RESEARCH ARTICLE

Genome-wide association mapping reveals race-specific SNP markers associated with anthracnose resistance in carioca common beans

Caléo Panhoca de Almeida1*, Jean Fausto de Carvalho Paulino1*, Caio Cesar Ferrari Barbosa1, Gabriel de Moraes Cunha Gonçalves2, Roberto Fritsche-Neto3, Sérgio Augusto Morais Carbonell2, Alisson Fernando Chiorato2, Luciana Lasry Benchimol-Reis1

1 Centro de Pesquisa em Recursos Genéticos Vegetais, Instituto agronômico (IAC), Campinas, São Paulo, Brazil, 2 Centro de Grãos e Fibras do Instituto agronômico (IAC), Campinas, São Paulo, Brazil, 3 Departamento de Genética, Universidade de São Paulo (ESALQ-USP), Piracicaba, São Paulo, Brazil

* These authors contributed equally to this work.

* caleoalmeida@hotmail.com

Abstract

Brazil is the largest consumer of dry edible beans (*Phaseolus vulgaris* L.) in the world, 70% of consumption is of the carioca variety. Although the variety has high yield, it is susceptible to several diseases, among them, anthracnose (ANT) can lead to losses of up to 100% of production. The most effective strategy to overcome ANT, a disease caused by the fungus *Colletotrichum lindemuthianum*, is the development of resistant cultivars. For that reason, the selection of carioca genotypes resistant to multiple ANT races and the identification of loci/markers associated with genetic resistance are extremely important for the genetic breeding process. Using a carioca diversity panel (CDP) with 125 genotypes and genotyped by BeadChip BARCBean6K_3 and a carioca segregating population AM (AND-277 × IAC-Milênio) genotyped by sequencing (GBS). Multiple interval mapping (MIM) and genome-wide association studies (GWAS) were used as mapping tools for the resistance genes to the major ANT physiological races present in the country. In general, 14 single nucleotide polymorphisms (SNPs) showed high significance for resistance by GWAS, and loci associated with multiple races were also identified, as the *Co-3* locus. The SNPs ss715642306 and ss715649427 in linkage disequilibrium (LD) at the beginning of chromosome Pv04 were associated with all the races used, and 16 genes known to be related to plant immunity were identified in this region. Using the resistant cultivars and the markers associated with significant quantitative resistance loci (QRL), discriminant analysis of principal components (DAPC) was performed considering the allelic contribution to resistance. Through the DAPC clustering, cultivar sources with high potential for durable anthracnose resistance were recommended. The MIM confirmed the presence of the *Co-1* locus in the AND-277 cultivar which revealed that it was the only one associated with resistance to ANT race 81. Three other loci were associated with race 81 on chromosomes Pv03, Pv10, and Pv11. This is the first study to identify new resistance loci in the AND-277 cultivar. Finally, the same *Co-1* locus was also significant for the CDP at the end of Pv01. The new SNPs identified,
especially those associated with more than one race, present great potential for use in marker-assisted and early selection of inbred lines.

Introduction

Approximately 70 species have been described in the Phaseolus genus, only five of which are cultivated; Phaseolus vulgaris L. (bean) is considered the most important species in the Phaseolus genus for direct consumption in the human diet [1–3]. The species arose in Mexico [4,5], from where it spread to South America, giving rise to two distinct gene pools, called Mesoamerican and Andean [6,7]. In some African and American countries, beans are responsible for providing an average of 15% of total daily calories and 36% of protein content consumed [8]. World bean consumption has increased in recent years, with dry bean production reaching about 30 million tons in most recent analysis [9]. Most beans are produced by the countries of Asia and the Americas, which together account for approximately 75% of world production. Brazil stands out as one of the largest producer and consumer of beans in the world, and is responsible for 36% of the production on the American continent [9].

The estimated production of the 2020/2021 common bean crop in Brazil was about 3.2 million tons [10], with the carioca bean variety accounting for 70% of common bean consumption, followed by the black bean variety with 15% [11]. The first carioca cultivar was released in 1971 and, due to high yield (i.e., superiority of approximately 35% in relation to the varieties launched in the 60s), the new cultivar Carioquinha quickly came to predominate bean growing and consumption in Brazil [12]. The carioca commercial group cultivars are characterized by cream-colored grain with brown stripes and high yields [13] and they belong to the Mesoamerican gene pool [14]. Although the variety has high yield, it is far from its genetic potential since it is susceptible to diseases, such as anthracnose, which can cause losses of up to 100% of production [15].

Anthracnose caused by the ascomycetous Colletotrichum lindemuthianum (Sacc. and Magnus) Briosi and Cavara and considered to have a high pathogenicity, is characterized by small brown spots throughout the aerial part of the plant, frequent beginning in leaf veins, stems, and petioles [11,12]. Due to the high pathogenic variability of the fungus, Pastor-Corrales [16] proposed the classification of C. lindemuthianum isolates into physiological races based on the susceptible and resistant responses of the isolates to a differentiating series composed of 12 differential cultivars of common bean. A total of 1,590 isolates have already been characterized, resulting in identification of 182 races worldwide [15]. In Brazil, 474 isolates have been tested and 60 races described, with race no. 65 as the most frequent [15]. More specifically for the state of São Paulo, a study characterized 51 isolates and identified 10 races, with a predominance of race 65 and 81. The study furthermore indicated race 321 and 351 as the most pathogenic races [17]. Coelho et al. [18] also reported 65, 73, and 81 as some of the most frequent occurring races in Brazil.

The development of cultivars with durable resistance to disease is the most economical, efficient, and environmentally friendly resource as it avoids intensive use of pesticides, ensuring higher yields and less environmental contamination [19]. For that reason, cultivars with resistance to multiple races and the respective genes associated with this resistance need to be investigated and identified, enabling their use in common bean breeding programs. About 25 loci with multiple alleles of resistance to ANT, from both Andean and Mesoamerican origin, have already been identified [20–22]. The loci with the greatest and dominant effect are

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designated “Co”. The loci of Mesoamerican origin include Co-2, Co-3 (Co-3^2, Co-3^3, Co-3^4, and Co-3^5 alleles) Co-4 (Co-4^2 and Co-4^3 alleles), Co-5 (Co-5^2 alleles), Co-6, Co-11, Co-16, Co-17, Co-u, and Co-v, mapped on chromosomes Pv02, Pv03, Pv04, Pv07, Pv08, and Pv11, respectively [23–35]. The “Co” loci originating from Andean origin are Co-1 (Co-1^2, Co-1^3, Co-1^4, and Co-1^5 alleles), Co-12, Co-13, Co-14, Co-15, Co-x, Co-w, Co-y, Co-z, Co-Pa, Co-AC, and CoPv01_CDRK on chromosomes Pv01, Pv03, and Pv04 [30,31,35–49].

Other studies involving QRL mapping have also been reported. López et al. [50] identified five QRL on three chromosomes; Zuiderveen et al. [51] reported 14 QRL with greater effects on chromosomes Pv01, Pv02, and Pv04, and 2 others with lesser effects on Pv10 and Pv11. Wu et al. [52] mapped 9 QRL (on Pv01, Pv02, Pv04, Pv05, Pv06, Pv10, and Pv11) and Perseguini et al. [53] another 17 QRL (on Pv01, Pv02, Pv03, Pv04, Pv05, Pv06, Pv07, Pv08, and Pv11). Recently, Fritsche-Neto et al. [54] identified a QRL with a greater effect on Pv02, explaining 25% of the resistance observed for nine environments evaluated.

In addition to the resistance loci, several studies have reported many ANT resistance sources from mainly cultivars of Andean origin [43,45,48,49,55–58], and also some of Meso-american origin, belonging to the black or special bean commercial classes [30,33,34,40,59–63]. Currently, developing new carioca cultivars through breeding is still a challenge as the variety demands high grain quality [64]. Breeding programs routinely exploit only the elite germplasm, which results in a narrow genetic base [65]. It is difficult to use Andean accessions as a source of resistance due to the reproductive incompatibility during the hybridization [66]. Recently, Almeida et al. [67] demonstrated that it is feasible to use Andean accessions to improve inbred lines resistance to angular leaf spot and that marker-assisted selection (MAS) is an important tool. However, obtaining elite genotypes was only possible after backcrossing cycles. Thus, identification of loci associated with ANT resistance in carioca bean is extremely important in development/breeding of elite genotypes with resistance to multiple races.

The current study aimed at identifying carioca genomic regions associated with race-specific ANT resistance and recommendation of these cultivars in Brazil will lead to genetic improvement of the common bean. A CDP, previously validated for GWAS, was characterized in terms of resistance to three different physiological ANT races (65, 81, and 321) and genotyped using high-throughput genotyping technologies (i.e., BeadChip BARCBean6K_3). The results were corroborated by linkage mapping using a segregating carioca population derived from the cross between the IAC-Milênio and AND-277 cultivars.

**Materials and methods**

**Plant material and high-throughput genotyping**

The CDP used in the present study was composed of 125 bean genotypes, including landraces, advanced inbred lines, and commercial cultivars. The set represents genotypes from the main genetic breeding programs in the country, with cultivars released by 11 different institutions. These vary from the first cultivar carioca released by the Instituto Agronômico—IAC (Campinas, SP, Brazil) in 1971 (i.e., Carioquinha or Carioca comum) to more recent cultivars, such as IPR-Sâbia and IAC-1850, released in 2017 and 2019, respectively. The panel was genotyped using the BeadChip Illumina technology by BARCBean6K_3, with 5,398 SNPs [68], and validated for GWAS by Almeida et al. [69] and successfully used to identify QRL associated with angular leaf spot by Almeida et al. [70]. All the information on the CDP is given in S1 Table of the Supplementary Materials, including the phenotypic and genotypic data.

