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Exploring the quantitative resistance to *Pseudomonas syringae* pv. *phaseolicola* in common bean (*Phaseolus vulgaris* L.)

Ana M. González · Fernando J. Yuste-Lisbona · Luis Godoy · Antonia Fernández-Lozano · A. Paula Rodiño · Antonio M. De Ron · Rafael Lozano · Marta Santalla

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Abstract *Pseudomonas syringae* pv. *phaseolicola* is an important disease that causes halo blight in common bean. The genetic mechanisms underlying quantitative halo blight resistance are poorly understood in this species, as most disease studies have focused on qualitative resistance. The present work examines the genetic basis of quantitative resistance to the nine halo blight races in different organs (primary and trifoliate leaf, stem and pod) of an Andean recombinant inbred line (RIL) progeny. Using a multi-environment quantitative trait locus (QTL) mapping approach, 76 and 101 main-effect and epistatic QTLs were identified, respectively. Most of the epistatic interactions detected were due to loci without detectable QTL additive main effects. Main and epistatic QTLs detected were mainly consistent across the environment conditions. The homologous genomic regions corresponding to 26 of the 76 main-effect detected QTLs were positive for the presence of resistance-associated gene cluster encoding nucleotide-binding and leucine-rich repeat (NL) proteins and known defence genes. Main-effect QTLs for resistance to races 3, 4 and 5 in leaf, stem and pod were located on chromosome 2 within a 3.01-Mb region, where a cluster of nine NL genes was detected. The NL gene *Phvul.002G323300* is located in this region, which can be considered an important putative candidate gene for the non-organ-specific QTL identified here. The present research provides essential information not only for the better understanding of the plant-pathogen interaction but also for the application of genomic assisted breeding for halo blight resistance in common bean.

Keywords Halo blight · *Phaseolus vulgaris* L · Quantitative trait loci · Epistasis · Resistance gene clusters · NL genes

Introduction

The bacterium *Pseudomonas syringae* pv. *phaseolicola* infects a broad range of plant species and causes economically important yield loss in common bean (Saettler 1991; Prosen et al. 1993). The disease symptoms are classically recognized by the presence of water-soaked lesions surrounded by haloes, named halo blight (Murillo et al. 2010), which results from the action of a non-specific phytotoxin known as phaseolotoxin (Moore et al. 1984). Different defence mechanisms are activated in plants when halo blight infection occurs, leading to complete or partial resistance (Arnold et al. 2011). Complete resistance, developed in the case of an incompatible

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A. M. González · L. Godoy · A. P. Rodiño · A. M. De Ron · M. Santalla (✉)
Grupo de Biología de Agrosistemas, Misión Biológica de Galicia-CSIC, P.O. Box 28, 36080 Pontevedra, Spain
e-mail: msantalla@mbg.csic.es

F. J. Yuste-Lisbona · A. Fernández-Lozano · R. Lozano
Departamento de Biología y Geología (Genética), Centro de Investigación en Biotecnología Agroalimentaria (BITAL), Universidad de Almería, 04120 Almería, Spain

interaction, is usually governed by the gene-for-gene system and also called race-specific resistance. This form of resistance, inherited as a monogenic trait, is determined by the concomitant presence of a plant resistance (*R*) gene that recognizes the corresponding pathogen avirulence (*Avr*) gene, and that often results in a hypersensitive response, which leads to a rapid induction of host cell death at the site of the pathogen invasion (Flor 1971; Jones and Dangl 2006). In contrast, the so-called partial resistance is quantitative, presumably non-race-specific and polygenic (Keen 1990; Hulbert et al. 2001). It limits the extent of disease caused by virulent pathogens and constitutes an additional layer of resistance in the absence of *R* gene function during compatible interactions.

The largest group of *R* genes belongs to the nucleotide-binding site leucine-rich repeat (NL) gene family (Meyers et al. 2005; Collier and Moffett 2009). A number of NL genes have resistance to *P. syringae*, such as the soybean *RPG1* gene (*P. syringae* pv. *glycinea* race 1; Ashfield et al. 2003), the *Arabidopsis RPS2*, *RPS5* (*P. syringae* proteins 2 and 5; Mindrinos et al. 1994; Shao et al. 2003) and *RPM1* genes (*P. syringae* pv. *maculicola* race 1; Bisgrove et al. 1994), and the common bean *Rpsar-1* and *Rpsar-2* genes (*P. syringae AvrRpm* numbers 1 and 2; Chen et al. 2010). The second largest group of *R* genes contains a cytoplasmic serine-threonine kinase domain as the *Pto* gene for resistance to *P. syringae* pv. *tomato* (Martin et al. 1994). The third group are the receptor-like kinase (RLK) genes, which contain an extracellular leucine-rich repeat domain with a single transmembrane spanning region and a cytoplasmic kinase domain (Dievart and Clark 2004). Several examples of *RLK* genes involved in resistance to *P. syringae* are found in *Arabidopsis*, such as *CRK5*, *CRK11* and *CRK13* (cysteine-rich receptors 5, 11 and 13; Czernic et al. 1999; Chen et al. 2003; Acharya et al. 2007). In plant genomes, *R* genes can be distributed as single loci, such as *RPM1* (Grant et al. 1995), but are more often grouped into complex loci as in *Arabidopsis* where two thirds of them are organized in tightly linked clusters (Meyers et al. 2003; Leister 2004; McDowell and Simon 2006, 2008). Such clustering is seen both for *R* genes or allelic series of *R* genes specific for different races of the same pathogen (Islam et al. 1989; Hulbert and Bennetzen 1991) and for *R* genes conferring resistance to unrelated pathogens (Witsenboer et al. 1995). These observations are reflected in the molecular architecture of *R* gene loci, which often consists of multigene families of linked sequences (Hulbert et al. 2001). Clusters of *R* genes have also been observed in the common bean genome. In particular, three large clusters were located at the end of chromosomes 4, 10 and 11 (Schmutz et al. 2014).

Nine races of halo blight have been identified in common bean through the use of *Phaseolus* spp. differential sets (Taylor et al. 1996a). The halo blight races 1, 2, 6 and 7 have a global distribution; races 3, 4, 5 and 8 are found predominantly in East and Central Africa and race 9 has been identified in East Africa and South America (Taylor et al. 1996a). Unfortunately, although some genotypes appear to show reduced susceptibility, there are no bean varieties resistant to most of the races of the pathogen (Terán et al. 2009). Qualitative resistance has been associated with the presence of race-specific resistance genes (*Pse-1* to *Pse-6*), most of them are dominant, except for the recessive *pse-5* gene for resistance to race 8 (Teverson 1991). The *Pse-1* gene protects against races 1, 5, 7 and 9 (Walker and Patel 1964; Miklas et al. 2009) and the *Pse-2* gene against races 2, 4, 5 and 7, and both have been mapped on linkage group (LG) 10 of the common bean genetic map (Teverson 1991; Miklas et al. 2009, 2011). In addition, the *Pse-4* gene confers resistance solely to race 5 (Miklas et al. 2014) and it has also been located on LG10. The *Pse-3* gene protects against races 3 and 4, and it was mapped at the end of LG02 by the complete co-segregation observed with the *I* gene (Pérez-Vega et al. 2010). Recently, the *Pse-6* gene for resistance to races 1, 5, 7 and 9, and the unnamed *R Pse-Race* 1 and *Pse-Race* 7 genes (unofficial gene symbol for preliminary use), were mapped at the end of LG04, supporting the presence of a cluster of *R* genes with specificity for resistance to different halo blight races (Miklas et al. 2014). In addition, two independent *Rpsar-1* and *Rpsar-2* genes, which recognize the avirulence *AvrRpm1* gene isolated from *Pseudomonas syringae* pv. *maculicola*, were mapped on LGs 11 and 08, respectively, in the vicinity of *R* genes for resistance to anthracnose (Geffroy et al. 1998; Melotto et al. 2004; Chen et al. 2010).

There are a limited number of reports on quantitative resistance to halo blight in common bean despite the evidence of quantitative variation in resistance reactions (Taylor et al. 1996b). Seven quantitative trait loci (QTLs) for leaf reactions to halo blight races 2 and 7 were found on the LGs 02, 03, 04, 05, 09 and 10 (Zaiter and Coyne 1984; Ariyarathne et al. 1999). Two QTLs for resistance to race 5 were detected by Yaish et al. (2006). However, the lack of common markers in the integrated map did not allow for localization of these

QTLs. In addition, Trabanco et al. (2014) detected QTLs *Psp4*^{812XC}, *Psp6.1*^{812XC} *Psp6.1*^{684XC} and *Psp6.2*^{684XC} for resistance to halo blight races 6 and 7 located on LGs 04 and 06. The physical positions of QTLs for race 6 on the bean genome revealed 16 candidate genes that carried sequences homologous to the resistance *RPM1*, flagellin-sensitive 2 (*FLS2*), *RPG1* and *Pto* genes, all of which confer resistance to *P. syringae* in different species. These studies indicate that not only major *R* genes but also quantitative resistance factors are involved in halo blight resistance. The present work studies the genetic basis of quantitative resistance to the nine races of halo blight in four different aerial organs of a segregating common bean RIL from the cross PMB0225 × PHA1037. Using a multi-environment QTL mapping approach, main and epistatic QTLs for halo blight resistance were identified. These QTLs showed significant main additive effects in stem, pod, and primary and trifoliate leaf organs, and some of them were co-localized with NL and known defence genes. Thus, markers associated with QTLs reported here constitute useful tools for marker-assisted selection (MAS) breeding programs directed toward improved halo blight resistance. The present work studies the genetic basis of quantitative resistance to the nine races of halo blight in four different aerial organs of a segregating common bean RIL from the cross PMB0225 × PHA1037. Using a multi-environment QTL mapping approach, main and epistatic QTLs for halo blight resistance were identified. These QTLs showed significant main additive effects in stem, pod, primary and trifoliate leaf organs, and some of them were co-localized with NL and known defence genes. Thus, markers associated with QTLs reported here constitute useful tools for MAS in breeding programs directed toward improved halo blight resistance.

