Genetic Analysis of Phytophthora Root Rot Race-specific Resistance in Chile Pepper

Ariadna Monroy-Barbosa1,3 and Paul W. Bosland2

Department of Plant and Environmental Science, New Mexico State University, Las Cruces, NM 88003–8003

ABSTRACT. Phytophthora capsici Leon., causal agent of phytophthora root rot, is one of the most devastating pathogens attacking chile pepper (Capsicum annuum L.) plants. Many studies have tried to better understand phytophthora resistance, but the genetic behavior is not completely understood. To determine if phytophthora root rot resistance in chile pepper is controlled by multiple alleles at a few loci, or multiple genes at different loci, five recombinant inbred lines (RILs) were evaluated. The resistant accession, Criollo de Morelos-334, and the susceptible cultivar, Early Jalapeno, were hybridized to develop multiple RILs. After seven generations of selfing using the single seed descent method, four RILs were selected based on their phenotypic response to inoculation by five P. capsici isolates. The RILs were rehybridized to each other to obtain F1 and F2 populations. The F2 populations were inoculated with single and a pair of races of P. capsici. When the F2 populations were inoculated with a single race, ratios of three resistant:one susceptible were obtained in the majority of the populations, indicating the action of an independent single gene. When the F2 populations were inoculated with a combination of two races, segregation ratios of 15 resistant:one susceptible were observed in two populations out of the four populations. The presence of susceptible individuals in all of the F2 population indicates that the resistant genes for the different P. capsici races are located at different loci. However, the rejection of the segregation ratio in one of the F2 population under a single race inoculation and in two of the F2 populations challenged with a combination of two races suggest a linkage phenomenon between some of the R genes. None of the RILs evaluated in this study displayed allelism for phytophthora root rot resistance.

Chile pepper is a very important crop worldwide, being used in the food industry as well as a coloring agent for food and cosmetics, an ingredient in pain relief medicine, antimugger sprays, and so on (Bosland, 1996; Lucier and Jerardo, 2006; Morrison and Skaggs, 2004). Chile pepper production can be dramatically reduced by a soilborne disease called phytophthora blight caused by the oomycete Phytophthora capsici. In 1922, P. capsici was first described as the pathogen of chile pepper in New Mexico (Leonian, 1922). This pathogen can completely devastate a field of chile peppers (Sanogo and Carpenter, 2006). The pathogen causes multiple disease syndromes such as phytophthora root rot, fruit rot, stem blight, and foliar blight (Sy et al., 2005). Based on host differential studies, nine P. capsici races for root rot syndrome were reported by Oelke et al. (2003) and, after that, 11 more races were identified by Sy et al. (2008). Thus, the presence of different P. capsici races complicates the control of this pathogen even more.

To develop strategies to control P. capsici, it is necessary to understand the genetic interaction between the plant and the pathogen. A gene-for-gene theory explains the specificity of this interaction as the recognition of an elicitor encoded by an Avr gene in the pathogen by a receptor encoded by its complementary R gene in the host and that this matching gene pair has an epistatic effect over any other incompatible gene pair (Crute and Pink, 1996; Flor, 1955). The specific recognition of R genes and Avr genes results in the induction of a signal transduction in the host that will initiate host defense responses against all the pathogen races and the inhibition of the pathogen growth (Staskawicz, 2001; Tyler, 2002). According to the gene-for-gene model when a resistant trait is manifested by the effect of dominant alleles, it is not possible to observe segregation. The complete lack of segregation for susceptibility provides strong evidence that the resistant phenotype is located at one locus (Chen et al., 2001). On the other hand, when two independent loci control the resistance phenotype, a segregating F2 population should contain at least 1/16 susceptible segregants, which represent the presence of double homozygous recessives (Chen et al., 2001).

