Disease Resistance to Multiple Fungal and Oomycete Pathogens Evaluated Using a Recombinant Inbred Line Population in Pepper

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ABSTRACT

Incorporating disease resistance into cultivars is a primary focus of modern breeding programs. Resistance to pathogens is often introgressed from landrace or wild individuals with poor fruit quality into commercial-quality cultivars. Sites of multiple disease resistance (MDR) are regions or “hot spots” of the genome with closely linked genes for resistance to different pathogens that could enable rapid incorporation of resistance. An F₂-derived F₆ recombinant inbred line population from a cross between ‘Criollo de Morelos 334’ (CMM334) and ‘Early Jalapeno’ was evaluated in inoculated fruit studies for susceptibility to oomycete and fungal pathogens: *Phytophthora capsici*, *P. nicotianae*, *Botrytis cinerea*, *Fusarium oxysporum*, *F. solani*, *Sclerotinia sclerotiorum*, *Alternaria* spp., *Rhizopus oryzae*, *R. stolonifer*, and *Colletotrichum acutatum*. All isolates evaluated were virulent on pepper. Significant differences in disease

susceptibility were identified among lines for each of the pathogens evaluated. *P. capsici* was the most virulent pathogen, while *R. oryzae* and one *Sclerotinia* isolate were the least virulent. Quantitative trait loci associated with resistance were identified for *Alternaria* spp. and *S. sclerotiorum*. Positive correlations in disease incidence were detected between *Alternaria* spp. and *F. oxysporum*, *F. solani*, and *C. acutatum*, as well as between *C. acutatum* and *Botrytis* spp., *F. oxysporum*, *F. solani*, and *P. capsici*. No sites of MDR were identified for pathogens tested; however, positive correlations in disease incidence were detected among pathogens suggesting there may be genetic linkage among resistance genes in CM334 and Early Jalapeno.

Additional keywords: *Capsicum annuum*, host resistance.

Pepper (*Capsicum annuum*) is the third most important solanaceous crop in the United States and the fourth most important in the world. In the United States, approximately \$78 million of bell and chili pepper fruit were exported in 2016 (USDA Economic Research Service 2017). Worldwide, nearly 200 million and 33 million tons of green and dry pepper fruit, respectively, are produced annually. Plant pathogens (bacterial, fungal, and oomycete) cost pepper growers millions of dollars annually through indirect and direct expenses, including pesticide and antibiotic use, increased labor, and reduced yields and quality. Resistant varieties and improved cultural practices have been highly successful at reducing the prevalence of diseases caused by some bacterial pathogens in the United States (Pernezny et al. 2003). Pre- and postharvest diseases of pepper caused by fungal and oomycete pathogens can still result in devastating losses for individual growers (Pernezny et al. 2003). Common diseases of pepper include major and minor fungal diseases such as white mold (*Sclerotinia* spp.), Fusarium wilt and rot (*Fusarium* spp.), anthracnose rot (*Colletotrichum* spp.), Rhizopus rot (*Rhizopus* spp.), gray mold (*Botrytis cinerea*), and *Alternaria* rot (*Alternaria* spp.); and major oomycete diseases such as Phytophthora rot (*Phytophthora* spp.) (Anand et al. 2009; Kamara et al. 2016; Le et al. 2013; Pernezny et al. 2003; Singh et al. 2014; Zitter 2000). Major diseases caused by pathogens such as *Phytophthora capsici* can result in complete crop failure when left unmanaged (Granke et al. 2012). Minor pathogens cause fewer losses but still require chemical or cultural practices to

mitigate losses (Frans et al. in press; Tzortzakis and Chrysargyris 2017). The impact of these individual pathogens varies greatly among regions but each is responsible for pre- and postharvest loss of pepper.

Host resistance is frequently incorporated into cultivated species from related wild and weedy species. These sources often have undesirable commercial qualities such as indeterminate growth, poor fruit quality, uneven ripening, and small fruit size (Acquaah 2012; Khan et al. 2014). Resistance from wild and landrace pepper has been used to incorporate *Phytophthora* root rot, bacterial spot, *Tobacco etch virus*, *Cucumber mosaic virus*, and *Verticillium* wilt resistance into commercial varieties (Hibberd and Gillespie 1982; Hibberd et al. 1987; Holdsworth and Mazourek 2015; Padgett et al. 1990). To incorporate only traits of interest, plants must undergo intensive phenotypic selection to minimize negative attributes. Studies have shown that disease resistance can have linkage or pleiotropic effects on commercial traits such as yield or fruit shape (Cheng 2014; Haggard et al. 2013; Naegele et al. 2014). In addition to poor fruit-quality traits, it is possible for undesirable disease susceptibility traits to be transferred from the wild background into the cultivated pepper.

In pepper, ‘Criollo de Morelos 334’ (CM334), a landrace from Mexico, is the main source of *P. capsici* resistance used for developing commercial cultivars. Resistance to *P. capsici* is one of the most genetically mapped pathosystems in pepper. For fruit, root, and foliar resistance, the major quantitative trait loci (QTL) were localized on pepper chromosome 5 (Mallard et al. 2013; Naegele et al. 2014; Ogundiwin et al. 2005; Rehrig et al. 2014; Sy et al. 2008; Walker and Bosland 1999). Other *Phytophthora* spp. such as *P. nicotianae* can also infect pepper, and QTL for root rot resistance have been identified on pepper chromosomes 3, 9, and 11 (Bonnet et al. 2007). Resistance to anthracnose, caused by *Colletotrichum*

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gleosporoides and *C. capsici*, has also been mapped in pepper. One to three QTL were identified, depending on the *Colletotrichum* sp. (Kim et al. 2008; Voorrips et al. 2004). A single recessive gene was determined to control resistance in the pepper species *Capsicum chinense*. In these anthracnose studies, QTL were identified though not associated with chromosomes. However, in a recent study, resistance QTL for *Colletotrichum acutatum* were identified on pepper chromosome 5 (Sun et al. 2015).

In sunflower, soybean, and rapeseed, QTL for resistance to *Sclerotinia sclerotiorum* have been identified, suggesting that it is a multigenic resistance (Arahana et al. 2001; Mestries et al. 1998; Micic et al. 2005; Zhao and Meng 2002). Evaluation of pepper cultivars and germplasm identified sources of resistance to *S. sclerotiorum* but, to date, no QTL studies have been performed (Yanar and Miller 2003). Resistance to *Botrytis* spp. has been identified in tomato on chromosomes 3, 4, and 9 (Finkers et al. 2007a). However, no studies have identified QTL for resistance to *Botrytis* spp. in pepper. Similarly, no information on QTL contributing to resistance to *Alternaria* spp., *Fusarium oxysporum*, *Rhizopus stolonifer*, *R. oryzae*, or *F. solani* is currently available for pepper.

