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Comparative mapping of *Phytophthora* resistance loci in pepper germplasm: evidence for conserved resistance loci across Solanaceae and for a large genetic diversity

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Abstract *Phytophthora capsici* Leonian, known as the causal agent of the stem, collar and root rot, is one of the most serious problems limiting the pepper crop in many areas in the world. Genetic resistance to the parasite displays complex inheritance. Quantitative trait locus (QTL) analysis was performed in three intraspecific pepper populations, each involving an unrelated resistant accession. Resistance was evaluated by artificial inoculations of roots and stems, allowing the measurement of four components involved in different steps of the plant-pathogen interaction. The three genetic maps were aligned using common markers, which enabled the detection of QTLs involved in each resistance component and the comparison of resistance factors existing among the three resistant accessions. The major resistance factor was found to be common to the three populations. Another resistance factor was found conserved between two populations, the others being specific to a single cross. This comparison across intraspecific germplasm revealed a large variability for quantitative resistance loci to *P. capsici*. It also provided insights both into the allelic relationships between QTLs across pepper germplasm and for the comparative mapping of resistance factors across the *Solanaceae*.

Keywords *Capsicum annuum* L. · Disease resistance · *Phytophthora capsici* Leonian · QTL · Epistasis

Introduction

Despite the growing interest in quantitative resistance to diseases in plant breeding, one important question remaining is the variability and the organisation of resistance factors involved in polygenic resistance (Gebhardt and Valkonen 2001). Insights in this domain would contribute to a better understanding of the diversity of plant natural mechanisms of resistance. At present more information is available on major resistance genes (R genes), whose functions are generally related to pathogen recognition or to signal transduction for defence response (Hammond-Kosack and Jones 1997). R genes have been shown to map in 'clusters' (Witsenboer et al. 1995, de Jong et al. 1997). Events such as unequal crossing over, genic conversion and 'birth and death' processes would help in duplicating these regions and creating new resistance specificity for responding to fast pathogen adaptation (Michelmore and Meyers 1998; Ronald 1998).

With respect to quantitative resistance, recent results have shown that a large variability of distinct gene families and functions might be involved. The co-localisation of quantitative trait loci (QTLs) with R genes has provided a solid basis for making analogies between QTLs and R genes (Caranta et al. 1997a; Geffroy et al. 2000). This was strengthened by reports of the co-localisation of resistance QTLs and R gene analogs (RGAs) in several crops (Pflieger et al. 1999). Some resistance QTLs are supposed to be allelic to R genes and to have the same function (Robertson 1989). In other cases, many resistance QTLs co-localised neither with R genes nor with RGAs (Qi et al. 1998) but with genes involved in defence mechanisms (Leonards-Schippers et al. 1994; Faris et al. 1999; Pflieger et al. 2001).

We report here the genetic architecture of partial resistance to *Phytophthora capsici,* a soil-borne Oomycete, in three unrelated resistant pepper accessions (*Capsicum annuum* L.) originating from different centres of diversification. This plant-pathogen interaction is a suitable model for characterising the diversity of quantitative resistance. Firstly, screening *Capsicum* germplasm revealed only partial resistance that displayed a complex inheritance. Secondly, a wide diversity for resistance is likely to be found because of the important number of pepper diversification centres spread across Central and South America, Africa, and Asia. Thirdly, a narrow co-

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evolution has surely occurred between pepper and *P. capsici* because the pathogen is present in all areas where pepper is cultivated. Fourthly, this complex resistance can be dissected into four components revealing different aspects of the plant-pathogen interaction evaluated through two distinct phenotypic tests (Pochard et al. 1976). The stem inoculation test enables the measurement of three resistance components: receptivity, inducibility and stability (Pochard and Daubèze 1980). The root rot index was evaluated after root inoculation (Palloix et al. 1988). The three resistant accessions analysed displayed a wide phenotypic variability for the different resistance components. Moreover, accession CM334 displayed the particular characteristic of maintaining the induced resistance under conditions of high temperature (Pochard et al. 1983).

In the study reported here, the comparative QTL analysis was performed with three resistant accessions using a linkage map close to saturation due to the alignment of three intraspecific maps (Lefebvre et al. 2002). The first objective of our study was to compare the genetic basis of the resistance to *P. capsici* in intraspecific germplasm and to study the organisation of variability for a complex resistance. The second objective was to compare the genetic location of resistance factors to various diseases already mapped in pepper and, more largely, in Solanaceaeous crops.

Materials and methods

Plant and pathogen materials

The three partially resistant lines used in this study were Vania (Van), an inbred bell pepper line in which the resistance from PI201234 (accession from Central America) was introgressed, Perennial (Per), a small-fruited and pungent line from India, and Criollo de Morelos 334 (CM334), a small-fruited and pungent line from Mexico. Susceptible parents used were H3, an inbred hot pepper line from East Africa, and 'Yolo Wonder' (YW), an American bell pepper line. Three intraspecific *C. annuum* L. populations were used for QTL detection: HV (H3×Van) and PY (Per×YW) were two F_1-derived doubled-haploid (DH) populations composed of 101 and 114 lines, respectively. In the YC progeny (YW×CM334), 151 individual F_2 plants were used for DNA analysis and the corresponding F_3 families were used for resistance evaluation. Two *P. capsici* strains were used: a moderately aggressive one (S101) and a very aggressive one (S197) (Palloix et al. 1988). These were maintained as described by Clerjeau et al. (1976).

Phenotypic assays

Two independent artificial tests were performed on the three progenies in growth chambers. The root inoculation test was performed as described by Palloix et al. (1988). It enabled us to compute the root rot index (RRI), a semi-quantitative resistance criterion ranging from 0 (resistant) to 5 (susceptible). The stem inoculation test, performed as described by Pochard and Daubèze (1980), allowed us to calculate three resistance components. Receptivity (REC, mm day^{-1}) measured the pathogen spread in early infection process (3rd day post-inoculation, DPI). Inducibility (IND, mm day^{-2}) measured the deceleration of the necrosis length between the 3rd and the 10th DPI. Stability (STA, mm day^{-1}) measured the average speed of necrosis length between the 14th and the 21st DPI. Resistance assays were conducted at 22 °C, except for YC, which was tested at two temperatures – 22 °C and 32 °C, since the induced resistance of CM334 to *P. capsici* is maintained under high temperature. In each test, the five parents of the three populations studied were used as controls. The experimental design regarding phenotypic assays for the three populations is described in Table 1.

Genotyping

Procedures for DNA isolation and RFLP, RAPD, AFLP marker assays have been previously described by Lefebvre et al. (1993, 1995, 2001, 2002).

Map construction and alignment

The three intraspecific maps were constructed independently and then aligned using common markers (RFLP, RAPD and AFLP markers). The HV framework map is composed of 135 markers with an average distance between markers of 13 cM (±9.0) for a total length of 1,513 cM. The PY and YC framework maps are composed of 154 and 64 markers, respectively, for a total length of 1,668 cM and 685 cM respectively and an average distance of 12.5 cM (±9.1) and 13.7 cM (±8.6), respectively (Lefebvre et al. 2002).

Data analysis

Statistical analyses of raw phenotypic data were performed using SAS (SAS Institute 1989). Raw data were analysed even though they were not normally distributed since no transformation improved Normality in a significant manner. Normality was checked using PROC UNIVARIATE and the Wilk and Shapiro's test. The phenotypic correlation coefficients were computed with PROC CORR. Environmental and genetic variances were calculated using

Table 1 Experimental design[a] for phenotypic assays of the three mapping populations

Population[b]	Population size	Strain	Temperature	Root test Test × Block × Plant	Stem test Test × Block × Plant
HV	101	S101	22 °C	2 × 2 × 20	1 × 2 × 8
PY	114	S101	22 °C	2 × 2 × 20	1 × 2 × 8
YC-22 °C	151	S197	22 °C	2 × 2 × 20	2 × 2 × 5
YC-32 °C	100	S197	32 °C	–	1 × 1 × 6

[a] Test is the number of independent tests performed; Block is the number of blocks performed in each test; Plant is the number of plants tested in each block

[b] HV, H3 × Vania; PY, Perennial × Yolo Wonder; YC, Yolo Wonder × Criollo de Morelos 334

PROC GLM with the random factor option and broad-sense heritability was calculated using PROC VARCOMP.