Several inheritance studies on P. capsici resistance in chile pepper have produced different results such as single-gene, two-gene, or multiple-gene systems (Barksdale et al., 1984; Guerrero-Moreno and Laborde, 1980; Ortega et al., 1995; Saini and Sharma, 1978; Smith et al., 1967; Sy et al., 2005; Walker and Bosland, 1999). The disparity in the reports published could be because of the use of different isolates, the cultivars, or environmental conditions. Resistance to P. capsici in C. annuum is genetically and physiologically complex (Quirin et al., 2005). Thus, despite the existence of resistant accessions such as Criollo de Morelos-334 (CM-334) and extensive breeding efforts, no chile pepper cultivars with universal resistance to phytophthora root rot have been commercially released (Oelke et al., 2003). Thus, the main objective of this study was to determine whether different phytophthora root rot race-specific resistance phenotypes for P. capsici races 1, 4, 5, 6, and 12 are controlled by multiple alleles at few loci or multiple genes at different loci using a set of New Mexico recombinant inbred lines (NMRLs). This information will aid in characterizing the genotype of resistant materials and also facilitate the mapping of phytophthora root rot resistance in...
chile pepper. In addition, these results will increase the efficiency of breeding programs in developing phytophthora root rot-resistant cultivars.

**Materials and Methods**

**Plant materials**

A population of 67 NMRILs was developed by Sy et al. (2008) from the hybridization of the resistant line CM-334 with the susceptible commercial cultivar Early Jalapeno. The NMRILs were obtained through the single seed descent procedure (Lister and Dean, 1993). This procedure was repeated until the seventh generation. From the previous population, four NMRILs—NMRILA, NMRILB, NMRILK, and NMRILX—were hybridized to generate F1 and F2 populations. The four NMRILs were chosen based on their phenotypic reaction when inoculated with a given isolate (Table 1). The hybridizations evaluated were: NMRILA × NMRILX, NMRILB × NMRILX, and NMRILB × NMRILK.

In a greenhouse, two seeds of each NMRIL were sown per cell in plastic trays of 72 cells (TOD 1804; T.O. Plastics, Clearwater, MN). Trays were filled with a commercial peatmoss-vermiculite soil mixture (Sun Gro Redi-earth Plug and Seedling Mix; Sun Gro Horticulture, Bellevue, WA), and they were placed on propagation pads to maintain soil temperature at 28 °C to promote seed germination. The trays were watered twice per day and fertilized with a 14N–6.2P–11.6K slow-release fertilizer (Osmocote 14-14-14; Scotts, Marysville, OH) as needed until the four- to six-true-leaf stage.

**Inoculum**

For this study, five *P. capsici* races were used: race 1 (ATCC no. MYA-2289), race 4 (6021EPPWS), race 5 (6021EPPWS), race 6 (6022EPPWS), and race 12 (6534EPPWS). The first isolate is located at American Type Culture Collection (ATTC), the rest of the isolates were provided by S. Sanogo of New Mexico State University. The races were maintained separately on water agar plates at 24 °C (Oelke et al., 2003). For inoculation, a 0.5-cm diameter plug was cut from the water agar medium, transferred to V8 agar, and maintained in an incubator at 28 °C for a period of 4 to 8 d until sporangia formation. After this, the V8 agar was cut into 15 to 18 pieces and transferred to 15-mm petri plates partially filled with sterilized distilled water. The plates were maintained in an incubator for 2 d at 28 °C. To promote zoospore release, the water plates were incubated at 10 °C for 1 h and placed back into the 28 °C incubator for another hour. The zoospores were collected and counted using a hemacytometer. The inoculum concentration was adjusted to 2000 zoospores/mL. The seedling screening method used is described by Bosland and Lindsey (1991). Seedlings were inoculated at the four- to six-true-leaf stages.

**Inoculation with a single race.** Each cell was inoculated with 5 mL of the 2000 zoospores/mL inoculum, giving a final concentration of 10,000 zoospores per cell. The root area of the cell was kept in a flooded condition for 48 h. Propagation pads ensured a soil temperature of 28 °C.

**Inoculation with a combination of two races.** The combinations of races used in this study were races 1 + 4, races 1 + 5, races 1 + 12, and races 4 + 6. These combinations were chosen because they varied in their specific phenotypic reaction to the NMRILs (Table 1). Plants were inoculated with 2.5 mL of each race, giving a final inoculum concentration of 10,000 zoospores per cell, the same concentration as with the single race.