As genetic maps and genomic resources have advanced, clusters or “hot spots” of traits have been identified in plants (Frary et al. 2014; Quirin et al. 2012; Thabuis et al. 2003). In pepper, chromosome 5 has been identified as a hot spot for *Phytophthora* spp. resistance, as well as resistance to *C. acutatum*. Among potato, tomato, and pepper, Grube et al. (2000) demonstrated that regions of shared resistance exist in the family Solanaceae. In tomato and potato, colocalization of QTL for *P. infestans* have been identified between the two species, suggesting that homologous regions of resistance exist among solanaceous species (Brouwer et al. 2004). Few colocalization studies on disease resistance have been done in the family Solanaceae, and no studies have examined resistance across fungal and oomycete pathogens. In pepper, colocalization among virus resistance QTL were determined, suggesting that regions of multipathogen resistance exist, and that the viruses were controlled by the similar genetic regions (Caranta et al. 1997). In grasses, regions of multiple disease resistance (MDR) have been identified for corn (southern leaf blight, gray leaf spot, and northern leaf blight) and rye (gray leaf spot, crown rust, leaf spot, and stem rust) (Jo et al. 2008; Zwonitzer et al. 2010). When present, identifying regions of MDR can improve our understanding of disease resistance and facilitate breeding of commercial cultivars with enhanced resistance.

The objectives of this research were to (i) evaluate resistance, (ii) evaluate likelihood of infection in pepper to oomycete and fungal pathogens, and (iii) identify QTL associated with host resistance.

MATERIALS AND METHODS

Plant material and isolate selection. A set of 63 recombinant inbred lines (RIL) (an F₂-derived F₆) was used for all evaluations. The RIL, developed by Dr. Paul Bosland, were derived from hybridization of ‘Early Jalapeno’ (susceptible to *P. capsici*) and CM334 (resistant to *P. capsici*) to study resistance to *Phytophthora* root rot (Walker and Bosland 1999). Plants were grown in a glasshouse at the Michigan State University Plant Sciences Greenhouses in East Lansing in 1-gal. pots containing Suremix Perlite (Michigan Grower Products, Inc., Galesburg, MI).

In total, nine fungal and five oomycete isolates were collected from diseased plant material in Michigan or were donated by colleagues (Table 1). Isolates were inoculated onto and reisolated from jalapeño pepper fruit prior to fruit screens to ensure pathogenicity and promote virulence. Fungal isolates were maintained on potato dextrose agar (PDA) (39 g of PDA and 1 liter of distilled water) and grown under constant fluorescent light at room temperature. Oomycete isolates were maintained on unclarified V8 agar (UCV8) (16 g of agar, 30 mM CaCO₃, 160 ml of unfiltered V8 juice, and 840 ml of distilled water) and grown under constant fluorescent light at room temperature. *P. capsici* isolates were previously evaluated on the pepper population (Naegele et al. 2014).

Fruit inoculations and evaluations. Immature green pepper fruit (2 to 4 cm in length) were surface disinfested for 5 min in a 10% sodium hypochlorite solution, rinsed with distilled water, and air dried. Pepper fruit were inoculated with a single isolate or negative control and incubated in clear polystyrene boxes with lids (23 by 10 by 32 cm; Potomac Display, Hampstead, MD), which contained wet paper towels to maintain high relative humidity. Pepper fruit were incubated under constant fluorescent lighting at room temperature (approximately 25 ± 2°C) for 1 week, except for pepper fruit inoculated with *Rhizopus* spp., which were incubated for 4 days, and pepper fruit inoculated with *P. nicotianae* or *P. capsici*, which were incubated for 5 days. At the end of the incubation period, pepper fruit were rated for symptoms of fruit rot and pathogen sporulation on the fruit. Fruit that showed symptoms of disease or pathogen signs were rated as susceptible (= 1) and fruit showing no symptoms or signs at the end of the incubation period were rated as resistant (= 0). Five replicate fruit were used for each experiment and the experiment was conducted two times for each fungal pathogen. Isolations were performed on symptomatic and asymptomatic fruit to confirm the presence or absence of the pathogen if no active sporulation was observed. For isolations, the skin was peeled back using a scalpel, and three 1-mm sections of pepper fruit were plated onto a single plate of PDA for fungi or UCV8 amended with ampicillin and rifampicin for oomycetes and grown under constant fluorescent light in the lab at 25°C. The

TABLE 1. Fungal and oomycete isolates used in this study^z

Fungal species	Host of origin	Identifier	Source
<i>Alternaria</i> spp.	Eggplant	None	L. L. Granke
<i>Botrytis cinerea</i>	Geranium	None	B. R. Harlan
<i>Colletotrichum acutatum</i>	Tomato	None	L. M. Rodriguez-Salamanca
<i>Fusarium oxysporum</i>	Potato	None	E. Gachango
<i>F. solani</i>	Potato	None	E. Gachango
<i>Sclerotinia sclerotiorum</i>	Soil	Decatur	J. J. Hao
<i>S. sclerotiorum</i>	Soybean	Clarksville	J. J. Hao
<i>Rhizopus oryzae</i>	Sugarbeet	Rp08-1	L. E. Hanson
<i>R. stolonifer</i>	Sugarbeet	Rp07-1	L. E. Hanson
<i>Phytophthora capsici</i>	Pepper	12889	M. K. Hausbeck
<i>P. capsici</i>	Cucumber	OP97	M. K. Hausbeck
<i>P. capsici</i>	Bean	13709	M. K. Hausbeck
<i>P. nicotianae</i>	Water	1006	C. D. Smart
<i>P. nicotianae</i>	Water	1007	C. D. Smart

^z All isolates were collected from naturally infected plant material or soil in Michigan or surface water in New York by the authors or colleagues at Michigan State University and Cornell University.

pathogen was confirmed based on morphological characteristics. The experiment was a complete randomized design blocked by isolate. Due to limited fruit, three replicate fruit were used for each experiment and the experiment was conducted three times for each *P. nicotianae* species.