QTL detection

QTLs were detected by interval mapping (IM, Lander and Botstein 1989) and by composite interval mapping (CIM, Zeng 1994) methodologies with QTL CARTOGRAPHER software (Basten et al. 1997). When two QTLs were detected by CIM within less than 20 cM, only the most significant was retained. The percentage of phenotypic variation explained by all the QTLs for a given resistance component was obtained by multiple stepwise regression with the flanking markers of the QTLs (PROC REG, SAS Institute 1989). The *P. capsici* resistance of the PY population was previously dissected thanks to ANOVA (Lefebvre and Palloix 1996). The same set of data was used for the present QTL analysis.

Thresholds

For each resistance component in each progeny, empirical thresholds were computed by permutation tests (1,000 permutations) for IM and CIM with QTL CARTOGRAPHER. For a given population and a given method, no difference was observed for the threshold according to the trait. For IM, the LOD thresholds were 2.30 for HV, 2.95 for PY and 2.86 for YC, for a type-I-error = 0.10. For CIM, the thresholds were 2.70 for HV, 3.04 for PY and 3.20 for YC, for a type-I-error = 0.10. To avoid losing potentially valuable genetic information, QTLs with significance ranging from "the empirical threshold – 1" to the empirical threshold were reported as putative QTLs.

Digenic interactions

For the HV and PY populations, digenic interactions were evaluated using a two-way ANOVA with a fixed interaction component. Tests for interactions were performed with markers used in framework maps. A drastic P value ($P < 2.10^{-4}$) was retained because of the large number of tests performed. This analysis was not performed in the case of YC population because of the larger number of gametic classes in a F_2 population and the limited progeny size.

Dominance ratio

For the YC population, the dominance ratio (DR) was estimated from the results of CIM analysis. Because of phenotypic evaluation on F_3 families and genetic mapping on F_2 plants, DR equals 2|d/a| (Stuber et al. 1987; Pernet et al. 1999b) where a and d are the additive and dominance estimates, respectively.

Results

Phenotypic variation

For each resistance component, the means of the three resistant parents (Table 2) were significantly different (at $\alpha = 5\%$) from those of the susceptible parents. The means of the susceptible parents (H3 and YW) were not significantly different (at $\alpha = 5\%$) with respect to RRI, REC and STA, but H3 displayed a more resistant phenotype for the IND value than YW. Phenotypic variability was observed for the various resistance components among the three resistant parental lines. Indeed, in each test performed, the three resistant accessions were used as controls (data not shown): CM334 displayed the highest level of resistance for the four components and for both strains and both temperature conditions. Perennial and Vania showed the same level of resistance for RRI, REC and IND (at $\alpha = 5\%$). For STA, Perennial displayed a more resistant phenotype than Vania.

The phenotypic distributions for each resistance component in the three mapping populations indicated complex inheritance (data not shown). RRI showed a continuous distribution with a bimodal trend for the three populations, indicating that a major genetic factor as well as a few minor ones controlled this resistance component. For the REC trait, HV and YC (at 22 °C and at 32 °C) showed a continuous Normal distribution. PY displayed a continuous bimodal-shaped distribution; this led us to believe that the genetic determinism for this resistance component in Perennial was different from that of the two other accessions. For IND, all three populations displayed a Normal distribution, indicating a complex genetic inheritance for this resistance component. For STA, HV and YC-32 °C displayed an asymmetric distribution towards the resistant phenotype. Such distributions are often due to the existence of epistasis. PY displayed a continuous and bimodal distribution, whereas a Normal distribution was observed for YC-22 °C. For all populations and resistance components, transgressions towards resistance, susceptibility or both were commonly observed (except for RRI in YC), suggesting the presence of resistance factors in both parents. The comparison of YC-22 °C and YC-32 °C showed an effect of the temperature on the three stem resistance components. The REC mean of the progeny was higher at 32 °C than at 22 °C, but IND and STA indicated a more resistant phenotype at high temperature (significant at $\alpha = 10\%$).

The position of the F_1 hybrid compared to the parents indicated that IND behaved as a dominant trait, whereas RRI, REC and STA displayed dominance in HV and additive gene action in PY and YC (significant at $\alpha = 10\%$, Table 2).

The broad-sense heritability ranged from 0.678 to 0.976 and showed the same trend across the three populations for the resistance components (Table 2). Heritability was intermediate for IND (from 0.678 to 0.868) and higher for other resistance components. This indicated an important genetic variability between plants evaluated compared to a limited environmental variability in controlled inoculation conditions. However, the test at 32 °C for YC increased the environmental variance, leading to the decrease of the heritability estimation for the stem resistance components.

Correlation among resistance components

For all of the mapping populations, RRI, REC and STA were significantly correlated with each other (0.396–0.768), although correlation coefficients were stronger in

Table 2 Estimates of means[a], variance components[b], broad-sense heritabilities[c] and Normality test values[e] for the four resistance components in the three mapping populations

Population[d]	Root rot index	Receptivity	Inducibility	Stability
HV (S101)				
HV mean	2.387 (1.156)	6.593 (2.052)	–0.546 (0.258)	2.776 (2.853)
Van mean	1.583 (0.824)	4.625 (0.602)	–0.526 (0.189)	0.646 (0.390)
H3 mean	3.325 (0.789)	9.633 (0.823)	–0.019 (0.362)	7.617 (1.983)
F_1 mean	1.500 (0.579)	4.933 (1.225)	–0.500 (0.225)	0.629 (0.239)
σ^2_g	1.155	3.774	0.569	7.909
σ^2_e	0.538	3.171	1.812	1.853
h^2	0.893	0.920	0.753	0.976
W (Pr<W)[e]	0.926 (0.0001)	0.951 (0.0042)	0.981 (0.6076)	0.735 (0.0001)
PY (S101)				
PY mean	3.098 (1.141)	7.184 (2.211)	–0.403 (0.337)	5.242 (3.667)
Per mean	1.756 (0.571)	4.791 (1.564)	–0.688 (0.227)	0.191 (0.216)
YW mean	4.440 (0.555)	8.584 (0.526)	0.325 (0.201)	7.290 (1.116)
F_1 mean	3.625 (0.781)	6.376 (1.030)	–0.749 (0.185)	2.819 (1.599)
σ^2_g	1.266	4.727	0.098	12.983
σ^2_e	0.592	1.292	0.12	3.657
h^2	0.944	0.967	0.868	0.969
W (Pr<W)	0.925 (0.0001)	0.918 (0.0001)	0.956 (0.0139)	0.881 (0.0001)
YC-22 °C (S197)				
YC mean	2.571 (1.377)	6.134 (1.588)	–0.551 (0.175)	2.830 (1.185)
CM334 mean	0.821 (0.448)	4.950 (2.001)	–0.676 (0.286)	0.660 (1.000)
YW mean	5.000 (0)	8.020 (1.239)	–0.124 (0.188)	5.236 (2.123)
F_1 mean	3.725(0.954)	5.412 (1.773)	–0.502 (0.358)	3.549 (2.104)
σ^2_g	1.739	2.157	0.023	1.211
σ^2_e	0.425	2.011	0.074	1.483
h^2	0.944	0.917	0.765	0.894
W (Pr<W)	0.895 (0.0001)	0.972 (0.0882)	0.975 (0.1770)	0.972 (0.1000)
YC-32 °C (S197)				
YC mean	–	9.895 (2.538)	–0.821 (0.311)	2.157 (1.605)
CM334 mean	–	6.389 (2.123)	–0.926 (0.309)	0.181 (0.442)
YW mean	–	13.055 (0.800)	–0.139 (0.487)	–[f]
F_1 mean	–	8.611 (1.452)	–0.822 (0.347)	1.282 (1.536)
σ^2_g	–	5.632	0.066	2.912
σ^2_e	–	4.632	0.183	5.380
h^2	–	0.876	0.678	0.759
W (Pr<W)	–	0.965 (0.0620)	0.978 (0.4295)	0.935 (0.0001)

[a] Standard deviation is given in parenthesis
[b] σ^2_g, Genetic variance; σ^2_e, environmental variance
[c] h^2, Broad-sense heritability
[d] see footnote Table 1. Van, Vania; per, Perennial; YW, Yolo Wonder
[e] W is the Wilk and Shapiro's test value. In parenthesis is the probability associated to the Normality test
[f] At 32 °C, YW plants died between 14 DPI and 21 DPI

HV and PY than in YC (data not shown). IND showed a weaker or a non-significant correlation with the other resistance components in all crosses. Correlation coefficients were always positive between IND and STA, and negative between REC and IND, in HV and YC at 22 °C. Comparing the data obtained at 22 °C and 32 °C, we found that the correlation coefficients were significant with an intermediate value.

