was related to ETI, a mechanism that has long been recognized as an efficient plant defense response against viruses (Teixeira et al., 2019). PTI mediated by NIK1 in common bean is likely inhibited by the viral nuclear shuttle protein (NSP) of BDMV (Gouveia et al., 2017), which could enhance the pathogenicity of the begomovirus in its host. However, the defense response against BDMV was rather related to ETI, where NSP acts as an avirulence protein recognized by R proteins, eliciting HR in the plant (Garrido-Ramirez et al., 2000). A similar report demonstrated the virulence (suppressing PTI) and avirulence activity of NSP in the defense response to BDMV and BGYMV (Zhou et al., 2007). These studies show the involvement and activation of ETI in the response against begomoviruses in common bean, supporting the successful inhibition of PTI by the viral NSP. However, the candidate LRR-RLK is hypothesized to be involved in the response to BLCrV through PTI rather than ETI. In fact, *BLC7.1* identified in this study lacks any predicted resistance genes that can be related to ETI (Meziadi et al., 2017; Richard et al., 2018). These results suggest that the two defense mechanisms PTI and ETI can be complementary against BDMV, BGYMV, BCTV and BLCrV in common bean. This hypothesis could explain the co-location of significant QTL against BCTV, BDMV, BGYMV and now BLCrV in the proximal arm of Pv07, as well as the simultaneous identification of the loci *bgm-1*, *BGY4.1*, *BGY7.1* and *BGY8.1* with additive or epistatic performance against BGYMV (Soler-Garzón et al., 2021).

The genome region associated with BLCrV resistance reported here also harbors other QTL related to disease

resistance. For example, the QTL *ANT7.1* and *ANT7.3* for resistance against anthracnose, caused by the fungus *Colletotrichum lindemuthianum* (Sacc. & Magn.), were identified on the proximal arm of Pv07 at 462 361 – 3 404 535 bp (Perseguini et al., 2016; Vaz Bisneta & Gonçalves-Vidigal, 2020). In fact, a separate study reported the differential expression of Phvul.007G040400 after inoculation with *C. lindemuthianum* (Padder et al., 2016). Close to this region, the resistance loci *Co-5* (and its alternative allele *Co-5*^{2}) and *Co-6* display differential responses to several races of the same pathogen, and the markers associated with *Co-5* and *Co-6* are located at 6 984 298 – 9 624 142 bp on Pv07 (Kelly & Bornowski, 2018; Sousa et al., 2014; Vaz Bisneta & Gonçalves-Vidigal, 2020). Furthermore, two QTL related to *Sclerotinia sclerotiorum*, the causal agent of white mold, were mapped at approximately 2 Mbp and 5 Mbp on Pv07 (Vasconcellos et al., 2017). Additional QTL for common bacterial blight (*Xanthomonas axonopodis* pv. *phaseoli*) and ashy stem blight (*Macrophomina phaseolina*) resistance were reported about 12 and 15 cM upstream of the AS8 marker (Miklas et al., 2000), respectively, which maps in the same region as the SAS8 marker at 2.98 Mbp (Larsen & Miklas, 2004). These studies support the hypothesis of a resistance cluster on Pv07 as suggested by Miklas et al. (2009). The era of pangenomics, based on the comparison of multiple high-quality genome assemblies, will open the possibility to further explore to what extent resistance to geminiviruses and maybe even other diseases are physically linked.

Breeding for resistance to begomoviruses

Breeding for virus resistance in common bean has been a long effort in the breeding programs of the Americas and has benefited from the identification and characterization of the genetic basis behind the resistance responses (Beebe, 2012). These efforts have provided molecular tools to accelerate the selection of resistant germplasm through MAS (Meziadi et al., 2017). Recent studies underline the effectiveness of MAS for introgression of multiple disease resistances in breeding programs (Mondo et al., 2019; Rezene et al., 2019). In this study, we identified a set of SNP markers that can be readily used for MAS of BLCrV resistance and could be complementary in the breeding effort against other begomovirus-related diseases. We further explored the allelic diversity present in the ROI using available WGS data, identifying at least five haplotype groups with a differential phenotypic response to BLCrV. The effect of different allelic series has been reported for anthracnose (Vaz Bisneta & Gonçalves-Vidigal, 2020) and angular leaf spot (Nay et al., 2019) resistance in common bean, highlighting its potential use for breeding. In the present study, haplotype H1 showed the highest resistance to BLCrV. This group is composed mainly by Mesoamerican breeding lines. Many of them were derived from Porrillo Sintético (G4495) and A429, two of the most widely used sources for begomovirus resistance in breeding programs in Latin America (Beebe, 2012; Blair & Morales, 2008), emphasizing the impact that previous breeding for BGYMV could have on the emerging BLCrV. Despite that, selection solely for *bgm-1* has proven ineffective against BLCrV (Beebe, 2012), which also supports the hypothesis that the two loci in Pv03 and Pv07 are complementary against begomoviruses. The prospect of pyramiding genes rather than selecting single loci might further increase efficiency of MAS.