For *Alternaria* spp. and *B. cinerea*, a 7-day-old culture on PDA was flooded with sterile distilled water with 0.1% Tween 80. The resulting suspension was adjusted to 10^5 conidia/ml for *Alternaria* spp. and 10^6 conidia/ml for *B. cinerea*. For *Alternaria* spp. inoculations, each fruit was wounded in the center of the fruit using a pipette tip (200- μ l size), and 10 μ l of spore suspension or double-distilled (dd)H₂O (negative control) was pipetted directly over the wound. For *B. cinerea* inoculation, each pepper was gently wounded by lightly passing a scalpel across the stem scar, and 50 μ l of spore suspension or ddH₂O (negative control) was pipetted onto the wounded stem scar.

For *C. acutatum*, *F. solani*, and *F. oxysporum*, sterile distilled water was added to a 14-day-old culture on PDA. The resulting spore suspension was adjusted to 5×10^6 conidia/ml for *C. acutatum* and 10^6 conidia/ml for *Fusarium* spp. For *C. acutatum* and *F. oxysporum*, fruit were inoculated in the same manner as described above for *Alternaria* spp. For *F. solani*, fruit were wounded as described above for *B. cinerea*, and 10 μ l of spore suspension or ddH₂O (negative control) was pipetted onto the wounded stem scar.

For *Rhizopus* spp., a cork borer was used to remove a 7-mm-diameter agar plug from a 7-day-old culture growing on PDA. The agar plug was placed in the center of the pepper fruit. For *S. sclerotiorum* and *Phytophthora* spp., fruit were inoculated as described above for *Rhizopus* spp., except each PDA or UCV8 agar plug was covered with a 12-mm-diameter screw cap (Axygen Scientific, Union City, CA) that was affixed to the fruit surface using petroleum jelly. Negative controls (agar only) were included and inoculated as described above.

Genotyping. Each pepper line had previously been genotyped using single-position polymorphism detection coupled with the Affymetrix GeneChip array developed for pepper (Hill et al. 2013). The chip consisted of 6,473,556 genomic tiling probes representing 31,196 unigene sequences, with a 2-bp overlap for each probe (Hill et al. 2013).

Data analysis. Disease incidence (number of fruit displaying symptoms out of the total number of fruit) was calculated for each line and pathogen. Significant differences were detected among lines with the PROC LOGISTIC procedure in SAS statistical software (v9.3; SAS Institute, Cary, NC) using a reference parameter and the descending option. Pathogens were compared with *R. oryzae* and lines were compared with the cultivated parent Early Jalapeno. Mean infection values of each pathogen were calculated using a random effects model in PROC GLIMMIX. Then, the odds ratio of a line being infected by a particular pathogen, as compared with the average infection rate among lines by that pathogen, were obtained for each line using Wald square limits at a 95% confidence interval. This provided a measure of the risk of infection of a line by a particular pathogen, as compared with the average infection rate for that pathogen. Linkage analyses were performed using R/qtl software (Broman et al. 2003; R Development Core Team 2012) in R v3.0 on the pepper genetic map previously published and evaluated for quality control (Hill et al. 2013; Naegele et al. 2014). QTL were mapped using incidence data and disease threshold data. When mapping disease threshold, if disease incidence was $\geq 50\%$ based on the total number of pepper fruit evaluated, the individual line would receive a disease score of 1 and, if $< 50\%$, the line would receive a disease score of 0. Missing data were filled using the imputation method for incidence data and entropy method for disease threshold data. Putative QTL were detected using the scanone method implemented in R with the binary model for disease threshold data with the EM algorithm method. For disease incidence, QTL were detected using the multiple imputation method. Significance of loci was determined using logarithm of odds (LOD) thresholds calculated from 1,000 permutations for each pathogen. Correlations among pathogens in disease incidence were analyzed using the PROC CORR feature implemented in SAS.

In the CM334 $\times$ Early Jalapeno population, disease resistance for each pathogen evaluated varied among lines, and significant differences were detected (Fig. 1). Of the eight fungal pathogen species evaluated, a *Sclerotinia* sp. (Decatur isolate) and *R. oryzae* caused the least amount of disease on pepper fruit. Infection by *P. capsici* isolates resulted in the greatest number of lines that had a significantly higher likelihood of disease than the population mean.

The odds ratio (the odds that disease will occur given exposure to a particular variable) for pathogen ($P = 0.0037$) and the interaction between line and pathogen ($P = 0.0123$) were both significant. CM334 had a reduced incidence of fruit rot caused by *P. capsici* and *P. nicotianae*, whereas Early Jalapeno had high disease incidence, consistent with previous studies (Table 2) (Naegele et al. 2014; Sy et al. 2008). However, Early Jalapeno had low disease incidence when inoculated with *Alternaria* spp., *C. acutatum*, *F. solani*, *R. stolonifer*, and *R. oryzae*, whereas CM334 was susceptible to these pathogens (Fig. 2). Neither parent was susceptible to *Sclerotinia* spp. (Clarksville or Decatur isolates), although resistance segregated within the progeny and four lines had disease incidence $\geq 50\%$. In addition, both parents were moderately susceptible to *Botrytis* spp. and *F. oxysporum* but the progeny segregated for resistance. CM334, although resistant to *Phytophthora* spp., was not significantly more resistant to other pathogens evaluated in this study compared with the cultivated parent Early Jalapeno. In some instances, CM334 had increased susceptibility compared with Early Jalapeno.

When pepper lines were compared across all pathogens, C, M, K, B, AAS, and AA had the greatest number of pathogens (≥ 5) to which they significantly differed in susceptibility compared with the population mean. When individual pathogens were evaluated, large differences were detected in line susceptibility and mean disease incidence.

When inoculated with a *Sclerotinia* sp. (Decatur isolate), the two pepper parents were not different and had low disease incidence (0 to 10%). Infection within the progeny remained low, with line AF having the highest incidence (60%) and 22 lines exhibiting no symptoms (Table 2). The population mean was $16.5 \pm 16.3\%$. The likelihood of infection was significantly more probable for only two lines (G and N) as compared with the population average. The LOD threshold for disease incidence was determined to be 3.03 (10%) and 3.33 (5%). A significant QTL was identified on chromosome 11 (CAPS_CONTIG.6117) at position 90 (LOD = 3.38). The QTL explained 22% of the variation observed. When disease incidence was converted to a disease threshold, no QTL were detected above the 10% LOD threshold (1.44). The genetic map used to identify QTL in the pepper population was previously described in detail (Hill et al. 2013).