**Scoring**

In this study, two phenotypic reactions were evaluated: resistant and susceptible. A disease assessment described by Bosland and Lindsey (1991) was used for the scoring. Plants with no symptoms (0 to 1) were considered resistant, although plants with brown roots, slight stunting (2), and very small lesion from stems (3); brown roots, small lesions on stems, lower leaves wilted, and stunted plants (5); brown roots, large lesion on stems, girdling, whole plant wilted, and stunted (7) to death (9) were considered susceptible. Even numbers for levels were used for intermediate responses. CM-334 was used as the resistant control, and ‘Early Jalapeno’ was used as the susceptible control. The plants were scored 10 d after inoculation when the susceptible control, ‘Early Jalapeno’, was dead (9). According to Bosland and Lindsey (1991), any plant that displays symptoms will be considered susceptible because it will not be able to reach the reproduction stage. Resistant:susceptible ratios were given as result of the scoring.

A test for goodness-of-fit was developed for the F1 progeny and F2 populations against Mendelian ratios for inheritance of one gene (three resistant:one susceptible) and two independent dominant genes (15 resistant:one susceptible). The segregation ratios were evaluated using an α = 0.05. The hypothesis was considered rejected for that specific segregation ratio for any population with $P \leq 0.0001$.

**Results**

The resistant control, CM-334, had a resistant phenotype against all the races, and combination of races; CM-334 never displayed any disease symptoms. The susceptible control, ‘Early Jalapeno’, displayed disease symptoms against all the races and combination of races. Usually, 10 d after inoculation ‘Early Jalapeno’ scored between levels 8 and 9. When NMRILA, NMRILB, NMRILK, and NMRILX were challenged with a single race, the NMRILs displayed different phenotypic reactions. Thus, a host differential reaction was observed when the NMRILs were inoculated with the same race, and one displayed a resistant phenotype and the other displayed a

Table 1. Phenotypic response of four *Capsicum annuum* New Mexico recombinant inbred lines inoculated with five different *Phytophthora capsici* isolates.

| Race designation* | P. capsici isolates |
|-------------------|---------------------|
|                   | 1                  | 4                  | 5                  | 6                  | 12                 |
| NMRILA            | R                  | R*                 | R                  | S*                 | S                  |
| NMRILX            | R                  | S                  | R                  | R                  | S                  |
| NMRILB            | S                  | R                  | R                  | R                  | R                  |
| NMRILK            | R                  | R                  | S                  | R                  | R                  |

*Race designation according to Sy et al. (2008).*

*C. annuum* New Mexico recombinant inbred line.

*R = resistant phenotype: no lesions on roots. Scoring based on Bosland and Lindsey (1991) disease scale symptoms.

*S = susceptible phenotype: ranging from small lesions on roots to death of plants. Scoring based on Bosland and Lindsey (1991) disease scale symptoms.
susceptible phenotype (see phenotypic reaction of NMRILs in Table 2). The NMRILs’ phenotypic reactions obtained in this study agreed with the results obtained by Sy et al. (2008). The F₁ populations from all the hybridizations displayed complete resistance when a single race was used for inoculation (Table 2).

When the F₂ populations were inoculated with a single race, segregation ratios of three resistant:one susceptible were found in all the hybridization with the exception of NMRIL₉ × NMRIL₆ inoculated with race 4 (Table 2). In this case, the hypothesis was rejected (Table 2).

When the F₂ populations from the hybridizations NMRIL₄ × NMRIL₆ and NMRIL₉ × NMRIL₆ inoculated with the combination of two races, segregation ratios of 15 resistant:one susceptible were observed [i.e., races 1 + 5 and 4 + 6 (Table 2)]. On the contrary, when the F₂ populations from the hybridization NMRIL₉ × NMRIL₆ were inoculated with the combination of two races [i.e., isolates 1 + 4 and 1 + 12], segregation ratios of 15 resistant:one susceptible were rejected (P < 0.0001) segregation ratio in the F₂ population of the hybridization NMRIL₉ × NMRIL₆ under single inoculation of race 4 suggested a linkage phenomenon, in which linkage affects the frequencies of various gene combinations (Chahal and Gosal, 2002).