Materials and methods

Biological material

The RIL population of 185 F_7 lines was obtained from an F_2 population generated by single-seed descent from the cross between PMB0225 [a photoperiod-adapted common bean line with the *I* gene for resistance to the bean common mosaic necrosis virus (BCMNV), abbreviated as P1] and PHA1037 [a photoperiod-sensitive red nuña bean line with quantitative resistance to races 23 and 1545 of anthracnose, abbreviated as P2]. Both accessions belong to the Andean gene pool (González et al. 2015). Parents were assessed with halo blight races kindly provided by Dr. J. Murillo (Universidad Pública de Navarra, Spain): races 1 (strain 1281A), 2 (strain 1650), 3 (strain 1301A), 4 (strain 1385A), 5 (strain 1390), 6 (strain 1448A), 7 (strain 1449B), 8 (strain 2656A) and 9 (strain 2709A) (Fig. 1). Race identification was ascertained by inoculation of these races on the differential set: Canadian Wonder, Red Mexican UI-3, 1072, A43, Tendergreen, Guatemala 196-B, A52 and A53 (Taylor et al. 1996a).

Plant growth and inoculation conditions: halo blight reaction evaluation

Given that both parents showed different photoperiod (Ppd) behaviour, RIL lines were grown under more than 12 h of light in artificial photoperiod (166 μE s^{-1} m^{-2}, named A-Ppd) and with less than 12 h of light in natural photoperiod (Northwest Spain, 42° 24′ N, 8° 38′ W, 40 masl, named N-Ppd) conditions, with average day and night temperatures of 25 and 20 °C. Plants were grown in plastic pots containing a mixture of clay soil and organic compound (1:1; *v*/*v*) and irrigated according to water needs. The halo blight races were kept on King B's medium (King et al. 1954) at 19–21 °C in darkness. A suspension of 10^6 and 10^8 colony-forming units per milliliter was used for stem and pod and primary and trifoliate leaf according to the previous studies of Mills and Silbernagel (1992) and Taylor et al. (1996a), respectively.

Plants were inoculated according to the growth stages of Schwartz et al. (2004), using inoculation methods of Mills and Silbernagel (1992) and Taylor et al. (1996a). Plants were inoculated at vegetative hypocotyl emergence growth stage (VE; crook-neck stage) by placing a droplet of inoculum on the hypocotyl between the cotyledons, and stem (S) was punctured two times through the inoculum droplet using a 22-gauge hypodermic needle. Primary leaves (PL) were inoculated at vegetative cotyledonary (VC) growth stage, when unifoliate leaves are visible, by spraying the bacterial suspension with an atomiser at 15 psi (103 kPa) in two small areas (0.5-mm diameter) on either side of the mid rib onto the abaxial surface of the leaf, therefore forcing the bacteria into the leaf tissue; afterwards, the whole leaf area was sprayed until completely wet. Three trifoliate leaves (TL) per plant were inoculated at V4 branching and rapid vegetative growth stage, when the

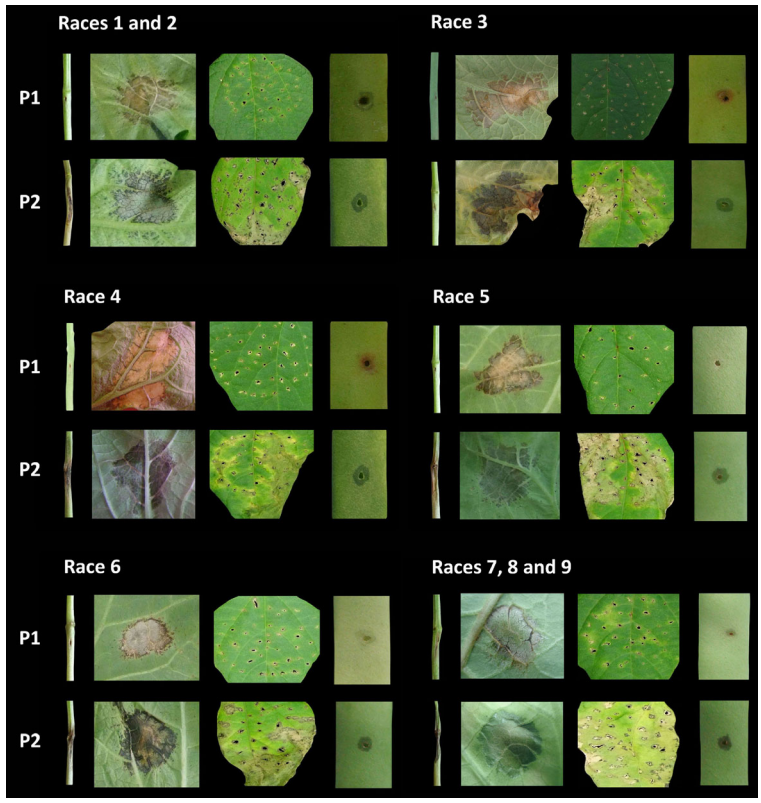

Fig. 1 Disease symptoms produced by the nine halo blight races in stem, primary and trifoliate leaf, and pod for PMB0225 (P1) and PHA1037 (P2) parents, at 10 dpi. Races 1 and 2, 7, 8 and 9 developed similar severe disease symptoms in both parentals

fourth trifoliate leaf is unfolded, by using a multiple-needle florist frog (2-cm square metal base supporting rows of needles 3 mm apart and 12-mm long) dipped in inoculum. Pods (P) were inoculated at R4 flowering and pod formation stage, when 50 % of the pods had reached maximum length, and were excised, washed three times with sterile water, inoculated with a toothpick dipped in inoculum and incubated in a pan lined cover with moist paper towels and sealed with paper wrap.

The infection phenotypes were assessed on visual appreciation of the percentage of symptom severity of each organ at intervals of 5, 7, 14 and 21 days post-inoculation (dpi), according to the 1–9 severity scale (Mills and Silbernagel 1992), where 1 = no visible symptoms (no stem collapse, no leaf halo development, no leaf and pod watersoak at inoculation point and no systemic chlorosis); 2 = traces (<1 mm) of watersoak at inoculation point in stem and leaf, no stem collapse, no leaf halo development, no watersoak at inoculation point with trace of necrosis in pod and no systemic chlorosis; 3 = slight (1–2 mm) watersoak at inoculation point in stem, leaf and pod, and turns necrotic in 24–48 h in pod, no stem collapse, no leaf halo development and no systemic chlorosis; 4 = slight (1–2 mm) watersoak at inoculation point in stem, leaf and pod, turns necrotic in 24–48 h in pod, slight stem constriction above or below inoculation point, slight (up to 1 mm beyond inoculation point) leaf halo development and transitory systemic chlorosis; 5 = moderate (2–3 mm) watersoak at inoculation point in stem, leaf and pod, turns necrotic in 48–72 h in pod, slight stem constriction above or below inoculation point, slight (up to 1 mm beyond inoculation point) leaf halo development and transitory systemic chlorosis; 6 = moderate (2–3 mm) watersoak at inoculation point in stem, leaf and pod, no necrosis in pod, moderate stem constriction (<1/2 diameter), moderate (1–2 mm beyond inoculation point) leaf halo development, and transitory systemic chlorosis; 7 = moderate to severe (3–4 mm) watersoak at inoculation point in stem and leaf, moderate water soak (2–3 mm) no necrosis in pod, moderate stem constriction (<1/2 diameter) and top wilting, moderate (1–2 mm beyond inoculation point) leaf halo development and slight permanent (<1/4 leaflet affected) systemic chlorosis; 8 = moderate to severe (3–4 mm) watersoak at inoculation point in stem, leaf and pod,

no necrosis in pod, moderate stem constriction (<1/2 diameter) and top dying, moderate to severe (2–3 mm beyond inoculation point) leaf halo development and moderate permanent (<1/4–1/2 leaflet affected) systemic chlorosis; and 9 = severe (>4 mm) watersoak at inoculation point in stem, leaf and pod, no necrosis in pod, stem collapse and top dead, severe (>3 mm beyond inoculation point) leaf halo development, and severe permanent (<1/2 leaflet affected) systemic chlorosis. The quantitative resistance traits were determined by: the numerical disease score (DC), which was based on measures at 21 dpi, and the area under the disease progress curve (AUDPC), that was calculated according to Shaner and Finney (1977) as AUDPC = $\sum_{i=1}^{n} [(x_i + x_{i+1})/2]t_j$, where x_i is the disease score on date i, n is the total number of evaluations made, and t_j is the time in days between evaluations x_i and x_{i+1}.

Experimental design and statistical data analysis

The experiment was set up as a randomized complete block design with four replicates for each Ppd condition. Each RIL genotype was represented by one plant in each replication. Independent replicated experiments were carried out for each race and organ.

Descriptive statistical (mean value, standard deviation and range of variation) and normality (Kolmogorov-Smirnov test) analyses were carried out for each quantitative trait and Ppd condition. Significant variation in the expression of traits through the Ppd conditions was analysed using PROC MIXED (SAS Institute Inc. 9.04, Cary, NC, USA). The estimates of variance components were obtained by the REML method with Proc MIXED in SAS9.04 and used to calculate the broad-sense heritability on a progeny-mean basis ($h^2 = \sigma^2{}_\lambda/[(\sigma^2{}_t/e) + \sigma^2{}_\lambda + (\sigma^2{}_e/re)]$ where $\sigma^2{}_\lambda$ = genetic variance of the trait, $\sigma^2{}_t$ = variance due to environmental factors, $\sigma^2{}_e$ = error variance, r = number of replications and e = number of environments). The harmonic mean of the number of replications and environments, where each experimental line was tested, was used for increased precision of the entry mean basis heritability estimate (Holland et al. 2003). Approximate standard errors of heritability estimates were obtained with the delta method (Holland 2006). Phenotypic Pearson correlation coefficients between traits were implemented using PROC CORR through the Ppd conditions in SAS9.04.