QTL analyses

For each population, results given by IM and CIM methods were in agreement with each other. The results are presented for CIM method and are summarised in Table 3. CIM enabled us to detect more QTLs than IM, as has been shown theoretically (Zeng 1994) and practically (Melchinger et al. 1998). Only in two cases (*rri.6.1* and *rec5.1* in YC), QTLs were found to be significant with IM and not with CIM. All the QTLs detected are represented in Fig. 1.

Comparison of the genomic regions involved in *P. capsici* resistance among the three crosses

A total of seven chromosomal regions displayed an additive effect on resistance to *P. capsici* in HV (Fig. 1). Two of these were found loosely linked on P3. Five chromosomal regions in PY and nine chromosomal regions in YC were found to display a significant additive effect on resistance. When focusing on P5, two linked loci were involved in resistance in PY and YC. In YC, two

Table 3 QTLs detected for the four resistance components in the three mapping populations

	Trait[a]	QTL	Mk[b]	Chr[c]	Psn[d]	Parent[e]	Composite interval mapping results			
							LOD [f]	Effect [g]	R^2 (%)[h]	2|d/a|[i]
HV	RRI	*rri.5.1*	CEX139v	P5	34.10	V	14.22	1.31	29.96	–
		rri.10.1	CMP087h	P10a	0.01	V	11.29	1.09	21.47	–
	ΣR^2								**58.83**	
	REC	*rec.3.1*	CRP083v	P3a	113.2	V	4.52	1.14	7.57	–
		rec.5.1	CEX139v	P5	34.10	V	25.95	3.07	53.97	–
		rec.10.1	CMP087h	P10a	0.05	V	8.12	1.41	11.72	–
		rec.11.1	*L*	P11	11.47	H	3.73	0.93	4.86	–
	ΣR^{2j}								**69.24**	
	IND	*ind.3.1*	TFE115h	P3b	178.80	H	3.41	0.18	11.13	–
		ind.5.1	TG123v	P5	26.30	H	4.99	0.23	17.71	–
		ind.12.1	GC148	P12	31.49	V	3.07	0.17	10.65	–
	ΣR^2								**42.67**	
	STA	*sta.5.1*	CEX139v	P5	34.1	V	6.86	2.39	16.93	–
		sta.7.1	CMO184h	P7	8.01	V	2.82	1.92	10.94	–
		sta.10.1	*GC082*	P10a	0.05	V	8.23	3.06	28.57	–
		sta.11.1	*L*	P11	11.47	H	2.47	1.50	6.64	–
	ΣR^2								**56.81**	
PY	RRI	*rri.2.1*	CAE351p	P2	127.95	P	2.63	0.49	4.43	–
		rri.5.1	CIA159y	P5	52.85	P	24.92	1.98		–
		rri.5.2	CSD_Hp	P5	88.58	P	3.41	0.60	5.50	–
		rri.10.1	CSO272y	P10	2.01	P	4.16	0.62	7.38	–
	ΣR^2								**64.66**	
	REC	*rec.5.1*	CIA159y	P5	48.85	P	22.96	3.86	73.38	–
	rec.5.2	R08_1.9	P5	87.23	P	6.40	2.31	26.31	–	
	ΣR^2								**58.41**	
	IND	*ind.5.1*	TG483	P5	32.03	P	2.28	0.21	9.54	–
	STA	*sta.2.1*	AF20_.2y	P2	121.75	P	2.10	1.75	5.52	–
		sta.5.1	TG586	P5	36.16	P	16.38	5.76	60.60	–
		sta.5.2	COA_Cp	P5	98.87	P	4.19	2.77	11.10	–
		sta.py2.1	Q04_0.2	PY2	0.01	P	2.18	1.70	5.20	–
	ΣR^2								**51.95**	
YC-22 °C	RRI	*rri.4.1*	CRP171y	P4	39.47	C	3.19	0.92	37.79	2.81
		rri.5.1	TG123y	P5	10.01	C	35.69	1.66	21.80	0.61
		rri.5.2	CMF183y	P5	38.43	C	15.29	0.90	42.43	2.52
		rri.6.1[k]	A07_.5c	P6	24.01	Y	2.82	0.80	1.00	5.36
		rri.11.1	PG263	P11a	2.53	C	2.53	0.34	1.47	1.48
		rri.12.1	CT138D	P12	6.01	C	4.16	0.49	13.33	2.69
	ΣR^2								**80.80**	
	REC	*rec.5.1*[k]	CIA159y	P5	14.91	C	4.42	1.30	67.05	0.71
		rec.6.1	A07_0.5c	P6	0.01	C	10.99	0.14	45.59	38.48
		rec.12.1	CMF237c	P12	117.78	C	2.63	0.25	1.10	12.95
		rec.yc1.1	CEX144y	YC1	14.01	C	2.82	0.44	2.72	10.37
	ΣR^2								**58.58**	
	IND	*ind.6.1*	A07_0.5c	P6	4.01	C	2.49	$3.6.10^{-3}$	15.43	$1.4.10^{-3}$
		ind.11.1	TG379	P11a	3.78	C	4.74	0.08	5.21	1.24
	ΣR^2								**22.22**	
	STA	*sta.6.1*	A07_0.5c	P6	0.01	C	6.16	0.11	25.01	37.68
		sta.6.2	CIA206c	P6	41.64	Y	4.26	0.96	6.63	2.89
		sta.12.1	CMF237c	P12	117.78	C	2.94	0.32	1.00	9.51
	ΣR^2								**68.5**	
YC-32 °C	REC	*rec32.5.1*	TG123y	P5	8.01	C	4.48	0.40	20.81	17.26
	STA	*sta32.4.1*	CRP171y	P4	23.47	C	3.26	0.80	14.07	0.08
		sta32.5.1	CIA159y	P5	12.91	C	6.67	1.43	69.88	0.80
		sta32.11.1	PG263	P11a	2.01	C	2.69	0.62	9.90	0.53
		sta32.12.1	CMF237c	P12	133.78	C	2.00	0.57	8.46	0.48
	ΣR^2								**68.50**	

[a] Trait, Resistance component used for QTL detection [b] Mk, Left flanking marker of the test position [c] Chr, Chromosome number as defined by Lefebvre et al. (2002) [d] Psn, Estimated position of the QTL using the CIM method along the chromosome [e] Parent, Parental allele which increased the resistance level. *H* H3, *V* Vania, *P* Perennial, *Y* YW, *C* CM334 [f] LOD, the value of the statistic test (values underlined indicate the putative QTLs as defined in Materials and methods) [g] Effect, Value of additive effect of the QTL (2a) with a being the additive estimate. It is expressed in the unit of the trait defined in the Materials and methods [h] R^2 (%), Percentage of variation explained by the QTL [i] 2|d/a|, The dominance ratio with a and d being the additive and dominance estimates, respectively. Its significance is: DR<0.2 (additive), 0.2<DR<0.8 (partially dominant), 0.8<DR<1.2 (dominant), DR>1.2 (overdominant) [j] ΣR^2 is the sum of the effects for a resistance component explained by markers linked to QTLs and calculated with a multiple stepwise regression. The estimated effect of the QTL on P5 thanks to CIM was probably over-estimated when compared to the results of multiple regression analysis [k] QTLs detected only thanks to the IM method

