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ORIGINAL PAPER

Are the polygenic architectures of resistance to *Phytophthora capsici* and *P. parasitica* independent in pepper?

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Abstract The pepper accession Criollo de Morelos 334 is the most efficient source of resistance currently known to *Phytophthora capsici* and *P. parasitica*. To investigate whether genetic controls of resistance to two *Phytophthora* species are independent, we compared the genetic architecture of resistance of CM334 to both *Phytophthora* species. The RIL population F5YC used to construct the high-resolution genetic linkage map of pepper was assessed for resistance to one isolate of each *Phytophthora* species. Inheritance of the *P. capsici* and *P. parasitica* resistance was polygenic. Twelve additive QTLs involved in the *P. capsici* resistance and 14 additive QTLs involved in the *P. parasitica* resistance were detected. The QTLs identified in this progeny were specific to these *Phytophthora* species. Comparative mapping analysis with literature data identified three colocations between resistance QTLs to *P. parasitica* and *P. capsici* in pepper. Whereas this result suggests presence of common resistance factors to the two *Phytophthora* species in pepper, which possibly derive from common ancestral genes, calculation of the colocation probability indicates that these colocations could occur by chance.

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Introduction

Phytophthora are currently re-emerging all over the world, like *P. ramorum*, which has a host range of over 40 plant genera and causes the dramatic sudden oak death in Europe and North America (Rizzo et al. 2005), and *P. infestans* and *P. parasitica* causing late blight and root-rot on tomatoes (Camele et al. 2005). *Phytophthora* spp. is responsible for some of the most destructive plant diseases in the world and is arguably the most devastating pathogens of dicotyledonous plants, including agronomically important Solanaceous crops. Misclassification of *Phytophthora* (Oomycete) as ''fungus'' induced incongruous crop management to control epidemics (Govers 2001).

Oomycetes form a unique lineage of eukaryotic plant pathogens that evolved independently from the true fungi and are closely related to heterokont (i.e. brown algae). There are ~60 described *Phytophthora* species (Erwin and Ribeiro 1996). Phylogenetic relationships among *Phytophthora* species were examined on the basis of the internal transcribed spacer sequence of genomic ribosomal DNA (Cooke et al. 2000). The *Phytophthora* genus forms a monophyletic group of eight clades. This cluster encompasses aquatic necrotrophs such as *P. capsici* and *P. parasitica*, as well as aerial biotrophs such as *P. infestans*. Looking closer permits to suggest that the biotrophic Oomycete species infecting a narrow host range may derive relatively recently from the hemi-biotrophic or necrotrophic broad host range *Phytophthora* ancestors (Cooke et al. 2000). For instance, *P. infestans*, the well-known causal agent of potato late blight, mainly infects potato and tomato, and occasionally some other Solanaceae genera, while *P. capsici* and *P. parasitica* infect a broader host range including several plant families. Previously grouped together by Waterhouse (1963), *P. capsici* and *P. parasi-*

tica were separated in two neighboring clades in the new molecular classification of Cooke et al. (2000).

The development of crops that possess durable genetic resistance provides the best prospect for efficient, economical and environmentally safe control of *Phytophthora*. Natural resistance to *Phytophthora* was found in some species. Some race-specific resistances are simply inherited, such as *R1* to *R11* genes from *Solanum demissum* introgressed in potato, and *Ph-1* to *Ph-3* genes from *Solanum pimpinellifolium* L. (formerly, *Lycopersicon pimpinellifolium* [L.] Miller) introgressed in tomato. But most of the *R* genes, largely deployed in cultivars, time and space, have already been overcome because of the emergence of virulent races of *Phytophthora*. Quantitative and polygenic resistance, also characterized and exploited in breeding, can confer an efficient control of disease severity (Palloix et al. 1988; Thabuis et al. 2004). Quantitative resistance is of high interest in disease management strategies because of differences in durability in the field (Ayme 2005). However, the extent to which durable partial resistance shares genetic components with *R* genes remains unclear.

Phytophthora capsici Leonian has for a long-time been considered responsible for the root-rot in pepper (*Capsicum* spp.). The symptoms are a sudden irreversible wilt followed by the death of the plant. More recently, *P. parasitica* has been described as a pathogen of several crops including *C. annuum*. Root rot caused by *P. parasitica* was first reported in Tunisia then in India and Spain (Allagui et al. 1995; Verma et al. 2001; Andres et al. 2003). Due to similarities of symptoms on roots and collar, *P. capsici* and *P. parasitica* may cause diagnostic confusion.

To date, no pepper accession showing complete resistance to *Phytophthora* wilt have been found. A number of *C. annuum* accessions were reported to be partially resistant to *P. capsici*. The accession Criollo de Morelos 334 (CM334), considered as being the most efficient source of resistance currently known, is largely used in breeding programs. Several conflicting genetic controls have been suggested, going from two recessive genes to QTLs with epistatic effects (Guerrero-Moreno and Laborde 1980; Pochard et al. 1983; Ortega et al. 1992; Reifschneider et al. 1992; Thabuis et al. 2003). Thabuis et al. (2003) demonstrated that the major effect QTL on the chromosome P5 confers resistance towards two *P. capsici* isolates with different levels of aggressivity. Comparative mapping (Pflieger et al. 2001; Thabuis et al. 2003) indicated colinearities between this QTL and resistance QTLs to *P. infestans* on potato chromosome IV (Leonards-Schippers et al. 1994). By assessing the CM334 resistance to both *P. parasitica* and *P. capsici* in 30 doubled haploid lines issued from crosses between susceptible lines and CM334, Allagui et al. (2001) showed significant correlations (r = 0.47–0.52), hypothesizing the presence of common genetic factors. But no molecular dissection of the CM334 resistance to *P. parasitica* has yet been achieved to corroborate this hypothesis.

Considering the monophyletic origin of *Phytophthora* and the QTL colinearity previously reported, one arising question is to know whether the genetic controls of the *Phytophthora* resistance in pepper could share any common genetic factor and more particularly whether the major effect QTL on chromosome P5 could have any effect on the resistance to *P. parasitica*. Answering these questions could help to decipher the molecular bases of the pepper resistance specificity to *Phytophthora* species and to investigate whether partial resistance loci could have a resistance spectrum including several *Phytophthora* species.

We report here the genetic map location of QTLs controlling the partial resistance to *P. capsici* and *P. parasitica* originating from the CM334 pepper genitor using the high resolution map published by Barchi et al. (2007). Comparison of the QTL locations issued from different experiments revealed colinearities between QTLs to *P. capsici* and *P. parasitica* in pepper, but probability calculation indicates that QTL colocations could occur by chance.