Halo blight resistance QTL mapping

The genetic linkage map described by González et al. (2015) was used for QTL analysis, which consisted of 229 loci (86 AFLPs, 98 SSRs, 42 SNPs, 2 SCARs and *P* locus) distributed on 11 LGs. The map spanned 858.4 cM, with an average distance of 3.7 cM between adjacent markers. QTLNetwork 2.0 software (Yang et al. 2008) was used to identify main-effect QTLs, epistatic QTLs (E-QTLs) and their environment interaction effects (QTLs × environment, QE and E-QTLs × environment, E-QE) through Ppd conditions. The mixed model based on composite interval mapping method (MCIM) was used for one-dimensional genome scan to detect putative main-effect QTLs and their environment interactions. A two-dimensional genome scan was also carried out to identify epistatic interaction effects. An experimental-wise significance level of $P < 0.05$ was designated for candidate interval selection, putative QTL detection and QTL effect. Both testing and filtration window size were set at 10 cM, with a walk speed of 1 cM. The critical F value to declare putative QTLs was determined by a 1000 permutation test at the confidence level of 95 %. The effects of QTLs and environment interactions were estimated by the Markov Chain Monte Carlo method (Wang et al. 1994). The genetic map and the QTLs detected were drawn using the MapChart 2.2 software (Voorrips 2002). QTL designations were made using abbreviations for the organs (PL, TL, S and P) and the quantitative trait (DC and AUDPC), with a prefix corresponding to the race, and followed by the LG number at which the QTL was mapped. If more than one QTL for the same race and organ was detected on an LG, a serial number was added.

Database searches of QTLs in common bean genome

Nucleotide sequences of the markers flanking the QTLs were used as queries for BLASTN search (Altschul et al. 1997) against the first chromosome (Chr) scale version of common bean genome (Phytozome v.10: Pv1.0; Schmutz et al. 2014). Those QTL physical intervals with equal or less than 3 millions of base pairs (Mbp) in length were selected for the identification of potential annotated genes associated with disease resistance. The annotated common bean protein sequences were used as queries for BLASTP search against the available

protein database from *Glycine max* (Wm82.a2.v1; http://www.soybase.org/), *Medicago truncatula* (Mt4.0 v1; http://www.jcvi.org/medicago/) and *Arabidopsis thaliana* (TAIR10; http://www.arabidopsis.org/), in order to identify putative homologous sequences. Only those sequences with an *E* value cutoff of $1e^{-10}$ were considered as positive matches.

Results

Race and organ halo blight resistance in the RIL population

PHA1037 bean accession was susceptible (values ≥7) to most of the halo blight races and organs tested except for race 3, which displayed intermediate resistance (values >3 and <7) in stem. The PMB0225 accession was fully resistant (values ≤3) to races 3 and 4 in all organs tested; race 5 in stem, trifoliate leaf and pod; and races 1, 2 and 6 in stem; and showed intermediate resistance to the other combinations of races and organs tested. In fact, PMB0225 and PHA1037 parents and RIL progeny were significantly different for halo blight reaction ($P \leq 0.001$), while the environment effect and the environment × RIL interaction were not significant for most of the resistance traits in each race and organ tested, which indicated a genetic origin for the different levels of resistance in the RIL population (Supplementary files 1 and 2: Tables S1 and S2). The RIL population showed a continuous distribution for the resistance traits studied in each race, organ and Ppd condition, which evidenced that halo blight resistance was quantitatively inherited (Supplementary files 3 and 4: Fig. S1 and S2). The observed transgressive segregation in the RILs toward resistance in stem for races 1, 2, 4, 5 and 6, suggested that resistance was conferred by several genes from both parents with an additive effect. Furthermore, the absence of transgressive segregation toward resistance in primary leaf for races 2, 5, 6 and 8; trifoliate leaf for races 1, 2 and 6; and pod for races 6, 7, 8 and 9, might imply that resistance is conferred by multiple genes with complementary additive effects from PMB0225.

The broad-sense heritability estimates were high (values ≥0.70) for most of the resistance traits for each given organ and race (Supplementary file 5: Table S3), except for the AUDPC resistance trait in primary leaf and pod for races 2 and 9, respectively, indicating that genetic variance accounted for a large portion of the phenotypic variance of resistance to halo blight. Significant and negative correlations were found for resistance to races 1, 3 and 5 between stem and other organs (e.g. $r = -0.45^{**}$ between resistance in stem and pod to race 3), while significant and positive values were found for the other races (e.g. $r = 0.35^{**}$ between stem and pod to race 4). Resistance values to races 3, 4 and 5 were significant and positively correlated, which suggests either linked or pleiotropic genes/QTLs could be involved in the genetic control of resistance of these races.

Mapping of main effect halo blight resistance QTLs

The evaluation of the RIL population developed from the cross PMB0225 × PHA1037 under two different Ppd conditions has led to the identification of 76 main-effect QTLs involved in resistance to nine halo blight races, ranged from 1 (race 6) to 13 (race 4) QTLs, which were mapped across the 11 common bean LGs (Fig. 2). However, 72 out of 76 QTLs detected showed significant genetic main effects and did not display significant additive-by-environment interaction effects (QE). A complete report of the main-effect QTLs detected for primary and trifoliate leaf resistance, and stem and pod resistance is given in Tables 1 and 2, respectively.

For primary leaf resistance: 4 (one for each race 3, 4, 5 and 8) and 9 (two for race 9 and one for each race 1, 2, 3, 4, 5, 6 and 7), main-effect QTLs were found for PLDC and PLAUDPC resistance traits, respectively. The total phenotypic variation explained for PLDC ranged from 5.51 % (race 5) to 20.78 % (race 3), whereas it ranged from 2.04 % (race 6) to 19.93 % (race 3) for PLAUDPC. The detected QTLs for races 3, 4 and 5 were co-localized on LG02.

For trifoliate leaf resistance: 11 (two for each race 2, 4, 5 and 8 and one for each race 1, 3 and 9) and 8 (two for each race 2 and 4 and one for each race 3, 5, 7 and 8) main-effect QTLs were identified for TLDC and TLAUDPC traits, respectively. The total phenotypic variation explained for TLDC ranged from 5.76 % (race 9) to 23.65 % (race 5); and it ranged from 4.93 % (race 7) to 20.12 % (race 2) for TLAUDPC. Some of the QTLs were co-localized in different genomic regions on LG02 for races 3 and 5, on LGs 06 and 11 for race 2 and on LG09 for race 4.

For stem resistance: 9 (two for each race 1, 2 and 4 and one for each race 3, 5 and 7) and 9 (one for each race 1, 2, 3, 4, 5, 8 and 9 and two for race 7) main-effect QTLs were found for SDC and SAUDPC, respectively.

The total phenotypic variation explained for SDC ranged from 7.57 % (race 5) to 22.04 % (race 7); while it ranged from 4.28 % (race 8) to 28.71 % (race 3, negative effects) for SAUDPC. Taken together, some of the QTLs were co-localized in different genomic regions on LG02 for races 3 and 5 and on LG07 for races 2 and 4.

For pod resistance traits, 12 (three for race 1; two for each race 2, 3, 4 and 8 and one for race 5) and 14 (three for each race 1 and 8; two for each race 1, 3 and 4 and one for each race 5 and 7) main-effect QTLs were identified for PDC and PAUDPC traits, respectively. The total phenotypic variation explained for PDC ranged from 13.86 % (race 8) to 40.46 % (race 4); whereas it ranged from 7.85 % (race 7) to 37.15 % (race 3) for PAUDPC. Some of the QTLs were co-localized in different genomic regions on LG02 for races 4 and 5, on LGs 02 and 09 for race 3, on LG06 for race 2, on LG08 for race 1 and on LG09 for race 8.

Detection of epistatic halo blight resistance QTLs

A total of 101 E-QTLs were mapped on the 11 LGs, ranging from 4 (race 7) to 22 (race 9) E-QTLs, and involved in 51 epistatic interactions (Tables 3 and 4). The percentage of phenotypic variance explained by the interaction of these E-QTLs ranged from 0.8 % (*E-PLAUDPC^{8}-7–E-PLAUDPC^{8}-9*) to 12.4 % (*E-TLAUDPC^{9}-2–E-TLADPC^{9}-9* and *E-SDC^{1}-5–E-SDC^{1}-7*). Thirty out of 101 E-QTLs had both individual additive and epistatic effects. The relative contribution of epistasis is also evidenced for those traits where the phenotypic variance is only explained by epistatic effects (e.g. trifoliate leaf resistance to race 6), compared to traits without E-QTLs (e.g. stem, trifoliate leaf and pod resistance to race 7). The positive and negative additive-by-additive E-QTLs values obtained in some of these epistatic interactions indicate that both parents might contribute to increasing resistance. Furthermore, most of the epistatic interactions detected did not display QE effects except for *E-SDC^{8}-3–E-SDC^{8}-11* and *E-PLDC^{6}-3–E-PLDC^{6}-7* interactions.

Location of major identified QTLs in common bean genome

The SNP and SSR markers flanking the main-effect and E-QTLs were located in silico in the bean genome using local BLAST analysis. QTL physical intervals with equal or less than 3 millions of base pairs (Mbp) in length were selected for the identification of potential annotated genes associated with disease resistance in common bean genome. The homologous regions spanning 26 of the 76 main-effect QTLs identified tested positive for the presence of NL and known defence genes. Six genomic regions deserve relevance: four regions containing QTLs for several races and organs and other two regions bearing specific QTLs for a particular race and organ. A total of 870 unique annotated genes were identified in these six genomic regions, most of them encoding uncharacterized proteins, or proteins with putative functions that are not known to be related to defence response against pathogens. However, 49 annotated genes encode proteins with domains that are known to be involved in defence response reaction against pathogens. The annotated potential candidate genes, their chromosome (Chr) location, the putative predicted function resulting from phytozome functional annotations and their homologues in other species are shown in Supplementary file 6: Table S4.