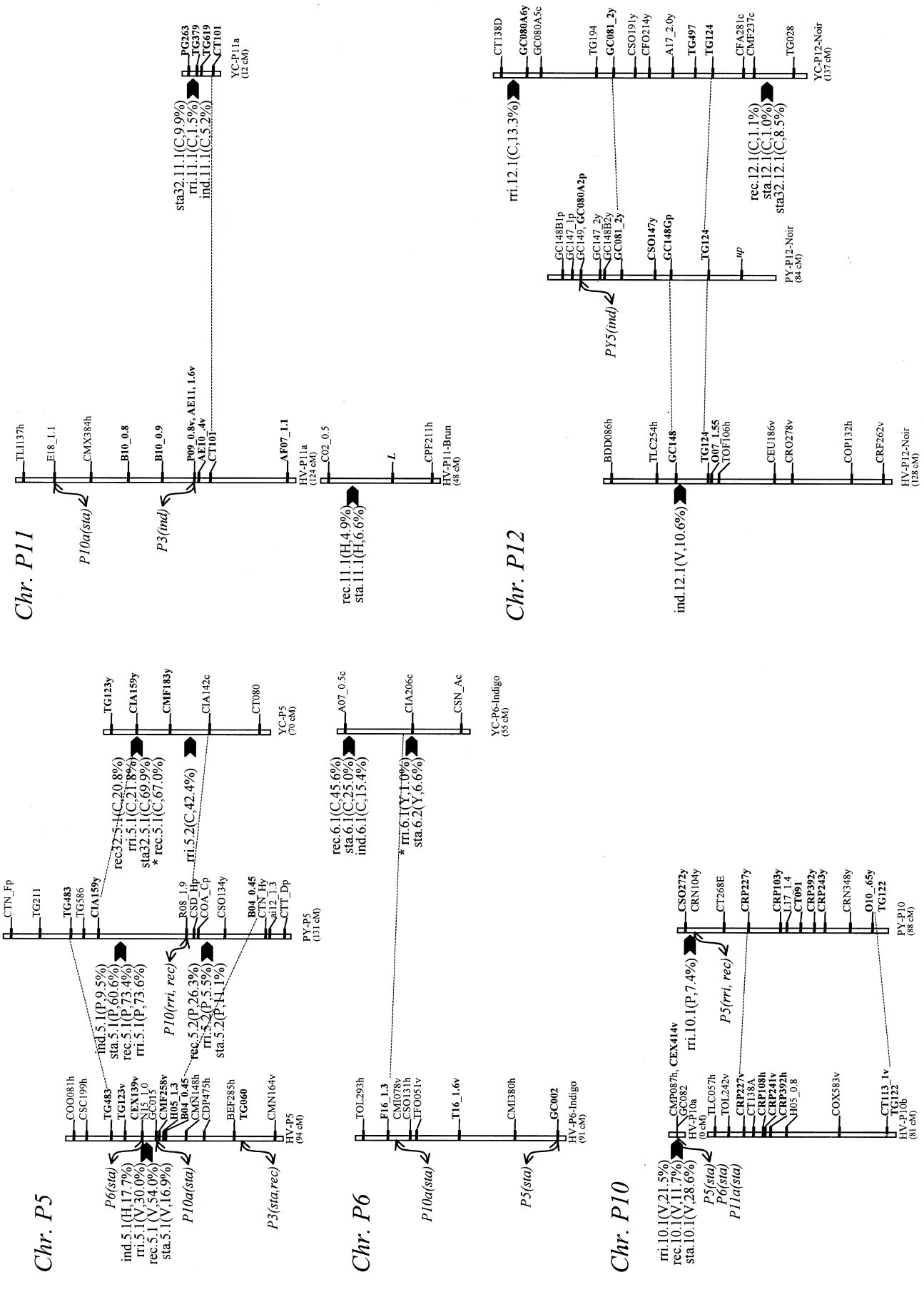

Fig. 1A, B Legend see page 1480

B **QTL carrier chromosomes specific to one cross**

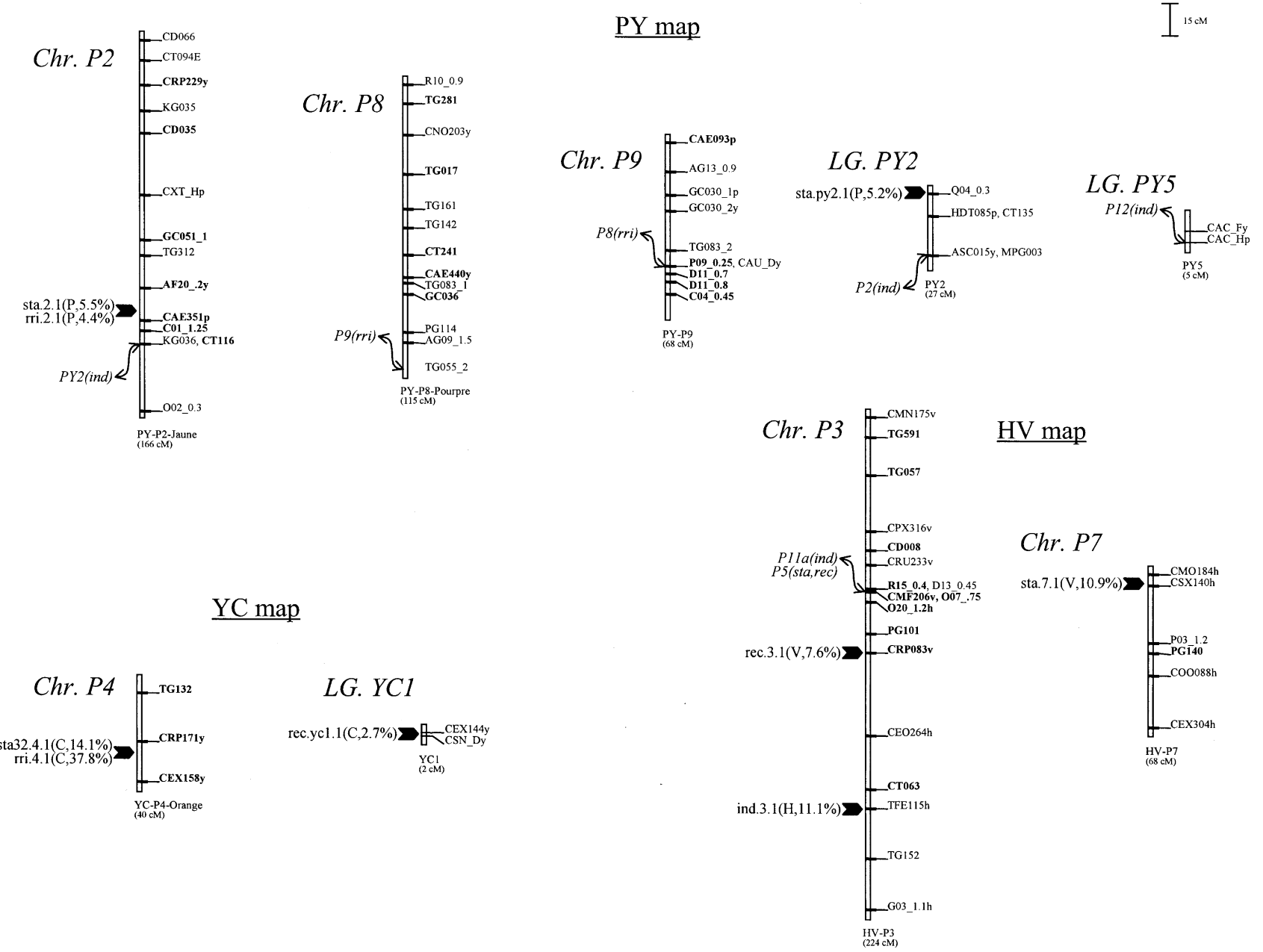

Table 4 Epistatic relationships detected in HV and PY populations

M1[a] HV	Chr/M1[b]	M2[a]	Chr/M2[b]	Trait[c]	>P[d]	>R^2 (%)[e]	Phenotypic mean of the four genotypic classes[f]			
							M1=V M2=V	M1= M2=H	M1=H M2=V	M1=H M2=H
GC015	P5	GC082	P10a	STA	$3.41.10^{-7}$	59.2	0.969	2.281	1.306	6.700
N15_1.0	P5	GC002	P6	STA	$9.7.10^{-5}$	40.9	1.682	2.609	1.883	7.102
TG060	P5	D13_0.45	P3	STA	$8.1.10^{-5}$	21.6	1.302	2.827	4.343	2.008
				REC	$1.3.10^{-5}$	29.8	4.936	6.839	7.655	6.120
CEX414v	P10a	E18_1.1	P11a	STA	$5.4.10^{-5}$	43.1	1.351	2.487	1.018	5.547
GC082	P10a	CMI078v	P6	STA	$2.4.10^{-5}$	52.5	1.155	1.273	2.284	6.068
AE11_1.6v	P11a	O07_.75	P3	IND	$1.9.10^{-5}$	25.6	–0.397	–0.643	–0.635	–0.357
PY							M1=Y M2=Y	M1=Y M2=P	M1=P M2=Y	M1=P M2=P
CT116	P2	MPG003	PY2	IND	$7.8.10^{-5}$	23.7	–0.510	–0.050	–0.370	–0.500
TG055_2	P8	CAU_Dy	P9	RRI	$9.8.10^{-6}$	42.7	3.700	1.800	2.670	3.900
CRN104y	P10	R08_1.9	P5	RRI	$1.3.10^{-4}$	29.2	3.378	2.916	3.776	1.958
				REC	$8.8.10^{-5}$	22.9	7.236	6.784	9.017	5.300
GC080	P12	CAC_Hp	PY5	IND	$5.4.10^{-5}$	19.5	–0.540	–0.300	–0.190	–0.570

[a] M1 and M2, Markers involved in the interaction
[b] Chr/M1 and Chr/M2 indicate on which pepper chromosomes the markers are mapped according to Lefebvre et al. (2002)
[c] Trait indicates the resistance component for which the interaction was found
[d] P is the P value of the interaction component in the two way ANOVA with an interaction component
[e] R^2 (%), Percentage of variation explained by the model (two-way ANOVA with an interaction component)
[f] Phenotypic means of the 4 genotypic classes were calculated. V, Vania allele; H, H3 allele; P, Perennial allele, Y, Yolo Wonder allele

resistance loci were found at both extremities of P12 and two were detected on P6.

One single chromosomal region on P5 was common to the three populations. Another chromosomal region was common to HV and PY on P10 (Fig. 1A). Other resistance factors appeared to be specific to the population studied (Fig. 1B). For most of the chromosomal regions, the resistance-increasing allele originated from the resistant parent for PY and YC (except *rri.6.1* and *sta.6.2*). In HV, resistant alleles originated from the susceptible parent (H3) in three cases (*ind.3.1, ind.5.1* and QTLs on P11).

QTLs involved in the different resistance components

RRI, REC and STA were the best components explained by markers (except for REC evaluated at 32 °C) as the global R^2 ranged from 51.95% to 80.80%. IND was the poorest component explained by markers (up to 42.67%). In the three populations studied; the number of QTLs for IND was the smallest (ranging from zero to three). With respect to the number of QTLs per resistance component, some appeared to be stable across the three populations, such as STA, where three to four QTLs were detected. Others were more variable, such as RRI where two QTLs were detected in HV and up to six in YC evaluated at 22 °C, and REC, where the number of QTLs ranged from one (YC at 32 °C) to four (YC at 22 °C and HV).