Materials and methods

Plant material and genetic map

The American bell pepper inbred line Yolo Wonder (YW) is susceptible to *P. capsici* and *P. parasitica*, while the Mexican chili line Criollo de Morelos 334 (CM334) is partially resistant (Pochard et al. 1983). The F5YC progeny, first described by Barchi et al. (2007), counts 297 recombinant inbred lines (RIL) derived from the cross Yolo Wonder × Criollo de Morelos 334. Led up to the F5 generation, this progeny displays a theoretical homozygous rate of 93.75%. The framework linkage map of the F5YC progeny (done on the 297 F5 lines) includes 323 molecular markers (AFLP, SSR, RFLP, SSAP) distributed on the 12 haploid pepper chromosomes covering 1553 cM, plus 26 unassigned small linkage groups covering 304 cM, for a total length of 1857 cM (Haldane) with an average inter-marker distance of 5.71 cM (SD: ±5.70 cM). The genome coverage of the F5YC map was estimated to 86.5%. Because Barchi et al. (2007) applied very stringent thresholds (LOD > 8, r < 0.1) for ensuring high confidence in marker placement during the map construction, 98 additional markers, unlinked to the map, were available for linear regression analyses. The framework linkage map and unlinked markers were used for further QTL analyses.

Each F5YC RIL was selfed, and the F6 families (F6YC) were used for phenotypic assessment. A set of 200 F5YC

RILs was selected as the most informative by applying the ''sampleexp'' command of the MapPop software (Brown and Vision 2000) on the F5YC linkage map as described by Barchi et al. (2007).

Phytophthora isolates and resistance assays

Two *Phytophthora* species were used for stem and/or root inoculations of the F6YC progenies, the parents YW and CM334, and the F1-hybrid. The *P. capsici* isolate *Pc*197 is the very aggressive isolate used by Thabuis et al. (2003). The *P. parasitica* isolate *Pp*329 was obtained from the INRA (Sophia-Antipolis) *Phytophthora* collection; it was isolated from tobacco and showed a very polyphagous behaviour on several plant species (F. Panabières, personal communication). Isolates were maintained and the inoculum was produced as described by Lefebvre and Palloix (1996). Resistance to *P. capsici* was evaluated by two independent artificial inoculation tests (root test and stem test), while resistance to *P. parasitica* was evaluated by the ''stem test'' only. Resistance tests were performed at 22°C for *P. capsici* and 24°C for *P. parasitica* in controlled growth chambers with 12 h light.

The ''root test'' was performed on 3-week-old plantlets held in glass container filled with a nutritive solution (Lefebvre and Palloix 1996). For each F6 family, two containers of 16 plantlets each were inoculated by dipping four ∅ 4-mm mycelium plugs of *P. capsici* into the container. Seven days post-inoculation (dpi), the root necrosis extension due to zoospore infection and mycelium extension in the root tissue was evaluated for each plantlet according to a semi-quantitative resistance criterion ranging from 0 (resistant) to 5 (susceptible), and a mean necrotic root rot index (RRI) was calculated for each container. Experimental design was arranged in two randomized complete blocks, with for each progeny a container of 16 plants per block. The 200 F6YC families were split out into 2 independent sets that were separately assessed for *P. capsici* resistance in the INRA-Montfavet laboratory. Parental lines and the F1-hybrid were included as control in each experimental set.

The ''stem test'' was performed on six-leaf stage plants beheaded and inoculated by depositing a ∅ 4-mm mycelium plug on the fresh stem section (Lefebvre and Palloix 1996). The length of stem necrosis induced by *Phytophthora* was measured at 3, 7, 10, 14, 17 and 21 dpi (L3 to L21 in mm) and the speed of the necrosis spread was calculated for each scoring date (S3 to S21 in mm/day). Four resistance components were considered: L21 (mm, Length 21 dpi); REC (mm/day, Receptivity) measures the speed of necrosis spread between the first and the third dpi (S3); S10 [mm/day, assimilated to the Inducibility as referred by Thabuis et al. (2004)] measures the speed of necrosis spread between the seventh and the tenth dpi; STA (mm/day, Stability) measures the average speed of necrosis spread between the 14th and the 21st dpi ([S14 + S17 + S21]/3). For the ''stem test'' to *P. capsici*, the 200 F6YC families were split out into five independent sets that were separately assessed in the INRA-Montfavet laboratory for two sets, and in the Vilmorin laboratory (Ledenon, France) for three sets. For the ''stem test'' to *P. parasitica*, the 200 F6YC families were split out into two independent sets assessed in the INRA-Montfavet laboratory. Common controls were included in each experimental set.

Data and QTL analyses

Statistical analyses were performed using the package for the general statistical software RGUI (R Graphical User Interface) version 2.2.1 (R. Development Core Team 2005). The ''laboratory'' (INRA vs. Vilmorin) and ''set'' effects were tested by analyses of variance (ANOVA) on phenotypic raw data of controls (the two parents and the F1-hybrid). The ''block'' and ''genotype'' effects were tested by ANOVA on raw data of RILs and controls. In order to merge raw data from the different sets in a single data set, raw data for each resistance component were standardized ($\mu = 0$ and $\sigma = 1$ on all the progeny data). Further analyses were performed with these values. Broad-sense heritabilities (h^2_{BS}) of the nine resistance components were calculated with the formula $h^2_{BS} = \frac{\sigma^2_g}{\sigma^2_g + \left(\frac{\sigma^2_e}{n}\right)}$ where σ^2_g is the genetic variance, σ^2_e the environmental variance, and n the number of replicates per genotype. Normality of distribution was checked by a Shapiro and Wilk test.

QTLs were detected by the composite interval mapping (CIM) method with the QTL Cartographer software (Basten et al. 1997). A maximum of five markers, selected by a forward–backward stepwise regression analysis, was used as cofactors, with a window size of 10 cM and a walking step of 2 cM. Significance thresholds were computed by 1000-permutation tests. The LOD score thresholds, calculated for a type-I-error of 0.10, were as followed: Pc_RRI = 2.80, Pc_L21 = 2.76, Pc_REC = 2.76, Pc_S10 = 2.80, Pc_STA = 2.79, Pp_L21 = 2.72, Pp_REC = 2.91, Pp_S10 = 2.98, Pp_STA = 4.05. When several QTLs were detected within less than a 20-cM interval, only the marker with the highest LOD value was retained. When several linked markers were significantly associated with the resistance, we considered the overall region as a single QTL and indicated the linked marker exhibiting the highest R^2 value.

To identify markers significantly associated with the resistance but outside the map coverage, we tested the effect of the 98 unlinked molecular markers on the 9

quantitative resistance components by a one-way linear regression analysis (LR). The threshold for declaring a significant association was determined by a 1000-permutation test to overcome problems of multiple testing (type-I-error of 0.01, LOD = 2.4).

