QTL Mapping of Fruit Rot Resistance to the Plant Pathogen *Phytophthora capsici* in a Recombinant Inbred Line *Capsicum annuum* Population

R. P. Naegele, H. Ashrafi, T. A. Hill, S. Reyes Chin-Wo, A. E. Van Deynze, and M. K. Hausbeck

First and sixth authors: Department of Plant, Soil and Microbial Sciences, Michigan State University East Lansing 48824; and second, third, fourth, and fifth authors: Seed Biotechnology Center, University of California, Davis 95616.

Accepted for publication 14 October 2013.

ABSTRACT

Naegele R. P., Ashrafi, H., Hill, T. A., Reyes Chin-Wo, S., Van Deynze, A. E., and Hausbeck, M. K. 2014. QTL mapping of fruit rot resistance to the plant pathogen *Phytophthora capsici* in a recombinant inbred line *Capsicum annuum* population. *Phytopathology* 104:479-483.

Phytophthora capsici is an important pepper (*Capsicum annuum*) pathogen causing fruit and root rot, and foliar blight in field and greenhouse production. Previously, an F6 recombinant inbred line population was evaluated for fruit rot susceptibility. Continuous variation among lines and partial and isolate-specific resistance were found. In this study, *Phytophthora* fruit rot resistance was mapped in the same F6

population between Criollo del Morelos 334 (CM334), a landrace from Mexico, and 'Early Jalapeno' using a high-density genetic map. Isolate-specific resistance was mapped independently in 63 of the lines evaluated and the two parents. Heritability of the resistance for each isolate at 3 and 5 days postinoculation (dpi) was high ($h^2 = 0.63$ to 0.68 and 0.74 to 0.83 , respectively). Significant additive and epistatic quantitative trait loci (QTL) were identified for resistance to isolates OP97 and 13709 (3 and 5 dpi) and 12889 (3 dpi only). Mapping of fruit traits showed potential linkage with few disease resistance QTL. The partial fruit rot resistance from CM334 suggests that this may not be an ideal source for fruit rot resistance in pepper.

Phytophthora capsici Leonian is an important pathogen of pepper (*Capsicum annuum*) in the United States and worldwide. This destructive pathogen is capable of infecting roots, stems, fruit, and foliage of pepper plants at any point during development (20,23,24,38,47). Disease management utilizes a combination of chemical and cultural controls to reduce losses but economic reductions in yield can still occur under conditions favorable for disease (18). The pathogen's biology and polycyclic reproduction and the development of thick-walled oospores support long survival in the soil, rapid development of fungicide resistance, and suppression of host resistance (18,20,27). Germplasm screening for root rot resistance has identified multiple lines with resistance to one or more isolates but few lines exhibit resistance to all isolates (10,25,29). *P. capsici*'s broad host range, persistent survival structures, and isolate diversity makes it incredibly difficult to manage.

Most studies and breeding have emphasized the identification and implementation of quantitative trait loci (QTL) for the root rot symptom of the disease. Studies have estimated the number of QTL for root rot resistance to vary drastically from a single dominant gene with modifiers to 14 QTL (3,28,32). A major QTL for root rot resistance was identified on chromosome (chr) 5, and molecular markers have been developed for marker-assisted selection (28,36). Development and incorporation of resistant varieties into commercial production systems has been slow. A few tolerant varieties have been bred but most are susceptible to highly virulent isolates of *P. capsici* (15). Criollo de Morelos 334 (CM334), a landrace from Mexico, is resistant to all isolates evaluated to date (10,15,17,43). Most breeding for *Phytophthora*

resistance and molecular evaluation of resistance has utilized this line (39).

Breeding for the other symptoms of the disease (fruit rot and foliar blight) has been limited. Comparative studies between root rot and foliar blight suggest that there is a single dominant gene controlling much of the foliar resistance (42). This gene was different from the major gene contributing to root rot resistance (11,43,47). This was later confirmed by Ogundiwin et al. (32) in a separate mapping population, where QTL were detected for foliar blight resistance with minimal overlap with root rot resistance. The independent associations between the foliar blight, stem blight, and root rot symptoms of the disease were also seen in a mass greenhouse evaluation by Candole et al. (10). Resistance to fruit rot is even less studied. Commercial cultivars are susceptible to *Phytophthora* fruit rot and no QTL for fruit rot resistance have been identified.

The inheritance of fruit rot resistance has been evaluated in a single study (40). In 1977, Saini and Sharma (40) looked at the inheritance of fruit rot in a mapping population and found that it segregated in a 3:1 Mendelian fashion. They concluded that a single dominant gene controlled fruit rot resistance. Previous work by Naegele et al. (31) in a recombinant inbred line (RIL) pepper mapping population with a different source of resistance demonstrated partial and isolate-specific resistance to *P. capsici*.

