The following is a postprint of the manuscript published at the Phytopathology APS journal with

DOI: 10.1094/PHYTO-06-19-0207-R

Available at the website:

https://apsjournals.apsnet.org/doi/10.1094/PHYTO-06-19-0207-R

Title

Natural variation in Portuguese common bean germplasm reveals new sources of resistance against *Fusarium oxysporum* f. sp. *phaseoli* and resistance-associated candidate genes

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Abstract

Common bean (*Phaseolus vulgaris* L.) is one of the most consumed legume crops in the world and fusarium wilt, caused by the fungus *Fusarium oxysporum* f. sp. *phaseoli* (*Fop*), is one of the major diseases affecting its production. Portugal holds a very

promising common bean germplasm with an admixed genetic background that may reveal novel genetic resistance combinations between the original Andean and Mesoamerican gene pools. In order to identify new sources of fusarium wilt resistance and detect resistance-associated SNPs, we explored, for the first time, a diverse collection of the underused Portuguese common bean germplasm using genome-wide association analyses. The collection was evaluated for fusarium wilt resistance under growth chamber conditions, using the highly virulent *Fop* strain, FOP-SP1 race 6. Fourteen of the 162 Portuguese accessions evaluated were highly resistant and 71 intermediate. The same collection was genotyped with Illumina BARCBean6K_3BeadChip and DArTseq arrays and SNP-resistance associations were tested using a mixed linear model accounting for the genetic relatedness among accessions. The results from the association mapping revealed nine SNPs associated with resistance on chromosomes Pv04, PV05, Pv07, and Pv08, indicating that fusarium wilt resistance is under oligogenic control. Putative candidate genes related to phytoalexins biosynthesis, hypersensitive response, and plant primary metabolism were identified. The results reported here highlight the importance of exploring underused germplasm for new sources of resistance and provide new genomic targets for the development of functional markers to support selection in future disease resistance breeding programs.

Keywords: *Phaseolus vulgaris* L., fusarium wilt, association mapping, GWAS, complete and incomplete resistance

Introduction

Common bean (*Phaseolus vulgaris* L.) is the most important food grain legume worldwide, with recognized benefits in health and nutrition (Câmara et al. 2013).

Fusarium wilts have a negative impact on the yield of several legume species and other crops (Okungbowa and Shittu 2012). The causal agent of fusarium wilt disease, *Fusarium oxysporum,* penetrates through root tips or wounds, growing in the plant vascular system. On susceptible plants, it may lead to vessel clogging, internal stem discoloration, and a rapid yellowing of foliage, followed by defoliation and ultimately plant death. Wilting may be caused by a combination of pathogen activity, such as the

accumulation of fungal mycelium and/or toxin and host defense responses, including the production of gels, gums, and vessels crushing (Di Pietro et al. 2003).

In common bean, fusarium wilt is caused by *F. oxysporum* (Schlecht.) f. sp. *phaseoli* Kendrick & Snyder (*Fop*) (Agrios 1997) and is among the most important fungal diseases affecting common bean production throughout the world (Alves-Santos et al. 2002; Niño-Sánchez et al. 2015; Schwartz and Pastor-Corrales 1980; Toledo Souza et al. 2012; Xue et al. 2015). At least six different races of *Fop* have been described (Alves-Santos et al. 2002; Salgado 1995) generally associated with a specific geographic area. Race 1 includes isolates found both in the USA and Italy; race 2, isolates found in Brazil; race 3, isolates found in Colombia; race 4, isolates found in the USA; race 5, isolates found in Greece; and race 6, isolates found in Spain. In Portugal, there is no history of the predominant *Fop* race(s) in the fields, but due to the country's geographical proximity to Spain, one may expect that *Fop* race 6 isolates may also be affecting common bean yields in Portugal.

The control of vascular wilt pathogens is not an easy task. Chemical fungicides are ineffective, especially for pathogens like *Fop* that have a soil-borne nature and possess structures that persist for long periods in the soil, even in the absence of host plants (Yadeta and Thomma 2013). Also, biocontrol using antagonistic bacteria or fungi cannot effectively limit these vascular diseases, since abiotic and biotic factors make their performance inconsistent. Consequently, the use of resistant cultivars is the most efficient, environmentally friendly, and economically viable strategy to provide effective fusarium wilt disease control (Dodds and Rathjen 2010).

A better understanding of the genetic basis of resistance mechanisms deployed by resistance sources is needed for more efficient resistance breeding, taking into consideration the pathogen's evolutionary potential. In common bean, the *Fop* resistances already described are controlled by either single major genes or polygenes according to the common bean geographical and genetic origin (Batista et al. 2017; Cross et al. 2000; Fall et al. 2001; Salgado 1995). Since *F. oxysporum* populations are not very large due to its relatively low potential for gene flow, asexual reproduction, and low mutation rate, the use of major resistance genes in breeding might be a sufficient strategy to achieve durable resistance provided that virulence is monitored and genes effectively deployed spatially (McDonald and Linde 2002). Nevertheless, because the fungus may be seed-transmitted, the risk of gene flow due to human activities is actually high. Under these circumstances, pyramiding of different major genes with the

complementary use of quantitative incomplete resistances is advisable. In this way, the continuous search for new sources of resistance is essential for development and deploying more durable resistances on new cultivars.

In Portugal, common bean represents about 70% of grain legumes consumed by humans ("Estatísticas Agrícolas 2017," www.ine.pt). A very diverse common bean germplasm, resulting from more than 500 years of cultivation and adaptation to the country's edapho-climatic conditions, is still preserved in farmers' fields, but underexploited by conventional breeding. An extended representative collection of this national diversity was recently characterized (Leitão et al. 2017). Genetic structure analysis divided this collection into three main clusters, one more related to the Mesoamerican gene pool and two more related to the Andean gene pool. Most of the Portuguese germplasm analyzed grouped with the Andean region race representatives and wild relatives. However, one-third of the national germplasm had an admixed origin and might represent putative hybrids between the original Andean and Mesoamerican gene pools.

As a result of co-evolutionary interactions between pathogens and their host plants, virulent isolates for each common bean gene pool have evolved. Accordingly, common bean resistance genes of Mesoamerican origin are more effective when transferred to bean varieties with Andean background, and vice-versa (Geffroy et al. 1999; Guzman et al. 1995; Miklas et al. 2006; Mkandawire et al. 2004). Thus, besides the resistance genes that may be detected in the Portuguese germplasm of Andean origin, the Portuguese gene pool admixed accessions may have novel resistance gene combinations, harder for the pathogen to overcome and, therefore, useful to enhance the durability of resistance. However, little is known about the response of the Portuguese germplasm against *Fop*. To the best of our knowledge, only one short report exists characterizing two Portuguese common bean cultivars (Tarrestre and Oriente) as very susceptible to a *Fop* strain (FA-15) isolated from a greenhouse in Portugal (Santos et al. 2017). Nevertheless, the genetic diversity found by Leitão et al. (2017) on the Portuguese common bean germplasm encouraged the exploitation of the natural variation for fusarium wilt resistance that might exist within a larger number of Portuguese accessions.

Until now, only a few bi-parental linkage mapping-based reports are available on common bean resistance to *Fusarium* sp., namely *Fop* (Fall et al. 2001) and *F. solani* f.sp. *phaseoli* (Hagerty et al. 2015; Nakedde et al. 2016). These studies were, however,

limited to the allelic diversity that segregates between the parental lines used in the cross, which eventually also restricts mapping resolution (Korte and Farlow 2013).

Genome-wide association studies (GWAS) are a powerful tool to identify polymorphisms underlying natural variation in genomic regions responsible for the expression of a given trait. This approach can provide higher resolution mapping, greater allelic diversity, and improved efficiency and accuracy in estimating marker effects for quantitative traits than bi-parental linkage mapping (Myles et al. 2009). GWAS has been successfully applied in common bean to analyze the genetic control of resistance to several diseases such as anthracnose, angular leaf spot, or bacterial blight (Choudhary et al. 2018; Perseguini et al. 2016; Wu et al. 2017; Zuiderveen et al. 2016).

The present study was designed to explore the Portuguese common bean natural variation to identify resistance against fusarium wilt and to detect SNP-resistance associations using a GWAS approach. For that, we evaluated a collection of 162 Portuguese common bean accessions for resistance to fusarium wilt under controlled conditions and performed a high-throughput SNP screening of the same collection. SNP-trait associations were tested using a mixed linear model accounting for the genetic relatedness among accessions to identify the genomic regions controlling fusarium wilt resistance. To our knowledge, this is the first time GWAS has been applied to a Portuguese common bean collection and for common bean fusarium wilt resistance.

Materials and Methods

Phenotypic data

Plant material and growing conditions

One hundred and sixty-two Portuguese common bean accessions, belonging to the national *P. vulgaris* germplasm collection, were used in this study. A complete list of the accessions studied along with their passport information is available in Supplementary Table 1. Two Mesoamerican lines – SER16 and Tio Canela-75 – kindly provided by the International Center for Tropical Agriculture (CIAT, Colombia) were also evaluated as international references for comparative purposes. SER16 is an improved bean line for drought resistance (Polania et al. 2016a) and Tio Canela-75 is a commercial variety noted for its resistance to *bean golden mosaic virus* (BGMV), common bacterial blight,

rust, and heat tolerance (Rosas et al. 1997). No previous information on the resistance levels of these accessions to fusarium wilt was available.

Common bean seeds were surface-sterilized for 20 min in a 20% solution of sodium hypochlorite, rinsed two times with sterile water for 20 min and sown in 0.5 L pots filled with sterile vermiculite (1–3 mm diameter). One seed was sown per pot and three to five pots per accession were used. Pots were placed in a growth chamber kept at 27 ± 2°C under a photoperiod of 14 h light (~250 $\mu mol.m^{-2}s^{-1}$) and 10 h dark, and with a relative humidity of 60-70%. In total, 1033 plants were evaluated.

Experimental design

Due to growth chamber space constraints, we used an incomplete block design and the 164 accessions (162 Portuguese, SER16, and Tio Canela-75) were assigned to three independent blocks or experiments. Three to five plants (average 4.5) per accession were evaluated in each experiment and averaged. To correct for the block (experiment) effect, 30 accessions were repeatedly evaluated in all experiments. Additionally, in each of the three experiments, three extra plants from eight accessions under evaluation (24 plants in total per experiment) were randomly chosen and used as non-inoculated controls for symptom comparison.

Fungal isolate and cultural conditions

Fusarium oxysporum f. sp. *phaseoli* isolate FOP-SP1 race 6 was kindly provided by Prof. José María Díaz Mínguez (University of Salamanca, Spain) and stored as micro conidial suspensions at -80ºC in 30% glycerol, for use in all the experiments. This fungal strain was identified in common bean cultivars in Avila, Spain, and classified as highly virulent (Alves-Santos, 2002).

For microconidia multiplication, a protocol adapted from Haglund (1989) and Lichtenzveig et al. (2006) was followed. Briefly, the fungal culture was grown in the dark at 28ºC under constant shaking (170 rpm), for four days, filtered using autoclaved cheesecloths to separate both micro and macro conidia from *F. oxysporum* mycelium and centrifuged at 6000 rpm for 10 min at room temperature. The conidial pellets were re-suspended and a suspension of $5.0x10^6$ $conidia.mL^{-1}$ was prepared to be used on the same day to inoculate the common bean seedlings.

Inoculation and disease assessment

For inoculation, seven-day-old seedlings were removed from the pots, vermiculite was cleaned from the roots, and the roots were then trimmed by a third and immersed for 5 min in the conidial suspension previously prepared, following a modified version of the dipping technique described by Haglund (1989). The non-inoculated control plants were similarly trimmed by a third but roots were immersed in sterile water instead of conidial suspension. Seedlings were replanted in the pots and maintained in the same growth chamber, under the same photoperiod and temperature conditions. All plants were well watered during the experiment using tap water, and once a week were irrigated with Hoagland nutrient solution (Hoagland and Arnon 1938).

Symptoms were assessed at leaf level every three days, from the 7th to the 30th day after inoculation (DAI), a total of eight time points, using a disease severity (DS) visual scale ranging from 1 (healthy leaf) to 5 (dead leaf) (adapted from Bani et al. (2012) and Rispail and Rubiales (2014)). In more detail: 1 – no symptoms; 2 – light to moderate chlorotic symptoms; 3 – leaves completely chlorotic and bright yellow; 4 – chlorotic leaves with wilt and necrosis symptoms; 5 – leaves and branches exhibiting wilt, chlorosis, necrosis, and defoliation, eventually with plant death (Figure 1). Intermediate scale values were given when appropriate. At 30 DAI, accessions were considered resistant if they had a mean DS score (DS30) of 1, intermediate if they had a mean DS score of 2 or 3, and susceptible if they had a mean DS score of 4 or 5.

The progression of fusarium wilt disease was monitored, and DS scores taken per plant at the eight time points were averaged per accession and plotted. Then, a linear regression was fitted to obtain the disease progress rate (DSr) given by the slope of the regression line. The accessions were grouped according to the disease progression profile obtained by the eight time points, and four trends were observed: a) accessions that maintained a low DS (1) along 30 DAI; b) accessions that reached and maintained an intermediate DS score (2-3); c) accessions that reached a high DS score (4-5) in the first two weeks of evaluation; and d) accessions that reached a high DS score (4-5) only 16 DAI or later.

To combine the multiple observations of fusarium wilt disease progress taken over time for each accession into a single value, the area under the disease progress curve (AUDPC) was calculated per plant, and then averaged per accession, using the formula:

$$\text{AUDPC} = \Sigma[(x_i + x_{i+1})/2] * (t_{i+1} - t_i)$$

where x_i = score of disease severity at time i, x_{i+1}= score of disease severity at time i+1, and $t_{i+1}-t_i$ = number of days between scoring times i and i+1. AUDPC scores provided a quantitative summary of fusarium wilt disease severity over 30 DAI for each accession, joining the progression with the extent of disease severity data assessed at multiple observations. Therefore, the higher the AUDPC value, the more susceptible the accession.

Phenotypic data analysis

The results from the three traits DS30 (disease severity score at the last time point 30 DAI), AUDPC, and DSr (disease progress rate) - were compared using Pearson's linear correlation.

To increase the accuracy and repeatability of the association study, the accessions showing DS30 and AUDPC standard deviations higher than 1.5 and 25, respectively, were excluded from the GWAS analysis. This resulted in the removal of 14 accessions.

The linear mixed model applied was *trait (Fusarium wilt DS30, AUDPC, DSr)* = *accession* + *block* + *error*. The assumptions of normal errors and homogeneous error variance were checked. *Accession* is the genotypic term and *block* is the term that identifies the three experiments needed to have all the 150 accessions evaluated. With the 30 accessions that were repeatedly evaluated in the three experiments, the experimental effect was estimated. In a first step, the model was fitted with all terms as random to obtain the best linear unbiased predictors (BLUPs). A restricted maximum likelihood (REML) procedure was conducted to estimate the variance components of the linear mixed model and the broad-sense heritability. In a second step, accessions were fitted as a fixed term and the best linear unbiased estimates (BLUEs) for each accession and trait were produced and used as input phenotypic data in the association mapping analysis. All analyses were performed in Genstat® software, 19th edition (VSN, 2017).

Genotypic data

DNA from one representative individual per accession was isolated from young leaves using DNeasy Plant Mini Kit from Qiagen®. The criteria to select the representative individuals were described in a previous work from our team (Leitão et al. 2017). DNA quantification was performed at 260 nm using a NanoDrop™ ND-2000C

spectrophotometer (Thermo Scientific, USA) and the concentration of all samples was set to values between 50 and 100 ng.μL^{-1}, in a volume of 30 µL. Wavelength ratios at 260/230 and 260/280 nm were examined to assess DNA purity. The DNA quality was also checked in 0.8% SeaKem® LE agarose gels (Cambrex Bio Science Rockland, Inc., USA) stained with SYBR® Safe (Invitrogen, USA).

DNA samples were genotyped using the Illumina Infinium BARCBean6K_3 BeadChip™ assay containing 5,398 SNPs (USDA-ARS, Maryland, USA), designed based on the sequence of *P. vulgaris* 14x and v0.9 *de novo* assemblies (Song et al. 2015), and DArTseq™ analysis (Diversity Arrays Technology sequencing, Canberra, Australia) (Kilian et al. 2012).

Genotypic data analysis

Quality control

Genotypic data quality control was performed by removing SNP markers and accessions with more than 25% of missing data. SNPs called as heterozygous were set as missing data. Moreover, markers with a minor allele frequency (MAF) smaller than 0.01 were removed.

Genetic structure

A subset of 454 SNP markers evenly distributed throughout the common bean genome (average distance between markers of 1.1 Mega base pairs, Mbp) was used to build a similarity matrix to estimate pairwise genetic relatedness among the accessions, as implemented in Genstat software, to calculate principal coordinate scores to study the population structure. The obtained SNP-based structure was compared with the three clusters (Mesoamerican related, Andean related and admixture nature) identified previously using SSR in the same collection (Leitão et al. 2017).