The QTL nomenclature indicates the initials of the *Phytophthora* species, followed by one digit indicating the pepper chromosome or linkage group number, and the rank number on the chromosome or linkage group separated by a dot. For QTLs associated with unlinked markers, the U letter replaces the chromosome or linkage group number.

Digenic interactions between all the available markers were tested using a two-way ANOVA with each resistance component, as described by Lefebvre and Palloix (1996). Significant epistasis was retained when $P < 10^{-5}$. Percentages of phenotypic variation explained by the individual markers (R^2) and by all the associated markers (GR^2) identified for a given trait were obtained by simple and multiple stepwise regressions.

To compare the map location of resistance QTLs to *P. capsici* and *P. parasitica* in pepper, we constructed a pepper synthetic map by compiling map information of the literature (data not shown). The comparison of QTL distribution issued from different experiments relies on the orthologous markers with which we virtually divided the genome in distinct chromosomal segments. Colinear segments were defined as the map regions bearing the same orthologous markers at its extremities. QTLs were assigned to segments by a homothetic projection process. Indeed, when two genetic maps share common loci, those loci can be considered as bridges between maps. Thus, projection of the remaining loci, including QTL, from the first map to the others is possible. We considered that QTLs colocated when they mapped in colinear segments. The probability P that QTL colocations between the F5YC QTLs and the QTLs reported in literature occurred by chance was calculated according to Lin et al. (1995) by the equation: $P = \frac{C_l^m}{C_n^s} x C_{n-l}^{s-m}$, where m, the number of colocations; l, the total number of QTL-carrier segments described in literature; s, the number of QTLs identified in our study; and n, the total number of colinear segments that could be compared. When $P > 0.05$, colocations could occur by chance.

Results

CM334 is partially resistant to both *Phytophthora* species

The length of stem necrosis after inoculation of *Pc*197 (*P. capsici*) and *Pp*329 (*P. parasitica*) increased differently during the 21 days post-inoculation in both parental genotypes and their F1-hybrid (Fig. 1). The necrosis lengths were significantly longer with *Pc*197 than with *Pp*329 for every genotype at every checking date ($P < 0.05$), indicating that *P. capsici* was more aggressive than *P. parasitica* in our experimental conditions on the tested pepper genotypes. Necrosis lengths were significantly different between CM334 and YW at every date with both *Pc*197 and *Pp*329 ($P < 0.05$), CM334 exhibiting partial resistance to both *Phytophthora* species. With *Pc*197, the F1-hybrid showed an intermediate response significantly different from both parents, indicating an incomplete dominance of the resistance response to *P. capsici*, while with *Pp*329, the F1-hybrid was not significantly different from CM334 ($P > 0.05$), suggesting a rather complete dominance of the resistance to *P. parasitica*.

Resistances to both *Phytophthora* species are polygenic

Two-hundred RIL F5 from the mapping population F5YC (Barchi et al. 2007) were assessed for resistance to the isolates *Pp*329 and *Pc*197 by progeny evaluation on F6 families. Nine resistance components were obtained from inoculation with both isolates: four components were measured with both *Pc*197 and *Pp*329 isolates (REC, S10, STA and L21) and one component was specific to *Pc*197 inoculation (RRI). Analyses of variance on the nine resistance components revealed that the ''laboratory'', ''set'', ''block'' and interaction effects were not significant on the standardized data.

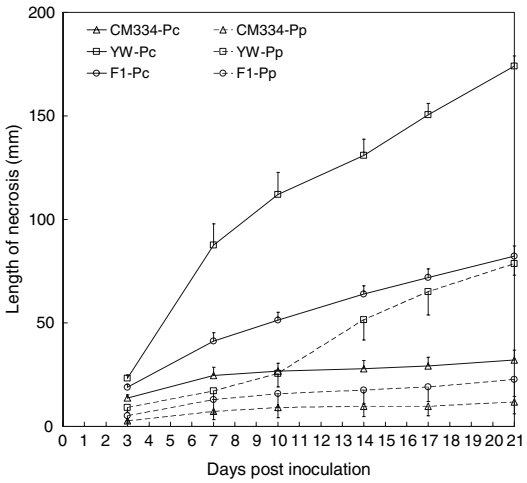

Fig. 1 Evolution of mean lengths of stem necrosis for the resistant accession CM334, the susceptible cultivar Yolo Wonder (YW) and the F1-hybrid inoculated by the isolate *Pc*197 of *P. capsici* (*continuous lines*) and the isolate *Pp*329 of *P. parasitica* (*dashed lines*) during 21 days post-inoculation. *Vertical bars* represent 95% confidence interval. For representation facilities, *bars* are turned up for *P. capsici* and down for *P. parasitica*. (YW *squares*, F1 *circles* CM334 *triangles*)

The Pearson's correlation coefficients (Table 1) between the resistance components to *P. capsici* ranged from 0.47 to 0.93 and were highly significant ($P < 0.001$). Correlations between *P. parasitica* resistance components were lower (from 0.01 to 0.66) but still significant ($P < 0.05$), except between Pp_REC and Pp_S10 ($P = 0.859$), and to a smaller extent between Pp_REC and Pp_STA ($P = 0.050$). Correlations between *P. capsici* and *P. parasitica* resistance components were lower, ranging from –0.01 to 0.34, and less significant than the latter. Low correlations indicate that genetic factors of resistance to *P. capsici* and *P. parasitica* are mostly independent.

The continuous distribution of standardized phenotypic data for the nine resistance components (Fig. 2) suggests that the resistances to *P. capsici* and *P. parasitica* are both polygenic. Transgressive segregants were observed for almost all the trait/isolate combinations. The traits related to *P. parasitica* resistance were generally biased towards resistance and showed less variation than traits related to *P. capsici*. This is consistent with the fact that YW was more susceptible to *P. capsici* than to *P. parasitica*. Conversely, the Pc_RRI trait was biased towards susceptibility. Despite the biased distribution of the phenotypic data, residues of the ANOVA model testing the genotype effect were normally distributed according to the W index of Shapiro and Wilks.

For each resistance component, the broad-sense heritabilities (h^2_{BS}) were computed on the merged standardized data (Table 2). They ranged from 0.80 to 0.96 indicating that phenotypic values are poorly affected by environmental effects, except for Pc_RRI (0.69) and Pp_S10 (0.47).

QTL mapping

QTL detection was performed for each trait independently. Results of the CIM method applied to the framework map and the LR applied to the unlinked markers are summarized in Table 2 and illustrated on Fig. 3.