Maintaining desirable fruit traits such as color, firmness, and gloss is important when breeding resistance into a cultivated background from landraces and wild relatives. Often, wild relatives are small-fruited, pungent, and have many characteristics not suitable for commercial production. Landraces, though more similar to their commercial counterparts, often contain a number of undesirable traits. Numerous studies have looked at correlations between fruit characteristics and *Phytophthora* fruit rot. Pungency, pericarp thickness, and fruit firmness were shown to not be associated with fruit rot (4,5,44). Reactive oxygen species

Corresponding author: M. K. Hausbeck; E-mail address: hausbec1@msu.edu

<http://dx.doi.org/10.1094/PHYTO-05-13-0143-R>
© 2014 The American Phytopathological Society

production had a negative correlation with disease susceptibility, and higher length/width ratio for fruit shape had a positive correlation with fruit rot (31,44). Linkage of fruit rot resistance with fruit-related traits would make incorporation of the resistance into a commercial background more difficult.

The objectives of this study were to identify genetic markers associated with general and isolate-specific resistance to *P. capsici*-induced fruit rot 3 and 5 days postinoculation (dpi) in an F6 RIL population and to identify genetic markers associated with fruit-phenotypic traits to test for co-localization with disease resistance.

MATERIALS AND METHODS

Plant material and inoculations. An F2-derived F6 ‘Early Jalapeno’ × CM334 RIL pepper population consisting of 63 greenhouse-grown individuals was previously evaluated for root rot, foliar blight, and fruit rot (12,31,43). As described by Naegle et al. (31), three isolates of *P. capsici* (12889, OP97, and 13709) from the long-term collection of Dr. Mary Hausbeck (Michigan State University) were used for inoculations. Cultures were maintained, in the lab on unclarified V8 agar (16 ml) under constant light at room temperature. In brief, three detached, immature, greenhouse-grown green pepper fruit per isolate from each line were surface disinfested in a 10% bleach solution for 5 min and rinsed in distilled water. Fruit were placed into a humidity chamber and inoculated with agar plugs (6 mm in diameter) from actively growing *P. capsici* colonies placed topside down on the pepper fruit and covered with sterile microcentrifuge caps. Control pepper plants were inoculated with a V8 agar plug and covered with a sterile microcentrifuge cap. Pepper fruit were kept in the humidity chambers under constant light at room temperature (25°C). Fruit were evaluated for early (3 dpi) and late (5 dpi) responses based on preliminary results of the earliest symptoms on commercial Serrano-type pepper and longest time to cover the fruit (data not shown). At 3 and 5 dpi, lesion width and diameter was measured for each individual fruit using a hand caliper. Nine replicate fruit were evaluated for each line and isolate. Pepper line and isolate were treated as main effects in the final experimental model.

Fruit phenotypic evaluations. Evaluations for fruit length, width, and shape were previously performed as described by Naegle et al. (31). In brief, 20 immature pepper fruit from each line were evaluated for fruit length (in centimeters), width (in centimeters), shape (maximum length/width in centimeters), and color (light green, green, dark green, or purple). In addition, 20 pepper fruit from each line were evaluated for gloss (low, medium, or high), firmness, (1 to 3), and pericarp thickness (in centimeters). Fruit length, width, shape, and pericarp thickness were measured using a hand caliper. Gloss and firmness were determined on a categorical scale from 1 (low) to 3 (high), as described by Chaim et al. (12).

Statistical analyses. Data were analyzed using the PROC MIXED function of the SAS v9.3 software using the LSmeans statement (SAS Institute, Cary, NC). Significant ($P = 0.05$) interactions between line and isolate were separated using the SLICE option. Line means (LSmeans output) for isolate-specific lesion area 3 and 5 dpi were used for QTL mapping. Line means for fruit gloss, firmness, and pericarp thickness were calculated in

SAS and used for QTL mapping. Narrow-sense trait heritability was estimated using the progeny mean basis method (14,26).