The main-effect QTLs for resistance to races 3, 4 and 5 in stem (*SDC^{3}-2*, *SAUDPC^{3}-2*, *SDC^{4}-2*, *SDC^{5}-2*, *SAUDPC^{5}-2*); trifoliate leaf (*TLAUDPC^{4}-2*, *TLDC^{5}-2*, *TLAUDPC^{5}-2*); primary leaf (*PLDC^{3}-2*, *PLAUDPC^{3}-2*, *PLAUDPC^{4}-2*) and pod (*PDC^{3}-2*, *PAUDPC^{3}-2*, *PDC^{4}-2*, *PAUDPC^{4}-2*, *PDC^{5}-2*, *PAUDPC^{5}-2*) covered 17.64 cM (67.94–85.58 cM) on LG02, while the corresponding genomic region spanned 3.0 Mb on Chr02 (45.5–48.5 Mb). Within this region, there is a cluster consisting of nine NL genes. Likewise, the stem and primary leaf resistance QTLs to races 1 and 9 (*SDC^{1}-5.1*, *PLAUDPC^{9}-5*, *E-SDC^{1}-5.2*, *E-PLDC^{9}-5.1*) detected on LG05 (0–16.79 cM) were located on Chr05 (31.8–31.9 Mb). The *Phvul.005G034100* gene is located in this region, which encodes a *Glycerol-3-P-DH* enzyme, a regulator of plant defence signalling in basal resistance (Venugopal et al. 2009; Yang et al. 2013). Furthermore, the stem, primary and trifoliate leaf resistance QTLs to races 3, 4 and 5 (*PLAUDPC^{5}-8*, *E-SDC^{3}-8*, *E-SDC^{4}-8*, *E-TLDC^{4}-8*) covered 1.51 cM (51.69–53.20 cM) on LG08, whereas the corresponding genomic regions spanned 2.6 Mb on Chr08 (45.7–48.3 Mb). Within this region, there are six NL genes, three *C3HC4-type zinc finger* transcription factors, one peroxidase involved in host-pathogen interactions (Saikia et al. 2004; Berrocal-Lobo et al. 2010; Wang et al. 2010), and the *Phvul.008G182700* gene which encodes for a tetraticopeptide repeat protein (*TRP*). In addition,

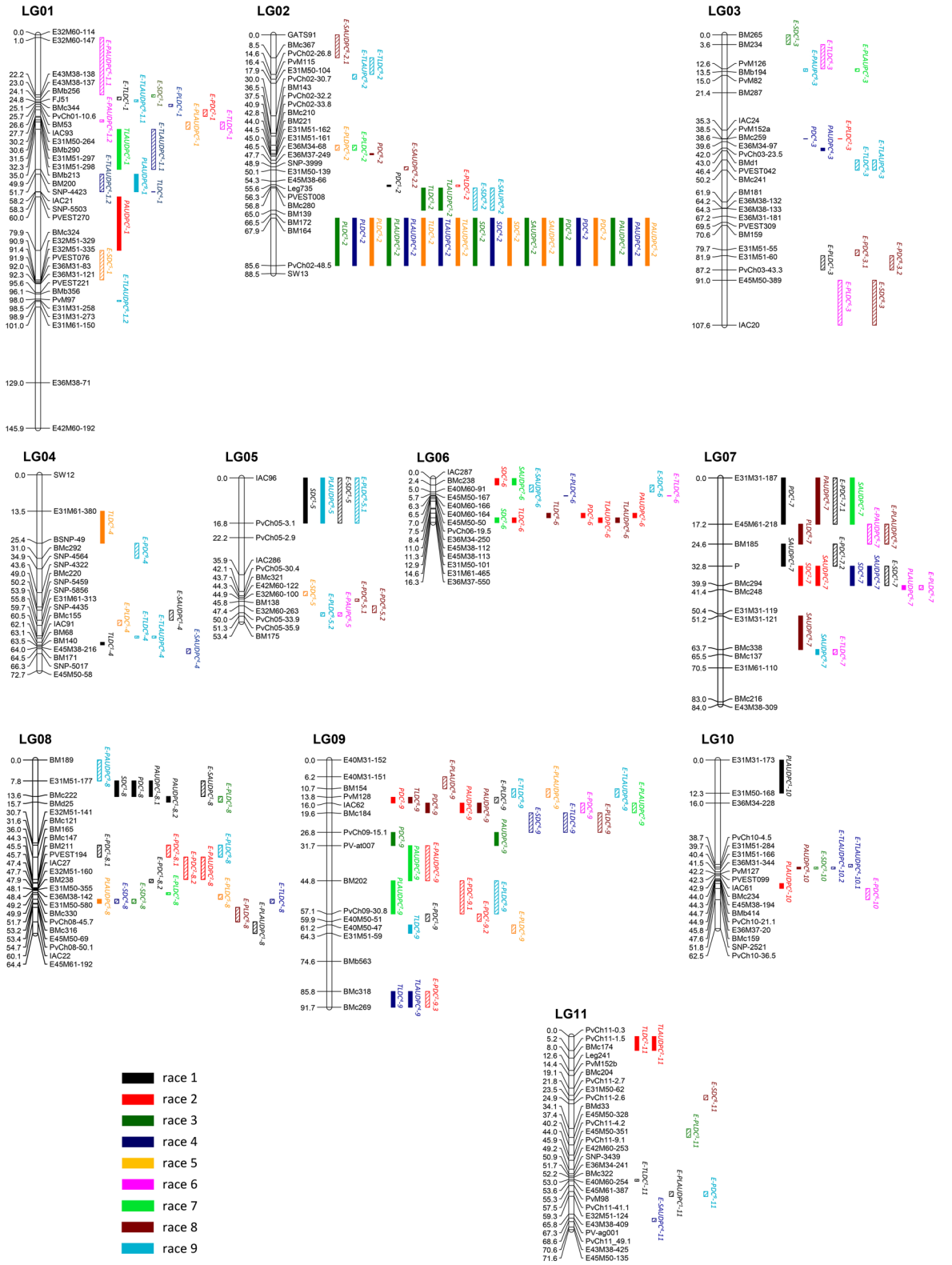
LG01
LG02
LG03
LG04
LG05
LG06
LG07
LG08
LG09
LG10
LG11
race 1
race 2
race 3
race 4
race 5
race 6
race 7
race 8
race 9

Fig. 2 Location of main-effect QTLs and E-QTLs for resistance to nine halo blight races in a genetic linkage map of common bean based on the RIL population developed from the cross PMB0225 × PHA1037. Distances among markers are indicated (in cM) to the *left* of the LGs; names of markers are shown on the *right*. QTLs are depicted as *vertical bars* to the *right* of the LG. Names of QTLs are listed in Tables 1, 2, 3 and 4. Main effect QTLs are indicated with *solid bars*, and E-QTLs are indicated with *hatched bars*. Different *colours* for each race are shown

the QTLs *TLDC*^{8}*-9*, *PDC*^{2}*-9.1*, *E-PLDC*^{1}*-9*, *PDC*^{3}*-9 PAUDPC*^{3}- cover two genomic regions of 3.63 cM (13.77–15.99 cM) and 4.9 cM (26.8–31.7 cM) on LG09, while the corresponding homologous regions spanned 1.74 Mb (65.5–79.4 Mb) and 1.64 Mb (15.09–16.59 Mb) on Chr09, respectively. The *Phvul.009G029700* and *Phvul.009G101900* genes are homologues of the *Arabidopsis* non-race-specific disease resistance 1 (*NDR1*) gene, which interacts with *RPM1*-interacting protein 4 (*RIN4*) for the activation of *Pseudomonas* resistance in *Arabidopsis* (Day et al. 2006), and *WRKY11* transcription factor that acts as negative regulators of basal resistance to *P. syringae* pv. *tomato* (Journot-Catalino et al. 2006). Main-effect trifoliate leaf resistance QTLs (*TLDC*^{2}*-11*, *TLAUDPC*^{2}*-11*) covered 5.2 cM (0–5.2 cM) on LG11, while the corresponding genomic region covered 1.5 Mb on Chr11 (0.03–1.50 Mb). Within this region, there is a cluster consisting of 10 NL genes, and the *Phvul.011G000400* gene is a homologue of the *Arabidopsis AIG1* (avirulence-induced protein) gene that confers resistance to *P. syringae* pv. *maculicola* (Reuber and Ausubel 1996).

Discussion

In common bean, the characterization of simply inherited halo blight *R* genes mediating race-specific recognition of the pathogen and complete resistance has been investigated (Teverson 1991; Taylor et al. 1996a, b; Miklas et al. 2009, 2011, 2014), whereas the genetic mechanisms that control quantitative or partial resistance are poorly understood. Therefore, there is little information available concerning quantitative genetics of halo blight resistance, only a few studies with races 2, 5, 6 and 7 (Ariyarathne et al. 1999; Yaish et al. 2006; Trabanco et al. 2014), in which the role of epistatic interactions in determining resistance has not been studied so far. Thus, the identification of halo blight resistance-related genes through multi-environment QTL mapping and the understanding of the action patterns of these QTLs might provide effective strategies for halo blight resistance. In this work, the gene action governing halo blight resistance was studied for a broad set of RILs generated from a cross between susceptible and resistant Andean accessions. Thus, insights into the number of quantitative resistance loci involved in halo blight resistance to nine races in four organs was provided, as well as their epistatic interactions.