Regarding the chromosomal locations of the QTLs detected, clusters of QTLs involved in distinct resistance components were observed. Such regions have been reported as 'generalist' (Lefebvre and Palloix 1996). In HV, three 'generalist' chromosomal regions were located on P5, P10 and P11. The number of resistance components involved ranged from two on P11 to four on P5. In PY, three 'generalist' regions were detected: one was located on P2 (two resistance components) and two on P5 (four resistance components). In YC, five 'generalist' regions on P4, P5, P6, P11 and P12 were reported. QTLs for RRI, REC and STA were the most frequently clustered in 'generalist' regions. Regions involved in a single resistance component were mentioned as 'specialist'. Four 'specialist' regions were found in HV, located on P3, P7 and P12. In PY, two 'specialist' regions were reported on P10 and PY2. In YC, four 'specialist' regions were found on P5, P6, P12 and YC1. No resistance component displayed preferentially 'specialist' effects.

Regarding the effects of the chromosomal regions involved in resistance, the 'generalist' common region on P5 displayed the strongest effect mainly for HV and PY for all resistance components. For YC, a major effect was observed for RRI at 22 °C and STA at 32 °C. The large effect for REC at 22 °C was only detected by IM. Other 'generalist' regions displayed strong to intermediate effects – such as regions on P10 for HV, P4 and P6 for

◀ **Fig. 1A, B** Linkage map locations of QTLs involved in resistance components to *Phytophthora capsici* on the three pepper maps. **A** QTL carrier linkage groups common to several crosses, **B** QTL carrier linkage groups specific to a single cross. Only linkage groups with QTL effects associated with the *P. capsici* resistance components are shown from linkage maps described by Lefebvre et al. (2002). Markers in *bold* are common to different genetic maps. *Dashed lines* align common markers of the linkage groups derived from the three populations indicated below as *HV*, *PY* or *YC*, followed by the chromosome assignation (*P1–P12*). Not assigned linkage groups are indicated without the *P*. *Thick arrows* indicate additive QTLs. QTLs are named following Table 3, with, in parentheses the parent from which they originate (*H* H3, *V* Vania, *P* Perennial, *Y* Yolo Wonder, *C* CM334) and the R^2 value (%). *indicates QTL detected by IM only. *Curved arrows* represent interactions between markers (epistatic effects)

YC. Some 'generalist' QTLs displayed a minor effect – such as regions on P11 for HV (allele increasing the level of resistance from H3), P2 for PY, P11 and P12 for YC. Effects of 'specialist' regions were low to intermediate, in the three populations and ranged from 2.72% (*rec.yc1.1* in YC) to 13.33% (*rri.12.1* for YC).

For YC, most of the QTLs displayed overdominant expression at 22 °C except for *rri.5.1* and *ind.6.1*. At 32 °C, QTL expression ranged from additive to overdominant. No generalisation regarding the effects could be made for a given resistance component or a given resistance factor. The analysis of resistance to *P. capsici* at high temperature from CM334 showed that no temperature-specific QTL were detected at 32 °C. Indeed, the four regions found involved in resistance at a high temperature on P4, P5, P11 and P12 were already reported at 22 °C.

Epistatic relationships

Significant digenic interactions were reported for HV and PY (Table 4, Fig. 1). Three types of epistatic relationship were observed. First, interactions between two chromosomal regions displaying additive effects were reported. One common interaction between P5 and P10 was detected in HV and in PY. Secondly, interactions were found between additive QTLs and QTLs specifically involved in epistasis. For such interactions, five cases were observed only for HV. Thirdly, interactions were detected between QTLs that did not display any additive effects. This was mainly observed for PY. Lastly, it was observed that some epistatic QTLs detected in one population mapped in the vicinity of additive QTLs of another population (Fig. 1A).