Genetic and QTL mapping. QTL mapping was performed using the R/QTL function in the statistical software R (9,37) using the genetic map previously built by Hill et al. (21) based on single-position polymorphisms (22). Prior to QTL mapping, the linkage map was evaluated for recombination frequency, marker order, segregation distortion, and switched genotypes. Problematic markers were removed from the map. The final map contained 3,814 markers, with 222 to 450 markers/chromosome and an estimated coverage of 1,267 centimorgans (cM) (Table 1). Missing genotypes were identified at each marker and the genotype was estimated using the imputation method implemented in R/QTL. QTL for each trait were identified using interval mapping by the multiple imputation method. Significance of QTL was determined using 1,000 replicates of the permutation test. After a single significant QTL was identified, additional additive and epistatic QTL were added using composite interval mapping. Multiple QTL, interactions, and effects of individual QTL were confirmed and estimated using a general linear model implemented in R/QTL, where y equals the sum of the individual QTL and their interactions ($y \approx Q1 + Q2 + Q1 \times Q2$). Confidence intervals were calculated for each resistance QTL using the 1.5-log of the likelihood ratio (LOD) support interval implemented in R/QTL.

RESULTS

A significant line–isolate interaction was detected at 3 dpi ($P = 0.02$) and 5 dpi ($P < 0.0001$) for the population and lines were evaluated separately for each isolate. The pepper population exhibited a skewed distribution at 3 dpi toward the more resistant phenotype and a more normal distribution at 5 dpi (Fig. 1). QTL for resistance to *Phytophthora* fruit rot were detected for isolates 12889, 13709, and OP97 at both 3 and 5 dpi. Significant LOD thresholds at $P = 0.05$ were 3.11 to 3.36 for disease responses. QTL for fruit characteristics had significant LOD thresholds at $P = 0.05$ for gloss, firmness, fruit shape, and pericarp thickness at 3.26, 3.32, 2.97, and 3.3, respectively. Confidence intervals are listed in parenthesis after each QTL.

Resistance to *P. capsici* 3 dpi. At 3 dpi, 24 to 48 lines were as resistant to *P. capsici* as the resistant parent CM334 and significantly more resistant than Early Jalapeno, depending on the isolate (data not shown). The mean lesion area for the population was 2.52 ± 1.63 , 3.72 ± 3.65 , and 3.06 ± 2.33 for isolates 13709, 12889, and OP97, respectively. Control fruit showed no symptoms at 3 or 5 dpi and were removed from analyses in order to not violate normality assumptions. Heritability of disease resistance to individual isolates was 0.68, 0.63, and 0.63 for isolates 12889, 13709, and OP97, respectively. All QTL were significant at $P = 0.01$ unless specified otherwise, and all interactions between QTL were positive unless specified otherwise. Isolate-specific QTL with varying effects were detected for each of the *P. capsici* isolates tested. For isolate OP97, two QTL were detected. One QTL located on chr 6 at 56 cM (53 to 66 cM) explained 14.8% of the variation seen and another on chr 5 at 21 cM (16.94 to 30 cM) explained 12% of the trait variation. The resistant allele on chr 6 was from the susceptible parent (Early Jalapeno) (Table 2). Resistance to 13709 was also explained, in part, by two QTL. One

TABLE 1. Distribution and marker density for the pepper recombinant inbred line genetic map^a

Chromosome	P1	P2	P3	P4	P5	P6	P7	P8	P9	P10	P11	P12
Number of markers	450	407	415	244	222	267	140	261	513	305	363	227
Distance (cM)	140	101	124	100	78.5	122	116	86	90	107	113	89.9
Markers/cM	3.2	4.1	3.3	2.4	2.8	2.2	1.2	3.0	5.7	2.9	3.2	2.5

^a Shown are the total number of markers per chromosome from P1 to P12 (number of markers), the centimorgan (cM) distance per chromosome (distance). The estimated coverage of the genetic map is 1,267.2 cM.

QTL on chr 6 at 69.3 cM (62 to 72 cM) explained almost 30% of the variation and came from the susceptible parent while the other at the end of chr 5 at 0 cM (confidence interval not calculable) was from the resistant parent and explained 17% of the variation. Isolate 12889 also had contributions for resistance from both parents on chr 6 at 58.1 cM (58 to 69.4 cM) and chr 2 at 13 cM (5 to 15.7 cM) explaining 38 and 16.4% of the variation, respectively. An interaction between the two QTL contributed another 13%.

