



Identification of QTLs and validation of markers associated with *Fusarium* wilt response in the common bean cultivar BRS Esplendor

Angelina Harmyans Ciappina¹ · Rosana Pereira Vianello¹ · Nayana Valéria Costa¹ · Saulo Muniz Martins¹ · Lucas Matias Gomes-Messias¹ · Leonardo Cunha Melo¹ · Thiago Lívio Pessoa Oliveira de Souza¹ · Helton Santos Pereira¹

Received: 9 September 2025 / Accepted: 25 December 2025 / Published online: 27 January 2026
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Abstract *Fusarium* wilt, caused by the fungus *Fusarium oxysporum* f. sp. *phaseoli*, is a soilborne disease that causes severe losses in the common bean (*Phaseolus vulgaris* L.) crop. The most effective control strategy is the use of resistant cultivars; however, few with high resistance are currently available. This study aimed to investigate the genetic control of resistance to *Fusarium oxysporum* f. sp. *phaseoli* and identify molecular markers associated with resistance alleles in the Mesoamerican cultivar BRS Esplendor. A total of 103 progenies from a

cross between BRS Esplendor (resistant) and BRS Horizonte (susceptible) were evaluated in a naturally infested field in Santo Antônio de Goiás, Goiás, Brazil. The experiments were conducted in the winter seasons of 2018 and 2019, and plants were evaluated regarding response to *Fusarium* wilt. F₂ progenies were genotyped using the DArTseq technology, resulting in SNP and SilicoDArT markers. A linkage map was constructed, and genetic mapping was carried out to identify regions of interest. Finally, the markers obtained were validated. Genetic variability was observed for *Fusarium* wilt response, with high estimates of heritability (97%) and of genetic gains (48%). A linkage map was constructed with 2,189 markers, spanning 3,026 cM. Three QTLs associated with *Fusarium* wilt response were identified on chromosome PV08, accounting for 51% to 66% of phenotypic variation. These QTL intervals overlapped, indicating a single stable QTL across the different years. The hydrolysis probe (AN33KKZ) developed from this QTL exhibited 97% selection efficiency, proving to be an effective tool for carrying out marker-assisted selection (MAS).

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s10681-025-03667-5>.

A. H. Ciappina
Centro Universitário Araguaia, Avenida T-10, nº 1047,
Setor Bueno, Goiânia, GO CEP 74223-060, Brazil

R. P. Vianello · N. V. Costa (✉) · L. C. Melo ·
H. S. Pereira
Embrapa Arroz E Feijão, Rodovia GO-462, Km 12,
Fazenda Capivara, Zona Rural, Santo Antônio de Goiás,
GO CEP 75375-000, Brazil
e-mail: nayanavcosta@gmail.com

S. M. Martins
Suzano S.A., Rodovia BR 158, Km 298, Fazenda Barra Do
Moeda, Zona Rural, Três Lagoas, MS CEP 79601-970,
Brazil

L. M. Gomes-Messias · T. L. P. O. de Souza
Limagrains Brasil S.A., Rua Pasteur, nº 463, Bairro Água
Verde, Curitiba, PR CEP 80250-104, Brazil

Keywords *Phaseolus vulgaris* L. · Molecular markers · SNPs · SilicoDArT

Introduction

Common bean (*Phaseolus vulgaris* L.) is a leguminous plant of great economic, nutritional, and social importance. Brazil, with annual production of 2.5 million tons in 2023, is one of the world's largest producers of this crop (Embrapa 2025). However, in areas with Fusarium wilt, a soilborne disease caused by the fungus *Fusarium oxysporum* f. sp. *phaseoli*, crop yield potential is impaired. The greatest losses are observed in areas of high agricultural intensity, particularly in winter crops grown using center-pivot irrigation, which favors the development of the pathogen. The use of resistant cultivars is the most effective and economical control method. Yet, despite the wide genetic variability in common bean, few cultivars with a high level of resistance to Fusarium wilt are available for growing (Pereira et al. 2019, 2020; Rocha et al. 2019).

Few studies have addressed genetic control of response to Fusarium wilt in common bean. Early studies based on heritability and segregation analyzes indicated an oligogenic pattern, with one or few dominant genes conferring resistance depending on the pathogen race and the genetic background of the host (Cândida et al. 2009; Pereira et al. 2009; Batista et al. 2017; Cavalheiro et al. 2023a). Subsequent mapping approaches, including QTL and genome-wide association studies (GWAS), revealed a more complex genetic basis, identifying multiple loci distributed across several chromosomes (Fall et al. 2001; Paulino et al. 2021; Torres et al. 2024; Chiwina et al. 2023). Collectively, these findings suggest that resistance to Fusarium wilt involves both major and minor effect genes, whose expression is influenced by environmental conditions, host genetic background, and pathogen variability. However, most of these studies were conducted under controlled conditions, which minimize environmental variation and provide limited insight into genotype performance under natural infection pressure (Azzimonti et al. 2014).

Among plant breeding strategies, the use of molecular tools has significantly accelerated breeding programs by enabling identification and localization of favorable alleles in resistant genotypes. These alleles can then be incorporated into crosses with elite cultivars, which reduces the time required to develop improved cultivars (Faleiro et al. 2018). Nevertheless, the use of molecular markers to identify QTLs

associated with Fusarium wilt resistance is still incipient, with few studies available (Torres et al. 2024; Chiwina et al. 2023; Paulino et al. 2021; Cavalheiro 2021). Initially, Fall et al. (2001), working under controlled conditions, identified a QTL on chromosome 10 that accounted for 64% of the phenotypic variation, using line A55 as the resistant parent. Paulino et al. (2021) applied GWAS analysis to a Mesoamerican panel and located SNPs associated with resistance on chromosomes Pv01, Pv03, Pv04, Pv05, Pv07, Pv10, and Pv11. Chiwina et al. (2023), analyzing the USDA Common Bean Core Collection, discovered 11 SNPs strongly associated with Fop races 1 and 4 on chromosomes Pv03, Pv04, Pv05, Pv07, Pv08, Pv09, Pv10, and Pv11.

When investigating sources of resistance in the Embrapa Arroz e Feijão germplasm of Mesoamerican origin, Torres et al. (2024) analyzed a population derived from the cultivars BRS FP403 (resistant, black bean) and BRS Horizonte (susceptible, carioca bean) in a field infested with *F. oxysporum* f. sp. *phaseoli*, and they identified four QTLs on chromosomes Pv01, Pv02, Pv03, and Pv04, which explained from 6 to 41% of resistance to Fusarium wilt. Particular emphasis was placed on the QTL FOP2.3^{403H} on chromosome Pv02, which showed stability over the years and accounted for 41% of the phenotypic variation in the data. Identification of the QTL2.3^{403H} made it possible to design the probes ANAAJK6 and ANCFDFU, which exhibited selection efficiency greater than 92%, favoring the use of MAS for resistance to Fusarium wilt in common bean. In an earlier study, Cavalheiro (2021) identified the QTL FOP7.1^{NS} on chromosome Pv07, which accounted for 45% of the variation in progenies from the cross between the cultivars BRS Notável (resistant, carioca bean) and BRS Supremo (susceptible, black bean), resulting in a hydrolysis probe with 88% selection efficiency for application in MAS.