When inoculated with the *Sclerotinia* isolate from Clarksville, the two pepper parents were not significantly different in the likelihood of infection, and disease incidence was low (10 and 20%). Likelihood of infection was significantly greater in four lines compared with the population average. Line AAB had the highest incidence (80%) when infected with the *Sclerotinia* isolate (Clarksville), whereas 14 lines did not become diseased (Table 2). However, multiple lines with low to moderate disease incidence when inoculated with a *Sclerotinia* sp. (Decatur) had high disease incidence when inoculated with isolate Clarksville or vice versa. The population mean was $22 \pm 20\%$. No QTL were observed for disease incidence above the 10% LOD threshold (2.92).

When inoculated with *R. oryzae* or *R. stolonifer*, the two pepper parents differed in disease incidence, with 10% (Early Jalapeno) and 100% (CM334) of fruit infected. Line differences were detected between *Rhizopus* spp., suggesting that resistance is species, if not isolate, specific. For *R. oryzae*, the likelihood of infection was higher for seven lines than the population average (Table 3). Line AC had the highest disease incidence (90%) when infected with *R. oryzae*, whereas 35 lines did not become diseased (Table 2). The population

mean was $10.3 \pm 16.9\%$. The LOD threshold for pathogen incidence was determined to be 2.40 (10%) and 2.66 (5%). No QTL were observed for disease incidence above the LOD threshold for disease incidence or disease threshold.

Line AU had the highest incidence (100%) when infected with *R. stolonifer*, whereas 13 lines did not display disease symptoms (Table 2). Likelihood of infection was significantly increased for lines J, AG, AR, AT, AU, AZ, and CM334 compared with the population average. The population mean was $24.6 \pm 22.6\%$. The LOD threshold for pathogen incidence was determined to be 2.81 (10%) and 3.10 (5%). No QTL were observed for disease incidence above the LOD threshold.

When inoculated with *F. oxysporum*, neither of the pepper parents had a high disease incidence (10% Early Jalapeno and 30% CM334). Seven lines had a significantly greater likelihood of infection compared with the population average (Table 3). Line D had the highest incidence (100%) when infected with *F. oxysporum*, whereas 12 lines did not become diseased (Table 2). The population mean was $25.5 \pm 23.5\%$. LOD threshold for disease incidence was determined to be 2.94 (10%) and 3.20 (5%). No QTL were observed for disease incidence or disease threshold above the LOD threshold.

When inoculated with *F. solani*, the two pepper parents differed in the likelihood of infection and varied in disease incidence, with 0% for Early Jalapeno and 70% for CM334. The likelihood of infection was significantly greater than the population average for eight lines and significantly less for eight (Table 3). Lines AM and AAE had the highest incidence (100%) when infected with *F. solani*, whereas six lines had no disease incidence (Table 2). The population mean was $42.6 \pm 29.1\%$. The LOD threshold for disease incidence was determined to be 3.04 (10%) and 3.45 (5%). No QTL were observed for disease incidence or disease threshold above the LOD threshold.

When inoculated with *C. acutatum*, the two pepper parents differed in disease incidence (70% for CM334 and 20% for Early Jalapeno). When compared with the population average, 7 lines had a significantly higher likelihood of infection and 10 lines had a significantly lower incidence (Table 3). Line AP had the highest incidence (100%) when infected with *C. acutatum*, whereas nine lines did not become diseased (Table 2). The population mean was

$40.9 \pm 28.7\%$. The LOD threshold was determined to be 3.02 (10%) and 3.38 (5%). No QTL were observed for disease incidence or disease threshold above the LOD threshold.

When inoculated with *Botrytis* spp., the two pepper parents differed in the likelihood of infection compared with the population average, despite minimal differences in incidence (50% for CM334 and 60% for Early Jalapeno). When compared with the population average, 8 lines had a significantly higher likelihood of infection and 10 had a lower likelihood (Table 2). Lines C and D had the highest incidence (100%) when infected with *Botrytis* spp. while line J had the lowest disease incidence (10%) (Table 2). The population mean was $60.8 \pm 20.1\%$. The LOD threshold was determined to be 2.93 (10%) and 3.38 (5%) for disease incidence. Only 7 of the 63 lines evaluated had a disease incidence $\leq 30\%$. No QTL were observed for disease incidence or disease threshold above the LOD threshold with visual separation of the disease phenotype.

When inoculated with *Alternaria* spp., the two pepper parents were different in disease incidence (0% for Early Jalapeno and 90% for CM334). Only two lines (AAC and J) had a significantly lower likelihood of infection than the population average. Seven lines had 100% disease incidence when infected with *Alternaria* spp. whereas lines AAG, AS, AP, AA, J, and I had no disease (Table 2). The population mean was $51.6 \pm 30.1\%$. The LOD threshold was determined to be 3.11 (10%) and 3.52 (5%). A single QTL was detected on chromosome 2 (LOD = 5.92, $P < 0.001$) located at position 63 centimorgans (cM) (marker ANOK.CO776125). When disease incidence was converted to a disease threshold, the LOD threshold was 2.95 (10%) and 3.24 (5%). The marker identified for disease incidence was redetected (Table 4).

When inoculated with *P. nicotianae* (isolate 1006), the two pepper parents differed in disease incidence (38% CM334 and 63% Early Jalapeno). Only line N had a significantly higher likelihood of infection compared with the population mean. Two lines had 100% disease incidence when infected with *P. nicotianae* (1006), whereas lines AAB, AQ, and AX had no disease symptoms (Table 2). The population mean incidence was $49.4 \pm 25.8\%$. The LOD threshold was determined to be 2.98 (10%) and 3.32 (5%). No QTL were

Fig 1. Variation in disease symptoms on pepper fruit 4 or 7 days postinoculation with a single isolate of *Alternaria* spp. on **A**, CM334 and **B**, Early Jalapeno; *Colletotrichum acutatum* on **C**, Early Jalapeno and **D**, AZ; or *Fusarium solani* on **E**, AAK and **F**, AF.