The quality of the genotypic data was analyzed using the Genome Studio 2.0 software (Illumina), which filtered markers with call frequency and GenTrain score < 0.6. The TASSEL 5.0 software [71] was used to eliminate SNPs with minor allele frequency < 3%,
heterozygosity > 5%, and missing data > 10%. The high-quality genotypic matrix was converted into a HAPMAP file format, with the reference allele represented by “A”, the alternative allele by “G”, the heterozygous by “R”, and missing data by “N”. The BARCBean6K_3 was developed based on the first common bean genome (i.e., *Phaseolus vulgaris* v1), and therefore, the flanking sequences of each SNP were blasted (e.g., BLASTN) against the most current reference genome, *Phaseolus vulgaris* v2.1 [8], and the position of each SNP was obtained. Markers of unknown position in the genome were removed, and “N” loci were imputed using the Beagle 5.0 software [72].

For linkage mapping, the AM genetic map estimated from 1,114 SNPs genotyped by GBS (genotyping by sequencing) in the AM segregating population BC2F3 ([(♀AND-277 ×♂IAC--Milênio) × IAC-Milênio] × IAC-Milênio)) was used. The AM population was developed and validated for genetic mapping by Almeida et al. [70] and was composed of 91 inter-pool lines selected according to the carioca grain ideotype. All the information on the genotypes of the AM population is presented in S1 Table in the Supplementary Materials, including the phenotypic and genotypic data.

**Phenotypic evaluation**

For evaluation of resistance, monosporic cultures of physiological races 65 (IAC fungal library code 14781), 81 (IAC fungal library code 14786), and 321 (IAC fungal library code 14831) of *C. lindemuthianum* were selected due to their importance for improvement of the variety as these are the most frequent occurring races in Brazil. The respective races were characterized by Ribeiro et al. [17] according to the differentiating series proposed by Pastor-Corrales [16]. Both sets, the CDP and the AM population, were evaluated individually for the severity of their reaction to each race, following a randomized block experimental design with three replications. The plot consisted of one row with five plants randomized in trays (36 × 26 × 7 cm) filled with sterile vermiculite.

A total of 15 seeds per genotype were pre-germinated in germination paper (Germitest) in BOD (Biochemical oxygen demand) at 25°C for three days. On the third day, the seeds were transplanted and grown in the greenhouse for nine days. The trays were then placed in a chamber with controlled humidity and temperature. The plants were inoculated by spraying on both leaf surfaces (concentration of 1.2 × 10^6 spores/mL). The inoculum was produced according to the method proposed by Cárdenas et al. [36] using test tubes containing sterile pods partially immersed in an agar-water medium.

The inoculated plants were maintained in high humidity (> 95%) for 48 h at 22°C, and disease severity was assessed 10 days after inoculation (DAI). The diagrammatic scoring scale proposed by Gonzáles et al. [73], based on the severity of the symptoms resulting from inoculation with ANT races, was used for evaluation. Plants with scores from 1.0 to 3.0 were considered resistant, from 3.1 to 6.0 were considered moderately resistant, and from 6.1 to 9.0 were considered susceptible. In all trials, the cultivar IAC-1850 was used as a resistance check, due to its high resistance to all races [74], and Rosinha G2 was used as a susceptible check [17].

**Statistical analysis**

Analysis of variance was performed for all phenotypic evaluations performed through the ExpDes package [75], and genetic parameters including selective accuracy which measures the precision of the experiment, were estimated by the RBio software [76]. The genetic resistance correlation of CDP for three races evaluated was done by Pearson’s correlation. For mapping analysis, genotypic values were estimated using the REML/BLUE (Restricted Maximum Likelihood/Best Linear Unbiased Estimator) model by the Be-Breeder package [77].
The Fixed and random model Circulating Probability Unification (FarmCPU) [78] implemented in the GAPIT 2.0 package [79] was used for association mapping due to its high statistical power and greater sensitivity to QRL with lesser effects. The FarmCPU uses the multilocus mixed model (MLM) and performs the analysis in two interactive steps: a fixed-effect model is applied first, followed by a random-effect model. Both models are repeated interactively until no significant SNP is detected. As demonstrated by Almeida et al. [14], the CDP does not require the use of a structuring matrix to correct type I errors (i.e., false positives), since there are no subgroups in the set. The p value threshold of each SNP for the first interactive steps in the model was determined by the permutation method using the function \textit{FarmCPU.P.Threshold} (500 repetitions). The Bonferroni [80] threshold method (cutoff $\alpha = 0.05$) was also used to determine significance in the Manhattan plot. In order to identify the QRL associated with the three races used together (i.e., the mixture of races), in addition to mapping the evaluations made, a fourth analysis was performed, considering the three evaluations together. For that reason, the genotypic value of each genotype was given by the highest adjusted average among the three ANT races used.

The AM genetic map, constructed by Almeida et al. [70], was used for the linkage mapping. The identification of QRL followed the approach described by the authors using the OneMap package [81]. Thus, the probabilities of the genotypes of the putative QRL were obtained by the hidden Markov chain, with steps every 1 cM. The adjusted values of the traits were used to fit the QRL mapping models in a progressive manner. First, markers with significant effects were identified using a fixed linear regression model, and the significant markers were used as cofactors in the composite interval mapping (CIM) model, proposed by Zeng [82]. The significance of the putative QRL were defined by the threshold obtained by 1,000 permutations [83], considering the significance level of 5%. The putative QRL identified were used as a starting point for MIM, based on the model proposed by Kao et al. [84]. The MIM mapping strategy involved three steps: searching for QRL, testing effects, and selecting the model. Starting from the model with the QRL identified in the CIM, new QRL were identified out, and the QRL with the highest likelihood of odds (LOD) values were inserted into the model. Then, all QRL were tested for conditional significance. Both models (complete and reduced) were also compared regarding the Akaiake information criterion—AIC [85]. If a QRL had a non-significant effect or an AIC value higher than the reduced model, it was removed from the model. The procedure was repeated until no QRL was added or removed from the model. Thus, the final model was selected, and the positions and effects of the QRL were re-estimated, as well as the variation explained by each QRL ($R^2$).

**Candidate genes**

The physical position of all the significant SNPs associated with resistance to ANT was used for a thorough search for candidate genes through genetic annotation using \textit{jbrowse} from Phytozome v11.0 [86] and the reference genome \textit{Phaseolus vulgaris} v2.1 [8]. For the search, we considered a confidence interval window of 0.59 Mbp, the average distance identified by Almeida et al. [14] for the CDP (i.e., distance to LD decay = $r^2 0.2$).

**Potential sources for bean resistance breeding**

With the goal of selecting the genotypes that exhibit the best multiple race resistance and have favorable alleles, resistant accessions for all the races evaluated were selected, as well as the significant markers for GWAS. The genotypic data of each genotype with high resistance was converted into a GenAlEx file, through which a favorable allele (i.e., an allele that leads to an increase in resistance) of each SNP was represented by “RR”, an unfavorable allele by “SS”, and
a heterozygous marker by "RS". A control genotype containing all "RR" alleles was inserted in the genotype matrix. To select accessions based on significant SNPs, the set was discriminated and grouped by DAPC proposed by Jombart et al. [87] and implemented in the ADEGENET v2.1.1 package [88]. The DAPC analysis is considered free of Hardy-Weinberg and LD, and consists of transformation of genotypic data by the PCA into components that better explain the genetic variance, and these components are used for linear Discriminant Analysis [87].

The number of clusters required was two—one group for the resistant cultivars (i.e., grouped by the presence of favorable alleles) and another for the susceptible cultivars.

**Results**

**Phenotypic resistance**

Characteristic symptoms of the disease (i.e., necrotic lesions on leaf veins and petioles) were observed for all experiments, from 7–8 DAI. As expected, the check cultivars showed highly contrasting resistance for all trials, and the entire AM population showed resistance to race 321. In addition, 92% and 34% of the AM population had a score ≤ 3 for race 65 and 81, respectively. In relation to the CDP, a total of 41.6% of the genotypes had high resistance to the three races (Fig 1A), with a higher degree of severity (40%) for race 81 (Fig 1B). The boxplot shows the difference in the degree of resistance of the CDP (Fig 1C), and it shows the smallest phenotypic variation for races 65 and 321.

Analysis of variance showed high phenotypic variability for all tests ($p < 0.001$), except for the AM population evaluation with race 321. However, there was no significance for the block effect, indicating the possibility of using a completely randomized design for future trials. The selective accuracy of all trials ranged from 0.96 to 0.99, and broad sense heritability was from 0.93 for CDP in the evaluation with race 65 to 0.99 for the AM population with race 81. Considering the CDP, the Pearson correlation was significant ($p < 0.001$) and positive for the three races, with the highest correlation between race 65 and 81 (60%), followed by race 81 and 321 (46%) and race 65 and 321 (40%).

**Genotypic resistance**

After SNP calling, 1,942 high-quality SNPs genotyped in the 125 accessions of the CDP by high-throughput genotyping technologies were used for association mapping. GWAS was performed for each of the three races individually, and a fourth association was performed using the adjusted average of the most pathogenic race for each genotype (mixture). Considering all analyses (Table 1), a total of 17 SNPs showed high significance (i.e., $p < 0.00002$) according to the Bonferroni test [80]. Among them, two were associated with race 65, six with race 81, five with race 321, and four with the mixture of races (Fig 2A). The QQ-plot represented the quality of the analysis through the good fit of the model used (Fig 2B).

On chromosome Pv01, two SNPs were significant for race 321: the first, SNP ss715648889, at the 43,062,012 bp position and the second, SNP ss715645285, at the 49,139,392 bp position. At 1.49 Mbp away from the SNP ss715645285 (Fig 2C), the SNP ss715645299 was associated with race 81, showing the presence of a QRL associated with the most pathogenic races in this region. On chromosome Pv02, the SNP ss715647887 (35,782,225 bp position) also showed significance for race 321, and a second SNP, ss715648710 (41,709,302 bp position), was associated with joint analysis (the mixture of races). Precisely at the beginning of the Pv04 chromosome, two SNPs had the greatest significance for the four analyses (Fig 2A), with the SNP ss715649427 (535,120 bp position) associated with race 65, and the SNP ss715642306 (373,157 position) associated with race 81, 321, and the mixture of races.
The SNPs on the Pv04 chromosome were at 0.16 Mbp from each other (Fig 2C), making it possible to identify the presence of a unique and important QRL in this region. Considering the allelic substitution test, the alternative “A” allele of SNP ss715649427 was responsible for the increase in resistance of 0.8 for race 65 considering the 1–9 scoring scale, while the alternative “C” allele of SNP ss715642306 led to susceptibility of -1.8, -0.78, and -1.15 for races 81 and 321 and the mixture of races, respectively (Fig 2D). The results indicated an important QRL associated with multiple ANT races at the beginning of the Pv04 chromosome, at the 0.45 Mbp position.