Discussion

The genetic basis of complex resistance to *P. capsici* was studied among three unrelated resistance accessions reflecting a part of pepper intraspecific variability. This study showed rare but strong co-localisations of QTLs: one resistance factor located on P5 occurred among the three unrelated accessions, one more co-localisation on P10 occurred between Vania and Perennial. Other QTLs were cross-specific. This comparative study was limited by the under representation of the YC population due to an incomplete genetic map.

Genetic architecture of partial resistance

The occurrence of one major resistance factor and several intermediate ones was found across the three crosses. This architecture has been frequently reported for other quantitative resistances (Young 1996). The number of additive regions involved in resistance varied among the resistant accessions, ranging from a rather oligogenic determinism (five additive regions) for Perennial to a more complex one (nine additive regions) for CM334. Moreover, strong interaction effects were detected. Genomic regions controlling resistance could display additive effects, epistatic effects or both. Such interactions have previously been reported in quantitative resistance of pepper to*P. capsici* (Lefebvre and Palloix 1996) but also to potyviruses (Caranta et al. 1997a) and to cucumber mosaic virus (CMV) (Caranta et al. 1997b), and in rice against rice yellow mottle virus (Pressoir et al. 1998). Resistant alleles originated more frequently from the resistant parent, but they occasionally originated from the susceptible parent, as observed in HV and in YC at 22 °C.

The dissection of partial resistance into distinct phenotypic resistance components was firstly achieved to facilitate the exhaustive selection of resistance factors in breeding programs. It also allowed a more precise QTL detection and a more exhaustive survey of allelic differences. QTLs involved in resistance components acting on different organs (root or stem) or at different stages of infection (early event as REC or late as STA) were frequently mapped in the same genomic regions (described as 'generalist'), thereby confirming what Lefebvre and Palloix (1996) observed for PY. The frequent clustering of QTLs detected for RRI, REC and STA explained the significant and positive phenotypic correlation observed among the resistance components. Conversely, QTLs for IND were either rarely associated with other components or, when associated, resistant alleles were in repulsion (for example, *ind.5.1* and *rri.5.1* in HV). This explained the negative correlation found in HV or YC at 22 °C. Co-localisations of QTLs involved in distinct resistance components have also been observed for other partial resistances, such as the resistance of barley to *Puccinia hordei* (Qi et al. 1998) or of sunflower to *Sclerotinia sclerotinium* (Mestries et al. 1998). One could speculate whether 'generalist' regions resulted from the clustering of 'specialist' QTLs or from the pleiotropic effect of a single gene. In our studies, the distinction between pleiotropy and tight linkage was impossible to make due to insufficient population sizes. However, clusters of QTLs acting on the same trait have been observed for various quantitative traits such as resistance to a pathogen, crop yield (Stuber et al. 1987), and horticultural traits (Saliba-Colombani et al. 2001; Ben Chaim et al. 2001). These results point out the role of selection that creates 'clusters' of genes acting on the same trait that might confer a selection advantage to the genotype.

A great variability of resistance factors maintained at the intraspecific level

Only two chromosomal regions involved in *P. capsici* resistance were shown to be common to resistant accessions. One located on P5 common to the three accessions displayed the strongest and the most 'generalist' effect. Another was reported on P10 common to

Vania and Perennial. Comparing the genetic basis of resistance of maize to maize streak virus in unrelated resistant lines, Pernet et al. (1999a) observed that only the QTL having the major effect was conserved. The review of Welz and Geiger (2000) regarding the quantitative resistance of three unrelated accessions of maize to *Exserohilum turcicum* points out three putative common chromosomal regions involved in resistance: the QTL with the major effect as well as two others co-localised in the three populations. Similarly, the conserved regions on P5 and P10 might be ancestral loci involved in resistance to *P. capsici* that pre-existed before divergence. They were conserved at the intraspecific level due to their essential function for resistance expression. Despite a conserved location, allelic differences among resistant accessions could be hypothesised for the level of resistance and the effect on resistance components. Furthermore, differences in genetic structure could be hypothesised as one QTL was detected on P5 in Vania while two were observed in PY and YC. Those divergences probably occurred in secondary diversification after domestication as hypothesised Geffroy et al. (1999) who found evidence of the diversification of multiple specificities from a single ancestral resistance cluster at the *Co-7* locus in bean. Allelic relationships between QTLs located on P5 and on P10 from different pepper accessions will be further analysed through fine-mapping studies.

Another issue is that most of the QTLs detected in this study were specific to individual progenitors. Lübbersted et al. (1998a, b) reported the genetic basis of four maize lines from a limited germplasm (European Flint population) to *Ustilago maydis* and *Puccinia sorghi*. No clear co-localisation among resistance factors was observed across the four populations studied, suggesting a great variability of resistance factors even within a limited germplasm. One reason for the existence of variability for resistance factors might be the involvement of different mechanisms underlying resistance factors involved in complex resistance, as proposed by Lübbersted et al. (1998a). Further arguments are provided by several authors (Geffroy et al. 2000, Pflieger et al. 2001) showing map coincidences between resistance QTLs and genes coding for different functions: RGAs, PR proteins, enzymes from metabolic pathways. It was shown that defence genes such as class I chitinases evolved in a similar manner to genes involved in plant pathogen recognition process (Bishop et al. 2000) and, hence, might easily generate new loci involved in defence responses. Such a variability of functions might explain the variability of loci involved in complex resistance.

To date, we can not assign functions to those numerous QTLs. However, their different map positions and favourable QTL/genetic background interactions could explain the occurrence of transgressive genotypes with an enhanced level of resistance that have been obtained through recurrent selection (Palloix et al. 1990). The diversity of quantitative resistance factors has opened new ways for both constructing genotypes and surveying new functions involved in plant defence mechanisms.

Highly conserved resistance factors across *Solanaceae*

Many co-localisations of resistance factors to *P. capsici* with resistance factors to other pepper diseases were observed. Within the same cross, a generalist resistance factor on P11 in HV was found to be linked to the *L* locus conferring resistance to tobacco mosaic virus (Lefebvre et al. 1995). Other resistance factors controlling quantitative resistance to CMV and potyviruses have been mapped in this region (Caranta et al. 1997a, b). Lefebvre and Palloix (1996), using ANOVA, reported this co-localisation between *P. capsici* QTL and *L* in PY. With the CIM method, this QTL was not detected in PY. A QTL conferring resistance to tomato spotted wilt virus (TSWV) from H3 has also been reported in this region (Moury 1997). Both QTLs detected on P3 in HV mapped close to QTLs involved in resistance either to CMV or to potyviruses (Caranta et al. 1997a, b).

Numerous examples in the literature indicate the non-conservation of map locations of resistance factors to a same parasite across several genera; for instance, an R gene conferring resistance to TSWV mapped in a non-orthologous position in pepper (*Tsw*) and tomato (*Sw-5*) (Jahn et al. 2000). The lack of map coincidences observed for resistance factors across genera was putatively explained by a rapid evolution of loci involved in resistance trait. It particularly concerns resistance genes having a nucleotide-binding site (NBS) and/or leucine-rich repeat (LRR) feature (Leister et al. 1998; Pan et al. 2000). On the contrary, co-localisation of resistance factors to the same pathogen across different genera was rarely observed. In our study, we observed the co-localisation between the major conserved *P. capsici* resistance factor on pepper chromosome P5 and QTLs involved in partial resistance to *Phytophthora infestans* in potato (Leonards-Schippers et al. 1994; Sandbrik et al. 2000) using anchor RFLP markers (TG123, TG483, TG586). Moreover, *R2* mapped in the vicinity of this region on the potato chromosome IV; *R2* is an R gene conferring resistance to the same pathogen (Li et al. 1998). The functional conserved synteny, observed for the resistance factor in pepper and potato, may indicate that loci involved in resistance to *Phytophthora* might be distinct from genes belonging to the NBS/LRR class. Indeed, the conservation of map location could indicate a slower evolutionary process than the rapid one observed for NBS/LRR genes. Pflieger et al. (2001) reported, in pepper, a co-localisation of this conserved resistance factor with a defence gene (class III chitinase). This candidate gene mapped in the expected orthologous region in potato (V. Lefebvre, unpublished data). Moreover, after an extensive RGA mapping in pepper, none were mapped close to the resistance factor on P5 (Pflieger et al. 1999). Such data strengthened the hypothesis of a new function for the conserved resistance factor on P5.