Genetic architecture of halo blight resistance

The present study indicated that the resistance to halo blight in common bean is a complex quantitative trait. Enhanced halo blight resistance level was found in the RIL progeny compared to the parents since resistant alleles came from the resistant parent PMB0225 more frequently, but they also originated from the susceptible parent PHA1037, as observed in stem resistance to races 4, 5 and 6. This result suggests that the susceptible parent also develops defence mechanisms, even though their activity could be insufficient to stop fungal progression, which agrees with previous evidences (Foulongne et al. 2003; Perchepied et al. 2005). Those genotypes more resistant than parental lines could be maintained and fixed through artificial selection. The halo blight resistance response was mainly consistent across the testing Ppd conditions (Tables 1 and 2), which evidenced that halo blight resistance is mostly influenced by genes rather than environmental conditions. Different kinds of resistance components, additive main effects, epistatic effects or both, were found. Most of the epistatic interactions detected were due to loci without detectable QTL additive main effects (Tables 3 and 4), which show the importance of the epistatic effects in genetic resistance to halo blight. In fact, phenotypic variation for resistance to race 6 is explained by six epistatic interactions ranged from 2.64 to 8.52 % (PLDC and PAUDPC, respectively) and one main-effect QTL (2.04 %, PLAUDPC); and resistance to race 9 is explained by 11 epistatic interactions ranged from 4.80 to 26.31 % (PLDC and TLAUDPC, respectively) and four main-effect QTLs, ranged from 5.03 to 11.49 % (SAUDPC and PLAUDPC, respectively. Epistatic interactions have also been previously reported in other crop species as having a key role in resistance to *Fusarium* in melon (Perchepied et al. 2005), *P. syringae* in *A. thaliana*

Table 1 Main-effect QTLs for primary and trifoliate leaf resistance to nine halo blight races of the RIL population, grown under artificial and natural photoperiod conditions (A-Ppd and N-Ppd)

QTL	Marker interval	LG (pos.)[a]	*F* value[b]	*A*[c]	$h^2(a)$[d]
Primary leaf					
PLDC threshold *F* value: race 3 = 8.03, race 4 = 7.91, race 5 = 8.32, race 8 = 8.07					
PLDC^{3}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	30.44	3.34***	20.78
PLDC^{4}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	18.17	2.80***	14.39
PLDC^{5}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	9.14	0.16***	5.51
PLDC^{8}*-7*	E45M61–218-BM185	07 (17.21–24.61)	12.72	0.39***	7.87
PLAUDPC threshold *F* value: race 1 = 8.50, race 2 = 8.14, race 3 = 7.99, race 4 = 7.80, race 5 = 8.46, race 6 = 8.07, race 7 = 8.19, race 9 = 8.16					
PLAUDPC^{1}*-10*	E31M31-173–E31M50-168	10 (0.00–12.32)	9.23	9.01***	5.71
PLAUDPC^{2}*-10*	E36M37-20–BMc159	10 (45.82–47.60)	8.60	13.22***	5.67
PLAUDPC^{3}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	28.53	421.14***	19.93
PLAUDPC^{4}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	18.48	412.22***	14.73
PLAUDPC^{5}*-8*	PvCh08–45.7-BMc316	08 (51.69–53.20)	8.55	37.19***	4.95
PLAUDPC^{6}*-7*	BMc294–BMc248	07 (39.85–41.39)	8.48	6.26*	2.04
PLAUDPC^{7}*-9*	BM202–PvCh09-30.8	09 (44.76–57.13)	8.81	−8.29***	4.85
PLAUDPC^{9}*-1*	SNP-4423–IAC21	01 (51.74–58.19)	8.34	12.91***	5.33
PLAUDPC^{9}*-5*	IAC96–PvCh05-3.1	05 (0.00–16.79)	10.03	7.97***	6.16
Trifoliate leaf					
TLDC threshold *F* value: race 1 = 8.30, race 2 = 8.49, race 3 = 8.50, race 4 = 8.55, race 5 = 8.18, race 8 = 8.20, race 9 = 8.31					
TLDC^{1}*-4*	IAC91–BM68	04 (62.11–63.13)	9.04	0.94***	7.38
TLDC^{2}*-6*	E31M61-465–E36M37-550	06 (14.56–16.35)	10.53	0.46***	8.80
TLDC^{2}*-11*	PvCh11-0.3–PvCh11-1.5	11 (0.00–5.19)	9.01	−0.28***	7.61
TLDC^{3}*-2*	BMc280–BM139	02 (56.81–65.01)	9.75	0.71***	8.89
TLDC^{4}*-1*	IAC21–SNP-5503	01 (58.19–58.31)	11.31	−0.88***	8.88
TLDC^{4}*-9*	BMc318–BMc269	09 (85.83–91.65)	8.99	−0.54***	6.30
TLDC^{5}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	32.34	3.16***	18.02
TLDC^{5}*-4*	E31M61-380–BSNP-49	04 (13.54–25.39)	10.55	0.46***	5.63
TLDC^{8}*-6*	E31M50-101–E31M61-465	06 (12.94–14.56)	10.63	0.53***	8.46
TLDC^{8}*-9*	PvM128–IAC62	09 (13.77–15.99)	8.35	−0.48***	6.59
TLDC^{9}*-9*	E40M50-47–E31M51-59	09 (61.16–64.29)	8.47	0.29***	5.76
TLAUDPC threshold *F* value: race 2 = 8.50, race 3 = 8.45, race 4 = 8.34, race 5 = 8.43, race 7 = 8.08, race 8 = 8.32					
TLAUDPC^{2}*-6*	E31M61-465–E36M37-550	06 (14.56–16.35)	12.74	13.95***	11.69
TLAUDPC^{2}*-11*	PvCh11-0.3–PvCh11-1.5	11 (0.00–5.19)	9.74	−6.44***	8.43
TLAUDPC^{3}*-2*	BMc280–BM139	02 (56.81–65.01)	8.80	15.76***	8.06
TLAUDPC^{4}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	8.78	38.29***	5.97
TLAUDPC^{4}*-9*	BMc318–BMc269	09 (85.83–91.65)	16.05	−13.91***	7.09
TLAUDPC^{5}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	33.65	91.53***	18.64
TLAUDPC^{7}*-1*	BMb213–BM200	01 (35.01–49.89)	8.23	−12.00***	4.93
TLAUDPC^{8}*-6*	E31M61-465–E36M37-550	06 (14.56–16.35)	8.59	16.13***	7.35

Predicted additive-by-environment interaction effect (QE AE) and estimated additive*environment effect [h^2 (ae)]: *PLAUDPC*^{6}*-7* = −9.09** A-Ppd (0.05) and 9.23* N-Ppd (0.05); *TLAUDPC*^{5}*-2* = 10.87** A-Ppd (0.06), −10.50** N-Ppd (0.06)

PLDC primary leaf disease score, *PLAUDPC* primary leaf area under the disease progress curve, *TLDC* trifoliate leaf disease score, *TLAUDPC* trifoliate leaf area under the disease progress curve

[a] Linkage group and the estimated confidence interval of QTL position in brackets (in Kosambi cM)

[b] *F* values of significance of each QTL

[c] Estimated additive effect. Positive values indicate that alleles from PHA1037 have a positive effect on the traits, and negative values indicate that positive effect on the traits is due to the presence of the alleles from PMB0225. Experiment-wide *P* value: *$P \leq 0.05$; **$P \leq 0.01$; ***$P \leq 0.001$

[d] Percentage of the phenotypic variation explained by additive effects

Table 2 Main-effect QTLs for stem and pod resistance to nine halo blight races of the RIL population, grown under artificial and natural photoperiod conditions (A-Ppd and N-Ppd)

QTL	Marker interval	LG (pos.)[a]	*F* value[b]	*A*[c]	$h^2(a)$[d]
Stem					
SDC threshold *F* value: race 1 = 8.59, race 2 = 8.51, race 3 = 8.10, race 4 = 8.02, race 5 = 8.01, race 7 = 8.41					
SDC^{1}*-5*	IAC96–PvCh05-3.1	05 (0.00–16.79)	10.49	−0.43***	4.12
SDC^{1}*-8*	E31M51-177–BMc222	08 (7.83–13.63)	9.59	0.59***	3.50
SDC^{2}*-6*	IAC287–BMc238	06 (0.00–2.37)	9.63	0.29***	5.74
SDC^{2}*-7*	P–BMc294	07 (32.76–39.85)	9.29	−0.32***	5.65
SDC^{3}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	27.85	−1.29***	17.21
SDC^{4}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	9.82	1.53***	6.18
SDC^{4}*-7*	P–BMc294	07 (32.77–39.85)	10.52	−0.60***	6.90
SDC^{5}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	13.20	−1.51***	7.57
SDC^{7}*-6*	E31M61-465–E36M37-550	06 (14.56–16.35)	32.01	0.83***	22.04
SAUDPC threshold *F* value: race 1 = 8.49, race 2 = 8.15, race 3 = 8.33, race 4 = 8.16, race 5 = 7.90, race 7 = 8.31, race 8 = 7.98, race 9 = 8.05					
SAUDPC^{1}*-7*	BM185–P	07 (24.61–32.76)	19.89	−32.43***	12.05
SAUDPC^{2}*-7*	P–BMc294	07 (32.76–39.85)	12.99	−18.51***	5.88
SAUDPC^{3}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	56.76	−146.50***	28.71
SAUDPC^{4}*-7*	P–BMc294	07 (32.77–39.85)	12.70	−38.04***	8.06
SAUDPC^{5}*-2*	BM164–PvCh02–48.5	02 (67.94–85.58)	17.43	−75.15***	9.94
SAUDPC^{7}*-6*	IAC287–BMc238	06 (0.00–2.37)	15.81	25.75***	12.19
SAUDPC^{7}*-7*	E31M31-187–E45M61-218	07 (0.00–17.21)	8.33	−16.63***	0.07
SAUDPC^{8}*-7*	E31M31-121–BMc338	07 (51.22–63.68)	9.01	−18.78***	4.28
SAUDPC^{9}*-7*	BMc338–BMc137	07 (63.68–65.52)	8.51	−15.26***	5.03
Pod					
PDC - threshold *F* value: race 1 = 6.49, race 2 = 6.38, race 3 = 6.48, race 4 = 6.35, race 5 = 8.10, race 8 = 8.38					
PDC^{1}*-2*	Leg735–PVEST008	02 (55.65–56.30)	8.66	−0.31***	4.18
PDC^{1}*-7*	E31M31-187–E45M61-218	07 (0.00–17.21)	8.11	0.39***	1.95
PDC^{1}*-8*	E31M51-177–BMc222	08 (7.83–13.63)	16.81	−0.58***	15.86
PDC^{2}*-6*	E31M50-101–E31M61-465	06 (12.94–14.56)	17.09	0.51***	11.10
PDC^{2}*-9*	PvM128–IAC62	09 (13.77–15.98)	11.27	−0.28***	8.18
PDC^{3}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	57.87	4.09***	32.03
PDC^{3}*-9*	PvCh09-15.1–PV-at007	09 (26.81–31.73)	8.92	−0.54***	8.32
PDC^{4}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	85.37	4.29***	35.03
PDC^{4}*-3*	PvM152a–BMc259	03 (38.48–38.60)	6.79	−0.38***	5.43
PDC^{5}*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	43.90	3.12***	22.17
PDC^{8}*-2*	BM221–E31M51-162	02 (44.03–44.54)	8.68	−0.35***	4.96
PDC^{8}*-9*	IAC62–BMc184	09 (15.98–19.60)	19.30	−0.40***	8.90
PAUDPC - threshold *F* value: race 1 = 6.51, race 2 = 6.44, race 3 = 6.31, race 4 = 6.32, race 5 = 8.00, race 7 = 8.04, race 8 = 8.26					
PAUDPC^{1}*-8.1*	E31M51-177–BMc222	08 (7.83–13.63)	9.3	−14.87***	10.87
PAUDPC^{1}*-8.2*	BMc222–BMd25	08 (13.63–15.71)	11.62	−14.35***	10.70
PAUDPC^{2}*-1*	PVEST270-BMc324	01 (59.99–79.94)	8.42	20.79***	2.55
PAUDPC^{2}*-6*	E31M50-101–E31M61-465	06 (12.94–14.56)	13.72	31.15***	12.99
PAUDPC^{2}*-9*	IAC62–BMc184	09 (15.98–19.61)	11.94	−18.20***	8.88
PAUDPC^{3}*-2*	BM164–PvCh02-8.5	02 (67.94–85.58)	51.35	146.74***	27.80
PAUDPC^{3}*-9*	PvCh09-15.1–PV-at007	09 (26.81–31.73)	9.67	−21.97***	9.35