Association mapping

Genome-wide association studies (GWAS) to reveal fusarium wilt DS30, AUDPC, and DSr associated SNPs were conducted using the quantitative trait loci (QTL) library procedures available in Genstat software. The adjusted means (BLUEs) of the three traits were tested for association with 9,825 SNP markers scored in 133 common bean accessions that passed the genotypic and phenotypic quality filters applied.

The association mapping was performed in the mixed-model framework of Genstat software, using the model *Phenotype* = *SNP* + *genotype* + *error*, fitting SNP as fixed and genotype as random terms using REML (Malosetti et al. 2007), with genotype random effects structured following a kinship matrix (*K* matrix). For each chromosome, a different kinship matrix was calculated using only the SNPs located on the remaining 10 chromosomes, as proposed by Cheng et al. (2013). The procedure was performed using the kin function of R package synbreed (Wimmer et al. 2012) and the Van Raden measure (Van Raden 2008). The genome-wide marker-trait association scan was conducted by testing the significance of the marker effect using a marginal Wald test at each SNP location. The observed $-\log_{10}$ (*P*-value) of each SNP was plotted against their chromosomal positions to produce a Manhattan plot. Using a threshold level of $-\log_{10}$ (*P*-value) = 3, the significant marker-trait associations were depicted. This threshold was set to discard the background noise obtained in the Manhattan plot without compromising the identification of potentially interesting regions, which would be missed by the overly stringent and conservative Bonferroni-corrected threshold of significance. However, as a "conservative" guidance, two additional approaches were followed. On one hand, a LD adjusted Bonferroni-corrected threshold (α/k), considering an α = 0.05 and setting the effective number of independent tests as the number of LD blocks per chromosome (*k* = 520) was calculated (Dugal et al. 2008). Additionally, adjusted *P*-values following the Benjamini and Yekutieli (B-Y) false discovery rate (FDR) method (Benjamini and Yekutieli 2001) were also calculated, in this case with α = 0.2 and *k* = 520, to control type I errors due to multiple testing.

For every SNP significantly associated with fusarium wilt DS30, AUDPC, and DSr progress rate, the effect of the allele variant in relation to the most frequent allele was calculated. The proportion of variance explained by each SNP-trait association was estimated using the formula V_{QTL}/V_{pheno}, where V_{QTL} = 2freq(1-freq)effect^2 and V_{pheno} is the phenotypic variance of the adjusted means of each trait (Resende et al. 2017). The relation between the frequency of each trait-associated SNP allele, the resistance level and the gene pool of origin of the accessions (Leitão et al. 2017) was also investigated.

Local linkage disequilibrium and candidate gene identification

A gene was considered a putative candidate gene for fusarium wilt DS30, AUDPC, or for DSr progress rate if it contained a significant associated SNP (threshold for significance $-\log_{10}$(*P*-value) ≥ 3) or if it was in linkage disequilibrium (LD) with a SNP

significantly associated with the trait. LD was calculated for each chromosome as a measure of the recombination history, using the squared coefficient of correlation between marker pairs, r^2, after correcting for population structure with the principal component scores from Eigenanalysis, as implemented in Genstat software. For this calculation, the entire set of SNPs was used. Average intra-chromosomal LD decay per chromosome was visualized by plotting r^2 against the physical mapping distance in Mb. To consider the existence of adjacent SNP markers in LD with the ones identified as significantly associated with the trait, the r^2 of the neighboring SNPs was investigated, bearing in mind a strict threshold of LD decay ($r^2 > 0.2$). The location of these adjacent SNPs in LD with the significantly associated ones was used to define an LD block and to browse for putative candidate genes mapped within those genomic regions.

The common bean genome sequence, from the Andean common bean accession G19833 (Schmutz et al. 2014), was investigated using the *JBrowse* tool in the *Phaseolus vulgaris* v2.1, available at the Phytozome v12 portal (DOE-JGI and USDA-NIFA, http://phytozome.jgi.doe.gov/). The functional annotation of the genes under the identified genomic regions was given by *KEGG*/*KOG*/*PFAM*/*PANTHER*/*Gene Ontology (GO)* databases identifiers, which were used to make inferences about the pathways involved and the possible role of the common bean candidate genes in the control of Fusarium infection.

Results

Fusarium wilt disease severity variation

To determine the progression of fusarium wilt symptoms among the common bean accessions, disease severity (DS) was scored eight different times during 30 DAI. By 10 DAI, 29 accessions presented leaves already completely chlorotic with visual symptoms of necrosis and were classified as DS 4 (susceptible), whereas 64 accessions still displayed DS 1 (resistant), and 71 DS 2-3 (intermediate). At 20 DAI all five DS scoring values were found among the collection showing the variability of responses among the Portuguese common bean germplasm. By 30 DAI 78 accessions displayed DS 4-5, with 55 of them dead. The Mesoamerican line Tio Canela-75 was included in this group (DS 4). On the other hand, at this final time point, 14 accessions (9%) were considered completely resistant (DS ≤ 1.5) and 72 accessions (44%) were considered intermediate, with leaves showing different levels of chlorosis but no necrosis (DS 2-3) (Figure 2). This last group included the Mesoamerican line SER16 (DS 3).

Additionally, the eight DS scores per accession were plotted (Supplementary Figure 1). Out of the 78 susceptible accessions, 60 reached a high DS score (4-5) within the first two weeks after inoculation, while 18 only showed high DS scores 15 DAI or later. On the other hand, the 14 accessions considered resistant (DS ≤ 1.5) maintained the low DS score during the entire experimental time frame. Finally, the 70 intermediate accessions reached and maintained DS values between 1.6 and 3.5. Fusarium wilt progress rate (DSr), given by the slope of the DS scores regression, ranged from a minimum of 0.000 (resistant accession)to a maximum of 0.571 (susceptible accession) (Supplementary Table 2).

With the fusarium wilt DS values scored every three days during 30 DAI, the area under disease progress curve (AUDPC) was calculated for each accession and their frequency distribution plotted (Figure 3).

Fusarium wilt AUDPC mean values per accession ranged from 27.8 to 105.1. The frequencies of AUDPC classes followed a bimodal distribution, with two AUDPC peaks, indicating a clear discrimination between resistant (low AUDPC values) and susceptible (high AUDPC values) accessions. Accessions having an AUDPC value below 30 were regarded as resistant, those with AUDPC between 31 and 69 intermediate, and those with AUDPC above 70 susceptible. A complete list of AUDPC values per accession is available in Supplementary Table 3.

The 14 accessions considered resistant – simultaneously with DS ≤ 1.5, AUDPC < 30, and DSr < 0.100 - were either of Andean or admixed origin. Nevertheless, within the intermediate and susceptible accessions, it was possible to identify both Andean and Mesoamerican gene pools in addition to the admixed origin (Figure 4 and Table 1).

Pearson's coefficients revealed strong pairwise correlations between DS measured at 30 DAI (DS30), AUDPC, and disease progress rate values (DSr) (Table 2).

Phenotypic data variance components and broad sense heritability

The examination of the histogram of residuals, residuals versus fitted values of the model and the expected versus normal quantiles (Q-Q) plot revealed a random pattern of residuals for the three traits (DS30, AUDPC, and DSr) further used in GWAS (Supplementary Figure 2).

The REML estimators of the variance components of the linear model were obtained with accession and block as random terms, and broad-sense heritability, calculated as the ratio of the genotypic variance to the total phenotypic variance (genetic plus error)

(Table 3). With accession term fixed, Wald statistics indicated very strong evidence for differences between accessions (*P*-value < 0.001) for DS30 and AUDPC, and less strong but still significant differences between accessions for DSr (*P*-value = 0.003).

Association panel genetic structure

Based on the 454 selected SNP markers, two main groups of accessions were visualized using principal coordinate analysis (Figure 5). The variance explained by the first two principal coordinates was 65.71%. The observed clustering on the 133Portuguese accessions was in accordance to their genetic proximity to the two original common bean gene pools in Mesoamerica and in the Andes. Intermediate resistant and susceptible accessions were identified within the accessions more related to the Andean and Mesoamerican gene pools and also among the accessions of admixture origin, whereas the resistant accessions were all of Andean or admixed origin.

Marker-traits associations

Illumina Infinium BARCBean6K_3 BeadChip™ assay and DArTseq™ analysis genotyped together 16,689 SNPs. After quality control, a total of 9,825 SNPs and 133 accessions were used in the association mapping study.

For the three traits under analysis - Fusarium wilt disease severity at 30 DAI (DS30), AUDPC, and disease progress rate (DSr) - the distribution of the $-\log_{10}$(*P*-values) from marginal Wald tests was investigated by Q-Q plots (Supplementary Figure 3). Some deflation of the test statistic was observed (*P*-values are slightly under the expected y=x line), but the points corresponding to the significant associations clearly stand out at the high end of the plots (Supplementary Figure 3).

The results from the association mapping revealed nine SNPs significantly associated (using $-\log_{10}$(*P*-value) ≥ 3) with resistance on chromosomes Pv04, PV05, Pv07 and Pv08 (Figure 6 and Table 4). The LD adjusted Bonferroni corrected *P*-value set the threshold as $-\log_{10}$(*P*-value) = 4.0. The use of this threshold would render the previously detected associations as suggestive. The Benjamini-Yekutiely *P*-values adjustment was found to be highly stringent for all the associations.

By inspecting the allelic variant effect on fusarium wilt resistance of the associated SNPs it was possible to identify the most promising SNPs for marker-assisted selection

(SNPs strongly associated and with higher effect on the trait variation). Nevertheless, each of the SNP-trait associations identified for DS30, AUDPC, and DSr only explained a small portion of the observed phenotypic variance (Table 4).

From the seven significant associations detected for both fusarium wilt DS30 and AUDPC on chromosomes Pv04, Pv07 and Pv08 (Figure 6), DART03480 on chromosome Pv04 had the highest $-\log_{10}(P\text{-value})$ = 3.79 and 3.84, respectively. The associated SNPs that explained the biggest proportion of variance (7.18% in DS30 and 7.02% in AUDPC) were SNP03304 and SNP03306 on chromosome Pv07 (Table 4).

The allelic variant of four of these seven associated SNPs caused a negative effect in fusarium wilt DS30, in relation to the most frequent allele, meaning that they contributed to an increase in disease resistance. The absolute value of the allelic variant effect was for all the DS30 associated SNPs close to 0.5 in the DS score scale. This corresponded to an increase (or decrease for the SNPs whose allelic variant had a negative effect) in 15% to the DS30 mean value (3.2) of the collection.

Similarly to DS30, the allelic variant of the associated SNPs located in chromosomes 7 and 8 caused a negative effect in fusarium wilt AUDPC, contributing to an increase in fusarium wilt disease resistance. The absolute value of the allelic variant effect was for all the AUDPC associated SNPs close to 11 AUDPC units. This corresponded to an increase (or decrease for the SNPs whose allelic variant has a negative effect) in 17% to the AUDPC mean value (63) of the collection.

The SNPs associated with fusarium wilt progression rate (DSr) on chromosomes Pv07 were the same associated with DS30 and AUDPC. However, the two associations on chromosome Pv05 were unique for DSr. From the five associations detected for DSr (Figure 6), DART04561 on chromosome Pv05 had the highest $-\log_{10}(P\text{-value})$ = 3.40. This SNP also explained the biggest proportion of variance (6.44%) in this trait (Table 4).

All the allelic variants of four out of five associated SNPs caused a negative effect in fusarium wilt DSr, in relation to the most frequent allele, meaning that they contributed to a decrease in the disease progress rate. The exception was DART04561 with a positive effect in DSr. The absolute value of the allelic variant effect for all the DSr associated SNPs was close to 0.05. This value corresponds to a decrease in 19% to the DSr mean value (0.264) of the collection.

SNP allelic variant frequency among gene pool of origin of accessions

The frequency of the favorable allele (providing an increase in resistance) in the nine associated SNPs was different within the gene pool of origin of the Portuguese accessions (Figure 7). The accessions of Mesoamerican origin had, on average, a higher frequency of the favorable alleles than the ones of Andean origin for the SNPs associated in chromosomes Pv04, and Pv07. The most contrasting frequency values were observed for SNP02051, located in chromosome Pv05, for which the frequency of the favorable allele was much higher (0.72) within the accessions of Mesoamerican origin, than within the accessions of Andean or admixed origin (freq. = 0.20). On the other hand, the favorable alleles of DART07926 associated in chromosome Pv08 and DART04561 in chromosome Pv05 were more frequent in the accessions of Andean origin. The average frequency of the favorable allele in the accessions of admixture origin was in most cases intermediate between the accessions of Andean and Mesoamerican origin. Additionally, the frequency of the favorable allele was always above 0.75 for the associated SNPs located on chromosome Pv04, regardless of the gene pool of origin of the accessions.

Candidate genes identification

The LD decay to r^2 = 0.1 per chromosome varied from 0.5 (on chromosome Pv07) to 1.8 Mb (on chromosome Pv09), with an average graphically estimated of 1.1 Mbp. Supplementary Figure 4 shows the LD decay, measured as r^2 values versus marker distance, and shade plots per chromosome with the correlation between markers highlighted using a color range code.

After identifying the SNPs significantly associated with fusarium wilt response – using the traits AUDPC, disease severity at 30 DAI (DS30), and disease progress rate (DSr) – and the neighboring SNPs in LD, their locations were used to search for putative candidate genes in the *P. vulgaris* genome v2.1 (Table 7). Candidate genes were identified for six of the nine SNP-trait associations.

Out of those six candidate genes identified, two (Phvul.004G006800 and Phvul.007G270000) encoded proteins involved in the inducible plant response to pathogens, such as phytoalexins biosynthesis and hypersensitive reaction. The others were related to amino acids and secondary metabolite biosynthesis (Phvul.004G000800), pre-mRNA splicing (Phvul.007G270500), signaling of plant immune receptors (Phvul.008G196600) and plant translational regulation and stress adaptation (Phvul.008G203200).

Discussion

The continuous search for new sources of resistance in underexplored plant germplasm collections and the study of their genetic basis is essential for the development of tools to support the breeding of new common bean cultivars with durable resistance to fusarium wilt. By exploring the natural variation of 162 accessions representative of the Portuguese common bean germplasm, we found 14 new sources of complete resistance and 71 new sources of incomplete resistance against the highly virulent *Fusarium oxysporum* f. sp. *phaseoli* isolate FOP-SP1 race 6. Complete and incomplete resistant sources were identified among accessions of Andean, Mesoamerican and genetic admixed origin that constitute the Portuguese germplasm.

Additionally, we identified nine SNPs with small effects associated with this natural variation and six candidate genes, suggesting an oligogenic control of the detected resistances. The identified favorable SNP alleles controlling fusarium wilt resistance will facilitate the resistance transfer into more productive elite cultivars using marker-assisted breeding schemes.

As far as we know, this was the first time that sources of resistance to this particular *Fop* isolate, classified as one of the most virulent in a pathogenicity screening of 16 isolates from Spain and Greece (Alves-Santos et al. 2002), were identified in European common bean germplasm. This was also the first GWAS dedicated to common bean fusarium wilt response and the first report of a GWAS using a panel of Portuguese common bean germplasm.

Different responses were detected among the Portuguese common bean accessions when inoculated with FOP-SP1 race 6, revealing the high variation present within the collection. Thirty days after inoculation, the accessions were categorized from completely resistant (9%) to susceptible (48%), with many intermediate cases (43%) that showed leaves with chlorosis that did not progress to necrosis. Two patterns of disease progression were observed among the susceptible accessions – a fast disease progress rate with accessions reaching high disease severity scores (4-5) within the first two weeks after inoculation, and a slower disease progression with accessions reaching the same high scores but only 16 DAI or later. There is a lack of information on how and where this delay takes place in the host-pathogen interaction (Garcés-Fiallos et al. 2017). In the case of the resistant accessions, no external disease symptoms were

observed and they kept their green leaves and a typical development throughout the experiment. Resistant and intermediate accessions have been described as either chemically inhibiting the hyphae growth or physically blocking the conidia spreading up the sap stream (Abawi and Pastor Corrales 1990; Garcés-Fiallos et al. 2017; Niño-Sánchez et al. 2015; Xue et al. 2015). This impairment may occur through the formation of papilla structure, cell wall strengthening, and vessels crushing, as it was described for other legume species (Bani et al. 2018; Cachinero et al. 2002; Castillejo et al. 2015; Grayer and Kokubun 2001). A histological analysis will be needed in the identified common bean resistant accessions to elucidate the underlying physiological mechanisms.

The identification of new sources of resistance to this common bean disease is of extreme importance since the existing ones provide only moderate or incomplete levels of protection to specific *Fop* races isolates (Buruchara and Camacho 2000; Pereira et al. 2013; Salgado 1995; Schwartz and Otto 2005). Moreover, previous studies on Spanish widely cultivated common bean cultivars (of Andean origin), and on other cultivars from CIAT that have been used for race determination in *Fop*, revealed the high virulence of FOP-SP1 race 6 isolate (Alves-Santos et al. 2002). All the screened cultivars in that study were susceptible to this isolate, even the ones that had been described as resistant against other *Fop* isolates.