Resistance to *P. capsici* 5 dpi. At 5 dpi, 29 to 40 lines evaluated were at least as susceptible to *P. capsici* as Early Jalapeno and significantly less resistant than CM334. Isolate-specific

responses were detected for each of the lines. The mean lesion area of the population plus or minus standard deviation was 16.8 ± 19.01 , 9.00 ± 6.94 , and 21.43 ± 25.60 for isolates 13709, 12889, and OP97, respectively. The resistant parent (CM334) was the only source of resistance at 5 dpi. Heritability for resistance to individual isolates was 0.76, 0.74, and 0.83 for isolates 12889, 13709, and OP97, respectively. Isolate OP97 at 5 dpi was the most virulent isolate and resistance to this isolate had the highest heritability. Resistant loci were detected on chr 4 at 13 cM (12 to 17.1 cM) and chr 5 at 29.8 cM (26.7 to 30 cM), explaining 19 and 50% of the variation observed, respectively. An interaction between the two QTL explained another 16% of the variation. Isolate 13709 had QTL located on chr 3 at 124 cM (76 to 128 cM) and chr 5 at 74 cM (70.5 to 78 cM), explaining 14.5 and 31% of the total variation observed, respectively. Interactions between the QTL on chr 5 and that on chr 6 explained an additional 7%. There were no isolate-specific QTL detected for isolate 12889 at 5 dpi.

Fruit trait QTL and co-localization with disease. Significant QTL were detected for fruit firmness, fruit shape, and pericarp thickness (Table 3). All QTL were significant at $P = 0.01$ unless otherwise specified. One QTL was detected for fruit firmness located on chr 12 at 55.4 cM. This QTL explained $\approx 29.8\%$ of the variation within the population. Fruit shape QTL were detected on chr 1 ($P = 0.05$), chr 2 ($P = 0.05$), chr 4, chr 5, and chr 10 ($P = 0.05$) at positions 124, 71.3, 32.2, 38, and 2.2 cM, respectively. Together, these QTL explained $>50\%$ of the variation observed. An epistatic interaction ($P = 0.05$) between the QTL located on chr 1 and chr 4 explained an additional 4.7%. Pericarp thickness resulted in a single QTL chr 3 (114 cM) that explained 26% of the variation. This QTL was tightly linked with a QTL for resistance to isolate 13709 at 5 dpi. QTL for fruit firmness did not show a tight linkage (≤ 10 cM) with any of the disease QTL mapped.

DISCUSSION

Host resistance to *P. capsici* is an important component for disease management in many areas of the world. Disease symptoms and the genetic factors controlling resistance to the pathogen can vary greatly depending on the site of infection. In addition to site-specific responses, isolate-specific interactions can be often host specific (10,15,29,35). Even resistant commercial pepper lines could succumb to highly virulent isolates when tested under greenhouse conditions (15). This combination of site-specific and isolate-specific responses makes breeding for resistance to *P. capsici* incredibly challenging.

Fig. 1. Frequency distribution of the average disease response of the pepper population combined across isolates at **A**, 3 and **B**, 5 days postinoculation. Mean lesion categories for resistant parent Criollo del Morelos 334 (CM) and the susceptible parent Early Jalapeno (EJ) are indicated by arrows.

TABLE 2. Fruit rot resistance quantitative trait loci (QTL)^a

Trait	Chromosome	Position	Marker name	LOD value	R ²	Source	Sig	Est. h ²
Additive effects								
12889 3 dpi	2	13	CAPS_CONTIG.10457	3.33	0.167	CM	**	0.68
	6	58.1	CAPS_CONTIG.3200	6.66	0.381	EJ	***	...
13709 3 dpi	5	0	CAPS_CONTIG.10639	3.36	0.164	CM	***	0.63
	6	69.3	CAPS_CONTIG.10555	5.51	0.292	EJ	***	...
13709 5 dpi	3	124	KS24039C09	4.4	0.207	CM	***	0.74
	5	71.2	CAPS_CONTIG.11780	7.49	0.330	CM	***	...
OP97 3 dpi	5	21.1	CAPS_CONTIG.11660	5.21	0.273	CM	***	0.63
	6	56	CAPS_CONTIG.1455	3.26	0.158	EJ	***	...
OP97 5 dpi	4	13	CAPS_CONTIG.9283	9.53	0.175	CM	***	0.83
	5	29.8	KS25046E04	4.05	0.500	CM	***	...
Epistatic effects								
12889 3 dpi	P2 × P6	13 × 58.1	CAPS_CONTIG.10457 × CAPS_CONTIG.3200	2.76	0.136	CM × EJ	***	...
13709 5 dpi	P3 × P5	124 × 71.2	KS24039C09 × CAPS_CONTIG.11780	1.24	0.070	CM × CM	**	...
OP97 5 dpi	P4 × P5	13 × 29.8	CAPS_CONTIG.9283 × KS25046E04	3.39	0.138	CM × CM	***	...