TABLE 2. Disease incidence of 14 isolates on a recombinant inbred line pepper population

Line	Pathogen													
								<i>Sclerotinia</i> sp.		<i>Phytophthora capsici</i>			<i>P. nicotianae</i>	
	<i>Alr</i> ^d	<i>Bot</i> ^r	<i>Col</i> ^s	<i>F.s</i> ^t	<i>F.o</i> ^u	<i>Rso</i> ^v	<i>Rss</i> ^w	Clk ^x	Dec ^y	12889	13709	OP97	1006	1007
A	40	70	56	63	0	0	0	0	10	100	100	100	88	100
AA	0	60	90	10	20	20	30	40	10	100	89	89	13	50
AAA	70	50	50	50	50	0	40	10	0	100	89	100	50	38
AAB	10	50	0	80	0	0	30	80	20	89	56	89	0	0
AAC	80	20	30	50	0	0	20	30	0	100	78	100	63	75
AAD	70	50	20	60	20	20	40	40	10	100	100	100	100	75
AAE	20	50	50	100	10	0	40	0	0	100	100	100	88	88
AAF	30	90	70	60	10	0	10	10	20	100	100	100	63	38
AAG	0	80	20	40	30	0	10	0	30	67	78	67	50	38
AAJ	50	60	60	30	10	20	0	20	0	100	100	100	63	75
AAK	30	30	0	0	0	0	10	10	10	89	100	89	13	25
AAM	30	80	50	10	10	0	0	50	20	100	89	100	75	25
AAN	30	50	30	10	0	0	0	50	20	78	56	100	63	75
AAO	60	60	50	30	0	10	0	10	40	100	100	100	63	63
AAP	100	70	90	90	70	0	30	70	40	100	100	100	75	75
AAQ	70	70	30	60	20	0	0	10	30	100	100	100	88	75
AAR	50	80	10	20	20	0	40	30	20	89	89	100	25	13
AAU	20	60	40	20	30	0	10	10	10	89	78	100	13	13
AB	50	90	70	80	10	0	10	20	20	100	100	100	63	75
AC	50	20	0	0	0	90	30	20	0	100	89	89	50	50
AD	100	30	50	50	60	0	30	70	20	89	89	100	88	100
AE	50	40	0	40	30	10	30	10	10	100	100	78	38	50
AF	30	50	30	40	0	0	20	50	60	100	100	100	63	75
AG	30	80	30	90	30	30	60	40	0	89	67	100	38	38
AI	60	90	50	30	0	0	20	10	40	100	100	100	75	63
AJ	80	60	50	60	30	60	40	10	0	78	89	89	88	75
AK	90	60	20	50	40	0	10	0	30	100	89	100	50	50
AL	100	60	30	60	30	10	10	20	40	78	67	100	88	38
AM	70	80	50	100	30	50	50	0	10	100	100	100	63	100
AN	80	60	40	70	70	20	40	0	40	100	100	100	63	63
AO	50	40	60	90	40	0	10	40	0	100	100	100	25	25
AP	0	70	100	20	40	0	50	40	20	100	100	100	63	38
AQ	80	80	20	20	60	10	0	0	10	89	78	100	0	25
AR	70	50	70	70	50	20	80	0	0	100	100	100	13	25
AS	0	60	30	20	0	0	0	0	40	89	100	100	50	50
AT	10	90	40	50	0	10	60	50	30	100	89	89	25	13
AU	100	80	90	50	60	30	100	10	0	100	78	100	50	25
AV	30	60	60	50	10	10	40	50	10	100	89	100	63	88
AW	30	50	10	30	10	10	30	0	0	78	100	78	25	50
AX	40	80	70	60	60	20	50	0	10	100	89	100	0	38
AY	70	60	50	30	30	0	20	10	0	89	100	100	50	63
AZ	80	50	60	0	10	10	80	40	20	100	89	89	75	63
B	50	60	50	40	50	10	0	0	30	89	100	100	63	88
C	70	100	80	40	20	20	20	10	0	100	56	89	50	50
CM334	90	50	70	70	30	100	100	20	10	100	89	78	38	50
D	100	100	100	90	90	20	10	50	10	100	100	100	25	100
E	60	90	80	10	10	10	10	30	40	100	100	100	88	63
EJ ^z	0	60	20	0	10	10	10	10	0	100	100	78	63	88
F	40	70	0	100	10	0	20	50	30	100	100	100	38	88
G	80	60	30	70	30	10	30	10	50	100	100	100	38	25
H	70	60	0	10	29	0	10	29	0	100	100	100	...	...
I	0	30	10	10	0	0	20	20	20	89	89	100	63	13
J	0	10	0	20	10	0	60	20	0	100	100	85	38	63
K	100	80	70	20	60	40	30	10	0	89	89	89	25	38
L	50	50	10	10	20	0	20	20	0	100	89	100	50	75
M	90	80	40	50	20	10	0	30	40	100	89	100	38	50
N	100	40	90	90	90	0	0	20	50	67	89	100	100	50
O	60	50	0	20	10	0	10	10	0	89	89	100	25	38
P	30	50	30	0	20	0	0	30	10	100	100	100	50	50
R	30	30	0	50	20	40	30	0	10	100	89	100	13	25
S	70	50	30	30	40	0	10	20	40	100	100	100	50	50
T	50	90	10	0	40	0	0	40	0	100	89	100	63	25
V	30	60	70	30	10	30	60	20	0	100	78	100	63	38
X	10	50	10	50	20	0	10	10	0	100	89	100	25	25
Z	50	50	40	0	10	0	20	0	10	78	67	100	13	13

^d *Alternaria* sp.^r *Botrytis* sp.^s *Colletotrichum acutatum*.^t *Fusarium solani*.^u *F. oxysporum*.^v *Rhizopus oryzae*.^w *R. stolonifera*.^x *Sclerotinia sclerotiorum* isolated from Clarksville, MI.^y *S. sclerotiorum* isolated from Decatur, MI.^z Early Jalapeno.

observed for disease incidence or disease threshold above the LOD threshold.

When inoculated with *P. nicotianae* (isolate 1007), the two pepper parents had little variation in disease incidence (50% CM334 and 88% Early Jalapeno). Four lines had a significantly higher likelihood of infection compared with the population mean based on odds ratios, whereas lines Q and AAB had a significantly lower likelihood of infection (Table 3). Lines A, D, AD, and AM had 100% disease incidence when infected with *P. nicotianae* (1007), whereas line AAB did not become diseased (Table 2). The population mean was $50.6 \pm 25.7\%$. The LOD threshold for disease incidence was determined to be 3.02 (10%) and 3.32 (5%). No QTL were observed for disease incidence or disease threshold above the LOD threshold.