The methods that were used to map *BLC7.1* display some limitations when trying to identify the causal polymorphisms that explain the observed phenotypic variation. Firstly, the proportion of explained variance by individual markers ranged between 8.22 (Pv07_3313494_G/A in Pra17) and 16.08% (Pv07_3644749_G/T in Pal14), which is significantly lower than the explained variance achieved by whole-genome regression models (ranging between 51.12 and 77.59%). This result indicates that, even though the resistance against BLCrV is mainly associated with *BLC7.1*, there are other small-effect loci in the genome that contribute to this response, displaying a more quantitative behavior. Secondly, the missing heritability ranged between 0.12 ($BLCrV_{AUDPC}$ – Pra17) and 0.32 (YdHa – Pal14), indicating that not all the causal variants were genotyped in the population (De los Campos et al., 2015). Despite these limitations, the results demonstrate the usefulness of these markers or the whole-genome prediction models for efficient selection of BLCrV resistance. The markers can be integrated in the breeding effort of small, publicly funded breeding programs against other begomovirus-related diseases that affect common bean.

CONCLUSION

The results presented in this study provide evidence that the locus *BLC7.1* is associated with resistance against the novel BLCrV. This locus contains the gene model Phvul.007G040400, which was independently identified in two separate populations and trials, using two complementary approaches. This gene encodes an LRR-RLK subfamily III member that is likely involved in the response against BLCrV through PTI of the plant, and thus is proposed as a candidate gene. The evidence reported in this study supports the hypothesis that previous breeding efforts against BGYMV and BGMV, two of the most important viral diseases affecting common bean in Latin America, contributed to the accumulation of minor-effect genes that play a fundamental role in the resistance against other begomoviruses, including BLCrV. This study provides SNP markers tagging the resistance locus in Pv07 and especially the resistant haplotype H1, which can be readily used in MAS applications against BLCrV. Considering the shared location with QTL reported for other geminiviruses, these

molecular tools can be complementary in the breeding efforts against them, including BGYMV and BGMV.

EXPERIMENTAL PROCEDURES

Plant material

A breeding population of 190 F_4-derived RILs that was developed by biparental crosses between breeding lines from the Andean (Red Canadian Wonder, CAL143, SUG131, PAN127, Natal Sugar) and Mesoamerican (SER8, SER16, SER22, SEC16, SEQ11) gene pools was used in this study. The development of this population was described by Mayor-Duran (2015) and Mayor-Duran et al. (2016). Together with the above-mentioned ten parental lines, the 190 F4-derived RILs form the AxM population of 200 lines as illustrated in Figure 1a (green).

The AxM population was extended with 120 additional breeding lines of Andean and Mesoamerican origin that have previously been characterized for the genetic response to different common bean diseases. Together with 197 lines of the AxM population, they form the extended AxM population, comprising in total 317 lines (Figure 1a, blue). Among these lines, there were several that have been used traditionally as resistance sources to BGYMV by different breeding programs (Beebe, 2012; Miklas et al., 2009), such as Tio Canela 75, DOR476, A429, Porrillo Sintético (G4495) and Garrapato (G2402).

Similarly, a panel of 185 breeding lines was assembled from the collection of elite germplasm of the Mesoamerican breeding pipeline at CIAT, forming the Mesoamerican panel as illustrated in Figure 1a (red).

Finally, two pre-breeding populations (P135 and P136) from the Andean breeding pipeline at CIAT were obtained from two- and three-way crosses between elite Andean lines and Mesoamerican resistance sources (36 parental lines in total). These resistance sources had been identified in previous field evaluations. P135 and P136 were composed of 111 $F_{4:6}$ lines intended to combine biofortification or drought tolerance with BLCrV resistance. Together with the 36 parental lines they form a panel of a total of 147 lines termed P135-136 (Figure 1a, yellow and Table S1).