Resistance to *P. capsici*

Eight QTLs involved in the *P. capsici* resistance were detected on the chromosomes P1, P4, P5, P6, and P11. For each QTL located on the framework map, the CM334 allele increased the resistance level. Four of these QTLs had an effect on several resistance components while the other 4 QTLs affected a single resistance component. The most consistent QTL, named *Pc_5.1* and located on the chromosome P5, displayed a major effect whatever the resistance component considered. It explained from 20.30 to 53.27% of the observed variation, depending on the considered resistance component. Another major effect QTL, named *Pc_5.2*, was located on the chromosome P5 around 44 cM below the former, and explained 23.63% of the variance of the Pc_RRI trait only. The plants carrying resistant alleles at both *Pc_5.1* and *Pc_5.2* QTLs were significantly more resistant (RRI mean = –0.56 for 70 plants) than those carrying a resistance allele at a single QTL (RRI mean = –0.18 with a resistant allele at *Pc_5.1* for 29 plants, RRI mean = 0.38 with a resistant allele at *Pc_5.2* for 18 plants). The other QTLs assigned to the framework map displayed weaker effects ranging from 2.83 to 9.21%.

The LR analyses identified 4 additional unlinked markers associated with the *P. capsici* resistance. For 3 of these QTLs, the CM334 allele increased the resistance level, whereas for *Pc_U4* QTL, the YW allele increased the resistance level. *Pc_U1* QTL linked to the e41/m54_353y marker was identified with the 5 *P. capsici* resistance components and explained from 8.05 to 18.18% of the trait variation. The 3 other QTLs were detected with 2 to 3 *P. capsici* resistance components and explained up to 13.74% of the trait variation. One single epistatic interaction was observed for the Pc_REC trait. Markers p19/m42_874c of LG41 and p14/m39_210c of P1 had significant effect ($P = 7.5$ 10–6) with R^2 value of 16%.

Multiple regressions revealed that the detected additive QTLs determined from 36.71 to 56.49% of the total phe-

Table 1 Pearson correlation coefficients between traits for *P. capsici* and *P. parasitica* resistance

	Pc_L21	Pc_REC	Pc_ S10	Pc_STA	Pc_RRI	Pp_L21	Pp_REC	Pp_S10
Pc_REC	0.69 ***							
Pc_S10	0.86 ***	0.57 ***						
Pc_STA	0.93 ***	0.53 ***	0.77 ***					
Pc_RRI	0.59 ***	0.47 ***	0.50 ***	0.56 ***				
Pp_L21	0.29 ***	0.22 **	0.25 ***	0.26 ***	0.09 NS			
Pp_REC	0.12 NS	0.08 NS	0.11 NS	0.11 NS	–0.01 NS	0.66 ***		
Pp_ S10	0.19 **	0.20 **	0.23 **	0.16 **	0.08 NS	0.33 ***	0.01 NS	
Pp_STA	0.34 ***	0.28 ***	0.32 ***	0.31 ***	0.20 **	0.64 ***	0.14 *	0.23 **

NS not significant ($P \geq 0.05$); * significant at $P < 0.05$; ** significant at $P < 0.01$; *** significant at $P < 0.001$

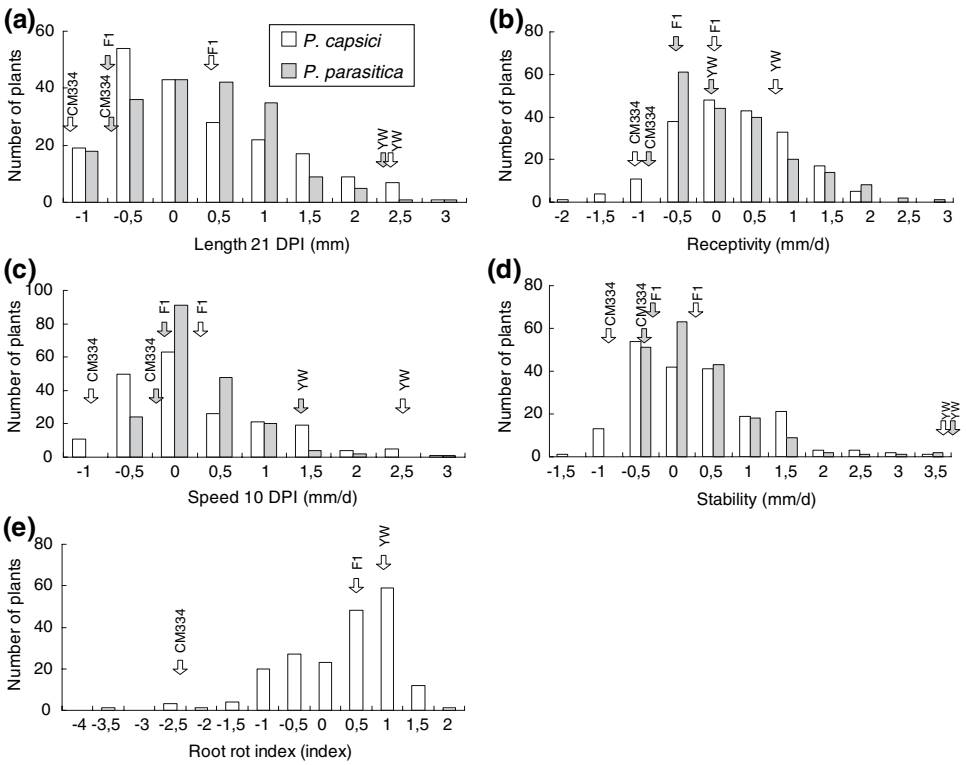

Fig. 2 Phenotypic distributions of the F6YC families for resistance components to *P. capsici* (Pc_L21 (**a**), Pc_REC (**b**), Pc_S10 (**c**), Pc_STA (**d**), Pc_RRI (**e**)) in *white bars*, and *P. parasitica* (Pp_L21 (**a**), Pp_REC (**b**), Pp_S10 (**c**), Pp_STA (**d**)) in *grey bars*. The *x*-axis indicates the standardized phenotypic values ($l = 0$ and $r = 1$). The *y*-axis indicates the number of F6YC families for each phenotypic class. *Arrows* indicate the positions of the mean resistance values of the parents (CM334 and YW) and the F1-hybrid (F1): *white* for *P. capsici* and *grey* for *P. parasitica*

notypic variation depending on the trait (Table 2). When adding the markers of the epistatic interaction, the global R^2 increased to 53.49% for Pc_REC. Taking into account heritability values, QTLs explained from 42 to 73% of the genetic variation of the *P. capsici* resistance components.

Resistance to *P. parasitica*

Eleven QTLs involved in the *P. parasitica* resistance were detected on four chromosomes (P3, P9, P10a, and P11) and five unassigned linkage groups (LG25, LG27, LG29, LG37, LG45). For each QTL of the framework map, the CM334 allele increased the level of resistance. The three QTLs located on P3, P11 and LG25 influenced two resistance components while the others affected a single resistance component. The QTL *Pp_25.1* displayed the major effect; it explained 24.14% of Pp_REC and 9.73% of Pp_L21. The other QTL effects ranged from 4.84 to 13.09%. Two QTLs, separated from each other by 10 cM, were detected on chromosome P9 and influenced distinct resistance components.