Table 2 (continued)

QTL	Marker interval	LG (pos.)[a]	*F* value[b]	*A*[c]	$h^2(a)$[d]
PAUDPC^4*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	64.90	167.95***	29.76
PAUDPC^4*-3*	PvCh03-23.5–BMd1	03 (42.04–42.99)	7.56	−20.47***	6.04
PAUDPC^5*-2*	BM164–PvCh02-48.5	02 (67.94–85.58)	16.99	70.05***	10.43
PAUDPC^7*-9*	PV-at007–BM202	09 (31.73–44.76)	11.97	−47.46***	7.85
PAUDPC^8*-7*	E31M31-187–E45M61-218	07 (0.00–17.21)	8.29	12.01***	5.86
PAUDPC^8*-9*	IAC62–BMc184	09 (15.98–19.60)	21.43	−19.06***	11.15
PAUDPC^8*-10*	E31M51-284–E31M51-166	10 (39.73–40.42)	8.42	−11.47***	3.84

Predicted additive-by-environment interaction effect (QE AE) and estimated additive*environment effect [h^2 (ae)]: *PAUDPC*^{3} *-2* = −34.41* A-Ppd (0.10), 31.14* N-Ppd (0.09) and *PAUDPC*^{4} *-2* = 41.43* A-Ppd (0.11), −34.54* N-Ppd (0.08)

Positive values (A and QE AE) indicate that alleles from PHA1037 have a positive effect on the traits, and negative values indicate that positive effect on the traits is due to the presence of the alleles from PMB0225

SDC stem disease score, *SAUDPC* stem area under the disease progress curve, *PDC* pod disease score, *PAUDPC* pod area under the disease progress curve

[a] Linkage group and the estimated confidence interval of QTL position in brackets (in Kosambi cM)

[b] *F* values of significance of each QTL

[c] Estimated additive effect. Positive values indicate that alleles from PHA1037 have a positive effect on the traits, and negative values indicate that positive effect on the traits is due to the presence of the alleles from PMB0225. Experiment-wide *P* value: $*P \leq 0.05$; $**P \leq 0.01$; $***P \leq 0.001$

[d] Percentage of the phenotypic variation explained by additive effects

(Kover and Cheverud 2007) and *Colletotrichum lindemuthianum* in common bean (González et al. 2015).

QTL mapping was used to localize genomic regions controlling variation in organ and halo blight race. The 76 main-effect QTLs detected were located on 37 genomic regions. QTL analysis indicated that none of the QTLs identified here were effective to all races tested. Most QTLs showed resistance in one or two of the four plant organs and for one or two of the nine races (Tables 1 and 2). For example, two genomic regions BMc280–BM139 (*TLDC*^{3}*-2*, *TLAUDPC*^{3}*-2*) and BMc318–BMc269 (*TLDC*^{4}*-9*, *TLAUDPC*^{4}*-9*) were effective against one race and one organ; and two genomic regions IAC287–BMc238 (*SDC*^{2}*-6*, *SAUDPC*^{7}*-6*) and P–BMc294 (*SDC*^{2}*-7*, *SDC*^{4}*-7*, *SAUDPC*^{2}*-7*, *SAUDPC*^{4}-7) were effective for two races and one organ. These results suggested that the QTLs affecting lesion organ and lesion race might not be the same. However, other studies of partial resistance in plants (Young 1996; Marcel et al. 2008; Poland et al. 2009; Chung et al. 2010; Kou and Wang 2010; St. Clair 2010) observed individual QTLs that may have different levels of specificity to pathogen races and plant growth stages, inoculation site or organ. In fact, in other genomic region, E31M50-101–E36M37-550, co-localized QTLs involved in resistance to race 2 (*TLDC*^{2}*-6*, *TLAUDPC*^{2}*-6*, *PDC*^{2}*-6* and *PAUDPC*^{2}*-6*); race 8 (*TLAUDPC*^{8}*-6* and *TLDC*^{8}*-6*) and race 7 (*SDC*^{7}*-6*) in trifoliate leaf, pod and stem. Previously, Trabanco et al. (2014) detected three halo blight resistance QTLs (*Psp6.1*^{812XC}, *Psp6.1*^{684XC} and *Psp6.2*^{684XC}) on LG06. However, the absence of common loci between both maps does not allow determining whether it is the same region. Furthermore, QTLs with opposite additive values co-localized in the same genomic region. Thus, QTLs with contrasting resistance effects for several races or organs were found at the same position on LG05 (*SDC*^{1}*-5* vs. *PLAUDPC*^{9}*-5*), on LG07 (*SAUDPC*^{7}*-7* vs. *PDC*^{1}*-7* and *PAUDPC*^{8}*-7*), and on LG08 (*SDC*^{1}*-8* vs. *PDC*^{1}*-8* and *PAUDPC*^{1}*-8.1*) (Tables 1 and 2). These results indicated that alleles from both parents may confer resistant or susceptible response to *P. syringae* infection depending on the inoculated organ or race.

Finally, only in one genomic region, BM164–PvCh02-48.5, were co-localized main-effect QTLs for resistance to races 3, 4 and 5 in all organs (*PLDC*^{3}*-2*, *PLDC*^{4}*-2*, *PLDC*^{5}*-2*, *PLAUDPC*^{3}*-2*, *PLAUDPC*^{4}*-2*; *SDC*^{3}*-2*, *SDC*^{4}*-2*, *SDC*^{5}*-2*, *SAUDPC*^{3}*-2, SAUDPC*^{5}*-2*; *PDC*^{3}*-2*, *PDC*^{4}*-2*, *PDC*^{5}*-2, PAUDPC*^{3}*-2*, *PAUDPC*^{4}*-2, PAUDPC*^{5}*-2*; *TLDC*^{5}*-2*, *TLAUDPC*^{4}*-2*, *TLAUDPC*^{5}*-2*).

Table 3 Epistatic QTLs (E-QTLs) for primary and trifoliate leaf resistance to nine halo blight races of the RIL population, grown under artificial and natural photoperiod conditions (A-Ppd and N-Ppd)