When the F₂ populations were inoculated with a combination of two races, segregation for susceptible individuals was observed. In many cases, a single R gene can provide complete resistance to one or more strains of particular pathogen (McDowell and Woffenden, 2003). Thus, the absolute absence of segregation for susceptibility indicates that the genes share a common locus with other alleles (Chen et al., 2001). On the other hand, the rejection of the 3:1

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**Table 2. Phenotypic response of Capsicum annuum New Mexico recombinant inbred lines (NMRILs) to Phytophthora capsici root rot resistance in parent, F₁ progeny, and F₂ populations.**

| Race | Populations | Plants (no.) | Expected ratio (R:S) | χ² | P value |
|------|-------------|--------------|----------------------|-----|---------|
| 1    | NMRIL₁      | 0 72         | 0:72                 | N/A | N/A     |
|      | NMRIL₆      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₉      | 72 0         | 72.0                 | N/A | N/A     |
|      | (B × K) F₁  | 12 0         | 12.0                 | N/A | N/A     |
|      | (B × X) F₁  | 10 0         | 10.0                 | N/A | N/A     |
|      | (B × K) F₂  | 44 17        | 3:1                  | 0.27| 0.60    |
|      | (B × X) F₂  | 149 60       | 3:1                  | 1.50| 0.22    |
| 4    | NMRIL₄      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₆      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₉      | 0 72         | 0:72                 | N/A | N/A     |
|      | (A × X) F₁  | 28 0         | 28.0                 | N/A | N/A     |
|      | (B × X) F₁  | 10 0         | 10.0                 | N/A | N/A     |
|      | (A × X) F₂  | 85 21        | 3:1                  | 0.52| 0.22    |
|      | (B × X) F₂  | 188 15       | 3:1                  | 33.58| 0.0001 |
| 5    | NMRIL₄      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₆      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₉      | 0 72         | 0:72                 | N/A | N/A     |
|      | (B × K) F₁  | 9 0          | 9.0                  | N/A | N/A     |
|      | (B × K) F₂  | 207 59       | 3:1                  | 1.13| 0.29    |
| 6    | NMRIL₄      | 0 72         | 0:72                 | N/A | N/A     |
|      | NMRIL₆      | 72 0         | 72.0                 | N/A | N/A     |
|      | (A × X) F₁  | 23 0         | 23.0                 | N/A | N/A     |
|      | (A × X) F₂  | 90 27        | 3:1                  | 0.23| 0.63    |
| 12   | NMRIL₉      | 72 0         | 72.0                 | N/A | N/A     |
|      | NMRIL₆      | 0 72         | 0:72                 | N/A | N/A     |
|      | (B × X) F₁  | 25 0         | 25.0                 | N/A | N/A     |
|      | (B × X) F₂  | 105 41       | 3:1                  | 0.74| 0.39    |
| 4 + 6| (B × X) F₂  | 207 44       | 15:1                 | 126.06| <0.0001 |
|      | (B × X) F₂  | 160 13       | 15:1                 | 0.47| 0.49    |
|      | (B × X) F₂  | 352 84       | 15:1                 | 54.55| <0.0001 |
|      | (B × X) F₂  | 569 42       | 15:1                 | 0.21| 0.52    |

*Phytophthora capsici* races (inoculum concentration = 10,000 zoospores/plastic tray-cell).
*C. annuum* New Mexico recombinant inbred line.
α = 0.05 was used for evaluation.
Not applicable.
Combination of two races (final inoculum concentration = 10,000 zoospores/plastic tray-cell).