^a Additive and epistatic effect QTL for general response resistance 3 and 5 days postinoculation (dpi) and isolate specific resistance for isolates 12889, 13709, and OP97 3 and 5 dpi. Chromosome and genetic map position (Position) of the marker. Log of the likelihood ratio (LOD) value of the QTL, percentage of the variation explained by the QTL (R²), and the donor parent of the resistant allele (Source). CM = Criollo del Morelos 334 and EJ = Early Jalapeno. Significance (Sig) of the QTL were determined by the general linear model implemented in R/QTL at $P = 0.05$, 0.01, and 0.001 (*, **, and ***, respectively). Est. h² = estimated narrow-sense heritability of the trait calculated using variance components.

TABLE 3. Fruit phenotype quantitative trait loci (QTL)^a

Trait	Chromosome	Position	Marker	LOD	R ²	Source	Sig
Additive effects							
Firmness	12	55.4	KS21041M02	4.486	0.298	EJ	***
Fruit shape	1	124	CAPS_CONTIG.816	1.98	0.055	CM	*
	2	71.3	CAPS_CONTIG.3755	1.83	0.051	CM	*
	4	32.2	KS20017D07	7.58	0.264	EJ	***
	5	38	KS17024G04	3.89	0.117	EJ	***
	10	2.2	KS26009E11	1.79	0.049	CM	*
Pericarp	3	114	KS17057E04	4.14	0.26	EJ	***
Epistatic effects							
Fruit shape	P1 × P4	124 × 32.2	KS20017D07 × CAPS_CONTIG.816	1.72	0.047	EJ × CM	*

^a Additive and epistatic effect QTL for fruit firmness and fruit shape. Chromosome and genetic map position (position) of the marker. Log of the likelihood ratio (LOD) value of the QTL, percentage of the variation explained by the QTL (R^2), and the donor parent of the allele (Source). CM = Criollo del Morelos 334 and EJ = Early Jalapeno. Significance (Sig) of the QTL were determined by the general linear model implemented in R/QTL at $P = 0.05, 0.01$, and 0.001 (*, **, and ***, respectively).

Breeding for all three symptoms of *Phytophthora* spp. infection (foliar blight, root rot, and fruit rot) is important for a sustainable and effective resistance. Previously, studies have mapped root rot resistance and foliar blight in multiple pepper populations but not fruit rot. Root rot and foliar blight are both estimated to be controlled by multiple QTL and to exhibit isolate-specific responses. Root rot is the most common symptom seen in the field but, under certain conditions, fruit rot can quickly decimate yields (4). Soil-applied systemic fungicides, a staple of any *P. capsici* management program, do not provide protection to fruit.

The idea of early and late *Phytophthora* resistance was first suggested on pepper roots by Pochard et al. (34), who observed that roots exhibited an early response termed receptivity, a late response termed stability, and the rate of response called inducibility. In our fruit study, we looked at early (3 dpi) and late (5 dpi) responses based on preliminary results of the earliest symptoms on commercial Serrano-type pepper and longest time to cover the fruit. At 3 dpi, approximately half of the lines exhibited few symptoms, suggesting that partial early resistance was present in a 1:1 ratio in the population, though multiple QTL were detected. Resistance at 5 dpi was less common (21 individuals) and the only QTL identified were from the resistant parent.

These results at 5 dpi contrast with previous results by Saini and Sharma (40) that found resistance to be controlled by a single dominant trait. In this study, resistance to *Phytophthora* fruit rot at 3 and 5 dpi was controlled by multiple genes of varying effects, with high overall heritability. In addition, the susceptible parent was also found to contribute a positive effect on disease resistance during early stages of response. However, it should be noted that, because these fruit were evaluated under ideal conditions for disease, some of the lines evaluated in this study with partial resistance may be more resistant under field conditions or as the fruit matures. Studies have shown that resistance to *P. capsici* increases for fruit and plants as tissues mature (4,16,23,24). Fruit rot resistance was controlled by a single dominant gene in 'Waxy Globe' but, in CM334, the trait is quantitatively inherited and highly influenced by the environment. The heritability of fruit rot resistance at 5 dpi was high, and the detection of major QTL controlling resistance may make marker-assisted selection useful.

Fruit traits evaluated also showed evidence of quantitative inheritance, consistent with previous studies (2,7,12,33,41,46, 48,49). QTL for fruit firmness did not show a tight linkage (≤ 10 cM) with any of the disease QTL mapped. The fruit shape QTL on chr 5 was linked with a major effect QTL for resistance to OP97 at 5 dpi (54% of the variation) and a moderate effect on fruit shape ($\approx 12\%$). The fruit shape QTL on chr 10 was a minor QTL ($< 10\%$) for both fruit rot resistance and fruit shape. This was consistent with the previous study that detected a small yet significant correlation between fruit shape and disease for at least one isolate (31). Pericarp thickness also had potential linkage with a disease resistance QTL with disease. This pericarp

thickness QTL was within 10 cM of a resistance QTL explaining 18% of the disease variation 5 dpi. The positive QTL for increased pericarp thickness was associated with Early Jalapeno (the susceptible parent), suggesting that the thinner pericarp may be associated with resistance. However, the linked QTL was only found in disease resistance to a particular isolate (OP97) and may not be useful when breeding for resistance to multiple isolates. A previous study (31) evaluated the correlation between fruit phenotype characteristics and pepper fruit susceptibility to *P. capsici* and found no correlation with resistance or susceptibility to pericarp thickness to any of the isolates evaluated.