A second SNP (ss715639578) on the Pv05 chromosome was significant for race 65 at 35,969,299 bp, and a single SNP (ss715646017, 37,347,362 bp position) on Pv07 was associated with race 81 and with the mixture of races. The Pv08 showed the highest number of associations; the first SNP (ss715647427, 15,431,263 bp position) was significant for race 81 and the second (ss715639361, 58,761,122 bp position) for the mixture of races. In the same region, at 3.2 Mbp, two other SNPs showed significance for race 81 (ss715646102, 61,452,776 bp position and ss715646115, 61,837,278 bp position) and one for the mixture of races (ss715639361, 58,761,122 position). Considering the last three SNPs on the same chromosome, the distance between them was not greater than 0.68 Mbp (Fig 3C), showing the presence of a single QRL associated with the races evaluated.

Fig 1. Phenotypic results from evaluation of carioca diversity panel (CDP) resistance to multiple races of anthracnose. (a) bar chart representing evaluation of the CDP regarding resistance to the *C. lindemuthianum* physiological races 65, 81, and 321. The bars represent the sum of scores of the phenotypic evaluation of each accession, with races 65, 81, and 321 being represented by colors (orange, green, and blue, respectively); (b) distribution of the number of accessions considered resistant (score ≤ 3), moderately susceptible (score from 4 to 6), and susceptible (score >7) for the three races evaluated; (c) boxplot for the three races evaluated.

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Regarding QRL analysis using the AM population (Table 2), three QRL showed significance for race 65: the ALS3.1\(^{AM}\) on chromosome Pv03 (estimated position 154 cM), explaining 9% of the phenotypic variance, flanked by “Marker244” and “Marker248”; the ANT10.1\(^{AM}\) on Pv07, explaining 1.47% of the phenotypic variance, flanked by “Marker244” and “Marker248”; and the ANT10.1\(^{AM}\) on Pv04, explaining 1.47% of the phenotypic variance, flanked by “Marker244” and “Marker248”.

### Table 1. Genome-wide association study results for the association between phenotypic evaluation of the carioca diversity panel (CDP) with multiple races of anthracnose and the high-throughput genotyping.

| Race | SNP     | Chr | Position v2.1 | Ref. allele | Alt. allele | p value   | MAF  | Effect |
|------|---------|-----|---------------|-------------|-------------|-----------|------|--------|
| 65   | ss715649427 | Pv04 | 535,120       | G           | A           | 1.8E-05   | 0.292| 0.80   |
|      | ss715639578 | Pv05 | 35,969,299    | T           | C           | 9.7E-06   | 0.096| 1.14   |
| 81   | ss715645299 | Pv01 | 50,635,589    | C           | T           | 5.4E-06   | 0.076| 1.74   |
|      | ss715642306 | Pv04 | 373,157       | T           | C           | 7.2E-13   | 0.336| -1.83  |
|      | ss715646017 | Pv07 | 37,347,362    | C           | T           | 2.5E-10   | 0.476| 1.47   |
|      | ss715647427 | Pv08 | 15,431,263    | C           | T           | 5.5E-06   | 0.084| -1.29  |
|      | ss715646102 | Pv08 | 61,452,776    | G           | A           | 2.1E-05   | 0.232| 0.84   |
|      | ss715646115 | Pv08 | 61,837,278    | C           | T           | 2.9E-06   | 0.328| -1.01  |
| 321  | ss715648889 | Pv01 | 43,062,012    | C           | T           | 2.5E-07   | 0.048| 1.51   |
|      | ss715645285 | Pv01 | 49,139,392    | C           | T           | 2.4E-06   | 0.344| 0.60   |
|      | ss715647887 | Pv02 | 35,782,225    | T           | C           | 1.3E-07   | 0.104| 1.13   |
|      | ss715642306 | Pv04 | 373,157       | T           | C           | 6.6E-08   | 0.336| -0.78  |
|      | ss715646756 | Pv08 | 62,139,343    | A           | G           | 9.8E-07   | 0.208| 0.80   |
| Mix  | ss715648710 | Pv02 | 41,709,302    | G           | A           | 5.9E-06   | 0.032| -2.38  |
|      | ss715642306 | Pv04 | 373,157       | T           | C           | 7.1E-07   | 0.336| -1.15  |
|      | ss715646017 | Pv07 | 37,347,362    | C           | T           | 8.0E-06   | 0.476| 0.98   |
|      | ss715639361 | Pv08 | 58,761,122    | T           | C           | 9.9E-06   | 0.192| -0.90  |

Race: *C. lindemuthianum* physiological races; Chr: Chromosomes; Ref. allele: Reference allele; Alt. allele: Alternative allele; MAF: Minor allele frequency; Effect: Allelic substitution effect.

Significant for association mapping using the CDP evaluated phenotypically for resistance to *C. lindemuthianum* physiological races 65, 81, and 321.

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Fig 2. Genome-wide association study analysis for resistance to multiple races of anthracnose with 1,942 high-quality simple nucleotide polymorphisms (SNPs) genotyped in the carioca diversity panel (CDP). (a) Manhattan plots showing the association between the SNP markers and the physiological races of *C. lindemuthianum*. Each color corresponds to a different race, with the colors orange, green, blue, and pink corresponding to races 65, 81, 321, and admixture, respectively; (b) QQ-plot for the races evaluated; (c) Position of significant SNPs in genomic regions with SNPs associated with more than one race, showing the distance between them; (d) Boxplots illustrating the relationships between alleles and phenotypes for the significant SNPs located at the beginning of the Pv04 chromosome and associated with all the races tested.

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Fig 3. Quantitative resistance loci (QRL) plots indicating the LOD score values for each marker position. Manhattan LOD scores obtained by multiple interval mapping analysis (y) using the molecular marker distances of the AM genetic map for *C. lindemuthianum* races 65 and 81. Black triangles indicate the position of significant QRL.

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Table 2. Quantitative resistance loci association results (QRL).

| Race | QRL<sup>1</sup> | P<sub>v</sub><sup>2</sup> | EPI (cM)<sup>3</sup> | Flanking Marker | Physical position<sup>4</sup> | LOD | R<sup>2</sup> (%) | AE<sup>5</sup> | SE<sup>6</sup> | p value |
|------|-----------------|------------------|-----------------|-----------------|------------------|-----|----------------|--------|--------|---------|
| 65   | ANT3.1<sup>AM</sup> | 3                | 154.42 (151.31–156.29) | Marker244       | 18.14            | 2.37| 9.12           | 0.59   | 0.18   | 0.001   |
|      |                 |                  |                 | Marker248       | 22.93            |     |                |        |        |         |
| 65   | ANT10.1<sup>AM</sup> | 10               | 35.89 (33.58–37.64) | Marker908       | 41.46            | 2.15| 5.28           | 0.86   | 0.27   | 0.002   |
|      |                 |                  |                 | Marker911       | 44.18            |     |                |        |        |         |
| 65   | ANT11.1<sup>AM</sup> | 11               | 8.98 (5.72–25.32) | Marker933       | 11.01            | 2.46| 9.52           | 0.77   | 0.23   | 0.001   |
|      |                 |                  |                 | Marker934       | 31.69            |     |                |        |        |         |
| 81   | Co-1<sup>h</sup> | 1                | 199.91 (191.52–207.48) | Marker34       | 47.81            | 2.07| 9.95           | 0.85   | 0.27   | 0.002   |
|      |                 |                  |                 | TGA1.1          | 50.02            |     |                |        |        |         |

<sup>1</sup>*C. lindemuthianum* race.

<sup>2</sup>QRL were named according to Pedrosa-Harand et al. [81].

<sup>3</sup>Chromosomes.

<sup>4</sup>Estimated position and interval QRL.

<sup>5</sup>Physical position (Mbp) according to the reference genome v2.1.

<sup>6</sup>Additive effect.

<sup>7</sup>Standard error.

Anthracnose QRL in multiple interval mapping for *C. lindemuthianum* races 65 and 81, using the segregating AM population (AND-277 × IAC-Milênio).

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Pv10 (estimated position 35.89 cM), with $R^2$ estimated at 5% and flanked by “Marker908” and “Marker911”; and finally, the ANT 11.1 AM on Pv11 (estimated position 8.98 cM) explaining 9.5% of phenotypic resistance. For race 81, a single QRL ANT 1.1 AM at the end of chromosome Pv01 showed high significance, explaining 10% of the resistance and flanked by “Marker34” and “TGA1.1”.

Resistance genes

Considering the average LD decay of the CDP, the SNPs ss715646102, ss7156461115, and ss715646756 on the Pv01 chromosome are in LD and belong to the same haplotype block, as well as the SNPs ss715642306 and ss715649427 on Pv04. Except for the SNPs ss715647887, ss715648710, ss715639578, ss715646017, ss715647427, ss715646115, and ss715646756, all the SNPs were close to the resistance genes (i.e., distance < 1 Mbp), whether R genes or genes encoding protein kinases (Fig 4).

The SNP ss715642306 (Pv04) significant for races 81 and 321 and for the mixture of races, which was in LD with the SNP ss715649427 (Pv04) significant for race 65, was in the first exon of the Phvul.004G005800 gene (functional annotation: alkane hydroxylase cyp96a15). A total of 16 resistance candidate genes were identified for the range of both SNPs (i.e., with 14 R genes and two encoding protein kinases). Another cluster of genes associated with resistance was placed in the confidence interval of the SNP ss715645285 (Pv01) associated with race 321, with a total of six genes encoding protein kinases.