The LR analyses identified three additional unlinked markers explaining from 8.11 to 14.52% of the Pp_STA and Pp_L21 traits. Remarkably, the resistant allele for these three QTLs was inherited from the susceptible parent YW. Two epistatic interactions were observed for the Pp_L21 trait. Markers e43/m54_256y of P3 and e33/m56_263y on P8 [visible on the Barchi et al.'s map (2007)] explained 12% of the variation ($P = 7.78$ 10–6); markers p14/m41_223c on P5 and p17/m39_244y (unlinked) explained 11% ($P = 8.3$ 10–6).

With multiple regressions, the 1–7 additive QTLs determining the four individual resistance components to *P. parasitica* accounted for 6.36–62.26% of the total phenotypic variance (Table 2). When adding markers of both epistatic interactions, the global R^2 increased to 68.20% for Pp_L21. Taking into account heritability values, QTLs explained from 14 to 78% of the genetic variation of the *P. parasitica* resistance components.

Discussion

In order to gain better insight into the genetic architecture determining the partial resistance of pepper to two *Phytophthora* species, a detailed QTL analysis of the resistance to *P. capsici* and *P. parasitica* was performed, using the high-resolution linkage map of Barchi et al. (2007). This study focus on the *Phytophthora* resistance carried by ''Criollo de Morelos 334'' (CM334), the most promising root-rot resistance source currently known in *C. annuum*.

CM334 is partially resistant to both *Phytophthora* species

In our experimental conditions, we succeeded to artificially reproduce compatible interaction between pepper and both *P. capsici* (*Pc*197) and *P. parasitica* (*Pp*329) isolates. For

Table 2 QTLs for *P. capsici* and *P. parasitica* resistance detected in F5YC progeny (detected by CIM) and broad sense heritability of the traits

Trait	QTL	Chr[a]	Marker[b]	Nb ind[c]	Position[d]	LOD value[e]	R^2(%)[f]	Resistant allele[g]	Additive effect[h]	GR^2(%)[i]	(h^2_{BS})[j]
Pc_L21	*Pc_5.1*	P5	Mfvt_M2	288	44,12	30.02	44.60	C	0.62		
	Pc_1.2	P1	EPMS_650	130	98,87	4.73	9.21	C	0.28		
	Pc_4a.2	P4a	Tntc01c	100	100,14	2.99	4.02	C	0.19		
	Pc_6.1	P6	e34/m53_091y	290	2,83	2.79	2.97	C	0.16		
	Pc_U1	U	e41/m54_353y	260	ND	4.85	11.10	C	0.31		
	Pc_U2	U	e36/m47_172c	274	ND	4.72	10.72	C	0.30		
	Pc_U3	U	e31/m58_311c	289	ND	3.51	7.78	C	0.26		
										55.23	0.96
Pc_REC	*Pc_5.1*	P5	Mfvt_M2	288	44,12	33.85	53.27	C	0.58		
	Pc_1.1	P1	e41/m61_199y	286	66,48	2.76	2.83	C	0.13		
	Pc_U1	U	e41/m54_353y	260	ND	5.11	11.10	C	0.28		
	Pc_U3	U	e31/m58_311c	289	ND	5.10	11.59	C	0.19		
										50.62	0.87
Pc_S10	*Pc_5.1*	P5	Mfvt_M2	288	44,12	21.15	35.14	C	0.49		
	Pc_U1	U	e41/m54_353y	260	ND	3.70	8.55	C	0.24		
	Pc_U3	U	e31/m58_311c	289	ND	3.63	8.11	C	0.23		
										36.71	0.88
Pc_STA	*Pc_5.1*	P5	Mfvt_M2	288	44,12	17.89	29.68	C	0.48		
	Pc_6.1	P6	e34/m53_091y	290	2,83	4.79	6.79	C	0.23		
	Pc_1.2	P1	EPMS_650	130	98,87	3.11	8.11	C	0.25		
	Pc_4a.1	P4a	e36/m52_392y	253	57,98	3.04	4.74	C	0.19		
	Pc_4a.2	P4a	Tntc01c	100	100,14	2.82	4.64	C	0.19		
	Pc_U4	U	p15/m40_314c	278	ND	5.46	12.10	Y	–0.30		
	Pc_U2	U	e36/m47_172c	274	ND	3.48	7.87	C	0.25		
	Pc_U1	U	e41/m54_353y	260	ND	3.40	8.05	C	0.25		
										56.49	0.93
Pc_RRI	*Pc_5.1*	P5	Mfvt_M2	288	44,12	14.33	20.30	C	0.46		
	Pc_5.2	P5	e41/m61_348c	261	88,25	11.53	23.63	C	0.43		
	Pc_11a.1	P11a	p25/m45_274y	294	0,01	4.16	8.14	C	0.25		
	Pc_U1	U	e41/m54_353y	260	ND	8.45	18.18	C	0.38		
	Pc_U2	U	e36/m47_172c	274	ND	5.86	13.74	C	0.32		
	Pc_U4	U	p15/m40_314c	278	ND	4.58	10.27	Y	–0.29		
										50.19	0.69
Pp_L21	*Pp_9.1*	P9	p15/m40_321c	282	103,10	6.83	12.38	C	0.28		
	Pp_25.1	LG25	e44/m51_646y	130	22,07	5.24	9.73	C	0.24		
	Pp_45.1	LG45	EPMS_402	248	17,54	4.35	9.14	C	0.25		
	Pp_10a.1	P10a	e34/m53_145y	289	13,22	3.43	7.73	C	0.22		
	Pp_10a.2	P10a	e36/m47_145y	277	36,92	3.47	8.37	C	0.23		
	Pp_3.1	P3	e40/m49_198y	268	164,49	3.20	5.81	C	0.20		
	Pp_29.1	LG29	p25/m45_434c	294	11,10	2.82	4.84	C	0.18		
	Pp_U1	U	p25/m42_227y	110	ND	3.77	14.52	Y	–0.34		
										62.26	0.87
Pp_REC	*Pp_25.1*	LG25	e44/m51_646y	130	21,26	14.49	24.14	C	0.40		
	Pp_9.2	P9	e44/m61_187y	288	96,81	6.08	9.88	C	0.25		
	Pp_3.1	P3	e40/m49_198y	268	164,49	3.41	5.21	C	0.18		
	Pp_37.1	LG37	p15/m40_319c	276	12,37	2.99	5.23	C	0.18		
										49.50	0.87