Most QTL identified at 3 and 5 dpi were located on chr 5, a known hot spot for *Phytophthora* resistance genes in several solanaceous species, including a major gene for root rot resistance to *P. capsici* (19,45). The quantitative inheritance of fruit rot is consistent with previous studies on root rot and foliar blight in pepper (6,13,30,32). The resistant parent used in this study, CM334, though more tolerant than Early Jalapeno, was not immune to *Phytophthora* fruit rot under our conditions, and may not be the best source of fruit rot resistance. In addition, the cultivated parent (Early Jalapeno) contributed two loci on chr 6 to resistance at 3 dpi, making commercial cultivars a potential source of early resistance to *P. capsici*. In this study, the QTL effects are likely to be slightly overestimated due to the limited number of individuals evaluated in this population (8) but provide a good first approximation. Other sources of resistance may have more complete resistance and more evaluation of lines is needed. For the QTL that were detected, further work with larger populations and more isolates will be needed to confirm the resistance QTL and their co-localization with fruit traits in different genetic backgrounds and to identify additional resistance QTL.

ACKNOWLEDGMENTS

We thank H. Gutting, J. Olsen, K. Stoeffel, W. Rehrig, and D. Wang for their technical assistance.

LITERATURE CITED

- Anonymous. 2012. Vegetable Summary. U. S. Dep. Agric. Natl. Agric. Summary Stat. 0884-6413. Online publication.
- Barchi, L., Lefebvre, V., Sage-Palioix, A., Lanteri, S., and Palloix, A. 2009. QTL analysis of plant development and fruit traits in pepper and performance of selective phenotyping. *Theor. Appl. Genet.* 118:1157-1171.
- Barksdale, T. H., and Johnston, S. A. 1984. Resistance to foliar blight and crown rot of pepper caused by *Phytophthora capsici*. *Plant Dis.* 68:506-509.
- Biles, C. L., Bruton, B. D., Wall, M. M., and Rivas, M. 1995. *Phytophthora capsici* zoospore infection of pepper fruit in various physical environments. *Proc. Okla. Acad. Sci.* 75:1-5.
- Biles, C. L., Wall, M. M., Waugh, M., and Palmer, H. 1993. Relationship of *Phytophthora* fruit rot to fruit maturation and cuticle thickness of New