Field trials and phenotyping

Three yield trials were conducted in Palmira, Colombia (3°30.270060′N, 76°21.397320′W, at 1001 meters above sea level) between 2013 and 2015 (labeled Pal13, Pal14 and Pal15). These trials were established in the field following an alpha-lattice design with three replicates. The experimental units consisted of two-row plots of 4.4 m^2. The trials were managed under rainfed (2013 and 2014) and irrigated (2015) conditions, following standard field practices such as the application of fungicide seed treatment and foliar insecticides. In these trials, DF and DPM were registered when 50% of the plants in the plot had at least one open flower and when 50% of the pods had turned color, respectively. The trait YdHa was calculated per plot, corrected for moisture content, and extrapolated to kg/ha. The three yield trials were carried out using the 190 RILs and the 10 parental lines of the AxM population (Figure 1a, green).

Three BLCrV resistance trials were conducted in Pradera, Colombia (3°23.266740′N; 76°16.712880′W, at 1147 meters above sea level), one in 2017 and two in 2020. The selected site has a high disease pressure from viruses transmitted by whiteflies. Prior to the establishment of each trial, staggered plantings were conducted in the field to maintain the whitefly populations. To monitor disease pressure, two different lines with known resistant and susceptible responses to BLCrV were included in these plantings (EMP496 and PVA773, respectively). The BLCrV trials in 2017 and 2020 followed a randomized complete block design with two and three replicates, respectively. The experimental units were one-row plots of 1.5 m^2 and a row-to-row distance of 0.6 m. To monitor the development of the disease in the field, the trials contained sentinel plots of EMP496 and PVA773 that were uniformly distributed in the field, taking up 15 to 20% of the total rows. The extended AxM population was tested in 2017 (Pra17, Figure 1a, blue). In addition, two separate trials were conducted using the Mesoamerican panel (Pra20M) and P135-136 (Pra20P) in 2020 (Figure 1a, red and yellow).

Plants were visually scored for disease severity 30, 40 and 50 days after planting, using a scale from 1 to 5 according to Morales (2000). A score of 1 indicates no or very low chlorotic mottling (resistant); a score of 3 indicates chlorotic mottling, leaf crumpling and growth arrest; and a score of 5 indicates that plants were fully affected by leaf crumpling and deformation of the pods (susceptible). These data were used to calculate the area under the disease progress curve (AUDPC), which expresses the susceptibility to BLCrV. Infection in the yield and BLCrV trials was confirmed thereafter by molecular diagnostics using generic primers (Rojas et al., 1993).

Statistical analysis of the phenotypic data

Data from the yield trials in Palmira (Pal13, Pal14 and Pal15) and data from the BLCrV trials in Pradera (Pra17, Pra20M and Pra20P) were modeled with the mixed linear model (MLM) approach with spatial correction implemented in the R package SpATS (v1.0–11) (Rodríguez-Álvarez et al., 2018). This model uses the arrangement of plots in the field as a row-column Cartesian plane. The genotypic term in the model was taken as a fixed effect to calculate the best linear unbiased estimators (BLUEs) for each trait and trial. In addition, the genotypic term was also taken as a random factor to calculate an estimate of the broad-sense heritability using the function 'getHeritability' of SpATS. To avoid bias in the estimation of heritability due to the presence of control lines in the Pradera trials (Pra17, Pra20M, Pra20P), the reference lines EMP496 and PVA773 were taken as a separate fixed effect factor within the model. Also, an additional random effects term for the number of harvested plants was included when modeling the seed yield (only for Pal13, Pal14 and Pal15). The phenotypic correlation between traits was expressed as Pearson's coefficients among BLUEs, and their significance was tested using a two-tailed *t* test.