Table 2 continued

Trait	QTL	Chr[a]	Marker[b]	Nb ind[c]	Position[d]	LOD value[e]	R^2(%)[f]	Resistant allele[g]	Additive effect[h]	GR^2(%)[i]	(h^2_{BS})[j]
Pp_S10	*Pp_11a.1*	P11a	e36/m52_335c	271	52,86	3.01	6.36	C	0.13		
										6.36	0.47
Pp_STA	*Pp_11a.1*	P11a	e36/m52_335c	271	52,86	6.33	13.09	C	0.26		
	Pp_27.1	LG27	e41/m61_169c	275	14,75	4.08	8.67	C	0.21		
	Pp_U2	U	p14/m33_262y	275	ND	4.09	9.87	Y	–0.25		
	Pp_U3	U	e36/m52_198c	292	ND	3.35	8.11	Y	–0.24		
										18.49	0.80

ND not determined

[a] Chr, Chromosome number (*P*), unassigned linkage group (LG), or U for unlinked markers

[b] Marker indicates the nearest upper flanking marker to QTL

[c] Nb ind indicates the number of RIL genotyped for the identified QTL-linked marker

[d] Position of the marker in cM on the chromosome

[e] LOD value, the value of the statistic test for the QTL detection

[f] R^2 is the proportion of variance explained by a QTL at the associated marker

[g] Resistant allele is the parental allele which contributed to the resistance. *C* = CM334 *Y* = YW

[h] Additive effect, value of additive effect of the QTL expressed in the unit of the standardized trait

[i] GR^2 is the global effects for a resistance component explained by all the markers linked to additive QTLs and calculated with a multiple stepwise regression

[j] h^2_{BS} is the broad sense heritability of the trait

both *Phytophthora* species, the pathogen penetrated the stem during the first several days of the interaction, and grew toward the bottom of the stem. Within several days, the plant cells involved in the interaction died, provoking a necrosis. The pathogens preceded the necrosis. The speed of the necrosis progression is considered to reliably represent the speed of pathogen invasion. For both species, the necrosis progression went faster in YW than in CM334 (where the necrosis progression was rapidly stopped). CM334 exhibited thus an inducible partial resistance to both *P. capsici* and *P. parasitica*. The necrosis lengths induced by the isolate *P*c197 were longer than those induced by *Pp*329 on all genotypes indicating a weaker ability of *P. parasitica* to colonize pepper than *P. capsici*. This difference of infection between *P. capsici* and *P. parasitica* was already reported (Allagui et al. 2001).

Genetic controls of both resistances differ since the resistance to *P. parasitica* is rather dominant and the resistance to *P. capsici* incompletely dominant. Both observations are in accordance with previously published data (Allagui et al. 2001; Thabuis et al. 2003). Correlations between resistance components to *P. capsici* and *P. parasitica* calculated on 200 RILs in our experiment (–0.01 to 0.34) were much lower than those calculated by Allagui et al. (2001) on 30 doubled haploid lines (0.47–0.52). However, comparison is not straightforward as Allagui's group did not use either the same plant materials or the same isolates of *P. parasitica*. Nevertheless, in opposition to the first presumptions, our observations suggest a rather different genetic control according to the considered *Phytophthora* species.

In the same mapping progeny, we identified 14 QTLs explaining up to 73% of the genetic variation for *P. capsici* resistance and 18 QTLs explaining up to 78% for the *P. parasitica* resistance. Both resistances are controlled by a major effect QTL in association with a few minor effect QTLs and QTLs in epistatic interaction.

Consistency of the resistance QTLs to *P. capsici* in pepper

Among the 12 additive QTLs for *P. capsici* resistance, 7 of the 8 QTLs located on the F5YC map were anchored to previously identified resistance QTLs to *P. capsici* (Fig. 4). The four QTLs associated to unlinked AFLP markers could not be anchored.

Thanks to the high-resolution pepper map, we identified two separate QTLs (*Pc_5.1* and *Pc_5.2*) on the chromosome P5 corroborating the presumptions of Thabuis et al. (2003) in two other cross populations. Sugita et al. (2006) detected a single major effect QTL (*Phyt-1*) covering the entire P5 chromosome in another cross population. Ogundiwin et al. (2005) also identified large effect QTLs (*Phyto-P* and *Phyto-Q*) on small linkage groups assigned to the P5 chromosome in two mapping progenies. The compilation of these studies demonstrated that this major effect