- Mexican-type peppers. *Phytopathology* 83:607-611.
6. Bonnet, J., Danan, S., Boudet, C., Carchi, L., Sage-Palloix, A. M., Caromel, B., Palloix, A., and Lefebvre, V. 2007. Are the polygenic architectures of resistance to *Phytophthora capsici* and *P. parasitica* independent in pepper? *Theor. Appl. Genet.* 115:253-264.
 7. Borovsky, Y., and Paran, I. 2011. Characterization of fs10.1, a major QTL controlling fruit elongation in *Capsicum*. *Theor. Appl. Genet.* 123:657-665.
 8. Broman, K. W., and Sen, S. 2009. A guide to QTL mapping with R/QTL. Statistics for Biology and Health. Springer Science + Business Media LLC. Online publication. doi:10.1007/978-0-387-92125-9_1
 9. Broman, K. W., Wu, H., Sen, S., and Churchill, G. A. 2003. R/QTL: QTL mapping in experimental crosses. *Bioinformatics* 19:889-890.
 10. Candole, B. L., and Conner, P. J. 2010. Screening *Capsicum annuum* accessions for resistance to six isolates of *Phytophthora capsici*. *Hortic. Sci.* 45:254-259.
 11. Candole, B. L., Conner, P. J., and Ji, P. S. 2012. Evaluation of *Phytophthora* root rot-resistant *Capsicum annuum* accessions for resistance to *Phytophthora* foliar blight and *Phytophthora* stem blight. *Agric. Sci.* 3:732-737.
 12. Chaim, A. B., Paran, I., Grube, R. C., Jahn, M., van Wijk, R., and Peleman, J. 2001. QTL mapping of fruit-related traits in pepper (*Capsicum annuum*). *Theor. Appl. Genet.* 102:1016-1028.
 13. Egea-Gilabert, C., Bilotti, G., Requena, M. E., Ezziyiani, M., Vivo-Molina, J. M., and Candela, M. E. 2008. Pepper morphological traits related with resistance to *Phytophthora capsici*. *Biol. Plant.* 52:105-109.
 14. Fehr, W. R., 1987. Principles of Cultivar Development. Macmillan, Inc., New York.
 15. Foster, J. M., and Hausbeck, M. K. 2010. Resistance of pepper to *Phytophthora* crown, root and fruit rot is affected by isolate virulence. *Plant Dis.* 94:24-30.
 16. Gevens, A. J., Ando, K., Lamour, K. H., Grumet, R., and Hausbeck, M. K. 2006. A detached cucumber fruit method to screen for resistance to *Phytophthora capsici* and effect of fruit age on susceptibility to infection. *Plant Dis.* 90:1276-1282.
 17. Glosier, B. R., Ogundiwin, E. A., Dihy, G. S., Sischo, D. R., and Prince, J. P. 2008. A differential series of pepper (*Capsicum annuum*) lines delineates fourteen physiological races of *Phytophthora capsici*. *Euphytica* 162:23-30.
 18. Granke, L. L., Quesada-Ocampo, L., Lamour, K., and Hausbeck, M. K. 2006. Advances in research on *Phytophthora capsici* on vegetable crops in the United States. *Plant Dis.* 95:1588-1600.
 19. Grube, R. C., Radwanski, E. R., and Jahn, M. 2000. Comparative genetics of disease resistance within the Solanaceae. *Genetics* 155:873-887.
 20. Hausbeck, M. K., and Lamour, K. H. 2004. *Phytophthora capsici* on vegetable crops: research progress and management challenges. *Plant Dis.* 88:1292-1303.
 21. Hill, T. A., Ashrafi, A. H., Reyes-Chin-Wo, S., Solano Romero, S., Van Deynze, A., and Kozik, A. 2011. Comparisons of high-density EST-based maps in pepper species. In: *Plant Biology*. American Society of Plant Biologists, Minneapolis, MN.
 22. Hill, T. A., Ashrafi, H., Reyes-Chin-Wo, S., Yao, J., Stoffel, K., Trucio, M.-J., Kozik, A., Michelmore, R. W., and Van Deynze, A. 2013. Characterization of *Capsicum annuum* genetic diversity and population structure based on parallel polymorphism discovery with a 30K unigene pepper GeneChip. *PLoS ONE* 10.1371/journal.pone.0056200.
 23. Hwang, B. K., Kim, Y. J., and Kim, C. H. 1996. Differential interactions of *Phytophthora capsici* isolates with pepper genotypes at various plant growth stages. *Eur. J. Plant Pathol.* 102:311-316.
 24. Kim, Y. J., Hwang, B. K., and Park, K. W. 1989. Expression of age-related resistance in pepper plants infected with *Phytophthora capsici*. *Plant Dis.* 73:745-747.
 25. Kimble, K. A., and Grogan, R. G. 1960. Resistance to *Phytophthora* root rot in pepper. *Phytopathology* 50:642-642.
 26. Knapp, S. J., Stroup, W. W., and Ross, W. M. 1985. Exact confidence intervals for heritability on a progeny means basis. *Crop Sci.* 25:192-194.
 27. Lamour, K. H., and Hausbeck, M. K. 2001. Investigating the spatio-temporal genetic structure of *Phytophthora capsici* in Michigan. *Phytopathology* 91:973-980.
 28. Lefebvre, A., and Palloix, A. 1996. Both epistatic and additive effects of QTL are involved in polygenic induced resistance to disease: a case study, the interaction pepper-*Phytophthora capsici* Leonian. *Theor. Appl. Genet.* 93:503-511.