Another important source of resistance in the Mesoamerican germplasm is the cultivar BRS Esplendor (black bean), which has shown high resistance over time (Cavalheiro 2021; Torres et al. 2024). The studies of Torres et al. (2024) and Cavalheiro (2021) indicate that BRS Esplendor carries the susceptibility alleles for the probes derived from the QTLs FOP^{403H} and FOP^{NS}. This therefore indicates that its resistance is conferred by a different gene. In light of the above, this study aimed to investigate the genetic control of

resistance to *Fusarium oxysporum* f. sp. *phaseoli* and to identify molecular markers linked to resistance alleles in the cultivar BRS Esplendor.

Materials and methods

Genetic material

The mapping population for resistance to Fusarium wilt was obtained by crossing the Mesoamerican origin cultivars BRS Esplendor (Costa et al. 2011) and BRS Horizonte (Melo et al. 2005). These parents were chosen based on the study conducted by Pereira et al. (2016) evaluating 30 genotypes in a field highly infested with *Fusarium oxysporum* f. sp. *phaseoli* over five consecutive years (2010 to 2014). BRS Esplendor is derived from the cross between the lines CB911863 and AN9123293 and is the cultivar with the greatest resistance to Fusarium wilt. It has black bean grains with a 100-seed weight of 21 g, upright plant architecture, and resistance to other diseases, such as anthracnose and bacterial wilt. In addition, the cultivar shows high resistance to common bacterial blight, with the molecular marker QTLSU91 recently validated for the disease (Coêlho et al. 2025). The cultivar BRS Horizonte, derived from the multiple cross EMP250 /4/ A769 /// A429 / XAN252 // PintoVII14, is highly susceptible to Fusarium wilt and has carioca bean grains, upright architecture, and anthracnose resistance.

Crosses were carried out in Santo Antônio de Goiás, Goiás, Brazil (16°30'17" S, 49°16'53" W, altitude 819 m), in May 2016 under controlled conditions, resulting in the F_1 generation. Subsequently, in December 2016, the F_1 plants were sown, and through self-pollination, the F_2 generation was obtained. The hybrid nature of the plants was phenotypically confirmed through a morphological marker for flower color, using BRS Horizonte (white flower) as the female parent. The F_1 plants with white flowers were discarded, and those with purple flowers, resulting from hybridization, constituted the F_2 population, with 103 progenies, which were sown in May 2017. Leaves were collected for DNA extraction and genotyping of individuals. At the end of the cycle, the seeds from each F_2 plant were harvested separately, giving rise to the $F_{2.3}$ progenies. Seeds from the $F_{2.3}$ generation were sown in October 2017

for seed multiplication, resulting in the $F_{2.4}$ generation. All these steps were carried out under controlled conditions (greenhouse), using pots with nutritional conditions suitable for growing. Progenies in the $F_{2.5}$ generation were obtained from the harvest of the $F_{2.4}$ progenies in the field.

Phenotyping

The 103 progenies in the $F_{2.4}$ and $F_{2.5}$ generations were sown in the field under center-pivot irrigation in 2018 and 2019, respectively, in the winter crop season (sowing in May), along with the parents BRS Esplendor and BRS Horizonte, the check cultivars BRS FP403 and IPR Tuiuiu, which show resistance to Fusarium wilt, and BRS Esteio and IPR Uirapuru, which are susceptible (Torres et al. 2024; Cavaleiro et al. 2023a, b). A randomized block experimental design was used with three replications, and each plot consisted of a single 3-m row. Sowing density was 12 seeds per meter, with a spacing of 0.50 m between rows. The experimental area used for phenotyping has been consistently employed in previous studies on resistance to Fusarium wilt in common beans, which demonstrated uniform and stable disease pressure over several years (Pereira et al. 2016, 2019; Torres et al. 2021, 2022, 2024; Cavaleiro et al. 2023a, b). The area is specifically managed to maintain a high and homogeneous level of natural inoculum and is used exclusively to evaluate resistance to Fusarium wilt. The trials are conducted during the winter, under center pivot irrigation, when mild temperatures combined with high soil moisture favor disease development. To maintain pathogen populations, susceptible genotypes are cultivated in the area during other growing seasons. This long-term management ensures consistent infection and reliable phenotypic expression over the years.

Response to Fusarium wilt was evaluated by two independent evaluators at 80 days after plant emergence (R8 stage), using a scoring scale ranging from 1 to 9 adapted from Pastor-Corrales & Abawi (1987), which classifies plots according to the incidence of diseased plants. A score 1 represents plots with no diseased plants (0% incidence, all plants healthy with green foliage), while a score 9 represents plots with an incidence of 90.1 to 100%, where most plants showed severe wilting, leaf necrosis, or death. Intermediate scores correspond to proportional increases

in disease incidence: score 2 (0.1–5%), score 3 (5.1–10%), score 4 (10.1–20%), score 5 (20.1–40%), score 6 (40.1–60%), score 7 (60.1–80%), and score 8 (80.1–90%).

Upon fulfilling the required assumptions, individual analyses of variance were performed for each year/generation, as well combined analysis of variance, according to the following model:

$$Y_{ijk} = \mu + g_i + a_k + b_{j(k)} + (ga)_{ik} + e_{ijk}$$

where Y_{ijk} is the observed value of the i -th genotype in the j -th block within the k -th year/generation; μ is the overall mean; g_i is the random effect of the i -th genotype; a_k is the fixed effect of the k -th year/generation; $b_{j(k)}$ is the effect of the j -th block nested within the k -th year/generation; $(ga)_{ik}$ is the random effect of the interaction between genotype and year/generation; and e_{ijk} is the random residual error.

The coefficient of variation (CV) and selective accuracy (SA) were estimated to assess experimental quality (Resende & Alves 2022). The genotypic (σ_g^2), and genotype $\times$ year/generation interaction (σ_{gy}^2) variance components were estimated from the mean squares obtained in the analysis of variance (Ramalho et al. 2012), and were used to calculate broad sense heritability (H^2) based on the mean input. For combined analysis the following formula was used:

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_{gy}^2}{e} + \frac{\sigma_e^2}{r}}$$

where e is the number of environments and r is the number of replications.

The percentage of expected gain from direct selection, at 20% selection intensity (21 progenies), was estimated using the expression:

$$GS(\%) = \left(SD \times \frac{H^2}{\bar{X}_0} \right) \times 100$$

where SD is the selection differential (the difference between the mean of the selected progenies and the mean of the original population), H^2 is the broad-sense heritability, and $\bar{X}_0$ is the mean of the original population.