Genotyping

The extended AxM population and the Mesoamerican panel were genotyped following the GBS protocol proposed by Elshire et al. (2011). DNA extraction and genotyping were carried out as described in Nay et al. (2019). Briefly, DNA was extracted from leaf tissue of germinated seedlings using liquid N_2 and following the urea buffer-based extraction miniprep protocol. The GBS library was prepared using the *Ape*KI restriction enzyme. The sequencing service was provided by the Institute of Biotechnology of Cornell University (https://www.biotech.cornell.edu/core-facilities-brc/services) and The HudsonAlpha Institute for Biotechnology (https://hudsonalpha.org/), sequencing 96-well plates on one or two lanes of an Illumina HiSeq 2500 instrument, using single-end reads of 101–151 bp. Sequence demultiplexing and SNP calling were performed using NGSEP (v4.0.3) (Tello et al., 2019), following recommended parameters for GBS data (Perea et al., 2016). Adapters and low-quality sequences at the ends of the reads were trimmed with Trimmomatic (v0.36) (Bolger

et al., 2014). The processed reads were mapped to the reference genome of *P. vulgaris* accession G19833 (v2.1) (Schmutz et al., 2014) using Bowtie2 (v2.2.30) (Langmead & Salzberg, 2012). The variant call format matrix was filtered for genotype calls with a quality score above 40, a minor allele frequency (MAF) above 0.05 and a maximum observed heterozygosity rate of 0.05 per SNP marker. Finally, those variants with less than 55% lines genotyped were removed to produce a genotype matrix with around 10% missing data. Missing marker information was then imputed using Beagle (v5.0) (Browning et al., 2018), setting the effective population size at 100 and using the genetic map reported by Diaz et al. (2020). The process to construct a genotypic matrix was performed independently for each population. In addition, a joint matrix was produced combining the sequencing data from both populations and the available sequencing data of the P135-136 parental lines.

The pre-breeding populations P135 and P136 were genotyped using the DArTag targeted genotyping service offered by Diversity Arrays Technology (DArT PL, Bruce ACT, Australia). To select the SNPs to set up this platform, the sequencing data from 1700 breeding lines and landraces that had been genotyped with GBS or WGS at the CIAT Bean breeding program was assembled into a large genotypic matrix (see Lobaton et al., 2018; Keller et al., 2020, Diaz et al., 2020; Diaz, Ariza-Suarez, et al., 2021; Diaz, Arredondo, et al., 2021 for details about the sequencing data used). The variants in this matrix were filtered to remove SNPs within or adjacent to (up to 300 bp) repetitive regions of the reference genome (Lobaton et al., 2018) and variants with a MAF below 0.01. In addition, the predicted recombination frequency between SNP variants was calculated using the genetic map reported by Diaz et al. (2020), fitting a non-parametric spline regression (implemented in R, v4.0.3) between the physical and genetic positions of the SNP variants. Using this information, one or two SNP variants per 0.5-cM window were selected across the reference genome. In addition, some markers that are regularly used in the breeding program at CIAT for MAS were included, producing a set of 1861 SNP-containing target sites that were used for amplicon sequencing within the DArTag pipeline (Table S2). The genotyping process consisted of drying leaf discs for each sample at 50°C overnight and shipping them to Intertek Australia for DNA extraction. Library preparation and DNA sequencing were performed at DArT PL (Bruce, ACT, Australia).

Linkage disequilibrium, population structure and association mapping

Pairwise measures of LD were calculated for each chromosome in sliding windows of 100 markers using the joint SNP matrix that was obtained from all available GBS data. The LD measures were corrected for kinship relationships in the population (r_V^2) as implemented in the R package LDcorSV (v1.3.2) (Mangin et al., 2012). The LD decay was estimated regressing the pairwise r_V^2 values on the physical distance of their markers using the locally estimated scatterplot smoothing implemented in the R function 'loess' (v4.1.0), with a span value of 0.5. The LD decay rate was defined as the physical distance where the predicted LD reached half of its initial value.

The population structure in the extended AxM population, the Mesoamerican panel and the parental lines of P135-136 was jointly assessed by calculating an identity-by-state distance matrix followed by a NJ tree using NGSEP (v4.0.3) (Tello et al., 2019). This tree was visualized using Splitstree (v4.15.1) (Huson & Bryant, 2006). In addition, a PCA was conducted on the SNP matrix using GAPIT (v3.0) (Wang & Zhang, 2021). A GWAS was carried out independently on the AxM population, the extended AxM population, the Mesoamerican panel and P135-136 using the data from each of the six trials (Pal13, Pal14, Pal15, Pra17, Pra20M and Pra20P) and four evaluated traits (DF, DPM, Yd and $BLCrV_{AUDPC}$). These analyses were conducted using the BLINK algorithm (Huang et al., 2019) implemented in GAPIT (v3.0) (Wang & Zhang, 2021), which uses a multiple-loci test method by combining a fixed effect model, Bayesian information criteria and LD between markers. Significant associations were defined when the *p* value for each SNP marker was smaller than the Bonferroni-corrected threshold, which was calculated with a genome-wide type I error rate $\alpha = 0.05$ ($P < 2.028 \times 10^{-6}$). Marker effects and proportion of variance explained by each marker were calculated using the MLM approach implemented in GENESIS (v2.24.0) (Gogarten et al., 2019) as follows:

$$y = W\alpha + x_i\beta_i + u + \varepsilon, \quad (1)$$

where y is the n-dimensional vector of BLUEs; α is the k-dimensional vector of covariate effects ($k = 3$ principal components); W is the corresponding $n \times k$ design matrix; β_i is the additive effect of the ith marker and x_i is the n-dimensional vector of marker coding; u is the n-dimensional vector of polygenic background effects that fit a normal distribution with mean 0 and a variance–covariance matrix defined as $u \sim N(0, \sigma_u^2 K)$, where K is the additive kinship matrix derived from the SNP markers and σ_u^2 is the polygenic additive variance; and ε is a vector of random residuals $\varepsilon \sim N(0, \sigma_\varepsilon^2 I)$. In this model, α and β_i are assumed to be fixed parameters.

Candidate gene identification

The ROI for candidate gene identification was defined by the genomic interval between SNP markers most significantly associated to BLCrV resistance as identified by GWAS. The functional annotation in this region was used to identify the gene models related to disease resistance mechanisms in plants. After the identification of the most promising candidates, the sequence variation of its coding region was analyzed using available WGS data (Lobaton et al., 2018; Wu et al., 2020), assembling a genotypic matrix of 699 accessions, which included 29 lines that were evaluated in the BLCrV trials (Figure 2). Using this matrix, the population structure in the coding region was inferred using the fastSTRUCTURE algorithm (v1.0) (Raj et al., 2014), identifying the optimal number of haplotypes. The effect of each haplotype on BLCrV resistance was modeled using the following MLM model:

$$y_{ij} = \mu + H_i + \varepsilon_{ij}, \quad (2)$$

where y_{ij} is the $BLCrV_{AUDPC}$ BLUE value for the jth genotype carrying the ith haplotype, μ is the overall intercept, H_i is the effect of the ith haplotype, and ε_{ij} is the error term corresponding to y_{ij}. In this model, the term H_i was treated as a random effect. We assumed that the random term H and the residual ε fit a normal distribution with mean 0 and independent variances $H \sim N(0, \sigma_H^2 I)$ and $\varepsilon \sim N(0, \sigma_\varepsilon^2 I)$. To test the significance of the haplotype effect, the previous MLM was compared to the reduced model $y_{ij} = \mu + \varepsilon_{ij}$ using a likelihood ratio test and the significance p value was adjusted following the work of Self and Liang (1987).

Genomic prediction

The genomic breeding values were calculated based on BLUEs and the SNP matrix using the BGLR package (Pérez & de los Campos, 2014) and the following model:

$$y_i = \mu + \sum_{i=1}^{p} x_{ij}\beta_j + \varepsilon_i, \quad (3)$$

where y_i is a vector of BLUEs for the ith line, μ is the overall phenotype mean, x_{ij} is the genotype of the ith line at the jth marker, β_m is the mth marker effect and ε_i is the error term. The assumptions were $\beta \sim N\left(0, I\sigma^2_\beta\right)$ and $\varepsilon \sim N\left(0, I\sigma^2_\varepsilon\right)$.The genomic prediction was assessed by cross-validation, randomly splitting the dataset into training (70%) and validation (30%) subsets.

The Pearson correlation coefficient between predicted and observed values of the validation subsets were calculated to quantify the prediction ability for each trait. This cross-validation process was repeated 100 times. The different Bayesian priors A, B and C, as well as BRR and Bayesian Lasso implemented in BGLR were evaluated. In addition, significant SNP–trait associations identified in the training set were added as fixed effects in the QTL-BRR model as described by Keller et al. (2020). Marker effects of the BRR model were averaged over the 100-fold cross-validation. The number of iterations per cross-validation step was 20 000, using the first 10 000 as burn-in. The genomic heritability was calculated using the remaining iterations thinned by a factor of 5 in the BRR model. It used the sample variance of genomic values at each iteration of the sampler, as described by De los Campos et al. (2015).