 29. McGregor, C., Waters, V., Nambeesan, S., MacLean, D., Candole, B. L., and Conner, P. 2011. Genotypic and phenotypic variation among pepper accessions resistant to *Phytophthora capsici*. *HortScience* 46:1235-1240.
 30. Minamiyama, Y., Tsuro, M., Kubo, T., and Hirai, M. 2007. QTL analysis for resistance to *Phytophthora capsici* in pepper using a high-density SSR-based map. *Breed. Sci.* 57:129-134.
 31. Naegle, R. P., and Hausbeck, M. K. Evaluation of pepper fruit susceptibility to *Phytophthora capsici* in a recombinant inbred line population and its correlation with fruit shape. *Plant Dis.* In press.
 32. Ogundiwin, E. A., Berke, T. F., Massoudi, M., Black, L. L., Huestis, G., Choi, D., Lee, S., and Prince, J. P. 2005. Construction of 2 intraspecific linkage maps and identification of resistance QTL for *Phytophthora capsici* root-rot and foliar-blight diseases of pepper (*Capsicum annuum* L.). *Genome* 48:698-711.
 33. Paran, I., and van der Knaap, E. 2007. Genetic and molecular regulation of fruit and plant domestication traits in tomato and pepper. *J. Exp. Bot.* 58:3841-3852.
 34. Pochard, E., Molot, P. M., and Dominguez, G. 1983. Characterization of two new sources of resistance to *Phytophthora capsici* Leon in pepper: further evidence for the existence of three resistance components. *Agronomie* 3:333-342.
 35. Quesada-Ocampo, L. M., and Hausbeck, M. K. 2010. Resistance in tomato and wild relatives to crown and root rot caused by *Phytophthora capsici*. *Phytopathology* 100:619-627.
 36. Quirin, E. A., Ogundiwin, E. A., Prince, J. P., Mazourek, M., Briggs, M. O., Chlanda, T. S., Kim, K. T., Falise, M., Kang, B. C., and Jahn, M. M. 2005. Development of sequence characterized amplified region (SCAR) primers for the detection of *Phyto.5.2*, a major QTL for resistance to *Phytophthora capsici* Leon. in pepper. *Theor. Appl. Genet.* 110:605-612.
 37. R Development Core Team. 2012. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna.
 38. Reifschneider, F. J. B., Boiteux, L. S., Della Vecchia, P. T., Poulos, J. M., and Kuroda, N. 1992. Inheritance of adult-plant resistance to *Phytophthora capsici* in pepper. *Euphytica* 62:45-49.
 39. Richins, R. D., Micheletto, S., and O'Connell, M. A. 2010. Gene expression profiles unique to chili (*Capsicum annuum* L.) resistant to *Phytophthora* root rot. *Plant Sci.* 178:192-201.
 40. Saini, S. S., and Sharma, P. P. 1977. Inheritance of resistance to fruit rot (*Phytophthora capsici* Leon.) and induction of resistance in bell pepper (*Capsicum annuum* L.). *Euphytica* 27:721-723.
 41. Stommel, J. R., and Griesback, R. J. 2008. Inheritance of fruit, foliar, and plant habit attributes in *Capsicum*. *J. Am. Soc. Hortic. Sci.* 133:396-407.
 42. Sy, O., Bosland, P. W., and Steiner, R. 2005. Inheritance of *Phytophthora* stem blight resistance as compared to *Phytophthora* root rot and *Phytophthora* foliar blight resistance in *Capsicum annuum* L. *J. Am. Soc. Hortic. Sci.* 130:75-78.
 43. Sy, O., Steiner, R., and Bosland, P. W. 2008. Recombinant inbred line differential identifies race-specific resistance to *Phytophthora* root rot in *Capsicum annuum*. *Phytopathology* 98:867-870.
 44. Tahboub, M. B., Sanogo, S., Bosland, P. W., and Murray, L. 2008. Heat level in chili pepper in relation to root and fruit infection by *Phytophthora capsici*. *HortScience* 43:1846-1851.
 45. Thabuis, A., Palloix, A., Servin, B., Baubeze, A. M., Signoret, P., Hospital, F., and Lefebvre, V. 2004. Marker-Assisted introgression of 4 *Phytophthora capsici* resistance QTL alleles into a bell pepper line: validation of additive and epistatic effects. *Mol. Breed.* 14:9-20.
 46. Tsaballa, A., Pasentsis, K., Darzentas, N., and Tsaftaris, A. 2011. Multiple evidence for the role of an *Ovate*-like gene in determining fruit shape in pepper. *BMC Plant Biol.* 11:46.
 47. Walker, S. J., and Bosland, P. W. 1999. Inheritance of *Phytophthora* root rot and foliar blight resistance in pepper. *J. Am. Soc. Hortic. Sci.* 124:14-18.
 48. Yarnes, S. C., Ashrafi, H., Reyes-Chin-Wo, S., Hill, T. A., Stoffel, K. M., and Van Deynze, A. 2013. Identification of QTL for capsaicinoids, fruit quality, and plant architecture-related traits in an interspecific *Capsicum* RIL population. *Genome* 56:1-14.
 49. Zygier, S., Chaim, A. B., Efrati, A., Kaluzky, G., Borovsky, Y., and Paran, I. 2005. QTL mapping for fruit size and shape in chromosomes 2 and 4 in pepper and a comparison of the pepper QTL map with that of tomato. *Theor. Appl. Genet.* 111:437-445.