The Mesoamerican lines from CIAT, SER16 and Tio Canela-75, used in our study for international comparison, were found intermediate and susceptible, respectively. This suggests that SER16, a recognized drought-tolerant elite line (Polania et al. 2016b), may also contain genes (common or not to drought tolerance) that confer resistance to this *Fop* race. Indeed, a transcriptomic analysis revealed that drought stress and vascular pathogen infection induced in chickpea shared differentially expressed genes associated to the cell wall and alkaloids biosynthesis, defense related-proteins and osmoprotectants (Sinha et al. 2017). This might indicate that some of the mechanisms induced by common bean in response to both stresses are coincident, but requires further investigation.

In the present study, we observed a range of plant responses to fusarium wilt inoculation from highly resistant to highly susceptible. Such continuity supports the existence of quantitative resistance mechanisms in common bean against *Fop* race 6. This quantitative nature was already suggested for *Fop* race 4 (Cross et al. 2000), with the involvement of several genes, each contributing a small to moderate effect in the

resistance level. Both dominant monogenic and oligogenic resistance to *F. oxysporum* have been observed in various legume species (Infantino et al. 2006; Rispail and Rubiales 2014; Sharma et al. 2005). In common bean, studies of the inheritance of resistance to fusarium wilt have been performed using segregating populations derived from contrasting cultivar crosses and *Fop* races isolated from particular geographical regions (Batista et al. 2017; Fall et al. 2001; Xue et al. 2015). Some major resistance genes and quantitative trait loci (QTLs) were identified against *Fop* races 1 and 3, while against race 4 recessive and polygenic resistance were also reported (Fall et al. 2001; Schwartz and Otto 2005). More recently, Batista and colleagues (2017) classified common bean resistance to a putative new *Fop* race as dominant and governed by a few major genes and polygenes.

In our study, we identified a total of nine different associated genomic regions using a $-\log_{10}(P\text{-value}) \geq 3$ (marginal Wald test). Three of the nine SNPs were associated with the three traits DS30, AUDPC and DSr; other four with both DS30 and AUDPC, and two only with DSr, totalizing 19 SNP-trait associations. Considering the more stringent LD-adjusted Bonferroni correction, these detected associations are to be considered only as suggestive associations. Nevertheless, looking at the Manhtattan plots, the threshold of $-\log_{10}(P\text{-value}) = 3$ was clearly above the associations background noise, and, on the other hand, the QQ plots didn't show much inflation, reassuring the interest of the nine detected associated genomic regions. Probably, the complexity of the measured traits, potentially controlled by multiple genes with small effects on the fusarium wilt resistance, together with the relatively small association panel has hampered the power to detect SNP-trait associations (Korte et al. 2013, Pasam et al 2012). Still, the associations detected in the present study were useful for identifying candidate loci related to disease resistance. These candidates need now to be validated by gene expression functional studies in contrasting accessions, and in follow-up studies using different genetic backgrounds or different environments, or through the development/use of segregating bi-parental populations (Ioannidis and Daly 2009).

Most of the associations were coincident for the three traits analyzed (AUDPC, DS30 and DSr), reflecting the high correlation between the traits. Two associations were unique for DSr both on chromosome Pv05. Although the proportion of the observed phenotypic variance explained by each significant SNP-trait associations ranged from 4.7% to 7.2%, the favorable allele of the associations with the highest effect corresponded to an increase in fusarium wilt resistance of 16% and a reduction in the

610 disease progress rate of 19%. This suggests that, even with moderate traits
611 heritabilities (0.72 for DS30, 0.70 for AUDPC and 0.41 for DSr) due to the high influence
612 of the environmental variability, improvements can be attained through selection within
613 this Portuguese germplasm.
614 The average frequency of the favorable allele of the nine SNPs associated with
615 fusarium wilt resistance varied according to the gene pool of origin of the common bean
616 accessions. For most of the resistance associated SNPs, the accessions of
617 Mesoamerican origin had higher frequencies of the allele conferring resistance, with the
618 exception of the two associated SNPs identified in chromosome Pv08. This indicates
619 that there is room within the accessions of Andean origin to improve their resistance, by
620 introgression of interesting resistance alleles from Mesoamerican lines into Andean
621 breeding germplasm. However, the smaller number of Portuguese accessions of
622 Mesoamerican origin in the association panel in relation to the accessions of Andean
623 origin (25 versus 97) could have biased these results. Although the Portuguese
624 common bean germplasm is predominantly constituted by accessions of Andean origin,
625 one-third of the accessions have admixed genetic origin and might represent putative
626 hybrids among gene pools from the two original centers of domestication (Leitão et al.
627 2017). Thus, not only the accessions of Andean or Mesoamerican origin identified as
628 resistant to *Fop* infection may be useful for common bean resistance breeding within
629 each particular gene pool, but also the resistant accessions with admixture nature may
630 contain novel and advantageous genetic combinations for both gene pool breeding. We
631 identified among the accessions of admixed genetic origin favorable SNP alleles for
632 fusarium wilt resistance that can reflect a positive selection contributing to adaptation to
633 the local environment. It is known that co-evolution of host and pathogens has led to the
634 development of isolates that infect mainly the common beans from one particular gene
635 pool (Geffroy et al. 1999; Kelly et al. 2003). The development of common bean cultivars
636 with pyramided genes for *Fop* resistance identified in common bean accessions from
637 different origins is accordingly an effective strategy for durable resistance because the
638 pathogen cannot easily overcome the resistance conferred by several genes (Batista et
639 al. 2017; Miklas et al. 2006).
640 Six of the nine resistance-associated SNPs were located within putative candidate
641 genes, according to the common bean reference genome (v2.1). DART03480 and
642 SNP01469, both on chromosome Pv04, were located within genes that code for a
643 pyruvate kinase protein and for a nuclear pore membrane glycoprotein (Nup210, gp210-

related), respectively. Pyruvate kinase is an enzyme that catalyzes the conversion of phosphoenolpyruvate and ADP to pyruvate and ATP in glycolysis and plays a role in amino acids and secondary metabolites (such as terpenes) biosynthesis (Ambasht and Kayastha 2002). Several studies reported the accumulation of amino acids such as valine, leucine, and tyrosine during plant defense responses; however, knowledge on the mechanisms behind the reconfiguration of the plant metabolism when facing a pathogen is still scarce (Rojas et al. 2014). On the other hand, the role of nuclear pore complex (NPC) in nucleo-cytoplasmic trafficking has been described not only in growth and developmental processes but also in plant response to biotic stresses (Cheng et al. 2009; Yang et al. 2017). For example, in *Nicotiana benthamiana* a nuclear pore protein (NbNup75) is involved in ethylene signaling and induction of defense responses such as the production of phytoalexins or programmed cell death that limits the pathogen spread (Ohtsu et al. 2014). Of note, the release of phytoalexins to inhibit fusarium wilt progression was already described in chickpea and pea (Bani et al. 2018; Cachinero et al. 2002). Furthermore, in Arabidopsis, the transmembrane nucleoporin CRP5 (Constitutive Expresser of Pathogenesis-Related Genes 5) associates with NPC and regulates an essential inhibitory mechanism of ETI/PCD (ethylene-triggered immunity/programmed cell death) (Gu et al. 2016), vital for host recognition of pathogen virulence effectors to induce defense. SNP03305, on chromosome Pv07, was also located within a candidate gene that codes for a protein involved in programmed cell death (pre-rRNA processing protein Rrp5). Programmed cell death is a well-described mechanism in plant-pathogen interactions (Huysmans et al. 2017) with an important role in resistance response. In fact, the hypersensitive response (HR), eliciting localized cell death at the site of the pathogen attack, is often triggered to restrict biotrophic and hemibiotrophic fungi growth and had already been observed in different *F. oxysporum*-plant interactions (Cachinero et al. 2002; Chen et al. 2014; Swarupa et al. 2014; Xue et al. 2015).

Interestingly, some of the loci associated with fusarium wilt detected in this study were located in genomic regions that have been previously associated with resistance to other diseases in common bean. For instance, the already referred SNP01469 and SNP01487 (with no candidate gene associated), co-localized, on chromosome Pv04, with a major QTL for bacterial resistance. This QTL (HB4.2) confers resistance to multiple races of *Pseudomonas syringae* pv. *phaseolicola*, the bacterium that causes halo blight (Tock et al. 2017). Within the mapping interval of this QTL, some genes were

identified and predicted to encode proteins with nucleotide-binding site and leucine-rich domains (NBS-LRR), known to enable pathogen detection and defense signaling and typically associated with hypersensitive cell death (Tock et al. 2017). In the same genomic region, the *Co-34/Phg-3* locus, which confers resistance to leaf angular spot caused by *Pseudocercospora griseola* (Sacc.), was also identified (Valentini et al. 2017). Among the candidate genes for the *Co-34/Phg-3* locus, one contains the serine/threonine kinase domain whose function has been correlated to HR and H_2O_2 accumulation (Cao et al. 2011). In our study, we did not microscopically analyze the roots after *Fop* inoculation. That could be a required follow-up histological task to monitor and confirm the presence of hypersensitive cell death in the resistant accessions of the Portuguese collection.

DART04561 on chromosome Pv05, and SNP03306 on chromosome Pv07 are located within candidate genes that code for pre-mRNA related proteins. While the first coded for a prp39-related protein of unknown function, the second is located within a candidate gene that codes for pre-mRNA splicing factor prp19-related. Alternative splicing has been described as an important mechanism in DNA damage response, plant immunity and defense (Lenzken et al. 2013; Shang et al. 2017; Yang et al. 2014). Moreover, in *Arabidopsis thaliana*, the role of the spliceosomal component prp19 was linked to pathogen defense (Meyer et al. 2015).

DART07926 (on chromosome Pv08) is located within a candidate gene that coded for a reticulon-like protein b1 (RTNLB1)-related, whose absence was described to increase susceptibility to pathogens in *Arabidopsis* by regulating the intracellular trafficking and activity of bacterial flagellin immune receptor (FLS2) (Lee et al. 2011). Downstream to FLS2, essential signal transduction events by mitogen-activated protein kinase (MAPK) cascades are well known to confer resistance to both bacterial and fungal pathogens (Asai et al. 2002), including *F. oxysporum* (Wang et al. 2015).

In addition to the already mentioned SNP01487, SNP2051 (on chromosome PV05) and SNP03304 (on chromosome Pv07) had no associated candidate gene. Nevertheless, these SNPs might still be useful to select for resistance to *Fop* in common bean breeding. The absence of annotated candidate causal genes at these loci might be due to genetic variability between the Portuguese common bean accessions and the Andean accession whose genome was used as reference (accession G19833).

The nine identified SNP-trait associations provided valuable insights into the genetic basis of fusarium wilt resistance but only explained a fraction of the total phenotypic

712 variance. The success of association mapping in identifying markers effectively
713 associated with the trait under study relies on how well the population structure is
714 corrected in the association model and on the existing levels of linkage disequilibrium
715 (LD). Linear mixed models can successfully correct for genetic relatedness between
716 individuals in a population by incorporating a kinship matrix into the model and have
717 been widely used in genome-wide association studies (Kang et al. 2010; Korte et al.
718 2012; Zhang et al. 2010). In common bean, LD levels were found to be stronger within
719 the Mesoamerican gene pool and decay more rapidly within the Andean gene pool
720 (Blair et al. 2018). In the Portuguese common bean collection analyzed here, the
721 average intra-chromosomal LD decayed to 0.1 r^2 within 1.13 Mbp. A similar LD decay
722 to 0.1 r^2 within 1 Mbp was reported recently for a common bean panel constituted by 27
723 Andean and 153 Mesoamerican accessions and using 10,326 SNPs (Diniz et al. 2018).
724 Using 9,825 SNP markers, a significant part of the genome was covered (1 SNP/55.3
725 kbp), although increasing the number and distribution of markers would increase the
726 probability of identifying additional markers in high LD with any QTL linked to the trait.
727 Additionally, since we are likely dealing with a polygenic trait with trait-associated
728 variants each with a small effect, increasing the sample size, and thus maximizing the
729 phenotypic diversity amongst accessions, would improve the power to recover
730 meaningful associations. In spite of that, most of the associated SNPs detected in our
731 study were located inside or near candidate genes related to resistance, which reinforce
732 the usefulness of the association panel used.
733 The associated SNPs and putative candidate genes identified in the current study
734 increase the number of functional markers available to facilitate resistance breeding in
735 this major crop. Next steps will include the validation of the usefulness of the SNPs
736 associated with fusarium wilt resistance identified here, in controlled conditions in the
737 field using multi-locations and different years. It will be also interesting to evaluate the
738 level of resistance of this germplasm against other strains of *Fop* than FOP-SP1 race 6
739 to enhance the insights on the resistance mechanisms and genetic control against
740 fusarium wilt on this underused germplasm. That information is needed to understand if
741 the putative candidate genes found here are only involved in the resistance to this
742 specific *Fop* isolate or if, on the other hand, they present broader resistance to different
743 isolates. We chose FOP-SP1 race 6 since this isolate was already well described and
744 characterized in our neighbor country Spain with a proven high virulence. However, it

745 would be of interest to monitor the presence of fusarium wilt in Portuguese fields and
746 perform characterization and pathogenicity tests using the local isolates.
747 With the present study, we unveil the potential of the natural variation of the Portuguese
748 common bean germplasm for fusarium wilt resistance. New sources of resistance and
749 incomplete resistance to a highly virulent *Fop* strain were identified on this germplasm
750 under an oligogenic control. The associated functional molecular markers detected will
751 support an effective marker-assisted common bean breeding for more durable
752 resistance against fusarium wilt.
753

754 **Acknowledgments**

755

756 The authors are grateful to Steve Beebe, from CIAT Genetic Resources Unit, for
757 providing seeds from the common bean lines SER16 and Tio Canela-75. We also
758 acknowledge Maria Manuela Veloso, from Research Unit of Biotechnology and Genetic
759 Resources, INIAV, Oeiras, Portugal, for the multiplication of the Portuguese common
760 bean collection. Sincere thanks are due to Moustafa Bani and Nicolas Rispail, from IAS-
761 CSIC, Spain, for help and valuable advice during the fungal inoculations. We are also
762 grateful to Perry Cregan, from USDA-ARS, for providing support on Illumina BeadChip
763 technology. Financial support by Fundação para a Ciência e Tecnologia (FCT),
764 Portugal, is acknowledged through grant SFRH/BD/92160/2013 (STL), IF/01337/2014
765 FCT Investigator contract (MCVP), research project BeGeQA (PTDC/AGR-
766 TEC/3555/2012) and research unit GREEN-IT (UID/Multi/04551/2013). Short-Term
767 Scientific Missions in the scope of the European Framework Programme COST Actions
768 FA1208 and FA1306 are also acknowledged.
769

770 **Authors' contributions**

771

772 STL performed the DNA isolation, conducted the fungal inoculations and symptoms
773 evaluation, the genotypic and phenotypic data analysis, participated in the genome-wide
774 association analysis and drafted the manuscript. MM and FvE participated in the
775 molecular and phenotypic data processing and performed the genome-wide association
776 analysis. FvE also participated in the revision of the manuscript. QS developed the
777 Illumina Infinium BARCBean6K_3 BeadChip™ assay and provided the SNP
778 genotyping. DR coordinated the fungal inoculation experiments, contributed to the

779 interpretation of the results and in the revising of the manuscript. MCVP designed and
780 coordinated the study, participated in the discussion of results and in the drafting and
781 revising of the manuscript. All authors read and approved the final manuscript.
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Tables

Table 1: Response of Portuguese common bean accessions to fusarium wilt according to the gene pool of origin. The minimum and maximum AUDPC values and disease severity scored at 30 DAI are shown.

Gene pool of origin[a]	AUDPC range	Number of common bean accessions		
		DS30 = 1 Resistant	DS30 = 2-3 Intermediate	DS30 = 4-5 Susceptible
Andean	27.8 – 104.8	11	40	50
Mesoamerican	32.1 – 102.4	0	14	13
Admixed	27.8 – 105.1	3	12	8

[a]Gene pool of origin resulting from the structure analysis performed together with gene pool representatives (Leitão et al. 2017)

1063 **Table 2:** Pearson's linear correlations between disease severity scored at 30 DAI
1064 (DS30), AUDPC, and disease progress rate (DSr), measured in 162 Portuguese
1065 common bean accessions.

	DS30	AUDPC	DSr
DS30	-		
AUDPC	0.9683	-	
DSr	0.8152	0.7019	-

1066

Table 3: Variance components and broad-sense heritability for the three traits measured in 148 Portuguese common bean accessions.

	Variance components			h^2 heritability (%)
Trait	$\sigma^2_{genotype}$	σ^2_{block}	$\sigma^2_{residual}$	
DS30	1.0517	0.3812	0.475	71.5
AUDPC	406.9	143.1	201.5	69.6
DSr	0.00877	0.00000	0.0185	40.8

Table 4: SNP associations ($-\log_{10}$(*P*-value) ≥ 3) with fusarium wilt DS30, AUDPC, and DSr, marker position within chromosomes, allelic reference and allelic variant for the associated SNP, minor allele frequency, the effect of the allelic variant, and the proportion of phenotypic variance explained by each associated SNP detected using a panel of 133 Portuguese common bean accessions.