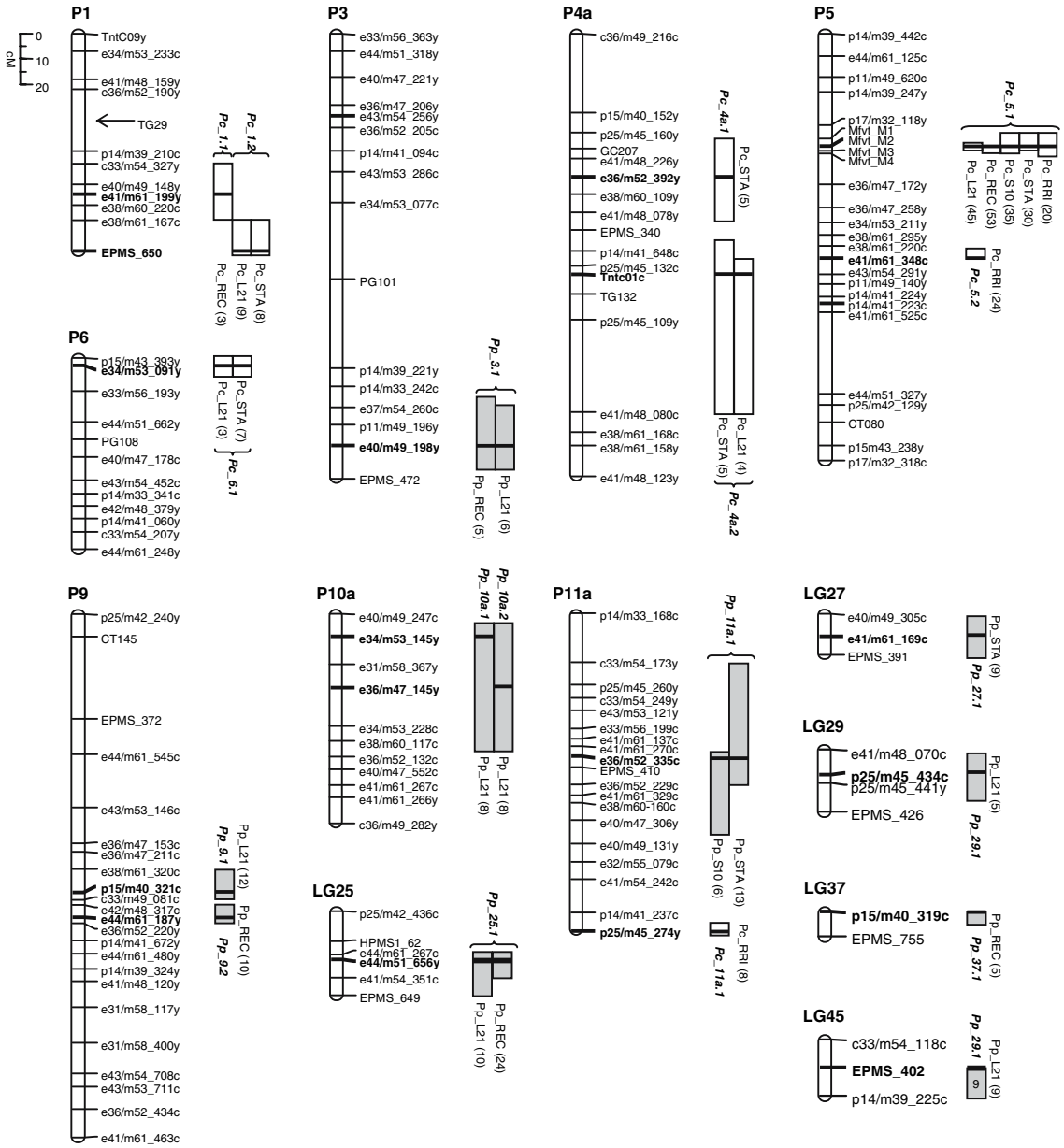

Fig. 3 Map location of *P. capsici* and *P. parasitica* resistance QTLs on the pepper F5YC framework map. Only chromosomes and linkage groups carrying additive QTLs are shown. Markers names refer to Barchi et al. (2007). Distances in cM (Haldane) are shown by the scale. The *boxes* to the right of linkage groups indicate the position of the QTLs (white for *P. capsici* and grey for *P. parasitica*). Lengths of *boxes* represent the confidence interval determined by LODmax-1. *Horizontal bold bars in boxes* indicate the closest marker of the LOD peak. The QTL names are indicated in *bold* and *italic* characters. The component associated with each box is indicated in normal character followed by the corresponding R^2 within brackets

region, conserved across at least four distinct resistance accessions, confers a broad spectrum resistance to *P. capsici*, since it is efficient towards at least six isolates originating from France, California, New Mexico, Taiwan, and Japan.

The *Pc_1.1* and *Pc_1.2* QTLs were colinear to one minor effect QTL detected in a breeding population by Thabuis et al. (2004), and putatively colinear to three QTLs located on the P1 chromosome by Ogundiwin et al. (2005). The *Pc_4a.2* QTL is flanked or covered by the markers e38/m61_168c, e38/m61_158y and TG132, which flanked the QTLs *Phyto4.1* detected by Thabuis et al. (2003, 2004). The *Pc_6.1* QTL was also identified in the F2YC progeny (Thabuis et al. 2003). Thanks to the *L* locus, we anchored the *Pc_11a.1* QTL located on the bottom of P11a chromosome to QTLs identified in two distinct progenies