AUTHOR CONTRIBUTIONS

BR and VM conceived the study. AEP-B, HFB and VM developed the plant material and conducted the field trials and data collection in Palmira. JMB and AS conducted the field trials and data collection in Pradera. DA-S, BK and JSA performed the statistical analyses of the phenotypic data. DA-S, BK and AS performed the genetic analyses, in consultation with BS. The first draft of the manuscript was written by DA-S and BK and improved by BS and BR. All authors contributed to and approved the final version of this manuscript.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article.

Figure S1. Pattern of linkage disequilibrium (LD) decay calculated genome-wide (black-dashed line) and for each chromosome separately (colored lines) using the joint genotypic matrix. The pairwise measures of LD were calculated in sliding windows of 100 markers and corrected for kinship relationships in the population (r^2_V). Each line corresponds to a locally estimated scatterplot smoothing (LOESS) regression on the LD measures.

Figure S2. Principal component analyses to assess the population structure for (a) the extended AxM population, (b) the Mesoamerican panel and (c) P135-136 analyzed in this study using genotyping-by-sequencing (a and b) or DArTag (c) data. The location of each genotype is represented by a point in the two-dimensional space defined by the eigenvectors of the first and second principal components.

Figure S3. Results of the genome-wide association analysis for days to flowering (DF), days to physiological maturity (DPM) and seed yield (YdHa) evaluated in three yield trials between 2013 and 2015 in Palmira, Colombia. These results are presented as individual Manhattan plots showing the significance of the SNP markers (colored dots) used in this study and their physical location on each of the 11 chromosomes of the reference genome of the accession G19833 (v2.1). The horizontal red-dashed lines represent the Bonferroni-corrected threshold, which was calculated with a genome-wide type I error rate $\alpha = 0.05$ ($P < 2.028 \times 10^{-6}$). The corresponding quantile-quantile plots to the right compare the deviation between the observed and the expected significance of the SNP markers from a theoretical χ^2 distribution.

Figure S4. Combined results of the genome-wide association analysis in the region of interest (ROI) for seed yield in Palmira in 2014 (YdHa – Pal14) and BLCrV susceptibility in Pradera ($BLCrV_{AUDPC}$ – Pra17 and Pra20M). The most significantly associated SNP markers are indicated with vertical gray lines. The horizontal red-dashed line represents the Bonferroni-corrected threshold, which was calculated with a genome-wide type I error rate $\alpha = 0.05$ ($P < 2.028 \times 10^{-6}$). The colored square matrix below represents the pairwise linkage disequilibrium (LD) measurements (r^2_V) between each pair of markers in the ROI. Gray lines connect the physical location of the SNP markers in the Manhattan plot with their corresponding position in the square matrix.

Figure S5. Population structure analysis in the coding region of the candidate gene Phvul.007G040400 using 69 sequence variants. This analysis was performed using fastSTRUCTURE and indicates that the optimal number of groups/haplotypes is between five and eight. The colors represent each one of the five haplotypes that were identified in a collection of 699 accessions with whole-genome resequencing data.

Table S1. List of lines and the trials in which they were included. In addition, the genotyping strategy as well as pedigree information is given for each common bean line used in this study.

Table S2. List of SNP markers and target regions selected for DArTag genotyping.

Table S3. Results of the genome-wide association analysis for days to flowering (DF), days to physiological maturity (DPM) and seed yield (YdHa) evaluated in three yield trials between 2013 and 2015 in Palmira, Colombia, and three BLCrV trials in Pradera between 2017 and 2020. PVE indicates the proportion of explained variance from each marker, while Est. Effect indicates the estimated effect.

OPEN RESEARCH BADGES

This article has earned an Open Data badge for making publicly available the digitally-shareable data necessary to reproduce the reported results. The data is available at https://doi.org/10.7910/DVN/9JSMED.

DATA AVAILABILITY STATEMENT

The datasets generated and analyzed in this study are available in the Harvard Dataverse repository under the title 'Replication data for: Genetic analyses for the response to Bean Leaf Crumple Virus (BLCrV) identify a

candidate LRR-RLK gene' (2021) (https://doi.org/10.7910/DVN/9JSMED).

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