The second-largest cluster with protein-encoding kinase genes (i.e., six genes in a range less than 0.58 Mbp) was at 2.3 Mbp of the SNP ss715639578 (Pv05) significant for race 65. Another three genes encoding protein kinases are close to the significant SNPs at the end of the Pv08 chromosome (i.e., ss715639361, ss715646102, ss715646115 and ss715646756). The SNPs ss715647887 and ss715648710 on Pv02 were at the greatest distance from the candidate genes and were in the last intron of the Phvl.002G193800 gene (functional annotation: encoding urease accessory protein) and in the second intron of the Phvul.002G245000 gene (functional annotation: encoding ATP-dependent protease cereblon), respectively.

Best cultivars: Phenotypic and genotypic resistance

Altogether, 52 cultivars showed high resistance to the three races in the phenotypic evaluations (Fig 1). To select the most promising accessions as sources of resistance for common bean breeding, DAPC was used and grouped 18 of the 52 accessions with 100% membership probability for the cluster containing the resistance pattern (i.e., genotype containing all RR alleles) when considering the 14 SNPs associated with ANT resistance (Fig 5A). The analysis did not show overlapping for both clusters (Fig 5B), except for the genotype H96A31, which presented only 20% of the membership probability for the green cluster (favorable alleles).

Among the 18 accessions clustered according to the presence of favorable alleles, the cultivars IAC-Akýt, IAC-Pytá, IAC-Imperator, IAC-Apuá, IAC-Týbatá, IAC-Aysó, IAC-Formoso, IPR-Curió, Carioca MG, and IAC-Ybaté which were most promising as sources of genetic resistance to ANT. Besides being resistant to the three ANT races, all of them are commercial cultivars with high yield potential [90].

Discussion

Several studies aiming at the identification of markers associated with ANT resistance loci in common bean have already been conducted, leading to the identification of genotypes showing the loci of greatest effect. However, most studies involve Andean accessions [30,33,34,40,59–63], and most of the studies with Mesoamerican accessions did not involve...
carioca bean varieties [43,45,48,49,55–58]. Although studies on characterization of ANT resistance and selection of carioca inbred lines and cultivars have been conducted [91–93], no previous association mapping study was conducted with only carioca accessions using a linkage mapping approach with the carioca variety as the resistant parent.

The characterization of new sources of ANT resistance is extremely important for carioca been breeding. However, the use of Andean accessions to improve Mesoamerican cultivars is extremely difficult [94–96], mainly due to reproductive incompatibility [66] and the breakdown of epistatic interactions specific to each gene pool [97]. An inter-pool cross involving parents from different commercial classes is considered a bottleneck for breeders, mainly due
to the segregation observed for the type of grain [98]. In the case of the carioca commercial class, the complexity is even greater since any change in the ideotype leads to a devaluation of the grain on the market. For that reason, Brazilian common bean breeding programs usually prefer crosses among elite carioca cultivars, due to the additive effects of quantitative traits, obtaining superior advanced bean lines [64,99].

Recent studies have shown that as a result of the limitation of the operational capacity of breeding programs, the right choice of parents for genetic improvement is extremely important [64,69,100]. Some authors have reported the possibility of selecting superior parents based only on genotypic information. However, for parental selection towards anthracnose resistance the best approach is to gather both phenotypic and genotypic information [64]. Therefore, the phenotypic characterization of the CDP for resistance to the main anthracnose races in the state of São Paulo (Brazil) and the genomic information on favorable alleles regarding ANT resistance loci represent a great advance in breeding of carioca common bean.

Association mapping

In the present study, we identified many QRL associated with *C. lindemuthianum* race-specific resistance. Among them, the SNPs ss715648889 (Pv01) and ss715647887 (Pv02) were significant only for the most pathogenic race (race 321), whereas ss715639578 (Pv05) and ss715647427 (Pv08) for the less pathogenic races 65 and 81, respectively. SNP ss715646017 (Pv07) also showed significance only for race 81; however, considering the mixture of races, it was also significant. Recently, Mungalu et al. [21] confirmed the race-specific resistance pattern, identifying a total of 14 QRL with nine different ANT races. Yet only two QRL were significant for all the races tested.

The identification of loci/markers associated with multiple races of any pathogen is extremely relevant within the aim of increasing ANT resistance in bean breeding programs.

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Fig 5. Discriminant analysis of the principal components (DAPC) carried out using the simple nucleotide polymorphisms (SNPs) associated with resistance to anthracnose classified by the resistance allele. (a) Complot showing clustering performed only with the 52 accessions considered resistant to all races (i.e., score ≤ 3), both groups being formed based on the frequency of resistance and susceptibility alleles, in green and red, respectively. (b) Graphical representation of the separation of both groups, showing the absence of overlapping for discriminant analysis.

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Such loci/markers can be used in marker-assisted selection (MAS) in an efficient way. From this perspective, the SNPs of greatest importance found in our study were ss715649427 and ss715642306 on the Pv04 chromosome, at 0.16 Mbp from each other and associated with the three of the races evaluated. We found 14 R genes and 2 encoding genes for protein kinases in the same region and within an interval of less than 0.6 Mbp (Fig 4). In addition to the candidate genes, a total of 14 markers have been associated with resistance loci in the same range as other studies, both by linkage mapping and GWAS [20,51,52,61,101]. In the same region, the Co-3 locus was identified by several authors, as well as its allelic series Co-3^2, Co-3^3, Co-3^4, and Co-3 [28,37,40,102,103]. However, among all the markers reported as associated with ANT resistance at the beginning of the Pv04 chromosome, the SNP ss715642306 showed greatest potential for use as a tool in MAS.

The same SNP ss715642306 that we identified as associated with the races evaluated was also mapped by Zuiderveen et al. [51] for race 7 in an Andean accession panel; by Costa et al. [101] for one of the five isolates characterized as race 65 on the Mesoamerican diversity panel; and by Valentini et al. [62] in a fine-mapping approach linked to the Ur-14, Co-3^4, and Phg-3 locus, responsible for conferring resistance to rust, ANT (race 73), and angular leaf spot, respectively. The annotation procedure demonstrated that this SNP is within the exon of the Phvul.004G005800 gene that encodes the Cytochrome P450 protein, which is known to play an important role in the catalysis of redox reactions [104]. For its part, P450 can trigger a plant hypersensitive disease reaction by oxidative degradation [51,101].

Considering the QRL associated with more than one race (races 81 and 321), a second locus on Pv08 composed of three different significant SNPs (ss715646102, ss715646115, and ss715646756) showed an interval of 0.69 Mbp. In the same region (i.e., between the 59.1 Mbp and 60.5 Mbp positions), there are six genes involved in disease resistance mechanisms, with three genes encoding NB-LRR (nucleotide-binding, leucine-rich repeat, R genes) and three encoding kinase proteins (Fig 4). The NBS-LRR sequences represent a crucial resource for combating pathogen attack [105]. Perseguini et al. [53] also reported a marker at the 56.7 Mbp position significant for race 7. Oblessuc et al. [56] identified a marker (PF5330, 61.2 Mbp position) in the same region linked to the ANT08.3 loci conferring resistance to race 38. The resistance locus linked to angular leaf spot (Phg-2), one of the five loci accepted by the Bean Improvement Cooperative Genetics Committee and the second identified in the Mesoamerican genotype [106], was also observed in the same genomic region [107].

Another QRL associated with more than one race (races 81 and 321) was identified at the end of chromosome Pv01 by the SNPs ss715645299 and ss715645285. In the same region on Pv01, between the 48.3 Mbp and 50.5 Mbp positions, is a large cluster of protein kinase-encoding genes that are involved in plant resistance mechanisms [89]. In the same interval, 15 markers were associated with ANT resistance in nine different studies [41,44,47,48,51,53,59,63,108]. In this region, several loci of greater effect have been reported, such as Co-1, Co-x, Co-w, Co-14, Co-Pa, Co-AC, and CoPv01CDRK [89]. However, previous studies identified this resistance locus in Andean genotypes, while our study was the first to find it using an exclusively Mesoamerican diversity panel. The results reinforce the hypothesis that the different loci reported are a single large locus composed of a cluster of resistance genes and, depending on the race or genotypes used, different genes with large effects from the same cluster are activated, enabling the identification of different loci in the same genomic region.

Linkage mapping

Unlike the PDC, the AM population is a set with resistance loci of Andean origin, since the AND-277 cultivar resistant to ANT was used as a donor parent to obtain the population. In a
previous study, the cultivar AND-277 showed resistance to races 64, 65, 73, 81, 87, 89, 453, and 2047. In addition, it carries an allele of the Co-1 locus designated Co-1\(^4\) [109]. Furthermore, our results confirmed that AND-277 is resistant to races 65 and 81 and that the cultivar is also resistant to race 321. Gonçalves-Vidigal et al. [63] mapped a single and dominant locus on Pv01 in cultivar AND-277 flanked by the CV542014450 and TGA 1.1570 markers, both associated with races 73 and 2047. The same Co-1 locus was identified in the present study for race 81 also flanked by the marker TGA1.1570. However, the locus was not significant for race 65, demonstrating that the locus is probably related to more pathogenic ANT races (e.g., 73, 81, and 2047).

For race 65, three QRLs were considered significant, one on each of the Pv03, Pv10, and Pv11 chromosomes. The QRL ANT3.1\(^{AM}\) on Pv03 was in the reference genome v2.1 between the 18.14 Mbp and 22.93 Mbp physical positions. On the same chromosome, two loci of greater effects have been reported in previous studies. The first Co-13 locus was identified by Lacanallo et al. [58] in the Andean landrace Jalo de Listras Pretas, linked to the marker OV20680 followed by the second Co-17 locus, identified by Trabanco et al. [33] in the Mesoamerican cultivar SEL1308, linked to the marker NDSU_IND_3_0.0441. In the same region, Peseguini et al. [53] identified the IAC-167 marker at the 13.43 Mbp position, associated with both ANT and angular leaf spot disease. Vaz Bisneta et al. [110] also identified SNP S03_13038972 at the 13.38 Mbp position, associated with the ANT 1545 race.