Genetic correlation between the genotypes in the two years/generations was also estimated (Ramalho

et al. 2012). All statistical analyses of the phenotypic data were performed using the GENES software (Cruz 2013).

Genotyping and construction of the linkage map

DNA was extracted from the leaves of F_2 plants together with their parents using the modified CTAB method (Hoisington et al. 1994). Individuals were genotyped by the company DArT Pty® (Canberra, Australia) on a HiSeq2500 sequencer (Illumina, USA), using the DArTSeq technology. This technology was described by Kilian et al. (2012), which uses frequent-cutting restriction enzymes in reducing complexity to identify the codominant SNP (Single Nucleotide Polymorphism) markers and the dominant SilicoDArT (Diversity Arrays Technology) markers.

After the codominant SNP markers and dominant DArT markers were obtained, a call rate $\geq 80\%$ was applied. The suitability of the selected markers to the Mendelian segregation hypothesis was evaluated through the chi-square test, with 1:2:1 ratio for SNP markers and 3:1 ratio for DArT markers at a 5% significance level. Analyses were performed on the R software (R Core Team 2022).

After the suitability of the data to the proportion expected for the F_2 population was tested using the chi-square test, FDR correction at a 5% probability level was applied to minimize false positive rates (Benjamini & Hochberg 1995). The markers that fit the null hypothesis tested were used to create the genetic map, and recombination frequency was estimated through pairwise comparison of the markers. The parameters set for construction of the map were a 30% recombination fraction and a minimum LOD score of 3. These thresholds are widely used in linkage mapping to ensure that only markers showing strong evidence of genetic linkage are grouped together. Subsequently, the “red” (rapid chain delineation) method and the “safe” command were used to determine the position and the order of each marker. The linkage map was constructed using the Onemap package (Margarido et al. 2007) in the R Studio software. Graphical representation of the linkage maps was created with the aid of the MapChart 2.32 program (Voorrips 2002).

QTL mapping

QTL analysis was carried out using the compound interval method (CIM) through Model 6 of the Windows QTL Cartographer vs. 2.5 – WinQTLCart software (Wang et al. 2012). The hypothesis of the presence of a QTL was tested based on “Forward and Backward” regression models at every 1.0 cM, with a window size of 10.0 cM. The significance threshold adopted, the LOD score, was estimated by the permutation test method (Doerge & Churchill 1996), using 1000 permutations to perform the analysis.

Candidate gene identification

Candidate genes were identified within the confidence intervals of the QTLs detected in this study using the *Phaseolus vulgaris* reference genome version 2.1 (Schmutz et al. 2014) available on the Phytozome platform (<https://phytozome-next.jgi.doe.gov/>). Genes located within these genomic regions were extracted based on their physical positions using JBrowse and analyzed according to their functional annotation. A gene was considered a potential candidate if it was located within the QTL interval and its function or putative role was associated with plant defense responses, such as recognition, signaling, or resistance mechanisms. This approach follows previously reported procedures for identifying positional candidate genes in common bean (Hamabwe et al. 2023).

Validation of markers associated with the resistance QTL

The SNP with the highest coefficient of determination (R^2) associated with response to Fusarium wilt was used in the marker validation process. To design the probes, the sequence containing the target SNP was aligned with the common bean reference genome (Schmutz et al. 2014) using the BLAST command; and a continuous search was performed for repetitive elements using RepeatMasker, both analysis resources available on the Phytozome platform (<https://phytozome-next.jgi.doe.gov/>). Subsequently, the probe was designed using the online Custom TaqMan Assay Design Tool (Thermo Fisher), available at <https://www.thermofisher.com/order/custom-genomic-products/tools/genotyping/>. Based on

development of the probe, the validation step was performed through locus-specific PCR using TaqMan® probes (ThermoFisher).

Validation of the TaqMan® SNP markers associated with Fusarium wilt was performed using 44 lines derived from crossing the resistant parent, BRS Esplendor, and the susceptible parents BRS Esteio, IPR Uirapuru, VP22, CNFP15171, and BRS Supremo, also from the black commercial group and of Mesoamerican origin. After analysis of polymorphism between BRS Esplendor and these parents, the lines were genotyped. The 44 lines, together with the parents and the check cultivars (BRS FC402, IPR Bem-te-vi, BRS FC415, BRS Notável, BRS FP403, and BRS Estilo), were evaluated in the winter crop season (sowing in May) of 2021 and 2022. The experiment was conducted in a randomized block design with three replications and plots consisting of a single 3-m row in the experimental area naturally infested with the pathogen, as previously described. Evaluation of response to Fusarium wilt and statistical analyses were carried out as reported for the phenotypic data collected in 2018 and 2019.

The selection efficiency of the marker (SE) was calculated through the following estimator: $SE(\%) = (1 - 4r^2) \times 100$, where r refers to recombination frequency (Liu 1998).

Results and discussion

Phenotypic analyses

The estimated coefficients of variation were 14.3% and 18.8% (Table 1), which were lower than those reported in the literature under field conditions (Cavalleiro et al. 2023a, b; Torres et al. 2021, 2022, 2024; Pereira et al. 2019), indicating high experimental accuracy. These results are satisfactory since natural occurrence of the pathogen in the field is not homogeneously distributed. The estimates of selective accuracy were very high in the two years (≥ 0.97), highlighting the reliability of the data obtained from the experiments.

Significant differences were observed among progenies in both years evaluated and in combined analysis (Table 1), indicating genetic variability among progenies for response to Fusarium wilt. Significant difference was observed between the years, as the

Table 1 Summary of analyses of variance for response to Fusarium wilt, evaluated in Santo Antônio de Goiás in the 2018 and 2019 winter crop seasons in progenies of the population BRS Esplendor×BRS Horizonte

Source of variation	DF	Mean squares		
		2018	2019	Combined
Genotypes	108	11.8**	11.2 ^{ns}	21.2**
Checks (CK)	5	13.3**	15.9**	28.6**
Parents (PR)	1	32.7**	32.7**	65.3**
Cultivars (C)	3	10.1**	15.4**	24.8**
PR vs C	1	3.4*	0.7 ^{ns}	3.6*
Progenies (P)	102	11.8**	11.0**	21.0**
CK vs P	1	0.1 ^{ns}	0.3 ^{ns}	0.4 ^{ns}
Years/generations (Y)	1	–	–	174.7**
Genotypes×Y	108	–	–	1.7**
CK×Y	5	–	–	28.6**
PR×Y	1	–	–	0.0 ^{ns}
C×Y	3	–	–	0.7 ^{ns}
(PR vs C)×Y	1	–	–	0.5 ^{ns}
P×Y	102	–	–	1.8**
(CK vs P)×Y	1	–	–	0.1 ^{ns}
Individual residue	216	0.6	0.7	–
Combined residue	432	–	–	0.7
Mean – progenies	–	5.5	4.5	5.0
Mean – resistant checks	–	3.8	2.6	3.2
Mean – susceptible checks	–	7.4	6.7	7.1
Correlation	–	–	–	0.84**
CV (%)	–	14.3	18.8	16.3
SA	–	0.97	0.97	0.98

CV, experimental coefficient of variation; SA, selective accuracy. * and **, denote significance at $p < 0.05$ and $p < 0.01$. ns, not significant.

mean value obtained in 2018 (5.5) was higher than the mean value in 2019 (4.5), which suggests that year-to-year environmental variation affected trait expression. The pathogen tends to develop more intensely in regions with intensive cultivation under center-pivot irrigation during the winter growing season, where mild temperatures combined with high soil moisture favor its multiplication and symptom expression (Benchimol-Reis et al., 2023).