Marker name	Trait	$-\log_{10}$(*P*-value)	Original *P*-value	[a]Adjusted BY *P*-value	[b]Chr	Position (Mbp)	Allelic reference	Allelic variant	Minor allele frequency	[c]Effect of the allelic variant	[d]V_{QTL}/V_{pheno}
DART03480	DS30	3.79	1.625×10^{-4}	5.630×10^{-5}	Pv04	0.0521	G	T	0.19	0.565	0.0602
	AUDPC	3.84	1.457×10^{-4}	5.630×10^{-5}						11.54	0.0610
SNP01469	DS30	3.37	4.262×10^{-4}	3.378×10^{-4}	Pv04	0.4735	C	A	0.18	0.545	0.0544
	AUDPC	3.32	4.779×10^{-4}	3.378×10^{-4}						11.16	0.0555
SNP01487	DS30	3.32	4.810×10^{-4}	3.941×10^{-4}	Pv04	2.040	C	A	0.16	0.605	0.0593
	AUDPC	3.25	5.673×10^{-4}	3.941×10^{-4}						12.13	0.0579
DART04561	DSr	3.40	3.975×10^{-4}	5.630×10^{-5}	Pv05	4.433	A	G	0.33	0.05631	0.0644
SNP02051	DSr	3.05	8.966×10^{-4}	2.251×10^{-4}	Pv05	4.781	A	G	0.27	-0.05003	0.0472
SNP03304	DS30	3.67	2.133×10^{-4}	1.689×10^{-4}	Pv07	39.04	C	T	0.23	-0.574	0.0718
	AUDPC	3.65	2.222×10^{-4}	1.689×10^{-4}						-11.59	0.0709
	DSr	3.19	6.395×10^{-4}	1.126×10^{-4}						-0.05689	0.0526
SNP03305	DS30	3.51	3.061×10^{-4}	2.815×10^{-4}	Pv07	39.11	G	A	0.24	-0.562	0.0702
	AUDPC	3.43	3.709×10^{-4}	2.815×10^{-4}						-11.37	0.0697
	DSr	3.00	9.987×10^{-4}	2.252×10^{-4}						-0.05502	0.0502
SNP03306	DS30	3.67	2.133×10^{-4}	2.252×10^{-4}	Pv07	39.17	C	T	0.23	-0.574	0.0718
	AUDPC	3.51	3.059×10^{-4}	2.252×10^{-4}						-11.59	0.0709
	DSr	3.19	6.395×10^{-4}	1.689×10^{-4}						-0.05689	0.0526
DART07926	DS30	3.71	1.974×10^{-4}	1.126×10^{-4}	Pv08	54.08	A	T	0.23	-0.571	0.0702
	AUDPC	3.65	2.221×10^{-4}	1.689×10^{-4}						-10.91	0.0621

[a]Adjusted *P*-value for multiple comparisons according to Benjamini-Yekutieli approach.[b]*P. vulgaris* chromosome. [c]A positive effect of the allelic variant represents an increase in susceptibility, while a negative effect represents an increase in resistance to fusarium wilt. [d]Proportion of the variance explained by each SNP-trait association, V_{QTL}= 2freq(1-freq)effect^2 and V_{pheno} = phenotypic variance of the adjusted means of each trait

Table 5: Putative candidate genes based on the gene annotation for the *P. vulgaris* genome v2.1, and on the reference resources for gene and protein annotation, grouped according to the positive or negative effect of the variant allele (SNP allele with minor

1083 frequency) in fusarium wilt disease response. The traits evaluated were fusarium wilt
1084 disease severity at 30 DAI (DS30), AUDPC and disease progress rate (DSr).

Trait	Marker name (location)	Associated Gene Model RefGen *Phaseolus vulgaris* v.2.1	Protein annotation (databases indicated)
	Markers whose minor allele frequency SNP variant ***increases fusarium wilt susceptibility***		
AUDPC, DS30	**DART03480** (Pv04: 52137 bp)	**Phvul.004G000800** Location (bp): Pv04:50533..54214	Pyruvate kinase family protein Pfam:PF00224, PANTHER:PTHR11817, KEGG_ENZYME:2.7.1.40
AUDPC, DS30	**SNP01469** (Pv04: 473538 bp)	**Phvul.004G006800** Location (bp): Pv04: 470002..498184	Nuclear pore membrane, glycoprotein Nup210 (NUP210, GP210), Pfam:PF02368, PANTHER:PTHR23019, KOG1833
AUDPC, DS30	**SNP01487** (Pv04: 2040423 bp)	no candidate gene	
DSr	**DART04561** (Pv05: 4432986 bp)	**Phvul.005G043100** Location (bp): Pv05: 4428398..4433095	F1C9.34 Pre-mRNA processing protein PRP39-related, PTHR17204:SF28
	Markers whose minor allele frequency SNP variant ***increases fusarium wilt resistance***		
DSr	**SNP02051** (Pv05: 4780996 bp)	no candidate gene	
AUDPC, DS30, DSr	**SNP03304** (Pv07: 39039345 bp)	no candidate gene	
AUDPC, DS30, DSr	**SNP03305** (Pv07: 39111049 bp)	**Phvul.007G270000** Location (bp): Pv07: 39106714..39126536 **Phvul.007G269900** (gene model within LD block) Location (bp): Pv07: 39100320..39101039	Programmed cell death protein 11, protein rrp5 homolog, rRNA biogenesis protein RRP5 (RRP5, PDCD11) transcriptional repressor PANTHER: PTHR23270:SF10; Expressed protein-related PANTHER: PTHR33057:SF33
AUDPC, DS30, DSr	**SNP03306** (Pv07: 39166109 bp)	**Phvul.007G270500** Location (bp): Pv07: 39159961..39168244	Pre-mRNA splicing factor prp19-related PANTHER:PTHR13889, Pfam:PF00400
AUDPC, DS30, DSr	**DART07926** (Pv08: 54083493 bp)	**Phvul.008G196600** Location (bp): Pv08: 54082363..54086721	Reticulon-like protein B1-related Pfam:PF02453, PANTHER: PTHR10994:SF27

Figure Captions

Figure 1: Progression of fusarium wilt disease in susceptible bean accessions inoculated with FOP-SP1 race 6. Numbers indicate the disease score based on a severity scale ranging from 1 (healthy leaf) to 5 (dead leaf).

Figure 2: Frequency distribution of disease severity (DS, scale 1-5) in 162 Portuguese common bean accessions caused by *Fusarium oxysporum* f. sp. *phaseoli* (FOP-SP1 race 6). Disease progression was monitored by assessing DS eight different times from the 7th until the 30th day after inoculation. The categorical bins in which SER16 and Tio Canela-75 lines fall are represented by a star and triangle, respectively.

Figure 3: Frequency distribution of the area under disease progress curve (AUDPC) for 162 Portuguese common bean accessions, 7 to 30 days after inoculation, discriminating resistant (low AUDPC values) and susceptible (high AUDPC values) accessions. The categorial bins in which SER16 (AUDPC = 67.2) and Tio Canela-75 (AUDPC = 80.4) lines fall are represented by a star and triangle, respectively.

Figure 4: Fusarium wilt AUDPC values of the Portuguese common bean accessions. The accessions are colored according to the clustering resulting from the structure analysis performed together with gene pool representatives (Leitão et al. 2017). Two groups of accessions were depicted (closed circles): one with Mesoamerican origin (in orange) and the other with Andean origin (in blue). Open circles (in green) refer to the accessions of admixed origin between the original gene pools.

Figure 5: Principal coordinate analysis (PCoA) of the Portuguese common bean collection based on the genotypic profile of 133 accessions using 454 SNP markers evenly distributed along the genome. In the top plot, the accessions are colored according to the structure analysis performed together with gene pool representatives (Leitão et al. 2017). Two groups of accessions were depicted (closed circles): one with the Mesoamerican origin (in orange) and another with the Andean origin (in blue). Open circles (in green) refer to the accessions of admixed origin between the original gene pools. In the bottom plot, the same PCoA is displayed but with the accessions colored according to their response to fusarium wilt infection (FOP-SP1, race 6): resistant in black, intermediate in dark grey and susceptible in light grey.

Figure 6: Manhattan plot depicting the genome-wide association results for fusarium wilt DS30, AUDPC and DSr using a panel of 133 Portuguese common bean accessions. The y-axis represents the −log10 (*P*-value) of 9,825 SNPs, and the x-axis shows their chromosomal positions. The horizontal red line indicates a threshold of significance of −log10 (*P*-value) = 3. The four highlighted vertical columns correspond to genomic regions with significantly associated SNPs.

Figure 7: Frequency of the favorable (conferring resistance) allele of the nine SNPs associated with fusarium wilt AUDPC, disease severity at 30 DAI (DS30) and disease progress rate (DSr) according to the main gene pool of origin of the Portuguese common bean accessions (previously determined in Leitão et al. 2017). Each SNP marker is identified in the x-axis by its name and position in the chromosome (in Mbp).

Figure 1: Progression of fusarium wilt disease in susceptible bean accessions inoculated with FOP-SP1 race 6. Numbers indicate the disease score based on a severity scale ranging from 1 (healthy leaf) to 5 (dead leaf).

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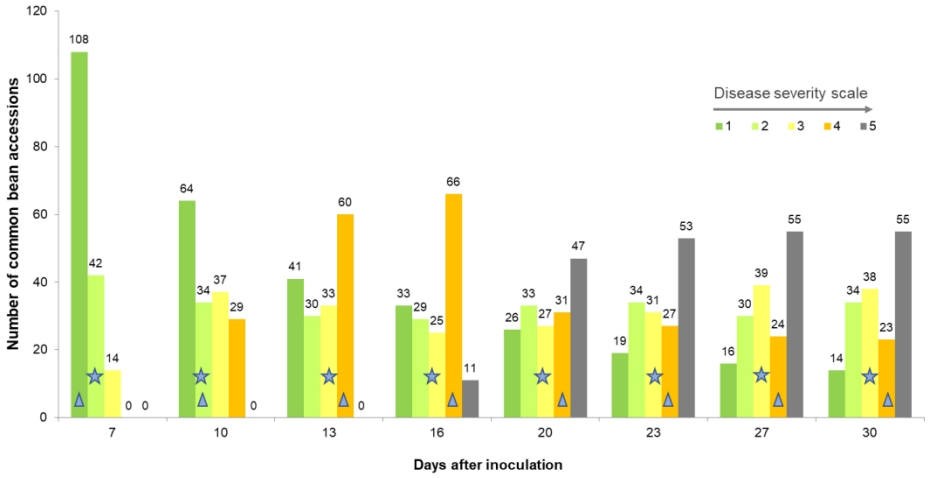

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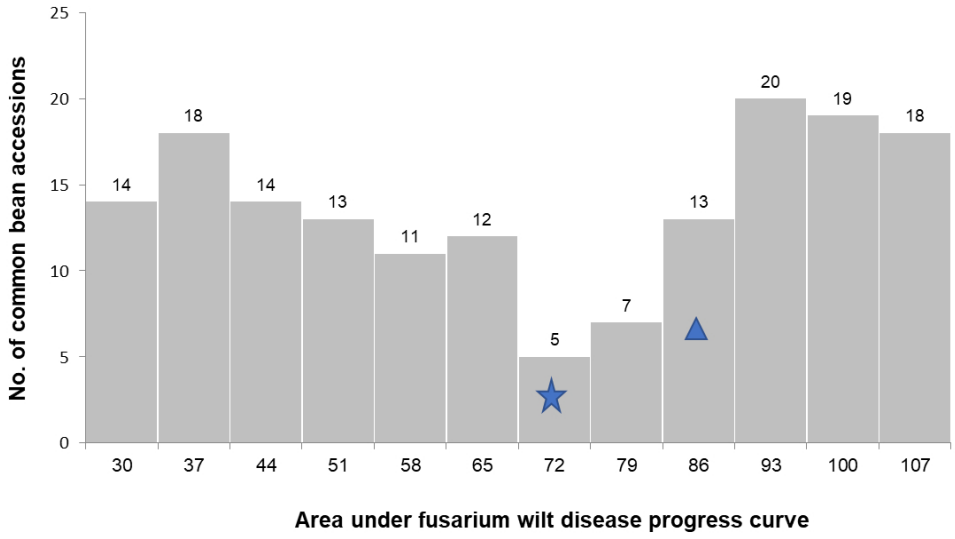

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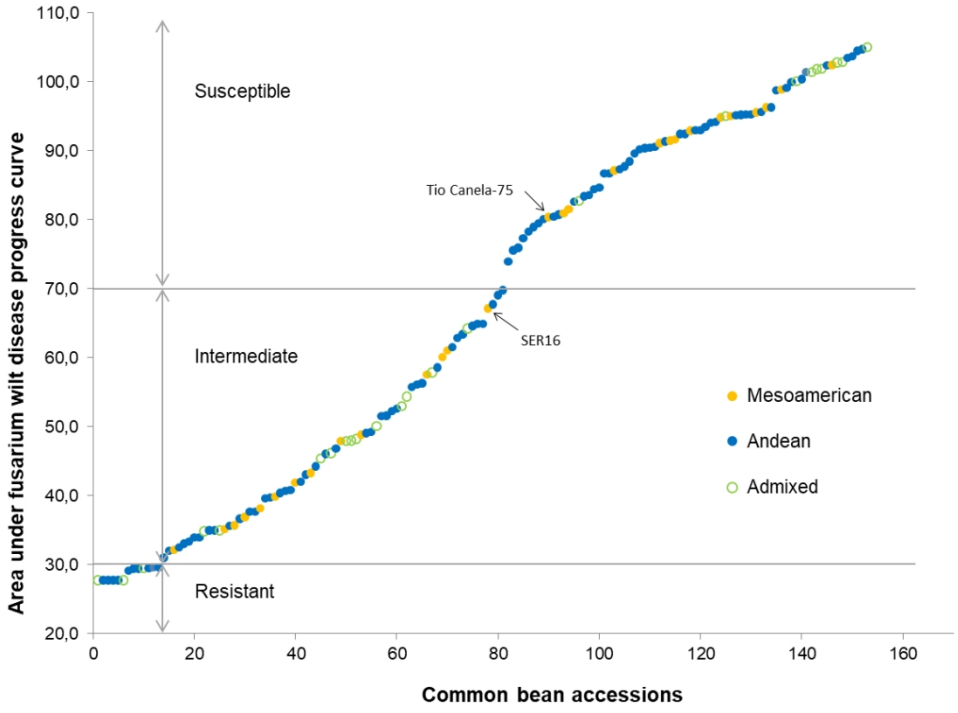

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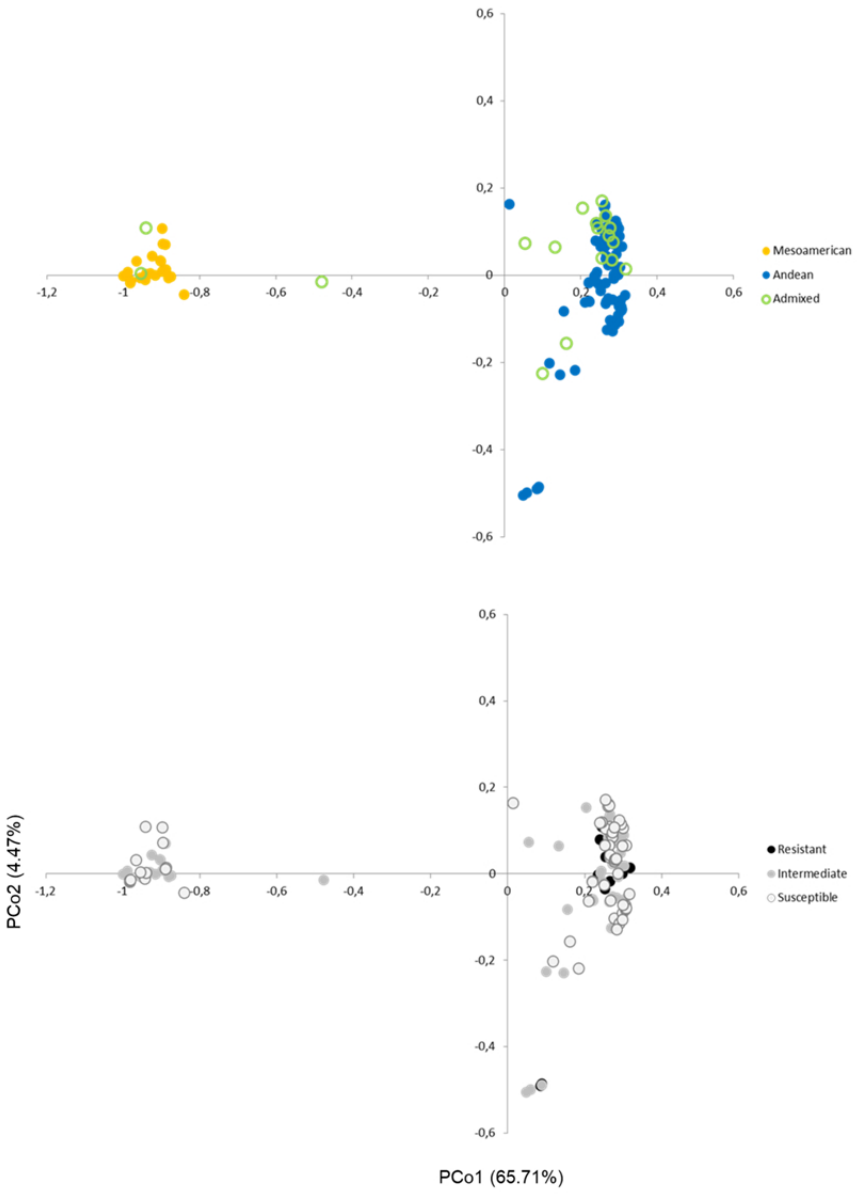