The ANT10.1\(^{AM}\) QRL was mapped between the 33.58–37.64 cM positions on the linkage map, and 41.46–44.18 Mbp on the physical map (reference genome v2.1). Mungalu et al. [21] mapped the ANT10.1\(^{AS}\) QRL significant for ANT race 1331 in a nearby position, between the 39.2–40.6 cM positions on the AS map (Solwezi × AO-1012-29-3-3). The SNP ss715648593 was the closest marker to ANT10.1\(^{AS}\) and was at 1.14 Mbp from Marker908 linked to ANT10.1\(^{AM}\). The presence of both QRL at the same position corroborates the presence of a single Andean locus of lesser effect at the end of the Pv10 chromosome. On Pv11, we identified the QRL ANT11.1\(^{AM}\) at the 8.98 cM position, a region in which the Co-2 locus in the Cornell cultivar was also mapped, by Geffroy et al. [29]. Rodriguez-Suárez et al. [111] identified another Co-2\(^{A252}\) locus in the A-252 cultivar and, more recently, Campa et al. [55] identified the Co-2\(^{AB136}\) locus on the AB-136 genotype. Several other studies involving GWAS have also identified markers associated with ANT resistance on Pv11 [51–53,89].

Common bean breeding for anthracnose resistance

Common bean has two main centers of origin [5], and in the case of some diseases such as angular leaf spot, due to the process of co-evolution, the pathogens have the same classification and may show a higher degree of pathogenicity to a specific gene pool [112,113]. In the case of ANT, it is common to identify some QRL specific to a certain gene pool. Co-1 is an example of a locus mapped exclusively in Andean cultivars, having been identified in Michigan Dark Red Kidney (Co-1), Kaboon (Co-1\(^2\)), Perry Marrow (Co-1\(^3\)), AND-277 (Co-1\(^4\)), Widusa (Co-1\(^5\)), Xana (Co-1\(^6\)), Pitanga (Co-14), Jalo EEP 558 (Co-x), Hongyundou (Co-1\(^{ADD}\)), Amendoin Cavalo (Co-AC), California Dark Red Kidney (CoPv01\(^{CDRK}\)), and Paloma (Co-Pa) [89]. Our results using the AM population validated the presence of Co-1\(^4\) in the Andean AND-277 cultivar; however, a locus in the same position showed a significant association with the resistance of the carioca panel.

In both cases, the loci were not significant for race 65, clearly showing that it is a single QRL for both sets. The identification of the resistance locus of Andean origin in the Mesoamerican group corroborates the results recently presented by Almeida et al. [14]. The authors identified a number of SNPs with 100% contrasting alleles between the Andean and Mesoamerican
genotypes, which enabled accurate identification of Andean allelic introgression events in Mesoamerican cultivars. They showed that the SNPs with the most prevalent Andean allele in the Mesoamerican group were close to the main loci associated with disease resistance, confirming the use of Andean genotypes as a source of resistance for the genetic improvement of new Mesoamerican cultivars. In the study, among the four SNPs found on Pv01 with the highest frequency of the Andean allele in the Mesoamerican cultivars, three were exactly at the position of the locus that we have identified in both the AM population and the CDP (i.e., position at 48.5 Mbp).

Our results show that although Andean accessions are extremely important for genetic improvement aiming to gain ANT resistance, it is possible to select elite cultivars as sources of resistance for obtaining new inbred carioca lines, without the need to face the challenge of using parents from another gene pool and/or commercial class. In this sense, the commercial cultivars IPR-Curió, Carioca-MG, IAC-Aytá, IAC-Apuá, IAC-Aysó, IAC-Imperador, IAC-Pyatá, IAC-Formoso, IAC-Ybaté and IAC-Tybatá showed the greatest potential for use by Brazilian common bean breeding programs. These cultivars not only fulfilled the minimum requirements of any commercial cultivar [114–116], but also showed high resistance (i.e., score < 3) to the three races evaluated. Molecular evaluation by DAPC, estimated with the significant SNPs from GWAS, showed that these cultivars have the highest number of favorable alleles among the 18 genotypes grouped in terms of resistance pattern (Fig 5). Among them, the cultivar IAC-Pyatá also showed high resistance to angular leaf spot in a recent study [69] and had the highest average yield among the 11 other commercial cultivars tested by Pompeu [116].

In addition, with recommendation of the cultivars, the markers associated with the resistance loci have considerable potential for screening germplasm and MAS. Recently, Paulino et al. [117], using the resistant cultivar IAC-Formoso, showed the efficiency of marker-assisted backcrossing. After two cycles of backcrosses with selection carried out by nine markers associated with QRL associated with ANT resistance by Oblessuc et al. [56], the authors obtained isogenic advanced lines with superiority to the recurrent parent BRS-Pérola in terms of ANT resistance and fusarium wilt and also with greater yield and tolerance to seed coat darkening.

**Supporting information**

S1 Table. Details of the 125 carioca cultivars: Name, grain size (mm), commercial classification, institution of origin, adjusted mean (BLUE) of the resistance evaluation to the anthracnose (physiological race of ANT 65, 81, and 321), genealogy, and genotypic matrix. (XLSX)

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**Author Contributions**

**Conceptualization:** Caléo Panhoca de Almeida.

**Data curation:** Caléo Panhoca de Almeida, Jean Fausto de Carvalho Paulino, Gabriel de Moraes Cunha Gonçalves, Roberto Fritsche-Neto, Sérgio Augusto Morais Carbonell, Alisson Fernando Chiorato, Luciana Lasry Benchimol-Reis.
Formal analysis: Calêo Panhoca de Almeida, Jean Fausto de Carvalho Paulino, Caio Cesar Ferrari Barbosa, Gabriel de Moraes Cunha Gonçalves, Roberto Fritsche-Neto, Luciana Lasry Benchimol-Reis.

Funding acquisition: Luciana Lasry Benchimol-Reis.

Investigation: Gabriel de Moraes Cunha Gonçalves.

Methodology: Calêo Panhoca de Almeida, Jean Fausto de Carvalho Paulino, Caio Cesar Ferrari Barbosa, Gabriel de Moraes Cunha Gonçalves, Luciana Lasry Benchimol-Reis.

Project administration: Luciana Lasry Benchimol-Reis.

Supervision: Sérgio Augusto Morais Carbonell, Alisson Fernando Chiorato, Luciana Lasry Benchimol-Reis.

Validation: Jean Fausto de Carvalho Paulino, Sérgio Augusto Morais Carbonell, Alisson Fernando Chiorato, Luciana Lasry Benchimol-Reis.

Visualization: Jean Fausto de Carvalho Paulino, Roberto Fritsche-Neto, Sérgio Augusto Morais Carbonell, Alisson Fernando Chiorato, Luciana Lasry Benchimol-Reis.

Writing – original draft: Calêo Panhoca de Almeida.

Writing – review & editing: Jean Fausto de Carvalho Paulino, Caio Cesar Ferrari Barbosa, Gabriel de Moraes Cunha Gonçalves, Roberto Fritsche-Neto, Sérgio Augusto Morais Carbonell, Alisson Fernando Chiorato, Luciana Lasry Benchimol-Reis.

References

1. Bellucci E, Bitocchi E, Rau D, Rodriguez M, Biagetti E, Giardini A, et al. Genomics of origin, domestication and evolution of Phaseolus vulgaris. In: Tuberosa R., Graner A. FE, editor. Genomics of Plant Genetic Resources: 1st ed. Dordrecht: Springer Netherlands; 2014. p. 483–507.
2. Bitocchi E, Rau D, Bellucci E, Rodriguez M, Murgia ML, Gioia T, et al. Beans (Phaseolus ssp.) as a model for understanding crop evolution. Front Plant Sci. 2017 May 8;8. https://doi.org/10.3389/fpls.2017.00722 PMID: 28533769
3. Broughton WJ, Hernández G, Blair M, Beebe S, Gepts P, Vanderleyden J. Beans (Phaseolus ssp.)—Model food legumes. Plant Soil. 2003; 252(1):55–128.
4. Bitocchi E, Nanni L, Bellucci E, Rossi M, Giardini A, Zeuli PS, et al. Mesoamerican origin of the common bean (Phaseolus vulgaris L.) is revealed by sequence data. Proc Natl Acad Sci U S A [Internet]. 2012 [cited 2020 Jul 2]; 109(14):E788–96. https://doi.org/10.1073/pnas.1108973109 PMID: 22393017
5. Bitocchi E, Bellucci E, Giardini A, Rau D, Rodriguez M, Biagetti E, et al. Molecular analysis of the parallel domestication of the common bean (Phaseolus vulgaris) in Mesoamerica and the Andes. New Phytol. 2013 Jan; 197(1):300–13. https://doi.org/10.1111/j.1469-8137.2012.04377.x PMID: 23126863
6. Gepts P, Bliss FA. Dissemination pathways of common bean (Phaseolus vulgaris, Fabaceae) deduced from phaseolin electrophoretic variability. II. Europe and Africa. Econ Bot. 1988 Jan; 42 (1):86–104.
7. Koinange EMK, Gepts P. Hybrid weakness in wild Phaseolus vulgaris L. J Hered [Internet]. 1982 [cited 2020 Jul 2]; 83(2):135–9.
8. Schmutz J, McClean PE, Mamidi S, Wu GA, Cannon SB, Grimwood J, et al. A reference genome for common bean and genome-wide analysis of dual domestications. Nat Genet. 2014; 46(7):707–13. https://doi.org/10.1038/ng.3008 PMID: 24908249
9. FAOSTAT. Food and agriculture organization of the united nations (FAO) [Internet]. Statistical database, Food and Agriculture. 2021 [cited 2021 Jan 12].
10. CONAB. Acompanhamento da safra brasileira de grãos: Safra 2020/21—Quarto levantamento [Internet]. Companhia Nacional de Abastecimento. 2021 [cited 2020 Jan 12].
11. Silva FC, Melo PGS, Pereira HS, Melo LC. Genetic control and estimation of genetic parameters for seed-coat darkening of carioca beans. Genet Mol Res. 2014 Aug 25; 13(3):6486–96. https://doi.org/10.4238/2014.August.25.12 PMID: 25158267

12. Almeida LD de Leitão Filho HF, Miyasaka S. Características do feijão Carioca, um novo cultivar. Bragantia. 1971; 30(1).

13. Chiorato AF, Carbonell SAM. O Melhoramento Genético do Feijoeiro no Instituto Agronômico IAC (1932 a 2014). Vol. 64. Campinas: O AGRONÔMICO; 2014. 6–13 p.