Air temperature conditions were similar between the two years (maximum of 33.5 °C in 2018 and 32.8 °C in 2019; minimum of 6.0 °C and 7.2 °C, respectively; and an identical mean temperature of 21.6 °C). In addition, accumulated rainfall during the growing season was low in both years, totaling 15.4

Table 2 Estimates of genotypic variance (σ_g^2), phenotypic variance (σ_p^2), environmental variance (σ_e^2), variance of the progeny×year interaction (σ_{gy}^2), heritability (h^2), and expected gain from direct selection (GS%) for response to Fusarium wilt based on individual and combined analyses, evaluated in 103 progenies from the BRS Esplendor×BRS Horizonte population

Parameter	2018	2019	Combined
σ_g^2	3.7	3.4	3.4
σ_p^2	3.9	3.7	3.5
σ_e^2	0.21	0.24	0.67
σ_{gy}^2	–	–	0.19
$h^2\%$	94.7	93.5	96.8
GS%	–48.1	–49.8	–48.4

mm in 2018 and 25.0 mm in 2019, which is expected for the winter growing season in the region. In irrigated winter cropping systems, Fusarium wilt symptom expression is mainly influenced by soil moisture conditions resulting from irrigation management. Thus, differences in soil moisture between years may help explain the higher disease level observed in 2018. These results contrast with those obtained by Torres et al. (2024) and Cavalheiro (2021), who found no significant differences between years in evaluations performed in the same experimental area.

The interaction between progenies and years/generations was significant (Table 1), indicating that the progenies exhibited different responses in the two years. However, the correlation between progenies in the two years was high and significant (0.84), indicating that this interaction is predominantly simple and that the genotypic responses were consistent across years.

The estimates of genetic parameters showed genotypic variance higher than environmental variance in both years (Table 2), confirming that the phenotypic variability observed is mainly attributable to genetic factors, which favors selection of progenies with greater resistance to Fusarium wilt. The high heritability estimates (higher than 93%) corroborate the occurrence of genotypic variation in the progenies, enabling successful selection, similar to that reported by Torres et al. (2022, 2024) and Cavalheiro et al. (2023b) in field experiments. These estimates are also consistent with results of studies performed under controlled conditions (Cândida et al. 2009; Pereira et al. 2009), which reported heritability estimates

ranging from 76 to 97%, suggesting the presence of genes with considerable effects on control of the trait.

Expected gains from selection for resistance to Fusarium wilt were high in both years (48% and 50%) and in combined analysis (48%) (Table 2). These gains exceeded those observed by Cândida et al. (2009) (27% and 36%) in progenies of populations inoculated with the FOP46 isolate in a greenhouse, as well as the gains reported by Torres et al. (2022) (34%), Cavalheiro (2023b) (23%), and Torres et al. (2024) (37%) in field trials. The high gains from selection are associated with high heritability estimates and with the contrast between the parents BRS Esplendor (score 2.3) and BRS Horizonte (score 7.0), resulting in a selection differential greater than the overall mean. The mean of the selected progenies (2.5) was similar to the mean of BRS Esplendor, which is the standard for Fusarium wilt resistance in black beans.

Linkage maps

The total number of markers obtained through DArTseq genotyping was 53,254, of which 26,462 were SNPs and 26,692 were SilicoDArT markers (Table S1). After removal of non-informative markers (call rate $\geq 80\%$), monomorphic markers ($\geq 95\%$), and those that did not fit the expected Mendelian segregation, 2473 markers remained for mapping analysis. The low percentage of polymorphic markers (14.2%) is due to the use of parents belonging to the same

gene pool, as also observed by Torres et al. (2024), Cavalheiro (2021), and Kamfwa et al. (2018), who reported 28%, 11%, and 14% polymorphic markers, respectively.

Of the 2473 markers included in linkage analysis, 2189 were mapped, resulting in 994 SNP markers and 1195 SilicoDArTs, distributed across 11 linkage groups formed (Table 3, Figure S1). Overall, the total map length was 2,854.39 cM, with an average distance between markers of 1.46 cM. The results presented here are consistent with current literature, which has reported construction of common bean linkage maps of varied lengths – maps from 754 cM to 4,474 cM can be found (Gomes-Messias et al. 2022; Langat et al. 2020). In addition, the markers spaced at distances of less than 5 cM accounted for 93% of the total, indicating high saturation of the constructed map (Langat et al. 2020).

QTL mapping

Mapping analysis identified three genomic regions associated with resistance to Fusarium wilt, all on chromosome Pv08 (Fig. 1). These regions correspond to the two years and to combined analysis (Table 4), with estimated critical LOD scores ($LOD_{critical}$) of 9.9 in 2018, 6.3 in 2019, and 7.8 in combined analysis. The QTL obtained based on combined analysis (FOP8.1^{EH}), which has the marker Ch_8_2819713_14794_SNP as the point of maximum magnitude, at position 71.37 cM, accounted

Table 3 Information on the genetic linkage map obtained from the F₂ population of the cross between the cultivars BRS Esplendor and BRS Horizonte

¹Number of SNP, DArT, and total (Total) markers allocated to each linkage group; ²Size of the linkage map for each linkage group and the total; ³Largest distance between the markers in the linkage group; ⁴Mean distance between the markers in the linkage map; ⁵Percentage of distances between markers that were ≤ 5 cM

Chromosome	No. of markers ¹			Size (cM) ²	Distances		
	SNP	DArT	Total		Largest (cM) ³	Mean (cM) ⁴	≤ 5 cM (%) ⁵
Pv01	58	58	116	197.28	47.87	1.71	95.69
Pv02	75	70	145	270.15	14.13	1.88	89.65
Pv03	87	82	169	341.61	33.91	2.03	85.80
Pv04	52	87	139	187.33	15.51	1.36	91.37
Pv05	96	92	188	210.20	11.98	1.12	96.28
Pv06	151	149	300	300.18	10.14	1.00	96.00
Pv07	116	96	212	280.97	15.84	1.33	91.98
Pv08	172	250	422	464.91	20.28	1.10	94.08
Pv09	39	32	71	178.30	38.16	2.55	90.14
Pv10	74	151	225	205.28	5.98	0.92	98.22
Pv11	74	128	202	218.18	25.03	1.08	96.53
Total	994	1195	2189	2854.39	–	–	–
Mean	90.36	108.64	199	259.49	21.71	1.46	93.25