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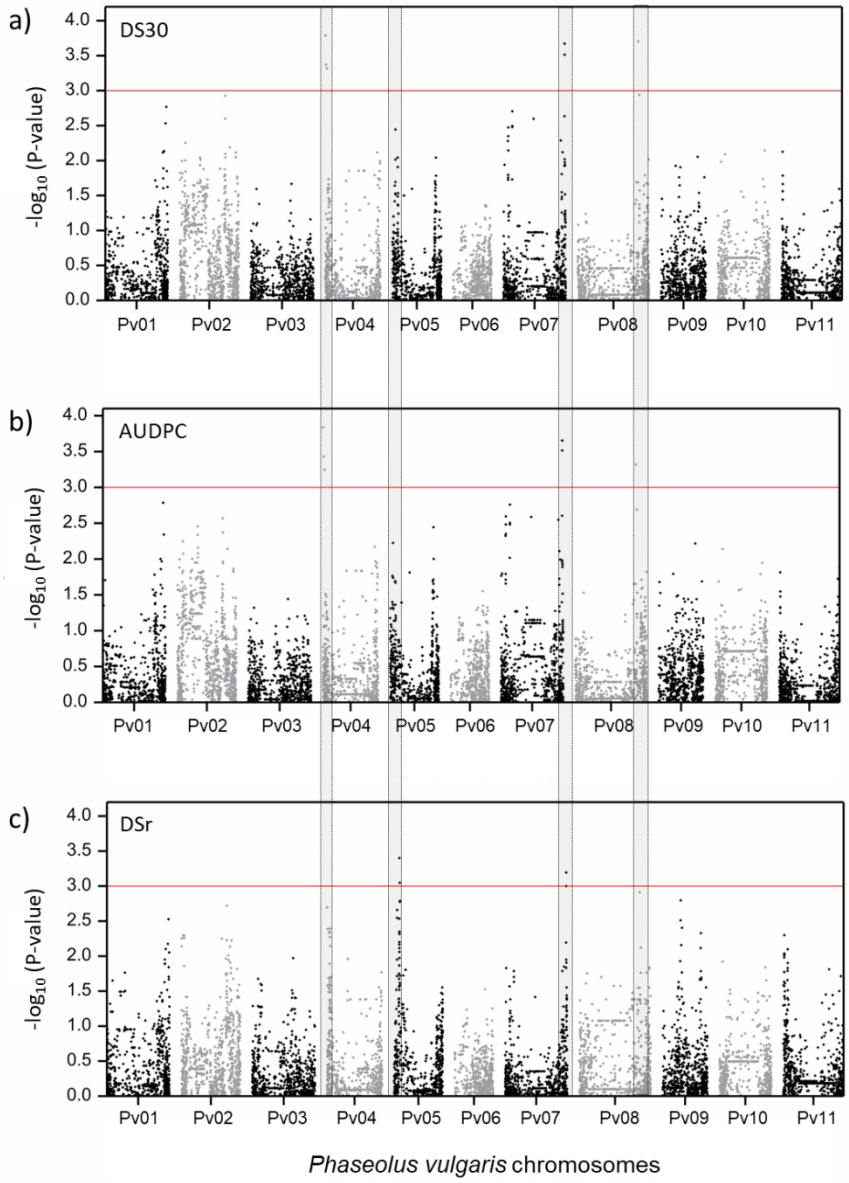

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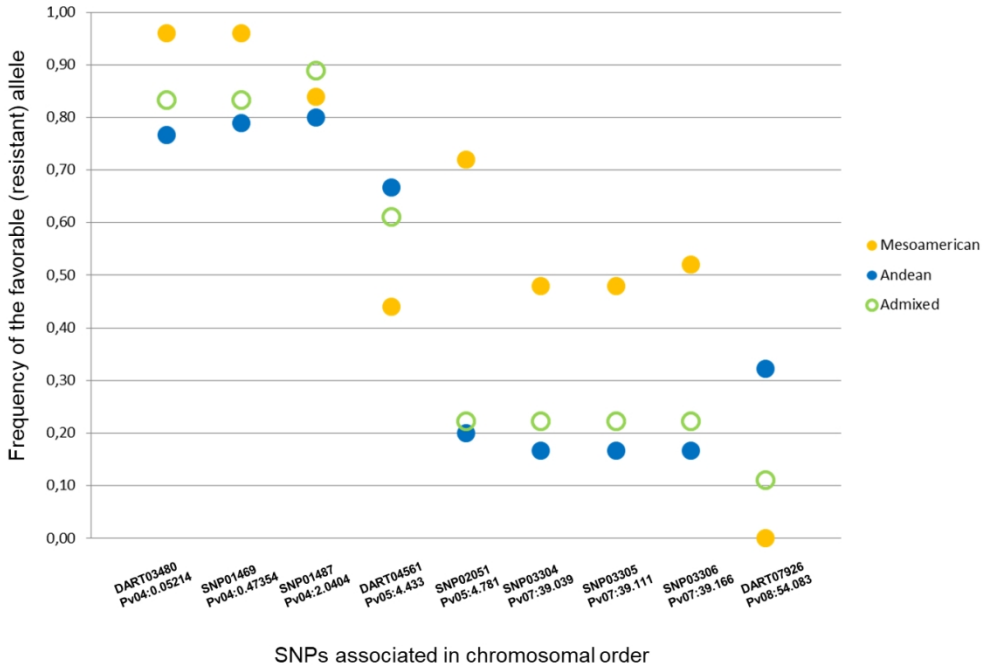
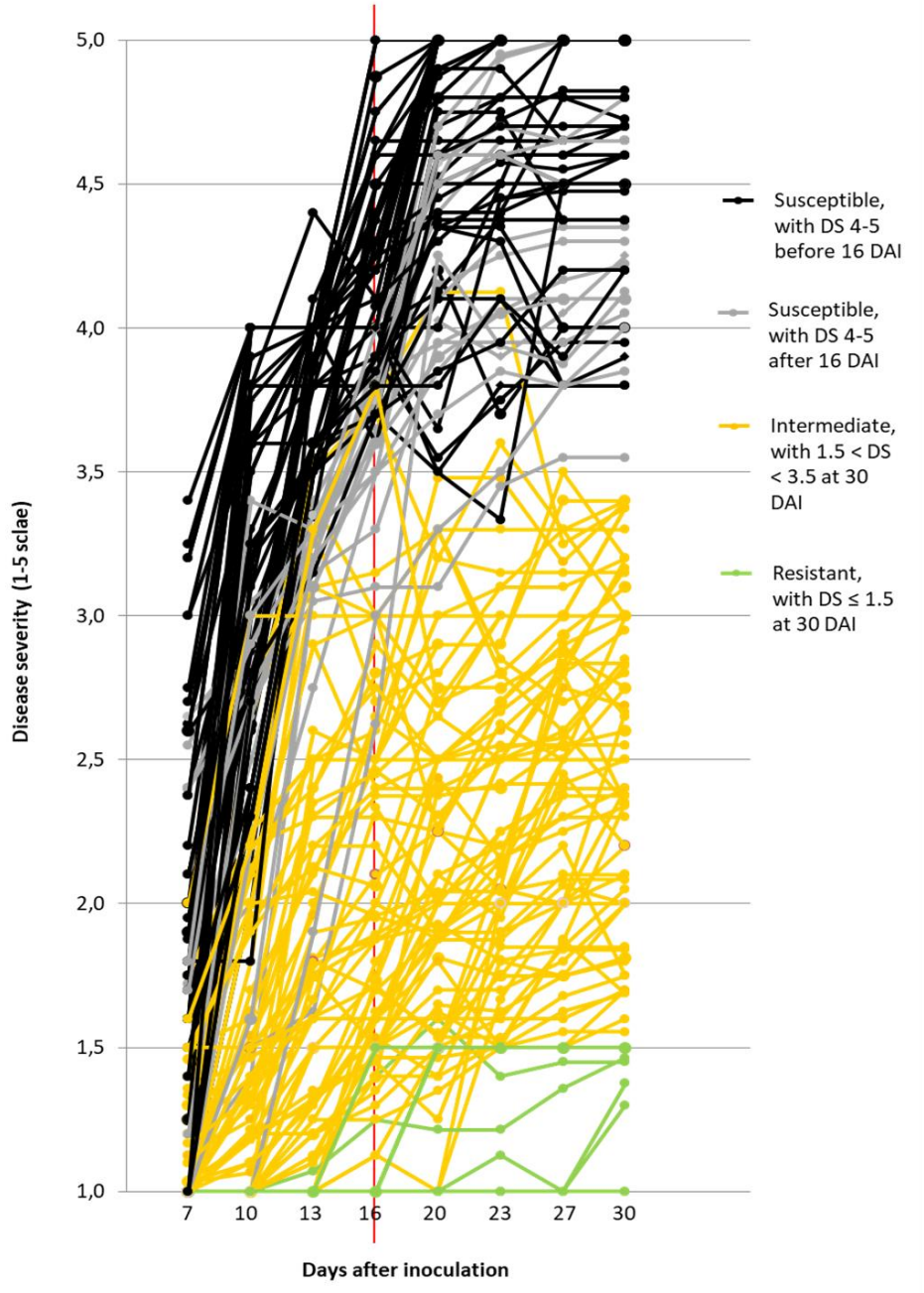


Figure 7: Frequency of the favorable (conferring resistance) allele of the nine SNPs associated with fusarium wilt AUDPC, disease severity at 30 DAI (DS30) and disease progress rate (DSr) according to the main gene pool of origin of the Portuguese common bean accessions (previously determined in Leitão et al. 2017). Each SNP marker is identified in the x-axis by its name and position in the chromosome (in Mbp).

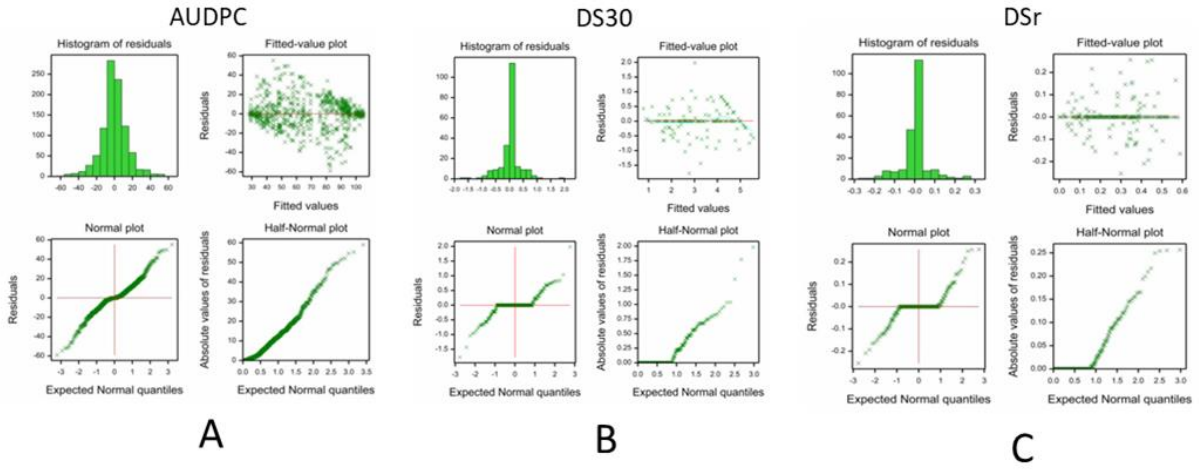
230x158mm (150 x 150 DPI)

Supplementary files of the manuscript entitled **"Natural variation in Portuguese common bean germplasm reveals new sources of resistance against *Fusarium oxysporum* f. sp. *phaseoli* and resistance-associated candidate genes"**
by Susana T. Leitão, Marcos Malosetti, Qijan Song, Fred van Eeuwijk, Diego Rubiales and Maria Carlota Vaz Patto

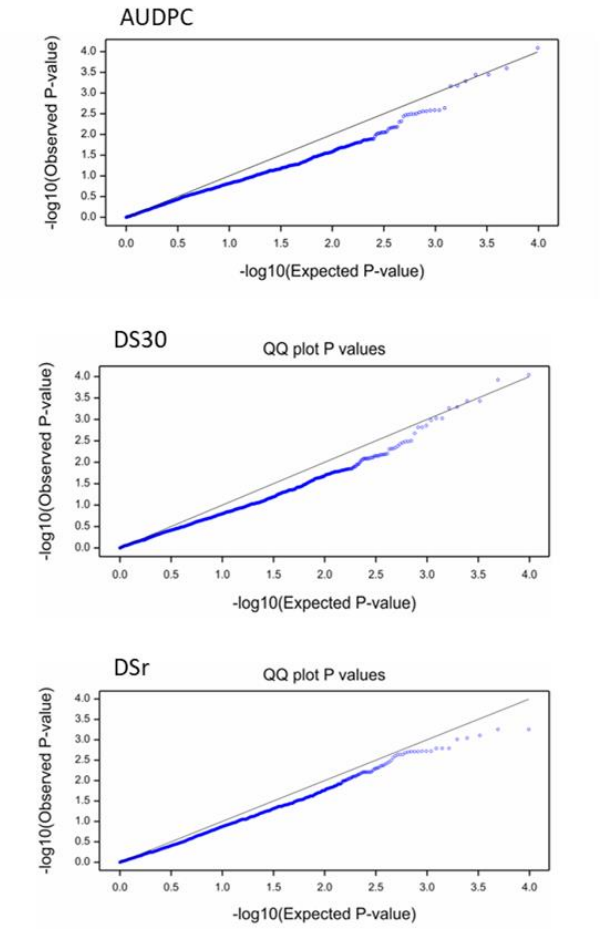
Supplementary Figure 1: Fusarium wilt disease severity (DS) progress for 162 Portuguese common bean accessions 7 to 30 days after inoculation (DAI). Accessions are colored according to their response. Susceptible accessions were divided into two groups: in black if a high DS (4-5) was scored early in time (in less than 16 DAI) and in grey if a high DS was scored later on (after 16 DAI). The 16-day threshold is highlighted with a red line.



Supplementary Figure 2: - Residuals plots for fusarium wilt AUDPC (A), for fusarium wilt disease score at 30 days after inoculation DS30 (B) and for fusarium wilt disease increase rate DSr (C).



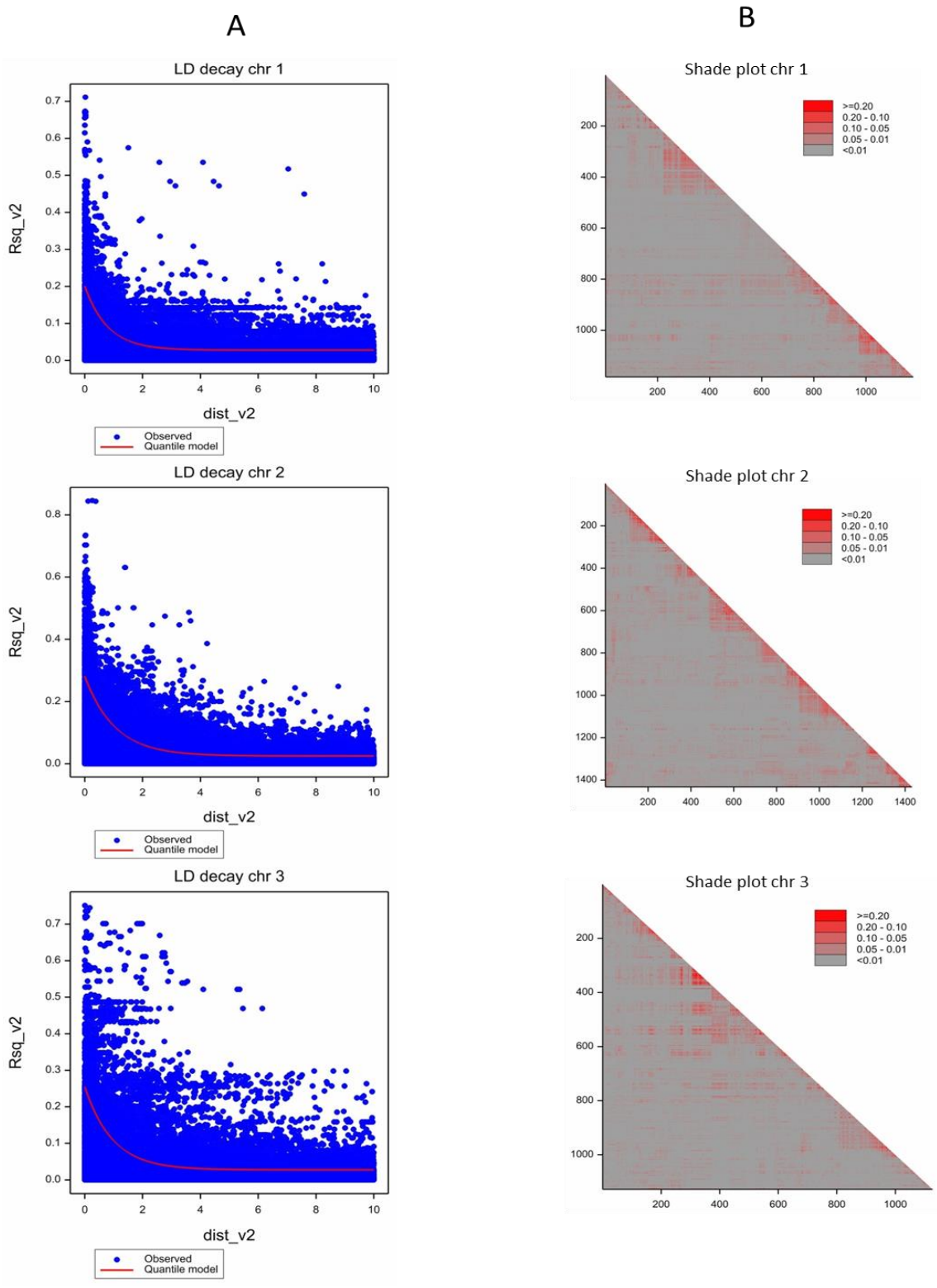
Supplementary Figure 3: Quantile-quantile (Q-Q) plot from the association mapping for fusarium wilt AUDPC, DS30 and DSr using the mixed model accounting for the genetic relatedness (kinship).