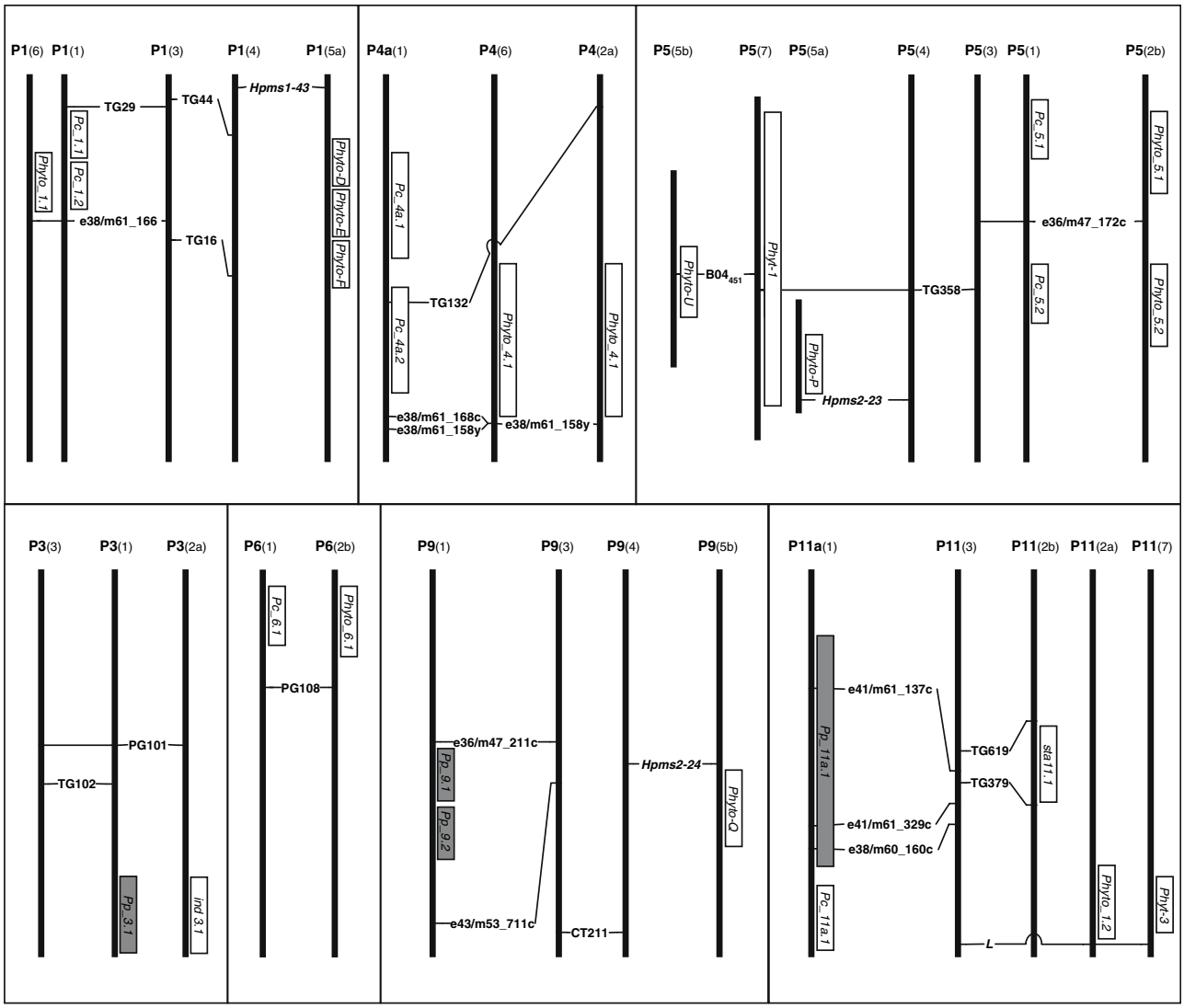

Fig. 4 Schematic comparative mapping in pepper for resistance QTLs to *P. capsici* and *P. parasitica*. Chromosomes are represented by *vertical bars* and named by their chromosome number (P1, P3, P4, P5, P6, P9 and P11). The pepper maps are referred by numbers at the top of chromosomes: (*1*) F5YC (this work), (*2a*) ''H3 × Vania'' and (*2b*) ''YW × CM334'' maps of Thabuis et al. (2003), (*3*) Paran et al. (2004), (*4*) Lee et al. (2004), (*5a*) ''PSP-11 × PI201234'' and (*5b*) ''JEP × CM334'' maps of Ogundiwin et al. (2005), (*6*) Thabuis et al. (2004), (*7*) Sugita et al. (2006). Anchor markers were shown by black thin full lines connecting maps. The marker order is respected according to their respective origin map. QTLs are represented by *boxes* (*white* for *P. capsici* and *grey* for *P. parasitica*) and named by the original QTL names

[*sta11.1* (Thabuis et al. 2003), *Phyt-3* (Sugita et al. 2006)]. This comparison lets us to conclude that most of the *P. capsici* resistance QTLs have a broad spectrum and are largely distributed among pepper genetic resources.

Colocations between minor effect resistance QTLs to *P. capsici* and *P. parasitica*

In the F5YC progeny, we did not identify any colocations between QTLs involved in the resistance to both species, although inoculation and environmental conditions were homogeneous between experiments. This result must be taken with care because of the LOD thresholds applied for QTL detection and the incomplete genome coverage (86.5%). Indeed, e44/m51_646y marker, linked to the major effect QTL on the *P. parasitica* resistance, *Pp_25.1*, had a LOD score just below the threshold (2.80) for the S10 resistance component to *P. capsici* (2.68, $R^2 = 3.76\%$). Moreover, this study concerns a single isolate per *Phytophthora* species considered. Thus some undetected QTLs might be effective against the both *Phytophthora* species. Notably, the major effect QTL *Pc_5.1* contributing to the

broad spectrum resistance to *P. capsici* was not significantly associated with *P. parasitica* resistance so far.

By extending the analysis to other published data on pepper, putative colocations between minor effect resistance QTLs to *P. capsici* and *P. parasitica* were identified (Fig. 4). They concerned the three genomic regions assigned to the P3, P9 and P11 chromosomes. Thanks to the anchor RFLP marker PG101, *Pp_3.1* QTL could be related to the QTLs *ind.3.1* affecting *P. capsici* resistance in the HV progeny (Thabuis et al. 2003). By using common markers on four pepper genetic maps, *Pp_9.1* and *Pp_9.2* QTLs were related to *Phyto-Q* QTL for resistance to *P. capsici* (Ogundiwin et al. 2005). *Pp_11a.1* QTL was related to the *Phyto11.1* QTL for *P. capsici* resistance (Thabuis et al. 2003, 2004). Whereas the resistant alleles to *P. capsici* and *P. parasitica* of the QTL on P3 originated from different genitors, the resistance alleles of the QTLs on P9 and P11 were always detected in the CM334 genitor. Whether they corresponded to a single gene or a cluster of resistance genes remains unknown.

Common resistance QTLs to different species of a same genus pathogen were previously reported. Risterucci et al. (2003) identified common resistance QTLs to two or three *Phytophthora* species in cocoa, and Voorrips et al. (2004) identified one common resistance QTL to two *Colletotrichum* species in pepper.

Are colocations between QTLs observed by chance?

Colinearities between resistance QTLs to *P. capsici* and *P. parasitica* in pepper might indicate the presence of common resistance factors to several *Phytophthora* species, the presence of allelic series for resistance to different *Phytophthora* species or the presence of resistance gene clusters. Conservation of function for *Phytophthora* resistance at these loci could infer that the ancestor locus must have undergone a positive selection pressure. On the contrary, if resistance loci are independent, they would have the same chance to colocate on common segments or to be found on independent segments. In our study, the eight *P. capsici* resistance QTLs that belonged to colinear segments colocated with *P. capsici* resistance QTLs previously identified in literature; for the 6 *P. parasitica* resistance QTLs that belonged to colinear segments, three QTLs colocated with *P. capsici* resistance QTLs. We tested whether colocations were observed by chance by applying the Lin et al. (1995) equation. The probability to obtain colocations by chance is $P = 0.0478$ $(=C_{20}^{5}/C_{35}^{5} \times C_{15}^{0})$ for *P. capsici* resistance QTLs between the different experiments in pepper, and $P = 0.3266$ $(=C_{20}^{3}/C_{35}^{4} \times C_{15}^{1})$ between *P. parasitica* resistance QTLs and *P. capsici* resistance QTLs. These probability values indicate that colocations between the *P. capsici* resistance QTLs from different crosses or studies did not occur by chance. The significant colocations between *P. capsici* resistance QTLs ($P < 0.05$) reinforces the validity of the seven QTLs concerned. On the contrary, the null hypothesis that colocations between the *P. capsici* and *P. parasitica* resistance QTLs occurred by chance is acceptable with the probability of ~33%. These calculations have to be taken with care. Indeed, the higher is the proportion of segments that were reported to carry QTLs in literature coupling with the high number of QTLs identified in our study, the higher is the number of colocations occurring by chance, and the more difficult is to conclude to independencies of genetic controls. Moreover, the more the genome is virtually divided in colinear segments, the more accurate is the comparison. If we could virtually divided the genome in 60 colinear segments as in Grube et al. (2000), the consistency of *P. capsici* resistance QTLs would be more significant, but also the independency of genetic factors for resistance to *P. capsici* and *P. parasitica* would be less likely. Our analysis was limited by the low number of anchor markers on the maps available in the literature. The more numerous are the orthologous markers, the more pertinent are the colocations. Additional anchor markers, tightly linked to QTLs are needed to confirm these results. Further investigations on comparative mapping by adding anchor markers and by using a meta-analysis (Goffinet and Gerber 2000) will be performed to identify the actually distinct and common QTLs detected in different experiments.

Conclusion

Two-third of the resistance additive QTLs to *P. capsici* identified in this study belonged to genomic regions colinear to previously reported resistance QTLs to *P. capsici* in pepper and one-fifth of resistance additive QTLs to *P. parasitica* colocated with previously reported resistance QTLs to *P. capsici*. The present comparative study suggests the independence between genetic controls of the resistance to *P. capsici* and *P. parasitica* in pepper. However, these assumptions still need to be consolidated by the addition of anchor markers in the compared maps.

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