14. Almeida CP, de Carvalho Paulino JF, Carbonell SAM, Chiorato AF, Song Q, Di Vittori V. et al. Genetic diversity, population structure, and andean introgression in Brazilian common bean cultivars after half a century of genetic breeding. Genes (Basel) [Internet]. 2020 Oct 30 [cited 2020 Nov 11]; 11(11):1–22. https://doi.org/10.3390/genes11111298 PMID: 33143347

15. Padder BA, Sharma PN, Awale HE, Kelly JD. *Colletotrichum lindemuthianum*, the causal agent of bean anthracnose. J Plant Pathol. 2017; 99(2):317–30.

16. Pastor-Corrella MA. Estandarización de variedades diferenciales y de designación de razas de *Colletotrichum lindemuthianum*. Phytopathology. 1991; 81:694.

17. Ribeiro T, Esteves JA de F, Silva DA, Gonçalves JGR, Carbonell SAM, Chiorato AF. Classification of *Colletotrichum lindemuthianum* races in differential cultivars of common bean. Acta Sci—Agron. 2016; 38(2):179–84.

18. Coêlho M, Gonçalves Vidigal MC, Vidigal Filho PS, Franzon RC, Martins VSR. Genetic diversity of *Colletotrichum lindemuthianum* races based on ITS-rDNA regions. Agron Sci Biotechnol [Internet]. 2020 Nov 27 [cited 2020 Dec 16]; 6:1–18.

19. Dillard HR, Cobb AC. Survival of *Colletotrichum lindemuthianum* in bean debris in New York State. Plant Dis. 1993; 77(12):1233–8.

20. Vidigal Filho PS, Gonçalves-Vidigal MC, Bisneta MV, Souza VB, Gilo TAS, Calvi AA, et al. Genome-wide association study of resistance to the anthracnose and angular leaf spot diseases in Brazilian Mesoamerican and Andean common bean cultivars. Crop Sci. 2020 Aug 18; 12:440–7.

21. Mungalu H, Sansala M, Hamabwe S, Mukuma C, Gepts P, Kelly JD. et al. Identification of race-specific quantitative trait loci for resistance to *Colletotrichum lindemuthianum* in an Andean population of common bean. Crop Sci. 2020.

22. Banoo A, Nabi A, Rasool RS, Mahiya-Farooq, Shah MD, Ahmad M, et al. North-Western Himalayan Common Beans: Population Structure and Mapping of Quantitative Anthracnose Resistance Through Genome Wide Association Study. Front Plant Sci [Internet]. 2020 Oct 6 [cited 2020 Dec 16]; 11:1. https://doi.org/10.3389/fpls.2020.00001 PMID: 32117356

23. Mastenbroek C. A breeding programme for resistance to anthracnose in dry shell haricot beans, based on a new gene. Euphytica. 1960; 9(2):177–84.

24. Bannerot H, Derieux M, Fouilloux G. Mise en évidence d’un second gène de résistance totale à l’anthracnose chez le haricot. Ann Amélior Plantes [Internet]. 1971 [cited 2020 Sep 25]; 21:83–5.

25. Young RA, Elly JDK. RAPD markers flanking the are gene for anthracnose resistance in common bean. J Am Soc Hortic Sci. 1996; 121(1):37–41.

26. Gonçalves-Vidigal MC, Cardoso AA, Vieira C, Saraiva LS. Inheritance of anthracnose resistance in common bean genotypes P.I. 207262 and AB 136. Brazilian J Genet. 1997; 20(1):59–62.

27. Gonçalves-Vidigal MC, Silva CR da, Vidigal Filho PS, Franzon RC, Martins VSR. Genetic diversity of anthracnose resistance gene cluster B4 in common bean. Euphytica [Internet]. 2014; 197(3):407–15. https://doi.org/10.1007/s00122-007-0678-y PMID: 18060540

28. Young RA, Melotto M, Nodari RO, Kelly JD. Marker-assisted dissection of the oligogenic anthracnose resistance in the common bean cultivar, “G 2333.” Theor Appl Genet. 1998; 96(1):87–94.

29. Geoffroy V, Cresusot F, Faquiot J, Sévignac M, Adam-Blondon AF, Bannerot H, et al. A family of LRR sequences in the vicinity of the Co-2 locus for anthracnose resistance in *Phaseolus vulgaris* and its potential use in marker-assisted selection. Theor Appl Genet. 1998; 96(3–4):494–502. https://doi.org/10.1007/s001220050766 PMID: 24710881

30. Geoffroy V, Sévignac M, Billant P, Dron M, Langin T. Resistance to *Colletotrichum lindemuthianum* in *Phaseolus vulgaris*: A case study for mapping two independent genes. Theor Appl Genet. 2008; 116(3):407–15. https://doi.org/10.1007/s00122-007-0678-y PMID: 18060540

31. Kelly JD, Vallejo VA. A Comprehensive Review of the Major Genes Conditioning Resistance to Anthracnose in Common Bean. HORTSCIENCE. 2004; 39(6).

32. Méndez-Vigo B, Rodríguez-Suárez C, Parieda A, Ferreira JJ, Giraldez R. Molecular markers and allelic relationships of anthracnose resistance gene cluster B4 in common bean. Euphytica [Internet]. 2005 Jan [cited 2020 Sep 25]; 141(3):237–45.
33. Trabanco N, Campa A, Ferreira JJ. Identification of a New Chromosomal Region Involved in the Genetic Control of Resistance to Anthracnose in Common Bean. Plant Genome [Internet]. 2015 Jul [cited 2020 Sep 25]; 8(2):plantgenome2014.10.0079. https://doi.org/10.3835/plantgenome2014.10.0079 PMID: 33228300

34. Coimbra-Gonçalves GK, Gonçalves-Vidigal MC, Coelho RT, Valentini G, Filho PSV, Lacanallo GF, et al. Characterization and mapping of anthracnose resistance gene in mesoamerican common bean cultivar Crioulo 159. Crop Sci. 2016; 56(6):2904–15.

35. Meziadi C, Richard MMS, Derquennes A, Thareau V, Blanchet S, Gratias A, et al. Development of molecular markers linked to disease resistance genes in common bean based on whole genome sequence. Plant Sci [Internet]. 2016 [cited 2020 Jul 22]; 242:351–7. https://doi.org/10.1016/j.plantsci.2015.09.006 PMID: 26566651

36. Cardenas F, Adams MW, Andersen A. The genetic system for reaction of field beans (Phaseolus vulgaris L.) to infection by three physiologic races of Colletotrichum lindemuthianum. Euphytica. 1964 Jul; 13(2):178–86.

37. Geoffroy V, Sicard D, De Oliveira JCF, Sévignac M, Cohen S, Gepts P, et al. Identification of an ancestral resistance gene cluster involved in the coevolution process between Phaseolus vulgaris and its fungal pathogen Colletotrichum lindemuthianum. Mol Plant-Microbe Interact. 1999; 12(9):774–84. https://doi.org/10.1094/MPMI.1999.12.9.774 PMID: 10494630

38. Melotto M, Kelly JD. An allelic series at the Co-1 locus conditioning resistance to anthracnose in common bean of Andean origin. Euphytica. 2000; 116(2):143–9.

39. Gonçalves-Vidigal MC, Kelly JD. Inheritance of anthracnose resistance in the common bean cultivar Widusa. Euphytica. 2006 Sep; 151(3):411–9.

40. Gonçalves-Vidigal MC, Cruz AS, Lacanallo GF, Vidigal Filho PS, Sousa LL, Pacheco CMNA, et al. Co-segregation analysis and mapping of the anthracnose Co-10 and angular leaf spot Phg-ON disease-resistance genes in the common bean cultivar Ouro Negro. Theor Appl Genet. 2013 Sep [cited 2020 Jul 2]; 126(9):2245–55. https://doi.org/10.1007/s00122-013-2131-8 PMID: 23760652

41. Gonçalves-Vidigal MC, Lacanallo GF, Vidigal Filho PS. A new gene conferring resistance to anthracnose in Andean common bean (Phaseolus vulgaris L.) cultivar “Jalo Vermelho.” Plant Breed. 2008 Dec; 127(6):592–6.

42. Gonçalves-Vidigal MC, Medeiros AF, Pastor-Corrales MA. Common bean landrace Jalo Listras Pretas is the source of a New Andean anthracnose resistance gene. Crop Sci. 2009 Jan; 49(1):133–9.

43. Gonçalves-Vidigal MC, Meirelles AC, Poletine JP, De Sousa LL, Cruz AS, Nunes MP, et al. Genetic analysis of anthracnose resistance in “Pitanga” dry bean cultivar. Plant Breed. 2012 Jun; 131(3):423–9.

44. Richard MMS, Pflieger S, Sévignac M, Thareau V, Blanchet S, Li Y, et al. Fine mapping of Co-x, an anthracnose resistance gene to a highly virulent strain of Colletotrichum lindemuthianum in common bean. Theor Appl Genet. 2014 May 25; 127(7):1653–66. https://doi.org/10.1007/s00122-014-2328-5 PMID: 24859268

45. Sousa LL, Gonçalves AO, Gonçalves-Vidigal MC, Lacanallo GF, Fernandez AC, Awale H, et al. Genetic characterization and mapping of anthracnose resistance gene in common bean landrace cultivar corinthiano. Crop Sci. 2015 Sep 1; 55(5):1900–10.

46. Gilio TAS, Hurtado-Gonzales OP, Valentini G, Castro SAL, Elias HT, Song QMC, et al. Fine mapping the broad spectrum anthracnose resistance gene in Amendoim Cavalo. Annu Rep Bean Improv Coop [Internet]. 2017.

47. Chen M, Wu J, Wang L, Mantri N, Zhang X, Zhu Z, et al. Mapping and genetic structure analysis of the anthracnose resistance locus Co-1HY in the common bean (Phaseolus vulgaris L.). PLoS One. 2017 Jan 1; 12(1).