E-QTLi[a]	Marker interval	LG (pos.)[b]	E-QTLj[a]	Marker interval	LG (pos.)	*F* value[c]	AA[d]	h^2(aa)[e]
Primary leaf								
PLDC threshold *F* value: race 1 = 6.97, race 2 = 7.10, race 3 = 5.99, race 4 = 6.35, race 5 = 7.57, race 6 = 6.35, race 7 = 8.24, race 8 = 5.51, race 9 = 7.14								
E-PLDC^{1}*-3*	E31M51-60–PvCh03-43.3	03 (81.87–87.18)	*E-PLDC*^{1}*-9*	PvM128–IAC62	09 (13.77–15.99)	9.69	2.54***	8.41
E-PLDC^{2}*–2*	Leg735–PVEST008	02 (55.65–56.30)	*E-PLDC*^{2}*-3*	PvM152a–BMc259	03 (38.48–38.60)	10.83	−0.47***	10.48
E-PLDC^{3}*-8*	BMc222–BMd25	08 (13.63–15.71)	*E-PLDC*^{3}*-11*	BMd33–E45M50-328	11 (34.15–37.42)	8.98	0.43***	3.79
E-PLDC^{4}*-1*	PvCh01-10.6–BM53	01 (25.70–26.57)	*E-PLDC*^{4}*-6*	E40M60-166–E40M60-164	06 (6.34–6.53)	13.27	−0.83***	6.36
E-PLDC^{5}*-2*	PvCh02-33.8–BMc210	02 (40.85–42.77)	*E-PLDC*^{5}*-4*	SNP-5856–E31M61–313	04 (53.86–55.85)	10.34	−0.81***	7.06
E-PLDC^{5}*-8*	BMc330–PvCh08-45.7	08 (49.93–51.69)	*E-PLDC*^{5}*-9*	E40M50-47–E31M51-59	09 (61.16–64.29)	9.29	0.29***	6.79
E-PLDC^{6}*-3*	E45M50-389–IAC20	03 (91.01–107.60)	*E-PLDC*^{6}*-7*	BMc294–BMc248	07 (39.85–41.39)	7.98	1.58***	2.64
E-PLDC^{7}*-2*	PvCh02-33.8–BMc210	02 (40.85–42.77)	*E-PLDC*^{7}*-8*	E31M50-580–BMc330	08 (49.20–49.92)	7.22	−0.64***	4.77
E-PLDC^{8}*-8*	PvCh08-50.1–IAC22	08 (54.66–60.11)	*E-PLDC*^{8}*-9*	BMc184–PvCh09–15.1	09 (19.60–26.80)	8.91	0.26***	4.72
E-PLDC^{9}*-5.1*	IAC96–PvCh05-3.1	05 (0.00–16.79)	*E-PLDC*^{9}*-9*	BM202-PvCh09–30.8	09 (44.76–57.13)	15.87	0.81***	4.80
E-PLDC^{9}*-5.2*	PvCh05-33.9–PvCh05-35.9	05 (49.99–51.33)	*E-PLDC*^{9}*-8*	BMc121–BM165	08 (31.63–35.95)	16.81	−0.57***	7.70
PLAUDPC - threshold *F* value: race 1 = 5.65, race 5 = 7.23, race 7 = 6.54, race 8 = 7.54								
E-PLAUDPC^{1}*-8*	IAC22–E45M61-192	08 (60.11–64.39)	*E-PLAUDPC*^{1}*-11*	PvCh11-41.1–E32M51-124	11 (57.48–59.35)	14.09	39.63***	8.4
E-PLAUDPC^{5}*-1*	E31M51–298–BMb213	01 (32.31–35.01)	*E-PLAUDPC*^{5}*-9*	BM154–PvM128	09 (10.73–13.77)	8.41	−25.03***	5.09
E-PLAUDPC^{7}*-3*	PvM126–BMb194	03 (12.58–13.54)	*E-PLAUDPC*^{7}*-9*	IAC62–BMc184	09 (15.98–19.60)	15.87	−11.78***	8.55
E-PLAUDPC^{8}*-7*	E45M61-218–BM185	07 (17.21–24.61)	*E-PLAUDPC*^{8}*-9*	E40M31-151–BM154	09 (6.23–10.72)	17.41	18.09***	0.81
Trifoliate leaf								
TLDC Threshold *F* value: race 1 = 5.10, race 4 = 6.31, race 6 = 7.12, race 9 = 7.05								
E-TLDC^{1}*-1*	E43M38-137–BMb256	01 (23.01–24.11)	*E-TLDC*^{1}*-11*	E40M60-254–E45M61-387	11 (53.04–53.65)	5.42	0.39***	4.48
E-TLDC^{4}*-8*	PvCh08-45.7–BMc316	08 (51.69–53.20)	*E-TLDC*^{4}*-9*	BMc184–PvCh09-15.1	09 (19.61–26.81)	13.63	0.63***	7.28
E-TLDC^{6}*-1*	E31M51-298–BMb213	01 (32.31–35.01)	*E-TLDC*^{6}*-6*	E40M60-166–E40M60-164	06 (6.34–6.53)	11.48	−0.63***	8.12
E-TLDC^{6}*-3*	BM234–PvM126	03 (3.56–12.58)	*E-TLDC*^{6}*-7*	BMc338–BMc137	07 (63.68–65.52)	14.23	−0.41***	6.52
E-TLDC^{9}*-2*	BMc367–PvCh02-26.8	02 (8.47–14.55)	*E-TLDC*^{9}*-9*	BM154–PvM128	09 (10.72–13.77)	18.10	−0.52***	10.12
E-TLDC^{9}*-3*	PVEST042–BMc241	03 (46.37–50.17)	*E-TLDC*^{9}*-4*	SNP-4435–BMc155	04 (59.74–60.47)	8.89	−1.78***	5.15
TLAUDPC threshold *F* value: race 4 = 6.70, race 9 = 7.63								
E-TLAUDPC^{4}*-1.1*	BMb213–BM200	01 (35.01–49.89)	*E-TLAUDPC*^{4}*-10.2*	E31M51-284–E31M51-166	10 (39.73–40.42)	9.15	−27.29***	6.04
E-TLAUDPC^{4}*-1.2*	SNP-4423–IAC21	01 (51.74–58.19)	*E-TLAUDPC*^{4}*-10.1*	PvCh10-4.5–E31M51-284	10 (38.72–39.73)	6.78	−16.49***	5.21
E-TLAUDPC^{9}*-1.1*	BMb256–FJ51	01 (24.10–24.80)	*E-TLAUDPC*^{9}*-1.2*	E31M31-258–E31M31-273	01 (98.52–98.94)	12.79	−52.46***	8.54

Table 3 (continued)

E-QTLi[a]	Marker interval	LG (pos.)[b]	E-QTLj[a]	Marker interval	LG (pos.)	*F* value[c]	AA[d]	h^2(aa)[e]
E-TLAUDPC^{9}*-2*	PvCh02-26.8–PvM115	02 (14.55–16.42)	*E-TLAUDPC*^{9}*-9*	BM154–PvM128	09 (10.72–13.77)	18.24	−16.29***	12.43
E-TLAUDPC^{9}*-3*	PVEST042–BMc241	03 (46.37–50.17)	*E-TLAUDPC*^{9}*-4*	SNP-4435–BMc155	04 (59.74–60.47)	9.14	−38.50***	5.34

Predicted additive-by-additive epistatic effect by environment interaction effect (E-QE AAE) and estimated additive-by-additive epistatic effect*environment interaction effect [h^2 (aaei)]: *E-PLDC*^{9}*-3* = −1.29* A-Ppd (0.31)

PLDC primary leaf disease score, *PLAUDPC* primary leaf area under the disease progress curve, *TLDC* trifoliate leaf disease score, *TLAUDPC* trifoliate leaf area under the disease progress curve

[a] E-QTLi and E-QTLj are the two QTLs involved in epistatic interaction

[b] Linkage group and the estimated confidence interval of QTL position in brackets (in Kosambi cM)

[c] *F* values of significance of each epistatic interaction

[d] Estimated additive-by-additive epistatic effect. Experiment-wide *P* value: *$P \leq 0.05$; **$P \leq 0.01$; ***$P \leq 0.001$. Positive values indicate that alleles from PHA1037 increase the trait value, and negative values indicate that the increase in the trait is due to the presence of the alleles from PMB0225

[e] Percentage of the phenotypic variation explained by additive-by-additive epistatic effects

Most of these QTLs had a positive additive value, indicating that the resistance alleles came from PMB0225, except for the QTLs detected for resistance to races 3 and 5 in stem (*SDC*^{3}*-2*, *SAUDPC*^{3}*-2*, *SDC*^{5}*-2* and *SAUDPC*^{5}*-2*), which showed that alleles from the susceptible parent PHA1037 also contribute to stem resistance. This result was supported by the significant and negative correlations found for resistance to races 3 and 5 between stem and other organs (Supplementary file 5: Table S3). Miklas et al. (2011) mapped the *Pse-3* gene at the end of LG02, responsible for resistance to races 3 and 4. *Pse-3* is linked to *I* gene and SW13 marker of BCMNV resistance (Melotto et al. 1996), which was included in our linkage map and located close to one of the flanking markers (PvCh02–48.5) of this genomic region. PMB0225 parent was fully resistant to races 3 and 4 in all organs, although also presented intermediate resistance to the other races in primary leaf. Fourie et al. (2004) found that certain genomic regions accumulate *R* genes and QTLs that confer complete and quantitative resistance, and Gebhardt and Valkonen (2001) observed that QTLs involved in quantitative or partial resistance were co-localize with weak or defeated *R* genes. Thus, it is not possible to conclude whether the resistance of this genomic region containing non-organ and non-race specific QTLs is provided by the *Pse-3* gene or by genes and/or QTLs with race-specific resistance, tightly linked to *Pse-3* gene. Therefore, for application in marker-assisted breeding of partial halo blight resistance into common bean cultivars, QTLs that contribute to the highest proportion of the phenotypic variation and are consistently detected using multiple isolates and different organs will be the most viable candidates.

Co-localization of QTLs with known resistance genes

The association between NL and defence genes and QTLs conferring resistance to halo blight species has been reported in this work. The homologous regions spanning 26 of the 76 main-effect QTLs identified tested positive for the presence of known resistance genes (Supplementary file 6: Table S4).