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**Discussion**

The resistance guided by a gene-for-gene relationship is also called race-specific resistance, in which the activation of the resistance depends on specific recognition on the invading pathogen by the plant (Keen, 1990). When the F₂ populations were inoculated with a single race, segregation ratios of three resistant:one susceptible were observed in the majority of the hybridizations, confirming that the resistant phenotypes are determined by a single dominant gene for each *P. capsici* race as reported by Sy et al. (2005) and Walker and Bosland (1999). Thus, the gene-for-gene relationship between *C. annuum* and *P. capsici* for root rot resistance was confirmed in this study, and one specific *R* gene was required for each *P. capsici* race to trigger a resistant reaction in the NMRILs.

The recognition of race-specific resistance in the *P. capsici–C. annuum* interaction will aid plant breeders in selecting appropriate resistant cultivars for future hybridizations (Sy et al., 2008; van de Weg, 1997). Because different geographical regions may have different races of *P. capsici*, plant breeders may need to breed for a specific production region, pyramiding a number of specific genes to confer resistance into a cultivar (Acquaah, 2007). On the other hand, the rejection of the 3:1 segregation ratio in the F₂ population of the hybridization NMRIL₉ × NMRIL₆ under single inoculation of race 4 suggested a linkage phenomenon, in which linkage affects the frequencies of various gene combinations (Chahal and Gosal, 2002).

When the F₂ populations were inoculated with a combination of two races, segregation for susceptible individuals was observed. In many cases, a single *R* gene can provide complete resistance to one or more strains of particular pathogen (McDowell and Woffenden, 2003). Thus, the absolute absence of segregation for susceptibility indicates that the genes share a common locus with other alleles (Chen et al., 2001). On the other hand, the presence of susceptible individuals in the F₂ populations is strong evidence that the genes involved in controlling the resistance in this study are not allelic (Boiteux, 1995). The segregation ratio of 15 resistant:one susceptible in the hybridization NMRIL₉ × NMRIL₆ against combination of races 1 + 5 and NMRIL₆ × NMRIL₉ against races 4 + 6 revealed the presence of two dominant independent *R* genes.
located at different loci (Ma et al., 2002; Zhen et al., 2006), each one providing resistance to a different P. capsici race. The segregation ratio of 15 resistant:one susceptible individuals in the F2 population indicates that the resistant genes act independently in the recognition of each specific P. capsici race. Digenic segregation was expected in the hybridizations evaluated as a result of differential responses of the NMRILs toward the different P. capsici isolates used. For the hybridization NMRIL B × NMRIL X against races 1 + 4 and races 1 + 12, the appearance of susceptible individuals in the F2 population is proof of two loci. However, the rejection of a 15:1 ratio suggests that the two loci are linked for the R genes of those specific races. Linkage is defined as the tendency of various genes to be inherited as a block because of being situated on the same chromosomal region (Chahal and Gosal, 2002). Several genetic and molecular studies have shown that plant pathogen-specific R genes are frequently linked within genome regions of various sizes (Grube et al., 2000; Huang et al., 2004; Moreau et al., 1998).

The results reported here for the R genes for races 1, 4, 5, 6, and 12 indicate they are located at different loci. However, resistant genes for races 1 and 4 are linked. Possible linkage was observed also between R genes for races 1 and 12. Other studies in chile pepper have shown the presence of linkage between different R genes. For instance, Yeam et al. (2005) reported for potyvirus resistance the presence of two dominant alleles that were tightly linked but clearly distinct.

The five R genes evaluated in this study segregated independently, indicating different loci for each resistant gene. Screening more P. capsici isolates, different from the five isolates used in this study, could display evidence of allelism for different resistant genes as was reported by Thabuis et al. (2004).

In the results reported here, all populations displayed segregation; thus, no evidence of allelism was observed. The presence of susceptible individuals in all of the F2 populations indicates that at least five loci for R genes to phytophthora root rot are present in the C. annuum genome. Having several loci for phytophthora root rot resistance will enable plant breeders to pyramid resistant alleles in Capsicum L. Pyramiding of genes involves the accumulation of several R genes into the same line to create a multiple gene-resistant cultivar (Chahal and Gosal, 2002). Because Capsicum is a diploid plant, it is only possible to introduce two resistant alleles per locus. Thus, the information obtained in this study will assist in introducing several phytophthora root rot R genes into chile pepper cultivars.

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