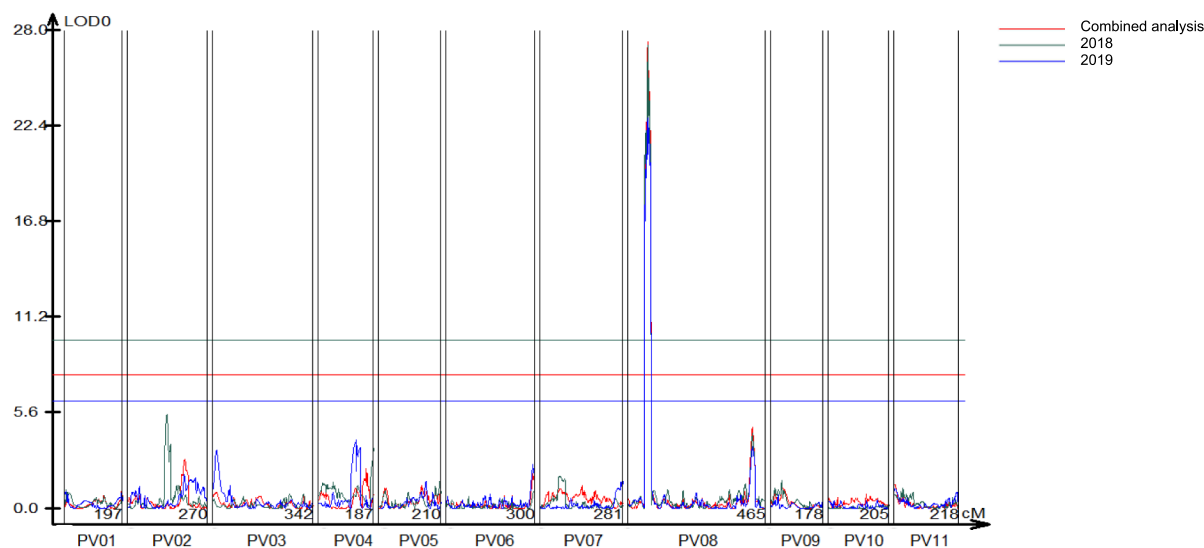


Fig. 1 Graphical analysis of the results obtained in QTL analysis for response to Fusarium wilt in the F_2 population of the cross between BRS Esplendor and BRS Horizonte for the different years/generations tested, using the composite interval

mapping (CIM) method of the QTL Cartographer software. The groups labeled Pv01 to Pv11 represent chromosomes 1 to 11

for 66% of the phenotypic variation, with a LOD of 25.63 (Table 4). The QTLs FOP8.2^{EH} and FOP8.3^{EH} were identified in 2018 and 2019, respectively, and both have the same SNP marker at the point of maximum magnitude and at the same position as that obtained in combined analysis. The QTLs FOP8.2^{EH} and FOP8.3^{EH} account for 62% and 55% of the phenotypic variation, with LODs of 25.18 and 22.25, respectively (Table 4).

The high LODs and coefficients of determination (R^2) indicate the presence of major-effect genes controlling the response to Fusarium wilt, a feature favorable for use in plant breeding. In addition, the fact that the three QTLs obtained were located at the same position indicates that they correspond to a single major-effect QTL, which remains stable across the different years evaluated. However, although the QTLs identified here are considered major-effect QTLs, the variability in the data has not yet been fully explained, suggesting the participation of other minor-effect regions in control of reaction to Fusarium wilt, indicating oligogenic or polygenic inheritance.

Few studies in the literature have identified genomic regions associated with resistance to Fusarium wilt. Fall et al. (2001) worked with RAPD

markers and identified a QTL on chromosome Pv10 explaining 64% of the resistance to race 4 of *F. oxysporum* f. sp. *phaseoli* from the cross between the Mesoamerican lines Belneb RR-1 and A55. In more recent studies, QTLs associated with resistance to Fusarium wilt were identified through GWAS analysis in common bean on several chromosomes. Paulino et al. (2021) used a Mesoamerican diversity panel and identified SNPs associated with resistance to the isolates FOP UFV01 and IAC18001 in Pv01, Pv03, Pv04, Pv05, Pv07, Pv10, and Pv11. Furthermore, Chiwina et al. (2023), analyzing the USDA Common Bean Core Collection, discovered 11 SNPs on chromosomes Pv03, Pv04, Pv05, Pv07, Pv8, Pv09, Pv10, and Pv11 associated with Fop races 1 and 4.

Cavalheiro (2021) used a population derived from a cross between the Mesoamerican cultivars BRS Notável (resistant) and BRS Supremo (susceptible) in a field naturally infested with the pathogen and identified the QTL FOP7.1^{NS} on chromosome 7, which explained 45% of the phenotypic variation. This QTL was used to develop a specific hydrolysis probe (ANKCHTP), which exhibited 88% selection efficiency in validation with other lines derived from crossing BRS Notável with other parents. Torres et al. (2024) analyzed a population derived from a

Table 4 Information on the QTLs (quantitative trait loci) identified for response to Fusarium wilt in common bean across different years/generations in the F₂ population derived from the cultivars BRS Esplendor and BRS Horizonte

QTL	Chromosome	Year	Interval (Mb)	NT ^a	Selected marker	Position (cM)	R ² (%) ^b	LR ^c	LOD ^d	LOD _e	AE ^f
FOP8.1	Pv 08	Combined	2.2–4.1	209	Ch_8_2819713_14794_SNP	71.37	66.07	118.18	25.63	7.8	-2.11
FOP8.2	Pv 08	2018	2.2–4.1	209	Ch_8_2819713_14794_SNP	71.37	62.04	116.08	25.18	9.9	-2.32
FOP8.3	Pv 08	2019	2.2–4.1	209	Ch_8_2819713_14794_SNP	71.37	55.03	102.59	22.25	6.3	-1.88

^a Number of transcripts; ^b Coefficient of determination; ^c Likelihood ratio test; ^d LODscore: Likelihood of odds score (LR/4.61); ^e LODcritical: minimum value for identification of QTL; ^f Additive effect of the QTL

cross between BRS FP403 (resistant) and BRS Horizonte (susceptible) in a field infested with the pathogen and identified four QTLs on chromosomes Pv01, Pv02, Pv03, and Pv04, explaining from 6 to 41% of resistance to Fusarium wilt. The QTL FOP2.3^{403H}, on chromosome Pv02, stood out for its stability and high percentage of explanation of phenotypic variation (41%), leading to development of the probes ANAAJK6 and ANCFDFDU, with selection efficiency greater than 92%, favoring marker-assisted selection for resistance to Fusarium wilt in common bean.