When inoculated with *P. capsici* isolates, the two pepper parents varied in disease incidence among isolates (Table 2). When compared with the population average, only line AC was significantly more likely to be infected for isolate OP97. Three lines (T, AI, and AAB) had a significantly lower likelihood of infection compared with the population mean when inoculated with isolate 13709. No lines were significantly different from the population average when inoculated with isolate 12889. The population mean was 73 ± 22.6 , 69.1 ± 28.5 , and $81.8 \pm 22.8\%$ for isolates 12889, 13709, and OP97, respectively. The LOD threshold for disease incidence was determined to be 2.91 (10%) and 3.24 (5%) for isolate 12889, 3.05 (10%) and 3.38 (5%) for isolate OP97, and 2.76 (10%) and 3.02 (5%) for isolate 13709. No significant QTL were identified, though small peaks were observed on chromosome 5 for all three isolates.

Correlations among pathogens were detected for disease incidence for most pathogens evaluated (Table 5). The *Sclerotinia* sp. (Clarksville isolate) had no correlation in disease incidence with any of the remaining pathogens (data not shown). *C. acutatum* resistance was correlated with resistance to the greatest number of other pathogens. Disease incidence among the three *P. capsici*

isolates was strongly positively correlated ($r > 0.5$, $P < 0.0001$). Disease incidence between the *P. nicotianae* isolates was strongly positively correlated ($r = 0.5754$, $P < 0.0001$).

DISCUSSION

Incorporating disease resistance into cultivars is a continuous process as pathogens evolve and host resistance is overcome. CM334, widely used in breeding programs across the United States for its durable *Phytophthora* spp. and *Potato virus Y* resistance, has no known resistance or susceptibility to other common pepper pathogens. In this study, we evaluated CM334, Early Jalapeno, and their F_6 progeny for susceptibility to minor (*Rhizopus*, *Colletotrichum*, *Fusarium*, and *Sclerotinia* spp.) and major (*Phytophthora*, *Botrytis*, and *Alternaria* spp.) pepper fruit pathogens. Our data suggest that breeding with CM334 could result in enhanced susceptibility to *Sclerotinia*, *Fusarium*, *Colletotrichum*, *Alternaria*, and *Botrytis* spp. while incorporating resistance to *Phytophthora* spp.

Resistance to *Sclerotinia* stem rot (a minor disease in the United States) has been identified in pepper accessions, although no commercial cultivars with resistance have been documented (Yanar and Miller 2003). Sources of fruit rot resistance, which may be inherited independently from stem rot, has not been identified in wild, unadapted, or cultivated pepper. Although neither of the parents was particularly susceptible to *Sclerotinia* spp., our results identified a single QTL at the end of chromosome 11. This is the first report of a resistance QTL to *Sclerotinia* spp. in pepper. The low incidence in the population suggests that *Sclerotinia* spp. susceptibility should not be a concern when breeding with CM334 but may also be the result of the particular isolates used in this study. The identified QTL did not colocalize with other disease resistance QTL or fruit QTL, and was not correlated with disease susceptibility to the other pathogens evaluated in this study. Further

Fig. 2. CM334 pepper fruit 7 days postinoculation with a single isolate of **A**, *Alternaria* spp.; **B**, *Botrytis* spp.; **C**, *Sclerotinia* isolate (Clarksville); **D**, *Colletotrichum acutatum*; **E**, *Sclerotinia* isolate (Decatur); **F**, *Fusarium oxysporum*; **G**, *F. solani*; or **H**, *Rhizopus oryzae*.

TABLE 3. Significant odds ratio from the average line within a pathogen for each pepper line^p

Line	Pathogen													
	<i>Alt</i> ^q	<i>Bot</i> ^r	<i>Col</i> ^s	<i>F.s</i> ^t	<i>F.o</i> ^u	<i>Rso</i> ^v	<i>Rss</i> ^w	<i>Sclerotinia</i> sp.		<i>Phytophthora capsici</i>			<i>P. nicotianae</i>	
								<i>Clk</i> ^x	<i>Dec</i> ^y	12889	13709	OP97	1006	1007
A	...	...	...	...	...	...	...	...	...	...	...	...	...	+
AA	...	-	+	...	...	...	...	...	...	...	...	...	...	...
AAA	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAB	...	-	-	...	...	...	...	+	...	...	-	...	...	-
AAC	-	...	...	...	...	...	...	...	...	...	...	...	...	...
AAD	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAE	...	...	...	+	...	...	...	...	...	...	...	...	...	...
AAF	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAG	...	-	...	...	...	...	...	...	...	...	...	...	...	...
AAJ	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAK	...	...	-	-	...	...	...	...	...	...	...	...	...	...
AAM	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAN	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAO	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAP	...	+	+	+	+	...	...	+	...	...	...	...	...	...
AAQ	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAR	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AAS	...	...	...	...	...	...	...	+	...	...	...	...	...	...
AAU	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AB	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AC	...	...	-	-	...	+	...	...	...	...	...	...	...	...
AD	+	...	...	...	+	...	...	+	...	...	...	...	...	+
AE	...	...	-	...	...	...	...	...	...	...	...	...	...	...
AF	...	...	...	...	...	...	...	...	+	...	...	...	...	...
AG	...	...	...	+	...	...	+	...	...	...	...	...	...	...
AI	...	...	...	...	...	...	...	...	...	...	-	...	...	...
AJ	...	...	...	...	...	+	...	...	...	...	...	...	...	...
AK	...	+	...	...	...	...	...	...	...	...	...	...	...	...
AL	...	+	...	...	...	...	...	...	...	...	...	...	...	...
AM	...	...	...	+	...	+	...	...	...	...	...	...	...	+
AN	...	...	...	...	+	...	...	...	...	...	...	...	...	...
AO	...	...	...	+	...	...	...	...	...	...	...	...	...	...
AP	...	-	+	...	...	...	...	...	...	...	...	...	...	...
AQ	...	...	...	...	+	...	...	...	...	...	...	...	...	...
AR	...	...	...	...	...	...	+	...	...	...	...	...	...	...
AS	...	-	...	...	...	...	...	...	...	...	...	...	...	...
AT	...	-	...	...	...	...	+	...	...	...	...	...	...	...
AU	...	+	+	...	...	...	+	...	...	...	...	...	...	...
AV	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AW	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AX	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AY	...	...	...	...	...	...	...	...	...	...	...	...	...	...
AZ	...	...	...	-	...	...	+	...	...	...	...	...	...	...
B	...	...	...	...	...	...	...	...	...	...	...	...	...	...
C	...	...	...	...	...	...	...	...	...	...	...	...	...	...
CM334	...	...	...	...	...	+	+	...	...	...	...	...	...	...
D	...	+	+	+	+	...	...	...	...	...	...	...	...	+
E	...	...	+	-	...	...	...	...	...	...	...	...	...	...
EJ ^z	...	-	...	-	...	...	...	...	...	...	...	...	...	...
F	...	...	...	+	...	...	...	...	...	...	...	...	...	...
G	...	...	...	...	...	...	...	...	+	...	...	...	...	...
H	...	...	-	...	...	...	...	...	...	...	...	...	...	...
I	...	-	...	...	...	...	...	...	...	...	...	...	...	...
J	-	-	-	...	...	...	+	...	...	...	...	...	...	...
K	...	+	...	...	+	+	...	...	...	...	...	...	...	...
L	...	...	-	...	...	...	...	...	...	...	...	...	...	...
M	...	...	...	...	...	...	...	...	...	...	...	...	...	...
N	...	+	+	+	+	...	...	...	+	...	...	...	+	...
O	...	...	-	...	...	...	...	...	...	...	...	...	...	...
P	...	...	...	-	...	...	...	...	...	...	...	...	...	...
Q	...	...	...	...	...	+	...	...	...	...	...	...	...	-
R	...	...	-	...	...	+	...	...	...	...	...	...	...	...
S	...	...	...	...	...	...	...	...	...	...	...	...	...	...
T	...	...	...	-	...	...	...	...	...	...	-	...	...	...
V	...	...	...	...	...	...	...	...	...	...	...	...	...	...
X	...	-	...	...	...	...	...	...	...	...	...	...	...	...
Z	...	...	...	-	...	...	...	...	...	...	...	...	...	...