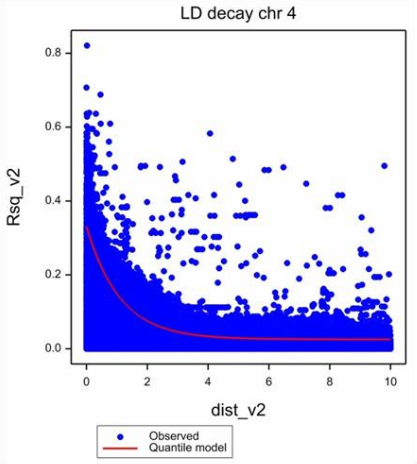


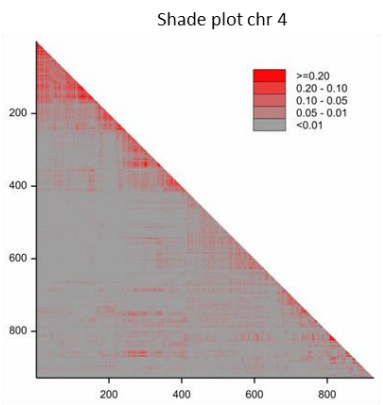
Supplementary Figure 4: Graphical output, performed with Genstat software 19th edition, from the LD analysis of the Portuguese common bean collection marker score data for all the 11 chromosomes.

A) LD decay plots per chromosome. R^2 between markers against markers distance.

B) Shade plots: the colors represent the strength of the LD between markers. The higher $\log_{10}$(*P*-value), the brighter the red, the stronger the LD.




LD decay chr 4
Rsq_v2
dist_v2
Observed
Quantile model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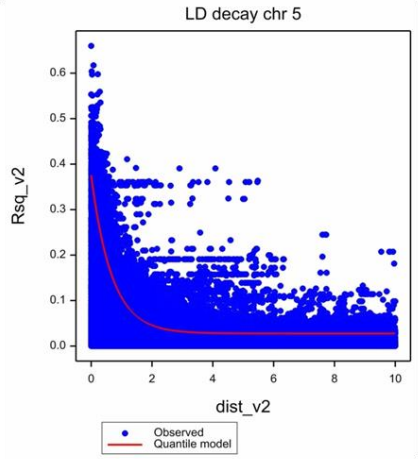
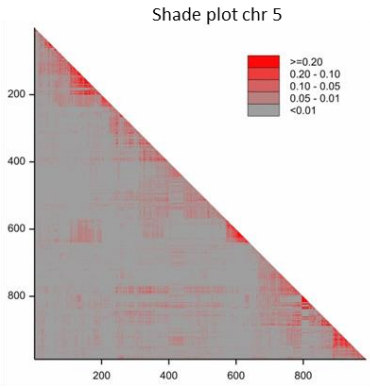
>=0.20
0.20 - 0.10
0.10 - 0.05
0.05 - 0.01
<0.01


LD decay chr 5
Rsq_v2
dist_v2
Observed
Quantile model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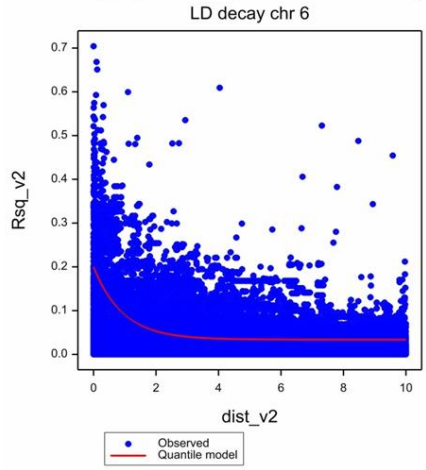
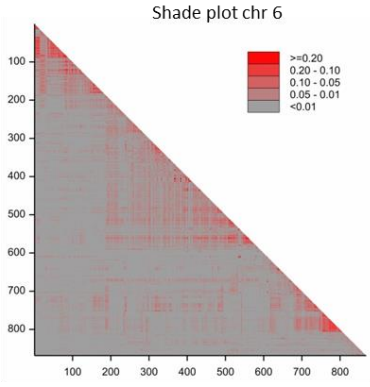
0.10 - 0.05
0.05 - 0.01
<0.01


LD decay chr 6
Rsq_v2
dist_v2
Observed
Quantile model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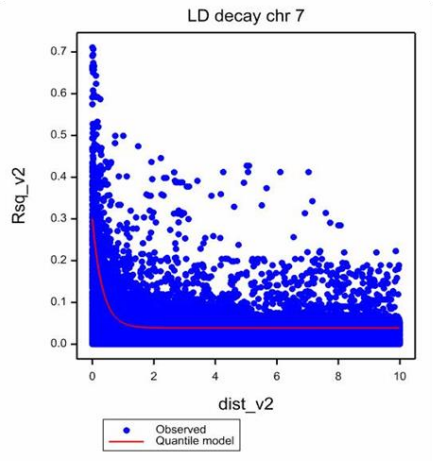
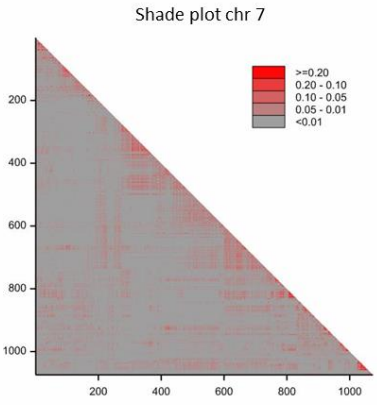
0.10 - 0.05
0.05 - 0.01
<0.01


LD decay chr 7
Rsq_v2
dist_v2
Observed
Quantile model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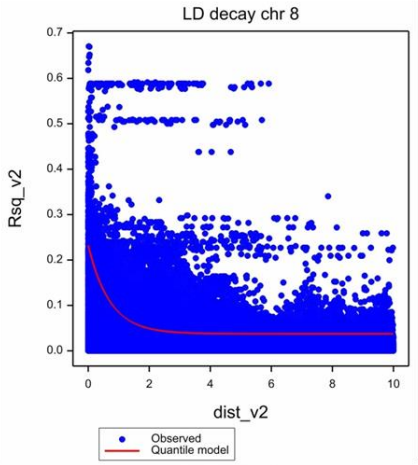
0.20 - 0.10
0.10 - 0.05
0.05 - 0.01
<0.01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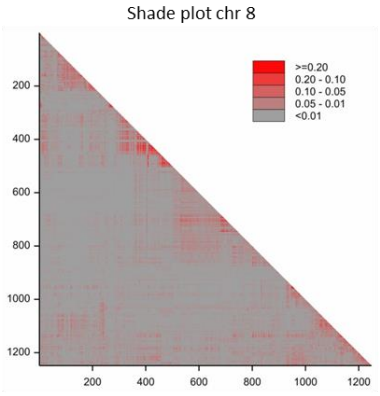
Rsq_v2
dist_v2
Observed
Quantile model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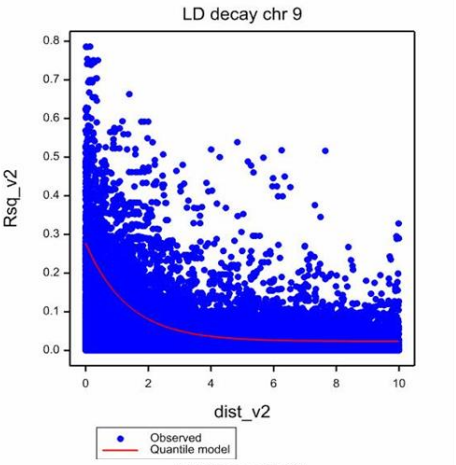
0.20 - 0.10
0.10 - 0.05
0.05 - 0.01
<0.01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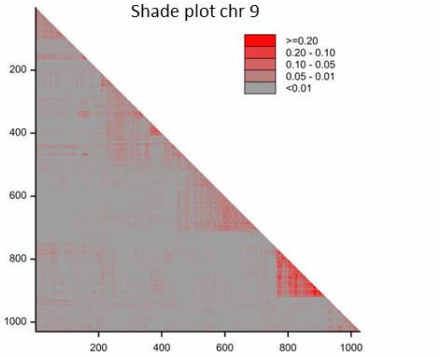
Rsq_v2
dist_v2
Observed
Quantile model


Shade plot chr 9
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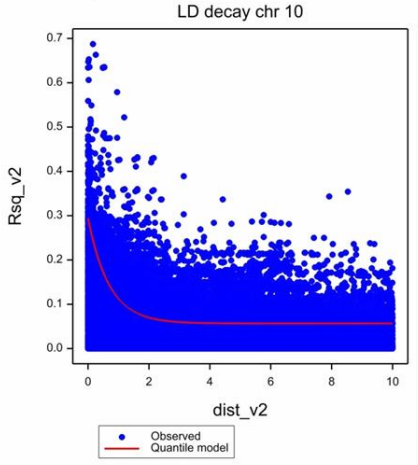
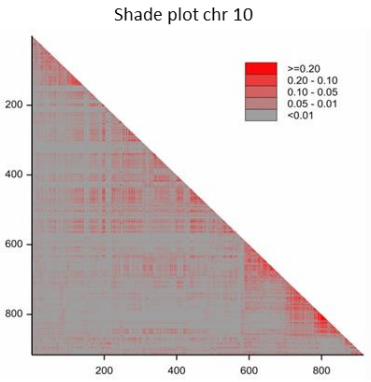
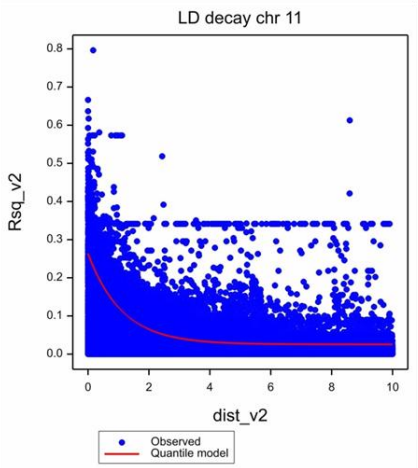
0.10 - 0.05
0.05 - 0.01
<0.01


LD decay chr 10
Rsq_v2
dist_v2
Observed
Quantile model


Shade plot chr 10
>=0.20
0.20 - 0.10
0.10 - 0.05
0.05 - 0.01
<0.01


LD decay chr 11
Rsq_v2
dist_v2
Observed
Quantile model

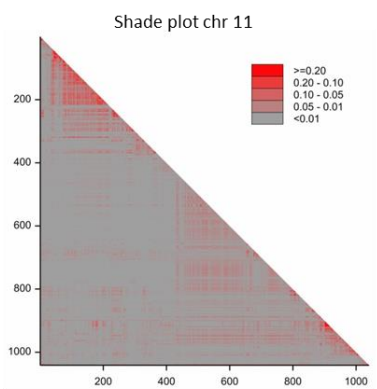

Shade plot chr 11
>=0.20
0.20 - 0.10
0.10 - 0.05
0.05 - 0.01
<0.01

Table S1: Passport information, including geographic origin, of the Portuguese common bean collection used in this study. The results from the structure analysis previously done (Leitão et al. 2017) is included.

Accession	Region of origin (Portugal)	District	Latitude	Longitude	Altitude (m)	Structure_K3Group[a]	
g0579	northern interior	Bragança	41°09'0.000"N	6°48'0.000"W	460	M	Mixed origin
g0583	northern interior	Bragança	41°09'0.000"N	6°48'0.000"W	460	M	Mixed origin
g0584	northern interior	Bragança	41°09'0.000"N	6°48'0.000"W	460	M	Mixed origin
g0587	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g0592	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g0600	northern interior	Bragança	41°20'0.000"N	6°43'0.000"W	749	M	Mixed origin
g0601	northern interior	Bragança	41°20'0.000"N	6°43'0.000"W	749	B	Andean
g0602	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	M	Mixed origin
g0610	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g0620	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g0621	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g0623	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	n/a	A	Mesoamerican
g0632	northern interior	Bragança	41°20'0.000"N	6°43'0.000"W	749	B	Andean
g0633	northern interior	Bragança	41°20'0.000"N	6°43'0.000"W	749	B	Andean
g0635	northern interior	Bragança	41°20'0.000"N	6°43'0.000"W	749	B	Andean
g0638	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g0639	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	M	Mixed origin
g0642	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g0644	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g0645	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g0648	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g0654	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	A	Mesoamerican
g0667	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g0670	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g0671	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g0675	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	M	Mixed origin
g0677	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g0695	interior north	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g0698	northern interior	Bragança	41°45'0.000"N	6°30'0.000"W	700	B	Andean
g0700	northern interior	Bragança	41°45'0.000"N	6°30'0.000"W	700	B	Andean
g0706	central north	Vila Real	41°44'0.000"N	7°28'0.000"W	368	B	Andean
g0735	northern interior	Bragança	41°50'8.000"N	7°00'0.500"W	687	B	Andean
g0736	northern interior	Bragança	41°50'8.000"N	7°00'0.500"W	687	B	Andean
g0737	central north	Vila Real	41°49'0.000"N	7°47'0.000"W	990	B	Andean
g0747	interior north	Bragança	41°29'2.400"N	7°10'3.900"W	218	B	Andean
g0748	northern interior	Bragança	41°29'2.400"N	7°10'3.900"W	218	A	Mesoamerican
g1628	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g1631	central south	Lisboa	38°47'0.000"N	9°23'0.000"W	180	B	Andean
g1636	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	A	Mesoamerican
g1644	central south	Lisboa	39°49'0.000"N	9°10'0.000"W	21	A	Mesoamerican
g1651	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	A	Mesoamerican
g1653	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	B	Andean
g1654	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	B	Andean
g1662	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	B	Andean

g1663	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	B	Andean
g1867	northern interior	Bragança	41°29'2.400"N	7°10'3.900"W	218	A	Mesoamerican
g1871	northern interior	Bragança	41°29'2.400"N	7°10'3.900"W	218	B	Andean
g1877	northern interior	Bragança	41°29'2.400"N	7°10'3.900"W	218	B	Andean
g1883	northern interior	Bragança	41°29'2.400"N	7°10'3.900"W	218	B	Andean
g1884	interior north	Bragança	41°29'2.400"N	7°10'3.900"W	218	B	Andean
g1889	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g1892	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	A	Mesoamerican
g1893	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	n/a	B	Andean
g1897	interior north	Bragança	41°20'0.000"N	6°43'0.000"W	749	A	Mesoamerican
g1911	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g1917	northern interior	Bragança	41°29'0.000"N	6°16'0.000"W	679	B	Andean
g1918	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	A	Mesoamerican
g1926	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1927	interior north	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1932	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	A	Mesoamerican
g1933	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1937	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1938	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	A	Mesoamerican
g1943	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1944	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g1948	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	M	Mixed origin
g1952	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	A	Mesoamerican
g1955	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	A	Mesoamerican
g1956	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	M	Mixed origin
g1961	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	B	Andean
g1964	northern interior	Bragança	41°32'0.000"N	6°57'0.000"W	570	A	Mesoamerican
g1966	central north	Vila Real	41°44'0.000"N	7°28'0.000"W	368	B	Andean
g1975	central north	Vila Real	41°44'0.000"N	7°28'0.000"W	368	B	Andean
g1976	central north	Vila Real	41°36'0.000"N	07°18'0.000"W	425	B	Andean
g1979	central north	Vila Real	41°36'0.000"N	07°18'0.000"W	425	A	Mesoamerican
g1984	central north	Vila Real	41°36'0.000"N	07°18'0.000"W	425	B	Andean
g2081	Madeira	Funchal	32°40'0.000"N	17°04'0.000"W	50	B	Andean
g2105	Madeira	Funchal	n/a	n/a	n/a	B	Andean
g2126	Madeira	Funchal	32°45'0.000"N	16°49'0.000"W	250	B	Andean
g2155	Madeira	Funchal	32°47'0.000"N	17°02'0.000"W	150	B	Andean
g2159	Madeira	Funchal	32°49'0.000"N	17°06'0.000"W	150	M	Mixed origin
g2179	Madeira	Funchal	32°43'0.000"N	16°57'0.000"W	690	A	Mesoamerican
g2189	Madeira	Funchal	32°43'0.000"N	17°01'0.000"W	500	B	Andean
g2192	Madeira	Funchal	32°43'0.000"N	17°01'0.000"W	500	B	Andean
g4038	central north	Viseu	40°53'7.600"N	07°42'5.000"W	862	B	Andean
g4044	central north	Viseu	40°53'9.700"N	07°43'3.900"W	844	B	Andean
g4048	central north	Guarda	40°53'0.100"N	07°48'4.000"W	774	B	Andean
g4049	central north	Guarda	40°53'0.100"N	07°48'4.000"W	774	B	Andean
g4050	central north	Guarda	40°53'0.100"N	07°48'4.000"W	774	B	Andean
g4051	central north	Guarda	40°53'0.100"N	07°48'4.000"W	774	B	Andean
g4064	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g4067	central north	Viseu	40°50'5.500"N	07°56'2.400"W	471	B	Andean