48. de Lima Castro SA, Gonçalves-Vidigal MC, Gilio TAS, Lacanallo GF, Valentini G, da Silva Ramos Martins V, et al. Genetics and mapping of a new anthracnose resistance locus in Andean common bean Paloma. BMC Genomics. 2017 Apr 18; 18(1). https://doi.org/10.1186/s12864-017-3685-7 PMID: 28420340

49. Gonçalves-Vidigal MC, Gilio TAS, Valentini G, Vaz-Bisneta M, Vidigal Filho PS, Song Q, et al. New Andean source of resistance to anthracnose and angular leaf spot: Fine-mapping of disease-resistance genes in California Dark Red Kidney common bean cultivar. Mir RR, editor. PLoS One [Internet]. 2020 Jun 29 [cited 2020 Jul 6]; 15(6):e0235215. https://doi.org/10.1371/journal.pone.0235215 PMID: 32598372

50. López CE, Acosta IF, Jara C, Pedraza F, Gaitán-Solís E, Gallego G, et al. Identifying resistance gene analogs associated with resistances to different pathogens in common bean. Phytopathology. 2003 Jan; 93(1):88–95. https://doi.org/10.1094/PHYTO.2003.93.1.88 PMID: 18944161
51. Zuiderveen GH, Padder BA, Kamfwa K, Song Q, Kelly JD. Genome-Wide association study of anthracnose resistance in andean beans (Phaseolus vulgaris). PLoS One. 2016 Jun 1; 11(6). https://doi.org/10.1371/journal.pone.0156391 PMID: 27270627

52. Wu J, Zhu J, Wang L, Wang S. Genome-wide association study identifies NBS-LRR-encoding genes related with anthracnose and common bacterial blight in the common bean. Front Plant Sci. 2017 Aug 9; 8. https://doi.org/10.3389/fpls.2017.01398 PMID: 28848959

53. Perseguini JMKC, Oblessuc PR, Rosa JRBF, Gomes KA, Chiorato AF, Carbonell SAM, et al. Genome-Wide Association Studies of Anthracnose and Angular Leaf Spot Resistance in Common Bean (Phaseolus vulgaris L.). PLoS One. 2016 Mar 1; 11(3). https://doi.org/10.1371/journal.pone.0150506 PMID: 26930078

54. Fritsche-Neto R, De Souza TLPO, Pereira HS, De Faria LC, Melo LC, Novaes E, et al. Association mapping in common bean revealed regions associated with anthracnose and angular leaf spot resistance. Sci Agric. 2019; 76(4):321–7.

55. Campa A, Trabanco N, Ferreira JJ. Identification of clusters that condition resistance to anthracnose in the common bean differential cultivars AB136 and MDRK. Phytopathology. 2017 Dec 1; 107 (12):1515–21. https://doi.org/10.1094/PHYTO-01-17-0012-R PMID: 28742459

56. Oblessuc PR, Baroni RM, da Silva Pereira G, Chiorato AF, Carbonell SAM, Brinex B, et al. Quantitative analysis of race-specific resistance to Colletotrichum lindemuthianum in common bean. Mol Breeds [Internet]. 2014 Jun 26 [cited 2020 Jul 3]; 34(3):1313–29.

57. Nanami DSY, Vidigal MCG, Castro SA de L, Frias AAT, Vidigal Filho PS, Elias HT. Characterization of genetic resistance in Andean common bean cultivar Amendoim Cavalo to Colletotrichum lindemuthianum. Agron Sci Biotechnol. 2017 Jun 8; 3(1):43.

58. Lacanallo GF, Gonçalves-Vidigal MC. Mapping of an andean gene for anthracnose resistance (Co-13) in common bean (Phaseolus vulgaris L.) Jalo Listras Pretas landrace. Aust J Crop Sci. 2015; 9 (5):394–400.

59. Murube E, Campa A, Ferreira JJ. Integrating genetic and physical positions of the anthracnose resistance genes described in bean chromosomes Pv01 and Pv04. PLoS One. 2019 Feb 1; 14(2). https://doi.org/10.1371/journal.pone.0221298 PMID: 30763410

60. Vallejo V, Kelly JD. New Insights into the Anthracnose Resistance of Common Bean Landrace G 2333. Open Hortic J. 2009 Apr 2; 2(1):29–33.

61. Rodríguez-Suárez C, Ferreira JJ, Campa A, Pañeda A, Giraldez R. Molecular mapping and intra-cluster recombination between anthracnose race-specific genes in the common bean differential cultivars Mexico 222 and Widusa. Theor Appl Genet. 2008 Apr; 116(6):807–14. https://doi.org/10.1007/s00122-008-0714-6 PMID: 18210079

62. Valentini G, . . . MG-V-T and A, 2017 U. High-resolution mapping reveals linkage between genes in common bean cultivar Ouro Negro conferring resistance to the rust, anthracnose, and angular leaf spot. Theor Appl Genet [Internet]. 2017.

63. Gonçalves-Vidigal MC, Cruz AS, Garcia A, Kami J, Filho PSV, Sousa LL, et al. Linkage mapping of the Phg-1 and Co-14 genes for resistance to angular leaf spot and anthracnose in the common bean cultivar AND 277. Theor Appl Genet. 2011; 122(5):893–903. https://doi.org/10.1007/s00122-010-1496-1 PMID: 21113774

64. Pereira HS, Mota APS, Rodrigues LA, de Souza TLPO, Melo LC. Genetic diversity among common bean cultivars based on agronomic traits and molecular markers and application to recommendation of parent lines. Euphytica [Internet]. 2019 Jan 1 [cited 2020 Jul 2]; 215(2):1–16.

65. Perseguini JMKC, Silva GMB, Rosa JRBF, Gazaffi R, Marçal JF, Carbonell SAM, et al. Developing a common bean core collection suitable for association mapping studies. Genet Mol Biol [Internet]. 2015 Jan 1 [cited 2020 Jul 9]; 38(1):67–78. https://doi.org/10.1590/S0100-83892015000100007

66. Davis DW, Frazier WA. The incidense of three abnormalities in F2 progeny of crosses between true bush beans and Blue Lake derived bush snap beans. Annual Report of the Bean Improvement Cooperative. 1964; 7:14–6.

67. Almeida CP, Paulino JF de C, Santos IL, Bajay MM, Gonçalves JGR, Carvalho CRL, et al. Marker-assisted backcrossing for disease resistance and agronomic traits in carioca beans. Crop Sci. 2021; accepted m.

68. Song Q, Jia G, Hyten DL, Jenkins J, Hwang EY, Schroeder SG, et al. SNP assay development for linkage map construction, anchoring whole-genome sequence, and other genetic and genomic applications in common bean. G3 Genes, Genomes, Genet [Internet]. 2015.

69. Almeida CP de, Arruda N, Paulino JF de C, Freitas GM de, Bonfant GFJ, Bajay MM, et al. Genetic diversity of Pseudocercospora griseola resistance loci in common beans. Trop Plant Pathol [Internet]. 2020 Sep 3 [cited 2020 Sep 8];1–10.
70. Almeida CP, Paulino JFC, Bonfante GFJ, Perseguini JMGC, Santos, González JGR, et al. Angular Leaf Spot Resistance Loci Associated with Different Plant Growth Stages in Common Bean. Front Plant Sci. 2021; 12:650. https://doi.org/10.3389/fpls.2021.647043 PMID: 33927738

71. Bradbury PJ, Zhang Z, Kroon DE, Casstevens TM, Ramdoss Y, Buckler ES. TASSEL: Software for association mapping of complex traits in diverse samples. Bioinformatics [Internet]. 2007 [cited 2020 Jul 9]; 23(19):2633–5. https://doi.org/10.1093/bioinformatics/btm308 PMID: 17586829

72. Browning BL, Zhou Y, Browning SR. A One-Penny Imputed Genome from Next-Generation Reference Panels. Am J Hum Genet. 2018 Sep 6; 103(3):338–48. https://doi.org/10.1016/j.ajhg.2018.07.015 PMID: 30100085

73. González AM, Yuste-Lisbona FJ, Paula Rodíguez A, De Ron AM, Capel C, García-Alcázar M, et al. Uncovering the genetic architecture of Colletotrichum lindemuthianum resistance through QTL mapping and epistatic interaction analysis in common bean. Front Plant Sci. 2015 Mar 17; 6:141. https://doi.org/10.3389/fpls.2015.00141 PMID: 25852706

74. Carbonell SAM, Chiorato AF, Bezerra LMC, González JGR, Silva DA da, Esteves JA de F, et al. IAC 1850: High yielding carioca common bean cultivar. Crop Breed Appl Biotechnol [Internet]. 2019 Sep [cited 2020 Nov 12]; 19(3):378–81.

75. Ferreira EB, Cavalcanti PP, Nogueira DA. ExpDes: An R Package for ANOVA and Experimental Designs. Appl Math [Internet]. 2014 [cited 2020 Sep 22]; 05(19):2952–8.

76. Bhering LL. Rbio: A tool for biometrical and statistical analysis using the R platform. Crop Breed Appl Biotechnol [Internet]. 2017 [cited 2020 Jul 3]; 17(2):187–90.

77. Matias FL, Granato I, Fritsch-Neto R. Be-Breeder: an R/Shiny application for phenotypic data analyses in plant breeding. Crop Breed Appl Biotechnol [Internet]. 2018 Apr [cited 2020 Nov 12]; 18(2):241–3.

78. Liu X, Huang M, Fan B, Buckler ES, Zhang Z. Iterative Usage of Fixed and Random Effect Models for Powerful and Efficient Genome-Wide Association Studies. PLoS Genet [Internet]. 2016 [cited 2020 Jul 9];12(2). https://doi.org/10.1371/journal.pgen.1005767 PMID: 26828793

79. Lipka AE, Tian F, Wang Q, Peiffer J, Li M, Bradbury PJ, et al. GAPIT: Genome association and prediction integrated tool. Bioinformatics [Internet]. 2012 [cited 2020 Jul 9]; 28(18):2397–9. https://doi.org/10.1093/bioinformatics/bts444 PMID: 22796960

80. Bonferroni C. Teoria statistica delle classi e calcolo delle probabilità. Pubbl del R Ist Super di Sci Econ e Commer di Firenze. 1963; 8:3–62.