Our study enabled us to describe three new inter-generic resistance clusters within the three Solanaceous crops (Grube et al. 2000b). The 'generalist' QTL mapped on P11 in YC is likely to be conserved with the R gene *Pto* in tomato (chromosome 5) using two common RFLP markers (TG379, TG619). Lefebvre and Palloix (1996) previously reported this co-localisation in PY using ANOVA. The common generalist resistance factor on P10 mapped in the vicinity of an R gene 'cluster'. Two potyvirus R genes, *Pvr4,* harboured by CM334 (Caranta et al. 1999), and *Pvr7,* originating from a *Capsicum chinense* accession (Grube et al. 2000a), were mapped in this region. *Tsw* conferring resistance to TSWV (Moury et al. 2000) and originating from *C. chinense* was also located in this region (Lefebvre et al. 2002). Moreover, this genomic region displayed a strong digenic interaction with the major resistance factor on P5. This interaction was the only one found to be conserved across both populations studied for epistasis. This resistance cluster is likely to be orthologous to a tomato region (chromosome 1) near CT268 where two R genes (*Cf-1* and *Cf-4*) conferring specific resistance to *Cladosporium fulvum* were located (Jones et al. 1992). In PY, *sta.py2.1* mapped close to an R gene conferring resistance to nematodes (*Me3* linked to CT135, Lefebvre et al. 2002). Thanks to a single common RFLP marker (CT135), a putative orthologous region in tomato and potato harboured R genes conferring resistance to nematodes, respectively *Mi3* and *Gpa2* (Djian-Caporalino et al. 2001).

All these co-localisations described suggest a conserved 'clusters' of resistance factors at an intraspecific, interspecific and inter-generic level. At an inter-generic level, clusters reported here might have evolved differently to engender various resistance factors to unrelated pathogens, and in few cases, to have evolved slowly within the *Solanaceae* family to maintain resistance factors to related pathogens such as *Phytophthora*.

Conclusion

This comparative study has provided a more complete knowledge of the pepper genome and its relationship within the *Solanaceae* family. Molecular markers were delivered for marker-assisted selection promoting genotype construction with conserved regions bearing major-effect QTLs and accession-specific QTLs with smaller effects (Thabuis et al. 2001). New genomic regions with relationships of synteny within *Solanaceae* were brought to evidence, although they generally displayed resistance factor clusters to different pathogens. And finally, the P5 genomic region involved in resistance in both pepper and potato might provide new insights into defence mechanisms against Oomycetes.

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References

Basten CJ, Weir BS, Zeng ZB (1997) QTL CARTOGRAPHER: a reference manual and tutorial for QTL mapping. Department of statistics, North Carolina State University, Raleigh, N.C.

Ben Chaim A, Paran I, Grube RC, Jahn M, van Wijk R, Peleman J (2001) QTL mapping of fruit-related traits in pepper. Theor Appl Genet 102:1016–1028

Bishop JG, Dean AM, Mitchell-olds T (2000) Rapid evolution in plant chitinases: molecular targets of selection in plant-pathogen coevolution. Proc Natl Acad Sci USA 97:5322–5327

Caranta C, Lefebvre V, Palloix A (1997a) Polygenic resistance of pepper to potyviruses consists of a combination of isolate-specific and broad-spectrum quantitative trait loci. Mol Plant Microbe Interact 10:872–878

Caranta C, Palloix A, Lefebvre, Daubeze AM (1997b) QTLs for a component of partial resistance to cucumber mosaic virus in pepper: restriction of virus installation in host-cells. Theor Appl Genet 94:431–438

Caranta C, Thabuis A, Palloix A (1999) Development of a CAPS marker for *Pvr4* locus: a tool for pyramiding potyvirus resistance genes in pepper. Genome 42:1111–1116

Clerjeau M, Pitrat M, Nourisseau JG (1976) La résistance du piment (*Capsicum annuum*) à *Phytophthora capsici*. IV. Etude de l'agressivitéde divers isolats au niveau des feuilles, des tiges et du collet des plantes résistantes et sensibles. Ann Phytopathol 8:411–423

de Jong W, Forsyth A, Leister D, Gebhardt C, Baulcombe DC (1997) A potato hypersensitive resistance genes against potato virus X maps to a resistance gene cluster on chromosome 5. Theor Appl Genet 95:246–252

Djian-Caporalino C, Pijarowski L, Fazari A, Samson M, Gaveau L, O'Byrne C, Lefebvre V, Caranta C, Palloix A, Abad P (2001) High resolution genetic mapping of the pepper (*Capsicum annuum* L.) resistance loci *Me3* and *Me4* conferring heat-stable resistance to root knot nematodes (*Meloidogyne* spp.). Theor Appl Genet 103:592–600

Faris JD, Li W, Gill BS, Liu D, Chen P (1999) Candidate gene analysis of quantitative resistance in wheat. Theor Appl Genet 98:219–225

Gebhardt C, Valkonen JP (2001) Organization of genes controlling disease resistance in the potato genome.Annu Rev Phytopathol 39:79–102

Geffroy V, Sicard D, de Oliveira JCF, Sevignac M, Cohen, Gepts P, Neema C, Langin T, Dron M (1999) Identification of an ancestral resistance gene cluster involved in the coevolution process between *Phaseolus vulgaris* and its fungal pathogen *Colletotrichum lindemuthianum*. Mol Plant Microbe Interact 12:774–784

Geffroy V, Sevignac M, de Oliveira JCF, Fouilloux G, Skroch P, Thoquet P, Gepts P, Langin T, Dron M (2000) Inheritance of partial resistance against *Colletotrichum lindemuthianum* in *Phaseolus vulgaris* and co-localization of quantitative trait loci with genes involved in specific resistance. Mol Plant Microbe Interact 13:287–296

Grube RC, Blauth JR, Arnedo-Andres MS, Caranta C, Jahn MK (2000a) Identification and comparative mapping of a dominant potyvirus resistance gene cluster in *Capsicum*. Theor Appl Genet 101:852–859

Grube RC, Radwanski ER, Jahn MK (2000b) Comparative mapping of disease resistance within the *Solanaceae*. Genetics 155:873–887

Hammond-Kosack KE, Jones JDG (1997) Plant disease resistance genes. Annu Rev Plant Physiol Plant Mol Biol 48:575–607

Jahn M, Paran I, Hoffman K, Radwanski ER, Livingstone KD, Grube RC, Aftergoot E, Lapidot M, Moyer J (2000) Genetic

mapping of the *Tsw* locus for resistance to the Tospovirus *Tomato spotted wilt virus* in *Capsicum* spp. and its relationship to the *Sw-5* gene for resistance to the same pathogen in tomato. Mol Plant Microbe Interact 13:673–682

Jones DA, Balint-Kurti PJ, Dickinson MJ, Dixon MS, Jones JDG (1992) Locations of genes for resistance to *Cladosporium fulvum* on the classical and reference maps of tomato. Rep Tomato Genet Coop 42:19–22

Lander ES, Botstein D (1989) Mapping Mendelian factors underlying quantitative traits using RFLP linkage maps. Genetics 121:185–199

Lefebvre V, Palloix A (1996) Both epistatic and additive effects of QTLs are involved in polygenic induced resistance to disease: a case study, the interaction pepper–*Phytophthora capsici* Leonian. Theor Appl Genet 93:503–511

Lefebvre V, Palloix A, Rives M (1993) Nuclear RFLP between pepper cultivars (*Capsicum annuum* L.). Euphytica 71:198–199

Lefebvre V, Palloix A, Caranta C, Pochard E (1995) Construction of an intraspecific integrated linkage map of pepper using molecular markers and doubled-haploid progenies. Genome 38:112–121