g4070	central north	Viseu	40°54'8.300"N	07°58'3.800"W	502	B	Andean
g4071	central north	Viseu	40°54'8.300"N	07°58'3.800"W	502	B	Andean
g4072	central north	Viseu	40°54'8.300"N	07°58'3.800"W	502	B	Andean
g4073	central north	Viseu	n/a	n/a	n/a	A	Mesoamerican
g4074	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g4081	central north	Viseu	40°57'1.300"N	07°54'8.400"W	867	M	Mixed origin
g4085	central north	Viseu	40°41'8.100"N	08°04'9.700"W	567	M	Mixed origin
g4088	central north	Viseu	40°41'8.100"N	08°04'9.700"W	567	B	Andean
g4097	central north	Viseu	40°41'8.100"N	08°04'9.700"W	567	B	Andean
g4099	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g4100	central north	Viseu	40°39'3.300"N	08°09'1.800"W	747	B	Andean
g4108	central north	Viseu	40°38'0.400"N	08°03'1.100"W	434	B	Andean
g4110	central north	Viseu	40°39'0.000"N	07°54'0.100"W	475	M	Mixed origin
g4119	central north	Viseu	40°39'0.000"N	07°54'0.100"W	475	B	Andean
g4120	central north	Viseu	40°39'0.000"N	07°54'0.100"W	475	B	Andean
g4127	central north	Guarda	40°45'7.500"N	07°34'4.400"W	609	B	Andean
g4133	central north	Guarda	40°45'0.500"N	07°32'1.700"W	544	B	Andean
g4135	centre-north	Guarda	40°45'0.500"N	07°32'1.700"W	544	A	Mesoamerican
g4144	central north	Guarda	40°51'3.200"N	007°30'1.900"W	618	A	Mesoamerican
g4149	central north	Guarda	40°19'5.100"N	007°41'1.800"W	794	B	Andean
g4150	central north	Guarda	40°19'5.100"N	007°41'1.800"W	794	B	Andean
g4162	central north	Guarda	40°31'5.300"N	007°34'2.300"W	459	M	Mixed origin
g4164	central north	Guarda	40°31'5.300"N	007°34'2.300"W	459	B	Andean
g4179	central north	Guarda	40°39'0.20"N	07°24'5.300"W	441	B	Andean
g4182	central north	Guarda	40°40'1.200"N	07°24'7.000"W	426	A	Mesoamerican
g4185	central north	Coimbra	40°19'9.200"N	07°50'5.500"W	269	B	Andean
g4189	central north	Coimbra	40°19'9.200"N	07°50'5.500"W	269	B	Andean
g4195	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g4295	south	Faro	37°18'0.000"N	08°48'0.000"W	36	B	Andean
g4300	south	Faro	37°00'0.000"N	07°56'0.000"W	9	B	Andean
g4306	south	Faro	n/a	n/a	n/a	B	Andean
g5249	central north	Viseu	40°53'1.920"N	08°05'9.830"	453	A	Mesoamerican
g5285	north coast	Braga	41°30'0.00"N	07°59'0.000"W	300	B	Andean
g5286	north coast	Braga	41°30'0.00"N	07°59'0.000"W	300	A	Mesoamerican
g5287	north coast	Braga	41°30'0.00"N	07°59'0.000"W	300	A	Mesoamerican
g5288	central north	Aveiro	40°38'0.00"N	08°39'0.000"W	8	B	Andean
g5289	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g5291	south	Faro	37°00'0.000"N	07°56'0.000"W	9	B	Andean
g5292	south	Faro	37°00'0.000"N	07°56'0.000"W	9	A	Mesoamerican
g5295	south	Faro	37°08'0.00"N	08°01'0.000"W	171	B	Andean
g5296	south	Faro	37°18'0.000"N	08°48'0.000"W	36	M	Mixed origin
g5297	south	Faro	37°08'0.00"N	08°01'0.000"W	171	M	Mixed origin
g5298	northern interior	Bragança	41°09'0.000"N	6°48'0.000"W	460	B	Andean
g5300	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	B	Andean
g5302	north-coast	Braga	341°16'0.00"N	08°16'0.000"W	300	M	Mixed origin
g5306	n/a	n/a	n/a	n/a	n/a	n/a	n/a
g5363	central south	Lisboa	38°56'0.000"N	9°19'0.000"W	242	B	Andean
g5366	central north	Guarda	40°32'0.000"N	07°16'0.000"W	540	B	Andean

g5367	central north	Guarda	40°32'0.000"N	07°16'0.000"W	540	B	Andean
g5368	central north	Guarda	40°32'0.000"N	07°16'0.000"W	540	M	Mixed origin
g5369	central north	Aveiro	40°38'0.00"N	08°39'0.000"W	8	B	Andean
g5370	north coast	Braga	41°32'0.000"N	08°36'0.000"W	34	A	Mesoamerican
g5371	north coast	Braga	41°32'0.000"N	08°36'0.000"W	34	B	Andean
g5372	central south	Leiria	39°21'0.00"N	09°09'0.000"W	51	B	Andean
g5376	south	Faro	37°00'0.000"N	07°56'0.000"W	9	M	Mixed origin
g5377	south	Faro	37°00'0.000"N	07°56'0.000"W	9	B	Andean
g5378	central south	Oeste	38°56'0.000"N	9°19'0.000"W	240	M	Mixed origin
g5379	northern interior	Bragança	41°48'0.000"N	6°45'0.000"W	673	M	Mixed origin
g5380	north coast	Braga	41°32'0.000"N	08°36'0.000"W	34	B	Andean
g5381	north coast	Braga	41°32'0.000"N	08°36'0.000"W	34	B	Andean
g5382	north coast	Braga	n/a	n/a	n/a	B	Andean
g5383	north coast	Braga	n/a	n/a	n/a	B	Andean
g5384	central south	Santarém	n/a	n/a	n/a	B	Andean
g5385	central north	Guarda	n/a	n/a	n/a	B	Andean
g5386	central north	Guarda	n/a	n/a	n/a	B	Andean
g5387	central north	Guarda	n/a	n/a	n/a	M	Mixed origin
g5388	central north	Viseu	n/a	n/a	n/a	M	Mixed origin
g5389	central north	Viseu	n/a	n/a	n/a	B	Andean
g5391	n/a	n/a	n/a	n/a	n/a	n/a	n/a
gTarrestre	north coast	Braga	n/a	n/a	n/a	A	Mesoamerican

[a]Results from Structure analysis with 21 SSRs and 10 individuals per accession, done together with accessions representative of the original Andean and Mesoamerican gene pools (Leitão et al., 2017)

K3Group	Description	Note
A	Q > 0.75 in Cluster A	Cluster A more related to the original Mesoamerican gene pool
B	Q > 0.75 in Cluster B	Cluster B more related to the original Andean gene pool
M	0.50 < Q < 0.75 in Cluster A or B	Mixed origin

Table S2: Fusarium wilt disease severity (DS) scores evaluated 7 to 30 days after inoculation with *Fusarium oxysporum* f. *phaseoli* FOP-SP1 race 6 and disease growth rate on 162 Portuguese common bean accessions, SER16 and Tio Canela-75.

	Days after inoculation								Linear regression	Disease progress rate (DSr)	Type of DS progression[b]
Accessions[a]	7	10	13	16	20	23	27	30			
g0579*	1,7	2,5	3,3	3,8	3,9	4,1	4,2	4,2	y = 0,3366x + 1,9504	0,337	d
g0583	2,0	2,8	4,0	4,4	5,0	5,0	5,0	5,0	y = 0,4271x + 2,2188	0,427	c
g0584	1,0	2,0	3,5	4,4	4,9	5,0	5,0	5,0	y = 0,5714x + 1,2723	0,571	c
g0587	1,0	2,6	3,6	4,4	4,9	5,0	5,0	5,0	y = 0,5321x + 1,5429	0,532	c
g0592	1,0	1,0	1,2	1,4	1,6	1,4	1,5	1,5	y = 0,0738x + 0,9804	0,074	a
g0600	1,0	1,3	2,0	2,5	2,5	2,7	2,8	3,2	y = 0,2937x + 0,9286	0,294	b
g0601	1,1	1,4	3,1	3,7	4,0	3,9	4,1	4,3	y = 0,4533x + 1,144	0,453	d
g0602	1,1	1,3	1,8	1,9	2,4	2,2	2,8	2,7	y = 0,2385x + 0,9386	0,239	b
g0610	1,0	1,0	1,0	1,0	1,0	1,7	2,0	2,7	y = 0,2222x + 0,4167	0,222	b
g0620	1,0	3,0	3,6	3,7	3,5	3,8	3,8	3,9	y = 0,294x + 1,9643	0,294	c
g0621	1,4	2,2	3,5	4,1	4,5	4,7	4,8	4,8	y = 0,4929x + 1,5384	0,493	c
g0623	1,0	1,0	1,0	1,1	1,0	2,0	2,5	3,0	y = 0,2902x + 0,2723	0,290	b
g0632	1,0	1,1	1,3	1,5	1,4	1,5	1,9	2,4	y = 0,1623x + 0,7821	0,162	b
g0633	1,0	1,0	1,0	1,8	2,5	2,6	2,8	3,2	y = 0,3565x + 0,3768	0,357	b
g0635	1,1	1,5	2,0	2,6	3,5	3,5	3,2	3,4	y = 0,3514x + 1,0201	0,351	b
g0638	2,2	3,6	4,0	4,4	5,0	5,0	5,0	5,0	y = 0,3595x + 2,6571	0,360	c
g0639	1,6	4,0	4,0	4,3	5,0	5,0	5,0	5,0	y = 0,3869x + 2,4964	0,387	c
g0642	1,0	2,6	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,5238x + 1,5929	0,524	c
g0644	2,0	3,8	3,8	3,8	4,2	3,7	4,0	4,0	y = 0,1798x + 2,8536	0,180	c
g0645	1,3	1,5	1,8	1,5	1,8	1,8	1,8	1,8	y = 0,0655x + 1,346	0,066	b
g0648	1,0	2,8	4,0	4,0	4,0	4,5	5,0	5,0	y = 0,4851x + 1,5982	0,485	c
g0654	2,4	3,3	3,6	3,7	4,7	4,8	4,8	4,7	y = 0,3423x + 2,4536	0,342	c
g0667	1,2	1,4	1,8	1,7	1,6	1,8	2,0	2,4	y = 0,1321x + 1,1304	0,132	b
g0670	2,6	3,1	3,3	3,9	4,4	4,7	4,7	4,8	y = 0,3387x + 2,3821	0,339	d
g0671	1,0	1,2	1,6	1,9	1,9	1,8	1,8	1,8	y = 0,1139x + 1,1256	0,114	b
g0675	2,3	3,6	3,4	2,8	2,5	2,5	2,5	2,5	y = -0,0845x + 3,1429	-0,085	b
g0677	1,0	2,6	3,5	3,8	4,1	4,1	3,3	3,4	y = 0,2619x + 2,0402	0,262	b
g0695	1,8	2,1	2,9	2,4	2,5	2,5	2,5	2,5	y = 0,069x + 2,0893	0,069	b
g0698	1,3	2,6	3,9	4,9	5,0	5,0	5,0	5,0	y = 0,4955x + 1,8482	0,496	c
g0700	1,3	2,7	3,4	3,0	3,0	3,1	3,1	3,2	y = 0,1714x + 2,0786	0,171	b
g0706	2,1	3,2	3,5	4,3	4,4	4,4	4,8	4,8	y = 0,3536x + 2,3464	0,354	c
g0735	1,0	1,0	1,8	1,9	2,0	2,3	2,4	2,4	y = 0,2158x + 0,8571	0,216	b
g0736	1,8	2,9	3,9	4,3	4,4	4,4	4,4	4,4	y = 0,3274x + 2,308	0,327	c
g0737	1,2	2,7	3,1	3,2	3,3	2,8	2,6	2,9	y = 0,1226x + 2,1607	0,123	b
g0747	1,0	2,6	4,0	4,1	5,0	5,0	5,0	5,0	y = 0,5226x + 1,6107	0,523	c
g0748	1,2	1,5	2,1	2,5	2,6	2,9	3,1	3,3	y = 0,3016x + 1,0429	0,302	b
g1628	1,0	1,0	2,5	2,6	3,2	3,6	3,3	3,0	y = 0,35x + 0,95	0,350	b
g1631	1,5	2,2	3,5	3,9	3,6	3,8	4,0	4,0	y = 0,3131x + 1,8786	0,313	c
g1636	1,8	2,0	2,8	3,5	4,5	4,9	5,0	5,0	y = 0,5394x + 1,2522	0,539	d
g1644	1,8	2,3	3,2	3,9	4,1	4,3	4,4	4,4	y = 0,3762x + 1,8446	0,376	d
g1651	1,0	2,4	3,4	3,8	4,8	5,0	5,0	5,0	y = 0,5571x + 1,2929	0,557	d
g1653	2,1	3,2	3,9	4,7	4,7	4,7	4,7	4,7	y = 0,3274x + 2,5768	0,327	c
g1654*	1,3	2,0	2,1	2,4	2,4	2,4	2,4	2,5	y = 0,1349x + 1,5804	0,135	b