As the QTL mapped in this study is different from the QTLs identified by Torres et al. (2024) and Cav-alheiro (2021), the availability of molecular tools will enable the combination of these QTLs in a single cultivar, thereby increasing resistance to Fusarium wilt. This therefore shows that common bean response to Fusarium wilt is complex, and the identified regions of interest may contain various genes that act through different defense pathways (Chen et al. 2019).

The regions identified here have alleles with additive effects ranging from -1.88 to -2.32, which contribute to increase resistance to Fusarium wilt, reducing the score of plants carrying these alleles (Table 4). These alleles, as expected, originate from the resistant parent (BRS Esplendor), such that plants carrying the nucleotide base G for the marker “Ch_8_2819713_14794_SNP” showed significantly greater resistance than those carrying base A (Fig. 2).

Annotated genes

A total of 209 transcripts were identified within the confidence interval that was defined based on the region selected in mapping analysis. The region analyzed on the physical map began at the 2.2 Mb position of chromosome eight (Pv08) in the *Phaseolus vulgaris* L. genome, version 2.1, and ended at the 4.1 Mb position, encompassing a total of 1.9 Mb. Of the 209 transcripts, 197 (94.2%) have an assigned function, and 50 (23.9% of the total) are related to common bean resistance to diseases.

Among the disease-related transcripts, 37 contain leucine-rich repeat (LRR) sequences and receptor-like kinases (serine/threonine protein kinases), both associated with defense responses against pathogen attack (Burt et al. 2015; Ashfield et al. 2012). Within this group, two LRR transcripts are associated with the RPP13 gene, which grants resistance to fungal

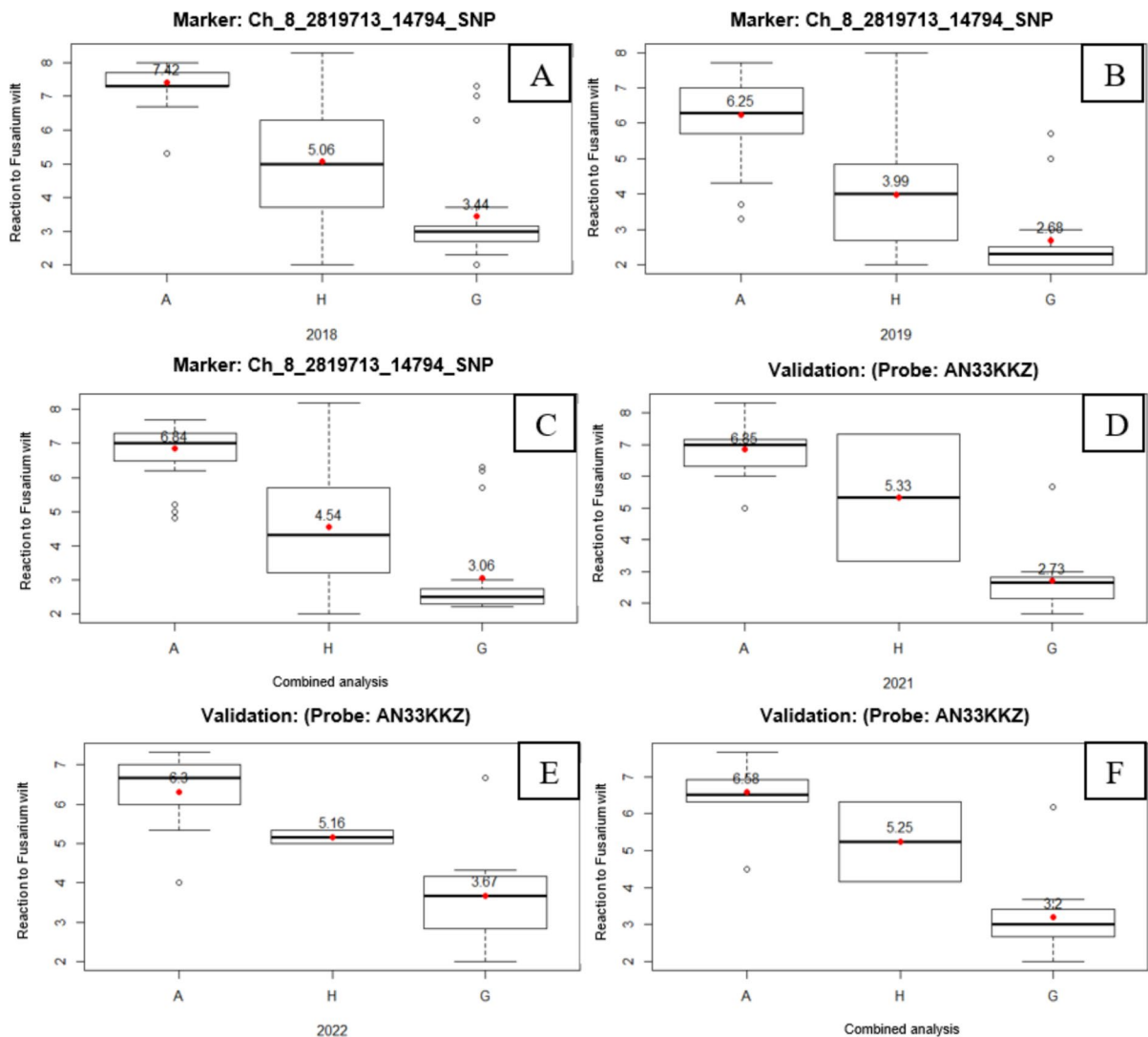


Fig. 2 Boxplots of the mean values of the progenies for the marker identified in QTL analysis located on chromosome Pv08 associated with reaction to Fusarium wilt in 2018 **A**, 2019 **B**, and combined analysis **C** together with the probe used in validation of the data (AN33KKZ) in 2021 **D**, 2022 **E**, and

in combined analysis **F**. The points represent the mean values. **A** and **G** correspond to the mean values of the homozygous progenies from the parents BRS Horizonte (susceptible) and BRS Esplendor (resistant), respectively, while H represents heterozygous genotypes

diseases in plants, such as downy mildew in *Arabidopsis* (Bittner-Eddy et al. 2000) and powdery mildew in barley (Wu et al. 2018) and in wheat (Wu et al. 2019). Ten other transcripts are associated with induction of the isoflavonoid pathway, a reaction associated with resistance to common bacterial blight in common bean (Cox et al. 2021). The remaining three encode endo-1,3- β -glucanase domains, which are also associated with resistance (Burt et al. 2015).