Lefebvre V, Goffinet B, Chauvet JC, Caromel B, Signoret P, Brand R, Palloix A (2001) Evaluation of genetic distances between pepper inbred lines for cultivar protection purposes: comparison of AFLP, RAPD and phenotypic data. Theor Appl Genet 102:741–750

Lefebvre V, Pflieger S, Thabuis A, Caranta C, Blattes A, Chauvet J-C, Daubèze A-M, Palloix A (2002) Towards the saturation of the pepper linkage map by alignment of three intraspecific maps including known-function genes. Genome 45:839–854

Leister D, Kurth J, Laurie DA, Yano M, Sasaki T, Devos K, Graner A, Schulze-Lefert P (1998) Rapid reorganization of resistance gene analogs in cereal genomes. Proc Natl Acad Sci USA 95:370–375

Leonards-Schippers C, Gieffers W, Schafer-Pregl R, Ritter E, Knapp S, Salamini F, Gebhardt C (1994) Quantitative resistance to *Phytophthora infestans* in potato: a case study for QTL mapping in an allogamous plant species. Genetics 137:67–77

Li X, van Eck HJ, Rouppe van der Voort JNAM, Huigen DJ, Stam P, Jacobsen E (1998) Autotetraploids and genetic mapping using common AFLP markers: the *R2* allele conferring resistance to *Phytophthora infestans* mapped on chromosome IV. Theor Appl Genet 96:1121–1128

Lübbersted T, Klein D, Melchinger AE (1998a) Comparative QTL mapping of resistance to *Ustilago maydis* across four populations of European flint maize. Theor Appl Genet 97:1321–1330

Lübbersted T, Klein D, Melchinger AE (1998b) Comparative quantitative trait loci mapping of partial resistance to *Puccinia sorghi* across four populations of European flint maize. Phytopathology 88:1324–1329

Melchinger AE, Utz HF, Schon CC (1998) Quantitative trait locus (QTL) mapping using different testers and independent population samples in maize reveals low power of QTL detection and large bias in estimates of QTL effects. Genetics 149:383–403

Mestries E, Gentzbittel L, Tourvieille de Labrouhe D, Nicolas P, Vear F (1998) Analyses of quantitative trait loci associated with resistance to *Sclerotinia sclerotinium* in sunflowers (*Helianthus annuus* L.) using molecular markers. Mol Breed 4:215–226

Michelmore RW, Meyers BC (1998) Clusters of resistance genes in plants evolve by divergent selection and a birth-and-death process. Genome Res 8:1113–1130

Moury B (1997) Evaluation de sources de résistance au *Tomato spotted wilt virus* chez le piment. Création d'outils d'aide à la sélection. PhD thesis, University of Rennes, France

Moury B, Pflieger S, Blattes A, Lefebvre V, Palloix A (2000) A CAPS marker to assist selection of *Tomato spotted wilt virus* (TSWV) in pepper. Genome 43:943–951

Palloix A, Daubèze AM, Pochard E (1988) Time sequences of root infection and resistance expression in an artificial inoculation method of pepper with *Phytophthora capsici*. J Phytopathol 123:12–24

Palloix A, Daubèze AM, Phaly T, Pochard E (1990) Breeding transgressive lines of pepper for resistance to *Phytophthora capsici* in a recurrent selection system. Euphytica 51:141–150

Pan Q, Liu YS, Budai-Hadrian O, Sela M, Carmel-Goren L, Zamir D, Fluhr R (2000) Comparative genetics of Nucleotide Binding Site-Leucine Rich Repeat Resistance Gene Homologues in the genomes of two cotyledons: tomato and Arabidopsis. Genetics 155:309–322

Pernet A, Hoisington D, Ditinger J, Jewell D, Jiang GC, Khairallah M, Letourmy P, Marchand JL, Glaszmann JC, Gonzales de Leon D (1999a) Genetic mapping of maize streak virus resistance from the Mascarene source. II. Resistance in line CIRAD390 and stability across germplasm. Theor Appl Genet 99:540–553

Pernet A, Hoisington D, Franco J, Isnard M, Jewell D, Jiang C, Marchand JL, Reynaud B, Glaszmann JC, Gonzales de Leon D (1999b) Genetic mapping of maize streak virus resistance from the Mascarene source. I. Resistance in line D211 and stability against different virus clones. Theor Appl Genet 99:525–539

Pflieger S, Lefebvre V, Caranta C, Blattes A, Goffinet B, Palloix A (1999) Disease resistance gene analogs as candidates for QTLs involved in pepper-pathogen interactions. Genome 42:1100–1110

Pflieger S, Palloix A, Caranta C, Blattes A, Lefebvre V (2001) Defense response genes co-localize with quantitative disease resistance loci in pepper. Theor Appl Genet 103:920–929

Pochard E, Daubèze AM (1980) Recherche et évaluation des composantes d'une résistance polygénique: la résistance du piment à *Phytophthora capsici*. Ann Amelior Plant 26:377–398

Pochard E, Clerjeau M, Pitrat M (1976) La résistance du piment, *Capsicum annuum* L., à *Phytophthora capsici* Leon. I. Mise en évidence d'une induction progressive de la résistance. Ann Amelior Plant 26:35–50

Pochard E, Molot PM, Dominguez G (1983) Etude de deux nouvelles sources de résistances à *Phytophthora capsici* Leon. chez le piment: confirmation de l'existence de 3 composantes distinctes dans la résistance. Agronomie 3:333–342

Pressoir G, Albar L, Ahmadi N, Rimbault I, Lorieux M, Fargette D, Ghesquière A (1998) Genetic basis and mapping of the resistance to rice yellow mottle virus. II. Evidence of a complementary epistasis between two QTLs. Theor Appl Genet 97:1155–1161

Qi X, Niks R, Stam P, Lindhout P (1998) Identification of QTLs for partial resistance to leaf rust (*Puccinia hordei*) in barley. Theor Appl Genet 96:1205–1215

Robertson DS (1989) Understanding the relationship between qualitative and quantitative genetics. In: Helentjaris T, Burr B (eds) Development and application of molecular markers to problems in plant genetics. Cold Spring Harbor Press, Cold Spring Harbor, pp 81–87

Ronald PC (1998) Resistance gene evolution. Curr Opin Plant Biol 1:294–298

Saliba-Colombani V, Causse M, Langlois D, Philouze J, Buret M (2001) Genetic analysis of organoleptic quality in fresh market tomato. 1. Mapping QTLs for physical and chemical traits. Theor Appl Genet 102:259–272

Sandbrik JM, Colon LT, Wolters PJCC, Stiekema WJ (2000) Two related genotypes of *Solanum microdontum* carry different segregating alleles for field resistance to *Phytophthora infestans*. Mol Breed 6:215–225

SAS Institute (1989) SAS/STAT user's guide, version 6, 4th edn. SAS Institute. Cary, N.C.

Stuber C, Edwards MD, Wendel JF (1987) Molecular marker facilitated investigations of quantitative trait locus in maize. II. Factors influencing yield and its component traits. Crop Sci 27:639–648

Thabuis A, Lefebvre V, Daubèze AM, Signoret P, Phaly T, Nemouchy G, Blattes A, Palloix A (2001) Introgression of a partial resistance to *Phytophthora capsici* Leon. into a pepper elite line by marker assisted backcrosses. Acta Hortic 546:645–650

Welz HG, Geiger HH (2000) Genes for resistance to northern corn leaf blight in diverse maize populations. Plant Breed 119:1–14

Witsenboer H, Kesseli RV, Fortin MG, Stranhellini M, Michelmore RW (1995) Sources and genetic structures of a cluster of genes for resistance to three pathogens in lettuce. Theor Appl Genet 91:178–188

Young ND (1996) QTL mapping and quantitative disease resistance in plants. Annu Rev Phytopathol 34:479–501

Zeng ZB (1994) Precision mapping of quantitative trait loci. Genetics 136:1457–1468