g1662	1,0	1,8	3,3	2,7	2,8	3,1	3,3	3,3	y = 0,275x + 1,425	0,275	b
g1663	1,4	2,7	3,3	3,6	4,2	4,3	4,3	4,3	y = 0,3804x + 1,7821	0,380	d
g1867	1,0	1,1	1,5	1,7	2,3	2,7	2,9	3,1	y = 0,3306x + 0,5583	0,331	b
g1871	1,0	2,2	3,6	3,7	3,2	3,2	3,2	3,2	y = 0,2137x + 1,9321	0,214	b
g1877	1,0	1,0	1,1	1,3	1,2	1,2	1,4	1,5	y = 0,0646x + 0,9056	0,065	a
g1883	2,0	2,8	3,6	3,9	4,4	4,5	4,5	4,5	y = 0,3446x + 2,1991	0,345	c
g1884	1,0	1,0	1,3	1,3	1,4	1,5	1,5	1,6	y = 0,0899x + 0,9018	0,090	b
g1889	1,6	2,9	3,8	3,9	4,6	4,6	4,6	4,6	y = 0,3881x + 2,0786	0,388	c
g1892	2,6	3,5	4,0	4,0	4,8	4,8	4,4	4,4	y = 0,2336x + 2,9955	0,234	c
g1893	1,6	3,3	3,8	4,5	4,5	4,5	4,5	4,5	y = 0,3381x + 2,3786	0,338	c
g1897*	2,4	2,8	3,2	3,3	4,0	4,0	4,0	4,1	y = 0,2452x + 2,3339	0,245	d
g1911	1,0	1,2	1,2	1,4	1,7	1,6	1,8	1,8	y = 0,1182x + 0,9152	0,118	b
g1917	1,4	3,6	4,0	5,0	5,0	5,0	5,0	5,0	y = 0,419x + 2,3643	0,419	c
g1918	2,6	2,9	3,4	4,5	4,8	5,0	5,0	5,0	y = 0,3875x + 2,4	0,388	c
g1926	1,0	1,7	2,1	2,7	2,5	2,6	2,6	2,6	y = 0,194x + 1,3268	0,194	b
g1927	1,0	1,2	1,3	1,5	1,5	1,5	1,6	1,6	y = 0,0748x + 1,0427	0,075	b
g1932	2,4	4,0	4,0	4,3	5,0	5,0	5,0	5,0	y = 0,3229x + 2,875	0,323	c
g1933	1,9	3,6	3,6	3,8	4,6	4,6	4,5	4,5	y = 0,3155x + 2,4679	0,316	c
g1937	3,2	4,0	4,0	4,2	5,0	5,0	5,0	5,0	y = 0,2548x + 3,2786	0,255	c
g1938	2,7	2,9	3,4	3,8	4,6	4,7	4,7	4,7	y = 0,3289x + 2,4232	0,329	d
g1943	1,6	4,0	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,3905x + 2,4429	0,391	c
g1944	1,0	2,4	3,6	3,7	4,6	4,7	4,7	4,7	y = 0,4952x + 1,4464	0,495	c
g1948	3,0	3,9	4,0	4,2	5,0	5,0	5,0	5,0	y = 0,2774x + 3,1393	0,277	c
g1952	2,0	3,5	4,0	4,0	4,1	4,4	4,4	4,4	y = 0,2649x + 2,6518	0,265	c
g1955	2,4	2,9	3,3	3,7	4,7	5,0	5,0	5,0	y = 0,4143x + 2,1232	0,414	d
g1956	1,8	3,8	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,3857x + 2,4643	0,386	c
g1961	1,0	3,2	4,0	4,0	3,5	3,3	4,5	4,5	y = 0,3413x + 1,9643	0,341	c
g1964	1,6	2,1	2,5	2,5	3,3	2,9	2,7	2,8	y = 0,1577x + 1,8339	0,158	b
g1966	2,6	3,9	4,0	4,6	5,0	5,0	5,0	5,0	y = 0,306x + 3,0107	0,306	c
g1975	1,7	2,8	3,2	3,7	4,1	4,1	3,8	3,8	y = 0,2732x + 2,16	0,273	c
g1976	1,0	1,2	1,5	1,5	1,5	1,6	1,6	1,7	y = 0,0857x + 1,0643	0,086	b
g1979	3,0	3,6	3,8	4,0	3,7	4,5	4,5	4,6	y = 0,206x + 3,0232	0,206	c
g1984	1,0	1,0	1,1	1,4	1,4	1,9	1,9	2,1	y = 0,1649x + 0,7143	0,165	b
g2081	1,0	2,8	3,6	3,8	4,4	4,4	4,0	4,0	y = 0,3548x + 1,8911	0,355	c
g2108	1,7	3,6	4,0	3,6	4,5	4,6	4,6	4,7	y = 0,3417x + 2,375	0,342	c
g2126	2,1	2,7	3,1	3,1	3,1	3,5	3,6	3,6	y = 0,1857x + 2,2393	0,186	d
g2155	1,0	1,0	1,0	1,5	1,5	1,5	1,9	1,7	y = 0,1272x + 0,8103	0,127	b
g2159*	1,0	1,0	1,1	1,5	1,9	2,3	2,4	2,4	y = 0,2411x + 0,6027	0,241	b
g2179	1,4	1,5	1,6	1,7	1,5	1,5	1,7	1,8	y = 0,0408x + 1,3967	0,041	b
g2189	1,0	2,0	3,1	3,0	2,7	2,8	3,0	3,0	y = 0,2119x + 1,6214	0,211	b
g2192	1,0	1,2	1,4	1,5	1,3	1,8	1,8	2,0	y = 0,1298x + 0,8911	0,130	b
g4038	3,2	4,0	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,2571x + 3,2429	0,257	c
g4044	3,4	4,0	4,0	4,4	5,0	5,0	5,0	5,0	y = 0,2357x + 3,4143	0,236	c
g4048	1,8	2,7	3,9	4,2	4,4	4,4	4,5	4,5	y = 0,3524x + 2,2143	0,352	c
g4049	1,3	2,3	3,5	3,9	4,1	4,1	4,1	4,1	y = 0,3643x + 1,7857	0,364	c
g4050	1,3	2,2	2,3	2,3	2,3	2,2	2,4	2,4	y = 0,0994x + 1,7214	0,099	b
g4051*	1,2	1,5	2,4	2,9	2,7	3,0	3,0	3,0	y = 0,2577x + 1,2964	0,258	b
g4064	3,3	4,0	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,253x + 3,2679	0,253	c

g4067*	1,3	2,2	2,4	2,8	2,9	2,9	3,4	3,4	y = 0,2655x + 1,4679	0,266	b
g4070*	1,4	2,1	2,9	3,0	3,3	3,3	3,3	3,4	y = 0,256x + 1,6857	0,250	b
g4071	1,9	3,7	4,4	3,9	3,6	4,8	4,8	4,8	y = 0,3179x + 2,5571	0,318	c
g4072	1,0	1,5	1,8	2,1	2,3	2,1	2,0	2,2	y = 0,1405x + 1,2304	0,141	b
g4073	2,1	3,1	4,1	4,6	4,9	4,9	4,7	4,7	y = 0,3411x + 2,5964	0,341	c
g4074*	1,8	2,3	2,5	2,5	3,0	3,0	3,0	3,4	y = 0,2039x + 1,7545	0,203	b
g4081	1,5	2,0	3,1	2,7	2,8	2,8	2,8	2,8	y = 0,1369x + 1,9214	0,137	b
g4085	1,5	1,9	2,3	2,5	2,3	2,5	2,6	2,8	y = 0,1591x + 1,5845	0,159	b
g4088	1,5	1,6	2,6	2,5	2,9	2,9	3,5	3,2	y = 0,2702x + 1,3714	0,270	b
g4097	1,5	2,3	3,5	3,9	4,8	4,8	4,8	4,8	y = 0,481x + 1,6357	0,481	c
g4099	1,0	1,0	1,3	1,6	1,8	1,5	1,5	1,7	y = 0,0976x + 0,9857	0,098	b
g4100	1,8	3,5	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,4077x + 2,3214	0,408	c
g4108	1,0	1,2	1,8	1,9	2,0	2,2	2,3	2,3	y = 0,1881x + 0,986	0,188	b
g4110	2,6	4,0	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,3071x + 2,9429	0,307	c
g4119	1,0	1,0	1,0	1,0	1,0	1,0	1,0	1,4	y = 0,0313x + 0,9063	0,031	a
g4120	1,0	1,0	1,2	1,3	1,5	1,9	2,0	2,1	y = 0,1786x + 0,6964	0,179	b
g4127	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g4133	1,0	1,5	1,6	2,6	4,3	3,9	3,9	4,1	y = 0,5037x + 0,6004	0,504	d
g4135	1,0	1,0	1,0	1,5	2,0	2,0	2,2	1,8	y = 0,1756x + 0,7661	0,176	b
g4144	1,3	2,1	2,5	2,8	2,5	2,5	2,5	2,8	y = 0,1443x + 1,7098	0,144	b
g4149	1,3	3,6	4,0	4,8	5,0	5,0	5,0	5,0	y = 0,433x + 2,2545	0,433	c
g4150	1,8	1,8	3,9	4,6	4,6	4,8	5,0	5,0	y = 0,4893x + 1,7357	0,489	c
g4162	2,4	4,0	4,0	5,0	5,0	5,0	5,0	5,0	y = 0,314x + 3,0089	0,314	c
g4164	1,8	3,1	3,6	4,3	4,5	4,6	4,6	4,6	y = 0,3604x + 2,2438	0,360	c
g4179*	1,2	2,7	3,2	3,5	3,9	4,1	4,1	4,1	y = 0,3613x + 1,7179	0,361	d
g4182	1,0	1,0	1,0	1,6	1,6	1,6	1,9	1,9	y = 0,1429x + 0,7946	0,143	b
g4185	1,0	1,4	1,8	2,0	1,9	1,9	2,1	2,0	y = 0,1272x + 1,1696	0,127	b
g4189	2,8	4,0	4,0	4,8	5,0	5,0	5,0	5,0	y = 0,2857x + 3,1518	0,286	c
g4195	1,9	4,0	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,3676x + 2,5804	0,368	c
g4295	1,0	1,0	1,0	1,0	1,0	1,0	1,0	1,3	y = 0,025x + 0,925	0,025	a
g4300	1,0	2,7	4,0	4,0	4,9	5,0	5,0	5,0	y = 0,5167x + 1,625	0,517	c
g4306	1,1	1,3	1,9	2,0	1,9	2,2	2,3	2,3	y = 0,1679x + 1,1071	0,168	b
g5249	1,5	2,0	2,0	2,0	2,0	2,0	2,1	2,1	y = 0,058x + 1,7024	0,058	b
g5285	1,8	3,4	3,3	3,5	3,7	3,9	3,8	4,0	y = 0,2292x + 2,3875	0,229	d
g5286	2,0	3,0	3,0	3,0	2,3	2,6	2,5	2,5	y = -0,0104x + 2,6563	-0,010	b
g5287	1,1	1,3	1,6	1,6	2,0	2,5	2,8	2,4	y = 0,2304x + 0,8696	0,230	b
g5288*	2,0	3,3	3,5	3,7	3,9	4,0	4,2	4,2	y = 0,2619x + 2,3964	0,262	c
g5289	1,0	1,0	1,0	1,0	1,0	1,0	1,0	1,0	y=1	0,000	a
g5291	1,0	2,2	3,2	3,8	4,6	4,6	4,7	4,7	y = 0,5095x + 1,2946	0,510	d
g5292	1,0	1,0	1,1	1,6	1,9	2,1	2,9	3,0	y = 0,3173x + 0,3911	0,317	b
g5295	1,0	1,0	1,0	1,0	1,0	1,1	1,0	1,0	y = 0,0045x + 0,9955	0,005	a
g5296	1,0	1,0	1,0	1,1	1,5	2,1	2,5	2,5	y = 0,2589x + 0,4286	0,259	b
g5297*	1,0	1,4	1,6	2,0	2,5	2,7	2,9	2,7	y = 0,272x + 0,8696	0,272	b
g5298	1,6	2,4	3,6	3,9	4,4	4,3	3,8	3,8	y = 0,2976x + 2,1232	0,298	c
g5300	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5302	1,5	1,9	2,2	2,2	1,9	2,1	2,8	3,0	y = 0,1673x + 1,4411	0,167	b
g5306*	1,0	1,0	1,9	3,0	3,3	3,5	3,8	3,9	y = 0,4649x + 0,5768	0,465	d
g5363	1,7	3,0	3,3	4,0	4,5	4,6	4,5	4,5	y = 0,375x + 2,075	0,375	d

g5366	1,0	1,0	1,2	1,4	2,4	2,4	2,6	2,6	y = 0,2804x + 0,5571	0,280	b
g5367	1,0	1,5	2,2	2,4	2,4	2,5	2,6	2,4	y = 0,1887x + 1,2696	0,189	b
g5368	1,0	1,2	1,3	1,8	2,1	2,0	2,5	2,2	y = 0,2012x + 0,8446	0,201	b
g5369	1,0	1,6	2,1	2,1	2,8	2,8	2,9	2,8	y = 0,2507x + 1,1138	0,251	b
g5370	1,5	1,5	1,6	1,6	2,1	2,2	2,3	2,4	y = 0,15x + 1,225	0,150	b
g5371	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5372*	1,0	1,6	3,1	3,6	3,9	4,1	4,1	4,1	y = 0,4446x + 1,1804	0,445	d
g5376	1,0	1,0	1,5	1,5	1,5	2,0	2,0	2,0	y = 0,1607x + 0,8393	0,161	b
g5377	1,2	1,3	1,7	2,3	2,0	2,5	2,8	2,8	y = 0,254x + 0,9405	0,254	b
g5378*	1,1	1,3	2,4	2,5	2,7	2,5	2,6	2,6	y = 0,203x + 1,2679	0,203	b
g5379	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5380	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5381	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5382	1,0	1,0	1,3	1,6	1,9	2,0	2,4	2,4	y = 0,2262x + 0,6696	0,226	b
g5383	1,0	1,0	1,5	1,5	1,6	1,5	1,6	1,7	y = 0,096x + 0,9978	0,096	b
g5384	1,0	1,0	1,5	1,5	1,5	2,0	2,0	2,0	y = 0,1607x + 0,8393	0,161	b
g5385	1,0	1,0	1,5	1,5	1,7	1,7	1,8	2,0	y = 0,1405x + 0,8929	0,141	b
g5386	1,0	3,8	4,0	4,0	5,0	5,0	5,0	5,0	y = 0,4554x + 2,0446	0,455	c
g5387	1,0	1,0	1,0	1,5	1,5	1,5	1,5	1,5	y = 0,0893x + 0,9107	0,089	a
g5388	1,0	1,0	1,0	1,0	1,5	1,5	1,5	1,5	y = 0,0952x + 0,8214	0,095	a
g5389	1,4	3,6	4,0	4,3	5,0	5,0	5,0	5,0	y = 0,4274x + 2,2393	0,427	c
g5391	2,7	3,8	4,4	4,1	4,3	4,5	4,5	4,5	y = 0,1976x + 3,2107	0,198	c
gTarrestre	1,6	2,2	3,3	3,8	2,7	3,0	3,0	3,0	y = 0,1405x + 2,1929	0,141	b
SER16	1,0	1,1	1,3	1,7	1,9	1,9	2,1	2,1	y = 0,175x + 0,85	0,175	b
Tio Canela-75	1,0	2,4	3,6	3,8	3,8	4,1	3,9	4,2	y = 0,3738x + 1,6679	0,374	c

[a]The 14 accessions highlighted with a star (*) had the highest standard deviations for DS30 and AUDPC among individual plants and were removed from the association analysis.
[b]Type of DS progression
a Resistant accessions: DS ≤1.5 at 30 DAI
c Susceptible accessions that scored a high DS (4-5) in the first 16 DAI
d Susceptible accessions that scored a high DS (4-5) only after 16 DAI
b Intermediate accessions: 1.6 < DS < 3.5

Table S3: Area under disease progress curve (AUDPC) mean values for 164 common bean accessions (162 Portuguese, SER16, Tio Canela-75) 30 days after inoculation with *Fusarium oxysporum* f. sp*. phaseoli* (FOP-SP1 race 6).

Common bean accession	Fusarium wilt AUDPC along 30 DAI
	Mean
g0579	82,7
g0583	95,1
g0584	101,9
g0587	95,2
g0592	29,5
g0600	53,0
g0601	77,4
g0602	46,2
g0610	31,3
g0620	79,0
g0621	92,5
g0623	35,2
g0632	35,7
g0633	46,1
g0635	64,6
g0638	101,5
g0639	101,5
g0642	95,3
g0644	86,8
g0645	36,7
g0648	90,5
g0654	96,3
g0667	37,7
g0670	93,0
g0671	40,4
g0675	64,3
g0677	78,3
g0695	56,1
g0698	95,3
g0700	67,8
g0706	89,7
g0735	43,1
g0736	90,3
g0737	65,0
g0747	95,7
g0748	57,6
g1628	61,0
g1631	75,6
g1636	87,2
g1644	80,9
g1651	91,5
g1653	93,0
g1654	52,3

g1662	63,4
g1663	84,7
g1867	48,8
g1871	69,8
g1877	29,1
g1883	90,6
g1884	29,6
g1889	91,4
g1892	95,6
g1893	93,5
g1897	81,6
g1911	33,0
g1917	102,4
g1918	98,9
g1926	52,6
g1927	34,0
g1932	102,4
g1933	92,5
g1937	103,5
g1938	92,9
g1943	100,4
g1944	88,5
g1948	102,9
g1952	91,1
g1955	94,9
g1956	100,1
g1961	83,4
g1964	60,1
g1966	103,7
g1975	78,3
g1976	32,5
g1979	91,7
g1984	39,6
g2081	84,5
g2108	93,0
g2126	69,1
g2155	31,0
g2159	45,4
g2179	38,2
g2189	61,6
g2192	33,4
g4038	102,8
g4044	104,5
g4048	87,4
g4049	79,5
g4050	49,2
g4051	56,3
g4064	102,9

g4067	62,9
g4070	65,0
g4071	94,2
g4072	51,6
g4073	95,1
g4074	62,2
g4081	57,9
g4085	54,4
g4088	58,6
g4097	87,8
g4099	39,4
g4100	99,1
g4108	44,3
g4110	101,9
g4119	29,6
g4120	40,7
g4127	29,0
g4133	76,0
g4135	35,7
g4144	55,8
g4149	101,4
g4150	90,4
g4162	105,1
g4164	94,1
g4179	80,1
g4182	32,1
g4185	39,7
g4189	104,8
g4195	100,8
g4295	29,5
g4300	95,3
g4306	42,1
g5249	47,9
g5285	80,8
g5286	61,1
g5287	43,3
g5288	82,6
g5289	29,0
g5291	83,6
g5292	39,9
g5295	29,4
g5296	34,9
g5297	48,0
g5298	80,5
g5300	27,8
g5302	48,3
g5306	69,6
g5363	86,7

g5366	40,8
g5367	49,1
g5368	48,0
g5369	51,5
g5370	41,9
g5371	27,8
g5372	74,0
g5376	35,0
g5377	46,8
g5378	50,1
g5379	27,8
g5380	27,8
g5381	27,8
g5382	37,7
g5383	32,1
g5384	35,0
g5385	34,0
g5386	98,8
g5387	29,5
g5388	27,8
g5389	100,0
g5391	96,3
gTarrestre	36,9
SER16	67,2
Tio Canela-75	80,4