The region of interest selected in this study was similar to that highlighted by Burt et al. (2015) in their work on identifying resistance to anthracnose in common bean. The authors reported 32 potential genes associated with disease resistance within the interval from 1.8 to 2.7 Mb, of which 27 transcripts corresponded to LRRs. In addition, studies have indicated the existence of an important cluster of resistance genes on chromosome Pv08 of common bean,

including gene SU91, associated with resistance to common bacterial blight (Coêlho et al. 2025); CPMMV.Pv08, which confers resistance to *Carlavirus* (Silva et al. 2024); and Phg-2, related to resistance to angular leaf spot (Lobaton et al. 2018; Gil et al. 2019). These genes also encode LRRs and proteins with multiple kinase domains, which play a central role in common bean defense against pathogens (Gil et al. 2019; Andersen et al. 2018). These findings suggest that this genomic region may comprise a set of resistance genes or a broad-spectrum resistance gene involved in defense against multiple pathogens, including *Fusarium oxysporum* f. sp. *phaseoli*.

Marker validation

The coefficients of variation obtained from phenotyping in the validation experiments for the two years and in combined analysis were 14% and 21%, with selective accuracy from 0.93 to 0.98, indicating good accuracy and high reliability of the data (Cavalheiro et al. 2023a, b; Torres et al. 2021, 2022, 2024). Heritability estimates (95%) and expected gain from selection (36%) in combined analysis confirmed the possibility of successful selection among these lines. The mean values varied widely in combined analysis, ranging from 2.2 to 7.8 (Table 5). Approximately 48% of the lines were considered resistant, and 12% of them proved to be as resistant as or more resistant than BRS Esplendor (score 2.5).

For the results of genotyping using the AN33KKZ probe, the BRS Esplendor parent carried GG alleles (Table 5). Among the lines, eleven were homozygous for the resistance allele (GG), representing 44% of the lines; twelve were homozygous for the susceptible allele (AA, 48%); and two were heterozygous (AG, 8%) (Table 5). The mean score of the resistant lines was 3.2, in sharp contrast with the mean score of the susceptible lines (6.6) (Fig. 2). The calculated selection efficiency was 97%, with a recombination fraction of 0.09, indicating that this marker is effective in selecting lines resistant to *Fusarium* wilt and can be used in plant breeding programs in MAS.

The resistant check cultivars (IPR Bem-Te-Vi, BRS FC415, BRS FC402, BRS FP403, and BRS Notável) were homozygous for the allele that confers susceptibility (AA) for the AN33KKZ probe (QTL FOP8.1^{EH}), suggesting that the resistance in these cultivars is conferred by a gene different from the one

that confers resistance in BRS Esplendor (Table 5). This result is consistent with the findings of Torres et al. (2024) and Cavalheiro (2021), who used the resistant genotypes BRS FP403 and BRS Notável and identified alleles different from BRS Esplendor at the loci of interest, which were located on chromosomes Pv02 and Pv07, respectively, and associated with resistance to *Fusarium* wilt (Table 6). Thus, it can be inferred that the cultivars BRS Esplendor, BRS Notável, and BRS FP403 have resistance to *Fusarium* wilt conferred by different genes, located on chromosomes Pv08, Pv07, and Pv02, respectively, supporting the use of marker-assisted selection for pyramiding the alleles into a single cultivar, so as to increase resistance and maximize their durability.

Additionally, Torres et al. (2024) observed that the cultivars BRS FC402 and BRS FC415 carry the resistance alleles of BRS FP403 (CC and TT) for the probes ANAAJK6 and ANCFDDU, respectively (Table 6), which are associated with the QTL FOP2.3^{403H}, suggesting that resistance in these cultivars may originate from the same gene. The study of Cavalheiro (2021) showed that the cultivar BRS Notável carries the resistance allele AA for the ANKCHTP probe, associated with the QTL FOP7.1^{NS}, differing from the cultivars BRS Esplendor, BRS FC402, BRS FC415, BRS FP403, and IPR Bem-te-vi, which, although resistant, had the GG allele (Table 6), indicating that the origin of the resistance comes from a distinct gene or that the markers are segregating independently of this gene in the genotypes mentioned. The check cultivars BRS Estilo and BRS Esteio, considered susceptible, showed alleles different from those obtained from the three sources of resistance discussed (BRS Esplendor, BRS Notável, and BRS FP403), which were AA for the AN33KKZ probe, GG for the ANKCHTP probe, GG for the ANAAJK6 probe, and AA for the ANCFDDU probe, as expected (Table 6).

Based on these results, it is possible to develop a cultivar that combines the different resistance alleles identified in the cultivars BRS Esplendor, BRS FP403, and BRS Notável through gene pyramiding. The cross between BRS Esplendor and BRS FP403 would result in black bean lines with resistance alleles from two genes located on chromosomes Pv08 and Pv02. For the carioca group, a cross can be made between BRS Notável and BRS FC415 or BRS FC402, resulting in lines with resistance alleles from

Table 5 Summary of genotyping for the probe AN33KKZ and validation of the results for response to Fusarium wilt in common bean lines derived from crosses of BRS Esplendor with BRS Esteio, IPR Uirapuru, and BRS Supremo.

Name	Parents	AN33KKZ	2021		2022		Combined	
			FOP	Class	FOP	Class	FOP	Class
BRS Esplendor / BRS Esteio.1	BRS Esplendor / BRS Esteio	A/A	7.0	S	6.7	S	6.8	S
BRS Esplendor / BRS Esteio.2	BRS Esplendor / BRS Esteio	G/G	2.3	R	4.0	R	3.2	R
BRS Esplendor / BRS Esteio.3	BRS Esplendor / BRS Esteio	G/G	3.0	R	4.3	R	3.7	R
BRS Esplendor / BRS Esteio.4	BRS Esplendor / BRS Esteio	G/G	2.0	R	2.3	R	2.2	R
BRS Esplendor / BRS Esteio.6	BRS Esplendor / BRS Esteio	G/G	1.7	R	3.3	R	2.5	R
BRS Esplendor / BRS Esteio.7	BRS Esplendor / BRS Esteio	—/—	6.3	S	6.3	S	6.3	S
BRS Esplendor / BRS Esteio.8	BRS Esplendor / BRS Esteio	A/A	7.0	S	7.0	S	7.0	S
BRS Esplendor / BRS Esteio.10	BRS Esplendor / BRS Esteio	G/G	2.0	R	2.0	R	2.0	R
BRS Esplendor / BRS Esteio.11	BRS Esplendor / BRS Esteio	A/A	6.0	S	6.7	S	6.3	S
BRS Esplendor / BRS Esteio.13	BRS Esplendor / BRS Esteio	A/A	6.3	S	7.0	S	6.7	S
BRS Esplendor / BRS Esteio.14	BRS Esplendor / BRS Esteio	A/A	7.3	S	5.3	S	6.3	S
BRS Esplendor / BRS Esteio.17	BRS Esplendor / BRS Esteio	G/G	5.7	S	6.7	S	6.2	S
BRS Esplendor / BRS Esteio.21	BRS Esplendor / BRS Esteio	G/G	3.0	R	2.7	R	2.8	R
BRS Esplendor / BRS Esteio.22	BRS Esplendor / BRS Esteio	A/G	7.3	S	5.3	S	6.3	S
BRS Esplendor / BRS Esteio.26	BRS Esplendor / BRS Esteio	A/A	7.0	S	6.0	S	6.5	S
BRS Esplendor / BRS Esteio.27	BRS Esplendor / BRS Esteio	A/A	7.0	S	6.0	S	6.5	S