^p Symbols indicate whether a line had significantly less (-) or more (+) likelihood of infection compared with the mean across lines for each pathogen.^q *Alternaria* sp.^r *Botrytis* sp.^s *Colletotrichum acutatum*.^t *Fusarium solani*.^u *F. oxysporum*.^v *Rhizopus oryzae*.^w *R. stolonifera*.^x *Sclerotinia sclerotiorum* isolated from Clarksville, MI.^y *S. sclerotiorum* isolated from Decatur, MI.^z Early Jalapeno.

research will need to be done to determine whether the identified QTL for fruit rot resistance corresponds to stem rot resistance. In other host–*Sclerotinia* spp. systems, studies have shown that isolate-specific interactions exist (Micic et al. 2005; Zhao and Meng 2002). Future QTL mapping studies should incorporate multiple isolates to ensure that general QTL are identified in addition to isolate-specific QTL.

Rhizopus spp. cause postharvest fruit rot on pepper and are considered opportunistic pathogens. The commercial parent (Early Jalapeno) was highly resistant to *Rhizopus* spp. and, due to the minor importance of the pathogen, one would expect most commercial cultivars to perform similarly. However, CM334 was highly susceptible to both *Rhizopus* spp. evaluated in this study. It should be noted that the population mean for disease incidence was low ($\leq 25\%$), suggesting that susceptibility to *Rhizopus* spp. is a recessive trait. Susceptibility to *Rhizopus* spp. was not correlated with any of the other pathogens evaluated in this study but susceptibility was strongly correlated between the two species. Due to the low incidence and lack of correlation with other diseases, *Rhizopus* fruit rot may not cause immediate problems when breeding *P. capsici*-resistant pepper but should be taken into consideration for downstream crosses. As with the *Sclerotinia* isolates evaluated in this study, the *Rhizopus* isolates collected from sugarbeet may have low virulence on pepper, which may have affected our results.

P. capsici is one of the major pathogens affecting solanaceous crops in the eastern United States. Chromosome 5 is the major QTL for *P. capsici* fruit, stem, and root rot resistance, and minor QTL have been identified on chromosomes 2, 3, 4, and 6 (Bonnet et al. 2007; Naegele et al. 2014; Ogundiwin et al. 2005; Sy et al. 2008). In this study, no significant QTL were identified for *P. capsici* resistance. The variation in *P. capsici* QTL is likely due, in part, to differences in

disease evaluations (qualitative versus quantitative). Qualitative variation in disease susceptibility was not observed, though quantitative variation (e.g., lesion size) was evident in some lines. Resistance to *P. capsici* was not correlated with resistance to any of the other pathogens evaluated, including *P. nicotianae*. This is in contrast with a previous study by Bonnet et al. (2007) in which QTL for resistance to *P. nicotianae*-induced root rot overlapped with resistance for *P. capsici*-induced root rot. However, this may also be due, in part, to the low disease incidence by the *P. nicotianae* isolate.

Of all the pathogens evaluated, *Alternaria* spp. had the greatest number of correlations in susceptibility. *Alternaria* resistance from Early Jalapeno was detected within 5 cM of a previously identified fruit shape QTL located in the same population on chromosome 2 (Naegele et al. 2014). This could indicate that *Alternaria* susceptibility in the CM334 background may be linked with the triangular fruit shape associated with ‘Serrano’ pepper (Naegele et al. 2014). In pepper, no studies are available; however, in tomato, QTL for resistance to *A. solani* have been identified on chromosomes 1, 2, 5 to 7, and 9, explaining 9 to 16% of the variation observed (Chaerani et al. 2006). However, these studies were conducted with mixed isolates, and no information is available on *Alternaria* isolate-specific QTL in the family Solanaceae. More isolates will need to be evaluated to confirm the general usefulness of this QTL. Because susceptibility to *Alternaria* spp. was positively correlated with susceptibility to *Colletotrichum* spp., it is possible that resistance to these two pathogens are linked or controlled by overlapping pathways.

Previous studies have found that resistance to *C. gloeosporioides* and *C. capsici* is highly dependent on the isolate and the source of resistance. Most sources of anthracnose resistance are identified from *Capsicum baccatum* and *C. chinense*. Resistance to *Colletotrichum acutatum* was controlled by a dominant cluster on chromosome 5 from *Capsicum chinense* accession PBC932 (Sun et al. 2015; Voorrips et al. 2004). In *C. annuum*, resistance to *Colletotrichum acutatum* has not been previously evaluated. Although only one isolate of *C. acutatum* was evaluated in this study, it is important to note that CM334 was more susceptible than Early Jalapeno. Because no QTL were detected in this study, future research will be needed to determine the genomic location contributing to susceptibility. If resistance to *C. acutatum* does reside on chromosome 5, similar to previous studies (Sun et al. 2015), this could be particularly important, because chromosome 5 contains the major QTL for *P. capsici* resistance.