The main-effect QTLs detected on LG02 for resistance to races 3, 4 and 5 in stem, pod, primary and trifoliate leaf were positioned within a 3.01-Mb region where the NL *Phvul.002G323300* gene is located. Based on BLAST homology search, it can be considered an important candidate gene for the non-organ and non-race-specific QTLs identified here. This candidate

Table 4 Epistatic QTLs (E-QTLs) for stem and pod resistance to nine halo blight races of the RIL population, grown under artificial and natural photoperiod conditions (A-Ppd and N-Ppd)

E-QTLi[a]	Marker interval	LG (pos.)[b]	E-QTLj[a]	Marker interval	LG (pos.)	*F* value[c]	AA[d]	h^2(aa)[e]
Stem								
SDC threshold *F* value: race 1 = 6.78, race 3 = 7.28, race 4 = 7.70, race 5 = 6.64, race 8 = 7.85, race 9 = 5.45								
E-SDC^1-5	IAC96–PvCh05-3.1	05 (0.00–16.79)	*E-SDC^1-7*	P–BMc294	07 (32.77–39.85)	25.41	−0.86***	12.37
E-SDC^3-1	E43M38-138–E43M38-137	01 (22.15–23.01)	*E-SDC^3-8*	PvCh08-45.7–BMc316	08 (51.69–53.20)	18.17	−0.58***	8.56
E-SDC^3-3	BM265–BM234	03 (0.00–3.56)	*E-SDC^3-10*	E31M51-284–E31M51-166	10 (39.73–40.42)	8.76	0.23***	7.28
E-SDC^4-8	PvCh08-45.7–BMc316	08 (51.69–53.20)	*E-SDC^4-9*	BMc184–PvCh09-15.1	09 (19.61–26.81)	13.03	1.03***	8.30
E-SDC^5-1	BMc324–E32M51-329	01 (79.94–90.92)	*E-SDC—5*	PvCh05-30.4–BMc321	05 (42.07–43.73)	9.44	−1.90***	7.33
E-SDC^8-3	E45M50-389–IAC20	03 (91.01–107.60)	*E-SDC^8-11*	PvCh11-2.7–E31M50-62	11 (21.77–23.48)	13.70	−0.42***	7.09
E-SDC^9-2	BMc280–BM139	02 (56.81–65.01)	*E-SDC^9-6*	BMc238–E40M60-91	06 (2.36–4.96)	14.43	−0.84***	7.94
SAUDPC - threshold *F* value: race 1 = 8.44, race 4 = 6.09, race 8 = 5.73, race 9 = 6.76								
E-SAUDPC^1-4	SNP-5459–SNP-5856	04 (50.19–53.86)	*E-SAUDPC^1-8*	E31M51-177–BMc222	08 (7.83–13.63)	12.84	−43.24***	3.23
E-SAUDPC^4-4	BM171–SNP-5017	04 (64.47–66.29)	*E-SAUDPC^4-11*	PV-ag001–PvCh11-49.1	11 (67.35–68.57)	9.48	186.82***	8.80
E-SAUDPC^8-2.1	GATS91–BMc367	02 (0.00–8.47)	*E-SAUDPC^8-2.2*	SNP-3999–E31M50-139	02 (48.87–50.12)	12.10	−20.23***	5.67
E-SAUDPC^9-2	BMc280–BM139	02 (56.81–65.01)	*E-SAUDPC^9-6*	BMc238–E40M60-91	06 (2.36–4.96)	10.33	−42.61***	4.97
Pod								
PDC threshold *F* value: race 1 = 6.23 race 2 = 6.30, race 6 = 7.59, race 8 = 5.51, race 9 = 6.44								
E-PDC^1-7.1	E31M31-187–E45M61-218	07 (0.00–17.21)	*E-PDC^1-8.1*	BMc121–BM165	08 (31.64–35.96)	15.59	−0.38***	6.57
E-PDC^1-7.2	BM185–P	07 (24.61–32.77)	*E-PDC^1-8.1*	BMc121–BM165	08 (31.64–35.96)	17.71	−0.35***	6.63
E-PDC^1-8.2	BMc147–BM211	08 (44.34–45.51)	*E-PDC^1-9*	PvCh09–30.8–E40M50–51	09 (57.14–59.86)	6.82	−0.88***	3.44
E-PDC^2-1	IAC93–E31M50-264	01 (27.66–30.19)	*E-PDC^2-9.3*	BMc318–BMc269	09 (85.83–91.65)	8.69	−0.33***	4.68
E-PDC^2-8.1	BMc121–BM165	08 (31.64–35.96)	*E-PDC^2-9.2*	PvCh09–30.8–E40M50-51	09 (57.14–59.86)	2.98	−0.65***	4.27
E-PDC^2-8.2	BM165–BMc147	08 (35.96–44.34)	*E-PDC^2-9.1*	BM202–PvCh09-30.8	09 (44.76–57.14)	8.20	−0.47***	3.05
E-PDC^6-9	IAC62–BMc184	09 (15.98–19.60)	*E-PDC^6-10*	BMc159–SNP-2521	10 (47.60–51.81)	13.46	−0.51***	6.36
E-PDC^8-3.1	E31M51-55–E31M51-60	03 (79.66–81.87)	*E-PDC^8-5.1*	E32M60-100–BM138	05 (44.91–45.76)	6.96	0.36***	3.45
E-PDC^8-3.2	E31M51-60–PvCh03-43.3	03 (81.87–87.18)	*E-PDC^8-5.2*	E32M60-263–PvCh05-33.9	05 (47.36–49.99)	12.73	−0.51***	6.01
E-PDC^9-4	BSNP-49–BMc292	04 (25.39–30.95)	*E-PDC^9-11*	PvCh11-41.1–E32M51-124	11 (57.48–59.35)	9.87	0.41***	5.51
PAUDPC - threshold *F* value: race 2 = 5.70, race 6 = 7.72, race 9 = 6.14								
E-PAUDPC^2-8	BM165–BMc147	08 (35.96–44.34)	*E-PAUDPC^2-9*	PV-at007–BM202	09 (31.73–44.76)	8.72	−24.98***	2.95
E-PAUDPC^6-1.1	E32M60-147–E43M38-138	01 (1.03–22.15)	*E-PAUDPC^6-7*	E45M61-218–BM185	07 (17.21–24.61)	9.61	65.92***	8.52
E-PAUDPC^6-1.2	E31M51-297–E31M51-298	01 (31.52–32.31)	*E-PAUDPC^6-5*	PvCh05-33.9–PvCh05-35.9	05 (49.99–51.33)	9.01	64.57***	4.31

Table 4 (continued)

E-QTLi[a]	Marker interval	LG (pos.)[b]	E-QTLj[a]	Marker interval	LG (pos.)	*F* value[c]	AA[d]	h^2(aa)[e]
E-PAUDPC^{9}*-3*	PvM126–BMb194	03 (12.58–13.54)	*E-PAUDPC*^{9}*-8*	BM189–E31M51-177	08 (0.00–7.82)	7.88	16.77***	4.43

Predicted additive-by-additive epistatic effect by environment interaction effect (E-QE AAE) and estimated additive-by-additive epistatic effect*environment interaction effect [h^2 (aae)]: *E-SDC*^{8}*-3* = −13.34* A-Ppd (0.29), 12.67* N-Ppd (0.29)

SDC stem disease score, *SAUDPC* stem area under the disease progress curve, *PDC* pod disease score, *PAUDPC* pod area under the disease progress curve

[a] E-QTLi and E-QTLj are the two QTLs involved in epistatic interaction

[b] Linkage group and the estimated confidence interval of QTL position in brackets (in Kosambi cM)

[c] *F* values of significance of each epistatic interaction

[d] Estimated additive-by-additive epistatic effect. Experiment-wide *P* value: *$P \leq 0.05$; **$P \leq 0.01$; ***$P \leq 0.001$. Positive values indicate that alleles from PHA1037 increase the trait value, and negative values indicate that the increase in the trait is due to the presence of the alleles from PMB0225

[e] Percentage of the phenotypic variation explained by additive-by-additive epistatic effects

gene showed homology to the *Arabidopsis TAO1* (target of *AvrB* operation) conditioning resistance to the *P. syringae* avirulence *AvrB* gene (Eitas et al. 2008). It is located within a cluster of nine NL genes (*Phvul.002G314200* to *Phvul.002G324600*), where the *I* gene for resistance to *BCMNV* and other related potyviruses are located (Schmutz et al. 2014; Bello et al. 2014). The *I* gene co-segregates with *Pse-3* gene for resistance to races 3 and 4 and it was mapped in an interval ~25 kb of the *Phvul.002G323800* gene (Teverson et al. 1991; Fisher and Kyle 1994; Collmer et al. 2000; Vallejos et al. 2006; Miklas et al. 2011). Seven of the NL genes (*Phvul.002G323000* to *Phvul.002G323800*) showed homology with two genes of *G. max* (*Glyma.01G033200* and *Glyma.01G033300*), which are involved in bacterial leaf resistance (Kang et al. 2012). However, since regions containing NL genes could be susceptible to chromosomal rearrangement and transposition or genomic duplication (Meyers et al. 2005), it is not possible to determine whether the detected non-organ and non-race specific resistance resulted from the pleiotropic effect of the *Phvul.002G323300* gene or from the clustering of different genes. Therefore, further studies on fine mapping of the target genomic regions would be necessary to draw definitive conclusions.

Most of the identified candidate genes showed conserved syntenic relationships with NL genes in other legumes such as *G. max*. Thus, seven (*Phvul.002G323000*, *Phvul.002G323100*, *Phvul.002G323200*, *Phvul.002G323300*, *Phvul.002G323400*, *Phvul.002G323500*, *Phvul.002G323800*) and four (*Phvul.011G014200*, *Phvul.011G014300*, *Phvul.011G014400* and *Phvul.011G014500*) NL genes located on Chr02 and Chr11, respectively, presented homology with two (*Glyma.01G033200*, *Glyma.01G033300*) and one (*Glyma.12G011700*) NL genes in the counterpart region of Chr01 and Chr12 of *G. max* genome, respectively, which are involved in bacterial leaf resistance in soybean (Kang et al. 2012); while one NL gene (*Phvul.008G172400*) on Chr08 appeared to be unique to the common bean species. These results suggested that said *R* gene clusters could arise by several duplication events in the common bean lineage after divergence of both legume species and that the number of *R* genes in the identified genomic regions did not proportionally increase in soybean genome according to the whole genome duplication event since its divergence from common bean (Shoemaker et al. 1996).

Concluding remarks

The results stated herein provide essential information not only for a better understanding of the common bean-*Pseudomonas syringae* pv. *phaseolicola* interaction but also for the application of genomic assisted breeding for halo blight resistance in common bean. This research has also shown the importance of the epistatic effects in genetic resistance to halo blight, which has not been studied so far. Thereby, both main and epistatic interaction effects of genes or QTLs should be considered for a successful application of MAS, which provides an opportunity to use a pyramiding strategy for durable resistance. As well as providing useful tools for MAS of halo blight resistance in common bean, this work also offers valuable clues for further study on cloning the candidate gene corresponding to the non-organ and non-race specific QTLs for resistance to races 3, 4 and 5 located on Chr02.

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