81. Margarido GRA, Souza AP, Garcia AAF. OneMap: Software for genetic mapping in outcrossing species. Hereditas. 2007 Jul; 144(3):78–9. https://doi.org/10.1111/j.2007.0018-0661.02000.x PMID: 17663699

82. Zeng ZB. Precision mapping of quantitative trait loci. Genetics. 1994; 136(4):1457–68. PMID: 8013918

83. Churchill GA, Doerge RW. Empirical threshold values for quantitative trait mapping. Genetics [Internet]. 1994 [cited 2020 Jul 9]; 138(3):963–71. PMID: 7851788

84. Kao CH, Zeng ZB, Teasdale RD. Multiple interval mapping for quantitative trait loci. Genetics. 1999; 152(3):1203–16. PMID: 1038834

85. Akaike H. A New Look at the Statistical Model Identification. IEEE Trans Automat Contr [Internet]. 1974 [cited 2020 Jul 10]; 19(6):716–23.

86. Goodstein DM, Shu S, Howson R, Neupane R, Hayes RD, Fazo J, et al. Phytozome: A comparative platform for green plant genomics. Nucleic Acids Res [Internet]. 2012 [cited 2020 Jul 9]; 40(D1). https://doi.org/10.1093/nar/gkr944 PMID: 22110026

87. Jombart T. Devillard S, Bailly F. Discriminant analysis of principal components: A new method for the analysis of genetically structured populations. BMC Genet. 2010 Oct 15;11. https://doi.org/10.1186/1471-2156-11-11 PMID: 20141624

88. Jombart T. Ahmed I, adegenet 1.3–1: New tools for the analysis of genome-wide SNP data. Bioinformatics [Internet]. 2011 [cited 2020 Jul 3]; 27(21):3070–1. https://doi.org/10.1093/bioinformatics/btr521 PMID: 21926124

89. Bisneta MV, González-Vidigal MC. Integration of anthracnose resistance loci and RLK and NBS-LRR-encoding genes in the Phaseolus vulgaris L. genome. Crop Sci [Internet]. 2020 Aug [cited 2020 Sep 25]; 20288.

90. Chiorato AF, Reis LLB, Bezerra LMC, Carbonell SAM. Global vision on common bean breeding cultivars. In: Phaseolus vulgaris: Cultivars, Production and Uses. 2018. p. 27–68.

91. Pereira LA, Costa LC, de Pádua PF, Ramalho MAP. Variability for angular leaf spot and anthracnose resistance among common bean progenies with different levels of endogamy. Trop Plant Pathol. 2019; 44(3):275–83.
92. Ragagnin VA, De Souza TLPO, Sangiardi DA, Arruda KMA, Costa MR, Alzate-Marín AL, et al. Development and agronomic performance of common bean lines simultaneously resistant to anthracnose, angular leaf spot and rust. Plant Breed [Internet]. 2009 Apr 1 [cited 2020 Jul 2]; 128(2):156–63.

93. Costa LC, Nalin RS, Ramalho MAP, De Souza EA. Are duplicated genes responsible for anthracnose resistance in common bean? PLoS One [Internet]. 2017 Mar 1 [cited 2020 Nov 27];12(3). https://doi.org/10.1371/journal.pone.0173789 PMID: 28296933

94. Arantes L de O, Ramalho MAP, Abreu Â de FB. Genetic control of incompatibility in crosses of andean and mesoamerican common bean cultivars. Cienc e Agropecuária [Internet]. 2008 May [cited 2020 Jul 3]; 32(3):978–80.

95. Singh SP, Gutiérrez AJ. Geographical distribution of the DL1 and DL2 genes causing hybrid dwarfism in Phaseolus vulgaris L., their association with seed size, and their significance to breeding. Euphytica [Internet]. 1984 Jun [cited 2020 Jul 3]; 33(2):337–45.

96. Vieira A, Ramalho M, Genética JS. Crossing incompatibility in some bean cultivars utilized in Brazil. Rev Bras Genética. 1989; 12:169–71.

97. Johnson WC, Gepts P. The Role of epistasis in controlling seed yield and other agronomic traits in an Andean × Mesoamerican cross of common bean (Phaseolus vulgaris L.). Euphytica. 2002; 125:69–79.

98. Bruzi AT, Ramalho MAP, Abreu Â de FB. Desempenho de famílias do cruzamento entre linhagens de feijões andinos e mesoamericanos em produtividade e resistência a Phaeoisariopsis griseola. Ciência e Agrotecnologia [Internet]. 2007 Jun [cited 2020 Jul 3]; 31(3):650–5.

99. Pereira R, Abreu Â de FB, Nalin RS, de Souza EA. Phenotyping for angular leaf spot severity and its implication in breeding common bean for resistance. Sci Agric. 2019 Sep 1; 76(5):415–23.

100. Filho JMC, Geraldi IO, Barona MAA. Heterose and genetic distances for productivity of common bean. Teor Aplic Genet [Internet]. 2020 Nov 1 [cited 2020 Dec 4]; 91(2):405–19. https://doi.org/10.1534/genetics.108.093583 PMID: 19087965

101. Costa LC, Nalin RS, Dias MA, Ferreira ME, Song Q, Pastor-Corrales MA, et al. Different loci control resistance to different isolates of the same race of Colletotrichum lindemuthianum in common bean. Euphytica. 2020; 198(23):13454–9. https://doi.org/10.1007/s10681-020-03713-x PMID: 3310954

102. Bannérot H. Résultat de l’infection d’une collection de haricots par six races physiologiques d’anthracnose. Ann Amélior Plantes. 1965; 15:201–22.

103. Fouilloux G. New races of bean anthracnose and consequences on our breeding programs. In: International symposium on diseases of tropical food crops [Internet]. 1979 [cited 2020 Dec 4]. p. 221–35.

104. Delledonne M, Zeier J, Marocco A, Lamb C. Signal interactions between nitric oxide and reactive oxygen intermediates in the plant hypersensitive disease resistance response. Proc Natl Acad Sci U S A [Internet]. 2001 [cited 2020 Dec 4]; 98(23):13454–9. https://doi.org/10.1073/pnas.231178298 PMID: 11606758

105. Geoffroy V, Macadré C, David P, Pedrosa-Harand A, Sévignac M, Dauga C, et al. Molecular analysis of a large subtelomeric nucleotide-binding-site-leucine-rich-repeat family in two representative genotypes of the major gene pools of Phaseolus vulgaris. Genetics [Internet]. 2009 [cited 2021 Apr 5]; 181(2):405–19. https://doi.org/10.1534/genetics.108.093583 PMID: 19087965

106. Sartorato A, Nietzsche S, Barros EG, Moreira MA. Inheritance of angular leaf spot resistance and RAPD markers linked to disease resistance gene in common beans. Annu Rep Bean Improv Coop. 1999; 42:21–2.

107. Nay MM, Souza TLPO, Raatz B, Mukankusi CM, Pastor-Corrales MA, Abreu AFB, et al. A review of angular leaf spot resistance in common bean. Crop Sci [Internet]. 2019 [cited 2020 Jul 2]; 59(4):1376–91. https://doi.org/10.2135/cropsci2018.09.0596 PMID: 33343018

108. Gilio TAS, Hurtado-Gonzales OP, Gonçalves-Vidigal MC, Valentini G, Elias JCF, Song Q, et al. Fine mapping of an anthracnose-resistance locus in Andean common bean cultivar Amendoim Cavalo. PLoS One. 2020 Oct 1; 15(10 October). https://doi.org/10.1371/journal.pone.0239763 PMID: 33027258

109. Alzate-Marín AL, Costa MR, Arruda KM, De Barros EG, Moreira MA. Characterization of the anthracnose resistance gene present in Ouro Negro (Honduras 35) common bean cultivar. Euphytica. 2003; 133(2):165–9.

110. Vaz Bisneta M, Vidigal Filho PS, Gonçalves-Vidigal MC, Valentini G, Lima LRL, Martiniano-Souza MC, et al. Association mapping reveals regions on chromosomes Pv03 and Pv05 related to anthracnose resistance in common bean. Annu Rep Bean Improv Coop. 2020; 63:75–6.

111. Rodríguez-Suárez C, Méndez-Vigo B, Pañeda A, Ferreira JJ, Giraldez R. A genetic linkage map of Phaseolus vulgaris L. and localization of genes for specific resistance to six races of anthracnose.
(Colletotrichum lindemuthianum). Theor Appl Genet [Internet]. 2007 Feb [cited 2020 Sep 25]; 114(4):713–22. https://doi.org/10.1007/s00122-006-0471-3 PMID: 17186216

112. Guzman P, Gilbertson RL, Nodari R, Johnson WC, Temple SR, Mandala D, et al. Characterization of variability in the fungus Phaeoisariopsis griseola suggests coevolution with the common bean (Phaseolus vulgaris). Phytopathology. 1995; 85(5):600–7.

113. Wagara IN, Mwang’ombe AW, Kimenju JW, Buruchara RA, Jamnadass R, Majiwa PAO. Genetic diversity of Phaeoisariopsis griseola in Kenya as revealed by AFLP and group-specific primers. J Phytopathol. 2004 Apr; 152(4):235–42.

114. Ramalho MAP, Abreu ADFB, Carneiro JES3. Cultivares. Inf Agropecuário. 2004;(223):21–32.

115. Chiorato AF, Morais Carbonell SA, Limonta Carvalho CR, de Barros VLN, Barbosa Borges WL, Ticeli M, et al. “IAC IMPERADOR”: Early maturity ‘carioca’ bean cultivar. Crop Breed Appl Biotechnol [Internet]. 2012 [cited 2020 Dec 9]; 12(4):297–300.

116. Pompeu AS, IAC-Maravilha, IAC-Unu, IAC-Carioca Pyatã, IAC-Carioca Aruã, IAC-Carioca Akytã e IAC-Bico de Ouro: Novos cultivares de feijoeiro. Bragantia [Internet]. 1997 [cited 2020 Jul 13]; 56(1):79–85.

117. Paulino JF de C, Almeida CP, Santos IL, Gonçalves JGR, Carbonell SAM, Chiorato AF, et al. Combining disease resistance and postharvest quality traits by early marker-assisted backcrossing in carioca beans. Sci Agric. 2021; in press.