Table 5 (continued)

Name	Parents	AN33KKZ	2021		2022		Combined	
			FOP	Class	FOP	Class	FOP	Class
BRS Esplendor / BRS Esteio.28	BRS Esplendor / BRS Esteio	A/A	8.0	S	7.3	S	7.7	S
BRS Esplendor / BRS Esteio.29	BRS Esplendor / BRS Esteio	G/G	2.7	R	4.0	R	3.3	R
BRS Esplendor / BRS Esteio.30	BRS Esplendor / BRS Esteio	A/A	8.3	S	7.0	S	7.7	S
CNFP 19714	BRS Esplendor/IPR Uirapuru	G/G	2.3	R	3.7	R	3.0	R
CNFP 19719	BRS Esplendor/IPR Uirapuru	A/A	6.3	S	6.3	S	6.3	S
CNFP 17959	BRS Esplendor/BRS Supremo	A/G	3.3	R	5.0	S	4.2	R
CNFP 17962	BRS Esplendor/BRS Supremo	A/A	5.0	S	4.0	R	4.5	R
CNFP 17973	BRS Esplendor/BRS Supremo	G/G	2.7	R	4.3	R	3.5	R
CNFP 17975	BRS Esplendor/BRS Supremo	G/G	2.7	R	3.0	R	2.8	R
BRS FC402	LM 96200246/LP 9632	A/A	4.3	R	3.0	R	3.7	R
IPR Bem-te-vi	-	A/A	3.7	R	4.0	R	3.8	R
BRS FC415	BRS Horizonte/LM202206076	A/A	4.0	R	3.3	R	3.7	R
BRS Esplendor	CB911863/AN9123293	G/G	2.7	R	2.3	R	2.5	R
BRS FP403	POT51 /// ICA PIJAO / XAN170 // BAC16 / XAN91	A/A	4.7	S	4.3	R	4.5	R
BRS Estilo	EMP250/4/A769///A429/XAN252//V8025/PINTOUI114	A/A	6.7	S	6.3	S	6.5	S
BRS Esteio	FT85-113 / POT51	A/A	6.7	S	4.7	S	5.7	S
CNFP 15171	–	G/G	7.0	S	6.0	S	6.5	S
VP22	–	G/G	7.0	S	7.0	S	7.0	S
IPR Uirapuru	–	A/A	5.0	S	5.0	S	5.0	S
BRS Supremo	W22-34/VAN163	A/A	7.7	S	8.0	S	7.8	S
Recombination fraction (rf): 0.0909		Selection efficiency (%): 96.7						

the genes located on chromosomes Pv07 and Pv02. It should be highlighted that better resistance sources are available in the black commercial group than in the carioca group. Thus, it is important to transfer the resistance allele from BRS Esplendor to the carioca

bean germplasm, and crosses could be made between BRS Esplendor and BRS FC415, or BRS Notável, for example. Genes pyramiding for resistance to Fusarium wilt can increase the durability and effectiveness

Table 6 Genotyping of the common bean check cultivars used in analysis of response to *Fusarium* wilt using the sources of resistance of BRS Esplendor (AN33KKZ), BRS FP403 (ANAAJK6 and ANCFDDU), and BRS Notável (ANKCHTP)

Check	Probe ^a			
	AN33KKZ	ANAAJK6	ANCFDDU	ANKCHTP
BRS Esplendor	G/G	G/G	A/A	G/G
BRS FP403	A/A	C/C	T/T	G/G
BRS FC402	A/A	C/C	T/T	G/G
BRS FC415	A/A	C/C	T/T	G/G
BRS Notável	A/A	G/G	A/A	A/A
IPR Bem-te-vi	A/A	-	-	G/G
BRS Estilo	A/A	G/G	A/A	G/G
BRS Esteio	A/A	G/G	A/A	G/G

^aThe AN33KKZ probe, identified in this study, is associated with the QTL FOP8.1^{EH} which confers resistance in BRS Esplendor; the ANAAJK6 and ANCFDDU probes, identified in the study of Torres et al. (2024), are associated with the QTL FOP2.3^{403H}, which confers resistance in BRS FP403; and the ANKCHTP probe, identified in the study of Cavaleiro (2021), is associated with the QTL FOP7.1^{NS}, which confers resistance in BRS Notável

of resistance, enabling the common bean crop to be grown in areas where the pathogen is present.

Using MAS to develop cultivars enables early selection of genotypes, allowing multiple selection cycles per year. This strategy also facilitates the integration of markers already validated for other traits of interest, such as slow seed coat darkening (Alvares et al. 2019), resistance to anthracnose (Gomes-Messias et al. 2022; Vieira et al. 2018) and common bacterial blight (Coêlho et al. 2025). This approach optimizes time use, increases efficiency, and reduces costs throughout the process of developing new lines.

Conclusions

The results demonstrate substantial genetic variability in the population derived from the cross between BRS Esplendor and BRS Horizonte for response to *Fusarium* wilt, indicating good potential for genetic improvement through selection. A major QTL, named FOP8.1^{EH}, which controls the response to *Fusarium* wilt in BRS Esplendor, was located on chromosome Pv08. This QTL was consistently detected over the years, explaining 66% of the phenotypic variation and confirming its stability. The AN33KKZ probe, strongly associated with this QTL, showed 97% efficiency in distinguishing between resistant and susceptible genotypes, corroborating its applicability for marker-assisted selection. Furthermore, the resistance gene identified in BRS Esplendor is distinct from

those previously reported in BRS Notável and BRS FP403, suggesting the presence of a new source of resistance to *Fusarium oxysporum* f. sp. *phaseoli* that can be explored in breeding programs.

Acknowledgements The authors are grateful to Embrapa Arroz e Feijão and its partners for providing infrastructure and labor support, to the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) for granting a doctoral scholarship to AHC (grant no. 88882.386276/2019-01), and to the Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) for granting a technological and industrial development scholarship to NVC, as well as for granting scholarships for technological development and innovative extension to HSP and LCM.

Author contribution A.H.C.: Data curation; formal analysis; methodology; validation; visualization; writing—original draft. R.P.V.: Conceptualization; funding acquisition; methodology; writing—original draft; writing—review and editing. T.L.P.O.S.: Conceptualization; methodology. L.C.M.: Funding acquisition; methodology. N.V.C. and S.M.M.: Formal analysis; visualization; writing—original draft. L.M.G.M.: Formal analysis; methodology; writing—original draft. H.S.P.: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; supervision; validation; visualization; writing—original draft; writing—review and editing.

Funding The Article Processing Charge (APC) for the publication of this research was funded by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - Brasil (CAPES) (ROR identifier: 00x0ma614).

Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interest The authors declare no competing interests.

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