In our study, CM334 was highly susceptible to *F. solani* and resistant to *F. oxysporum*, whereas Early Jalapeno was resistant to both species.

TABLE 4. Disease resistance quantitative trait loci (QTL) identified in an F₆ Criollo de Morelos × Early Jalapeno pepper population

Pathogen	Isolate	Chr ^x	Pos ^y	R ^{2z}	Data mapped
<i>Alternaria</i> sp.	...	2	63	0.35***	Threshold or incidence
<i>Sclerotinia</i> sp.	Decatur	11	90	0.22*	Incidence

^x Chromosome.

^y Centimorgan position on the chromosome.

^z Significance of the QTL: * and *** indicate $P \leq 0.05$ and 0.001, respectively.

TABLE 5. Pearson’s correlation coefficient in disease incidence among pepper pathogens^P

	<i>Alt</i> ^r	<i>Bot</i> ^s	<i>Col</i> ^t	<i>Fsol</i> ^u	<i>Foxy</i> ^v	<i>Rso</i> ^w	<i>Rss</i> ^x	<i>Sclerotinia</i> sp.		<i>Phytophthora capsici</i>			<i>P. nic</i> ^a
								Clark ^y	Decat ^z	12889 ^y	13709	OP97	1006
<i>Bot</i>	0.1320	...	0.4360***	0.1621	0.1868	−0.0420	−0.0820	0.0090	0.1624	−0.0557	−0.0986	−0.0896	0.0255
<i>Col</i>	0.2981*	0.4360***	...	0.2760*	0.4276***	0.1453	0.2394	0.0906	0.0872	0.1325	0.2965*	0.3249**	0.2381
<i>Fsol</i>	0.2958*	0.1621	0.2760*	...	0.3577**	0.1172	0.2108	0.0934	0.1591	−0.0207	0.1825	0.1343	0.1107
<i>Foxy</i>	0.5756***	0.1868	0.4276***	0.3577**	...	0.0933	0.1242	−0.0297	0.0407	0.1027	0.1158	0.1847	−0.04923
<i>Rso</i>	0.2423	−0.0420	0.1453	0.1172	0.0933	...	0.5014***	−0.1524	−0.2672*	−0.0391	0.1750	−0.0983	−0.0678
<i>Rss</i>	0.0903	−0.0820	0.2394	0.2108	0.1242	0.5014***	...	0.0437	−0.2661*	−0.0661	0.1028	−0.1036	−0.1323
Clark	−0.0175	0.0090	0.0906	0.0934	−0.0297	−0.1524	0.0437	...	0.1581	0.0336	−0.0117	−0.1910	0.1107
Decat	0.1359	0.1624	0.0872	0.1591	0.0407	−0.2672*	−0.2661*	0.1581	...	0.1461	0.0788	0.1983	0.2933*
12889	0.0510	−0.0557	0.1325	−0.0207	0.1027	−0.0391	−0.0661	0.0336	0.1461	...	0.5998***	0.5396***	0.1994
13709	0.1296	−0.0986	0.2965*	0.1825	0.1158	0.1750	0.1028	−0.0117	0.0788	0.5998***	...	0.6488***	0.2387
OP97	0.0828	−0.0896	0.3249**	0.1343	0.1847	−0.0983	−0.1036	−0.1910	0.1983	0.5396***	0.6488***	...	0.2197
1006	0.2349	0.0255	0.2381	0.1107	−0.0493	−0.0678	−0.1323	0.1107	0.2933*	0.1994	0.2387	0.2197	...
1007 ^a	0.2182	0.0109	0.2049	0.2566*	0.0268	0.0496	−0.1230	0.0853	0.0633	0.0139	0.3059*	0.1458	0.5754***

^P Asterisks indicate significance at 0.05 (*), 0.001 (**), or ≤ 0.0001 (***).

^a *Phytophthora nicotianae*.

^r *Alternaria* spp.

^s *Botrytis* spp.

^t *Colletotrichum acutatum*.

^u *Fusarium solani*.

^v *F. oxysporum*.

^w *Rhizopus oryzae*.

^x *R. stolonifera*.

^y *Sclerotinia sclerotiorum* isolated from Clarksville, MI.

^z *S. sclerotiorum* isolated from Decatur, MI.

No QTL were detected in the population for resistance to *F. oxysporum* or *F. solani*. However, quantitative studies may be more likely to identify genomic regions associated with resistance than this qualitative study. In a related species, tomato, as many as six genes have been identified as contributing to *Fusarium* resistance (Sela-Buurlage et al. 2001). This would suggest that *Fusarium* resistance in the Solanaceae family may have isolate-specific components, similar to results from a study conducted in bean (Park et al. 2001). *F. solani*, considered of minor importance in the United States, could become increasingly important as breeders incorporate *Phytophthora* resistance from CM334 into commercial cultivars. In pepper, resistance to *F. oxysporum* had not been previously mapped; this is likely due to the low occurrence of disease, as seen in this study.

Similarly, no QTL studies on *B. cinerea* have been performed in pepper but, in tomato, minor QTL (explaining $\leq 15\%$ of the variation) were discovered for resistance to leaf and stem gray mold on chromosomes 1, 2, and 4 (Finkers et al. 2007b). Because neither parent demonstrated resistance to the pathogen, any resistant lines are likely due to recessive, epistatic combinations or pleiotropic effects.

In this study, the landrace parent (CM334) was not resistant to most of the pathogens evaluated, and was more susceptible than the cultivated parent in some instances. Early Jalapeno demonstrated resistance to *Alternaria* spp., *C. acutatum*, *Rhizopus* spp., and *F. solani*. Although no MDR sites were detected among the pathogens, several correlations in disease incidence were noted between pathogens. This suggests that future studies using quantitative data may be able to further identify QTL and sites of MDR. Although CM334 is a useful source of resistance to diseases caused by *Phytophthora* spp., evaluation of the resulting progeny is needed to ensure that disease susceptibility to other common pepper pathogens is not incorporated into the resulting cultivars.

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