MANAGING PHYTOPHTHORA DISEASES OF VEGETABLES USING HOST RESISTANCE

By

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ABSTRACT

This project aimed to identify commercial cultivars of hard squash and tomatoes resistant to *P. capsici* and *P. infestans*, respectively, to improve disease management for Michigan's conventional and organic vegetable growers. In a two-year field study, we compared 12 commercial cultivars of hard squash, four Butternut types (*Cucurbita moschata),* two Hubbard types (*Cucurbita maxima*), and six Kabocha types (*C. maxima),* for crown rot resistance, and fruit characteristics relevant to processing including mesocarp soluble solids, percent dry matter, and average fruit weight. To evaluate crown rot, the plants were inoculated with *P. capsici* in replicated field trials. The *C. moschata* cultivars had significantly less plant death for both years. In non-inoculated field trials, mature fruits were assessed for fruit characteristics. Of the resistant *C. moschata* cultivars, only 'Ultra Butternut' exhibited similar °Brix than 'NK 580' in both years and had comparable or greater dry matter and fruit weight. Kabocha cultivars with moderate crown rot susceptibility (i.e., 'Thunder') exhibited higher °Brix, dry matter, and smaller fruit weight than 'NK 580' each year. In 2018 and 2019, tomato cultivars were tested under growth chamber, greenhouse, and field conditions. Plants were inoculated with an isolate of *P. infestans* clonal lineage US-23. In the growth-chamber study, the lowest disease severity at the final assessment (<20%) was observed in 'Matt's Wild Cherry' and 'Tomato Stellar,' with significantly less disease than all other cultivars.' In greenhouse experiments, 'Mountain Magic,' 'Tomato Stellar,' 'Mountain Merit', 'Iron Lady' and 'Defiant' had the least foliar disease severity (0 – 8.0%) for each observation date. For the field experiment, eleven of the cultivars included in the study had foliar disease severity <5% on the final observation date.' Results from this study can assist growers in selecting cultivars with genetic resistance which can be employed in conjunction with biorational and conventional fungicides.

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CHAPTER 1. LITERATURE REVIEW

INTRODUCTION

Vegetables are cultivated around the world under a wide variety of weather and soil conditions (Welbaum, 2015). In the United States, California leads vegetable production with approximately 40.5% of the national total, which represent incomes for $ 80 B in 2019 (USDA NASS, 2019). Michigan ranks second in crop diversity behind California, and ninth and sixth in the production of fresh and processing vegetables, respectively (USDA, NASS 2019). In 2019, Michigan ranked 12th for total production of dual-purpose (fresh market and processing) vegetables. The state ranked first for production of cucurbits (cucumber, pumpkin, melons, summer, and winter squash) and fourth in the production of tomatoes for processing (USDA, NASS 2019).

Phytophthora capsici

The genus *Phytophthora* belongs to the kingdom Stramenopila, phylum Oomycota, class Oomycetes, order Peronosporales and family Peronosporaceae (Ho, 2018; Hulvey et al. 2010). The genus *Phytophthora* includes some of the most problematic plant pathogens and has more than 100 described species. In the United States, crop losses caused by *Phytophthora* spp. have been estimated to be one billion USD each year (Erwin and Ribeiro 1996).

P. capsici causes fruit, stem, crown, and root rot and foliar blight on a wide array of crops (Erwin and Ribeiro 1996). Disease caused by *P. capsici* was initially reported on chili pepper in New Mexico causing blighted lesions initially on the fruits and expanding to branches and stems (Leonian 1922). Symptoms were reported as water-soaked lesions, which increased under warm and wet conditions (Leonian 1922). Later, *P. capcisi* was reported infecting watermelon in Arizona in 1932 (Brown and Evans 1933) and honeydew melon in California in 1935 (Tompkins and Tucker 1937). In 1938, watermelons from Arizona with *P. capsici* lesions were detected in

local markets in New York City (Wiant and Tucker 1940). In Michigan, an outbreak in 1997 threatened the Michigan vegetable industry when farms were unable to harvest their pumpkin and cucumber crops due to fruit rot caused by *P. capsici* (Hausbeck and Lamour 2004).

Host Range. *Phytophthora capsici* has been reported to infect approximately 49 woody and herbaceous plant species worldwide from different plant families including Cucurbitaceae, Fabaceae, and Solanaceae (Erwin and Ribeiro 1996; Quesada-Ocampo et al. 2011; Tian and Babadoost 2004). The pathogen has been detected in at least 19 U.S. states (Hausbeck and Lamour 2004). In Michigan, *P. capsici* can cause more than 50% of the total crop losses in susceptible vegetables including cantaloupe, bell and hot pepper, snap bean, cucumber, lima bean, eggplant, tomato, pumpkin, summer and winter squash, watermelon, and zucchini (Gevens et al. 2008; Hausbeck and Lamour 2004; Quesada-Ocampo et al. 2009).

Reports from Illinois indicated that *P. capsici* isolated from pumpkin was able to colonize beet (*Beta vulgaris*), carrot (*Daucus carota*), swiss-chard *(Beta vulgaris* var. cicla), turnip (*Brassica rapa*), spinach (*Spinacia oleracea*), velvetleaf (*Abutilon theophrasti*), and other crops for a total of 22 different crop species. Moreover, 50% of onion plants tested developed symptoms after inoculation with *P. capsici* (Tian and Babadoost 2004).

The variation in pathogenicity was investigated by Polach and Webster (1972) using a group of *P. capsici* isolates obtained from pepper, tomato, eggplant, squash, and fallow soil. Each isolate was used to inoculate pepper, tomato, eggplant, squash, pumpkin, and watermelon plants to observe disease development. Pathogenicity responses for a single isolate were diverse, ranging from non-pathogenic on all hosts, pathogenic to a single host, pathogenic to some of the hosts, or pathogenic on all hosts. Granke et al. (2012) evaluated 126 *P. capsici* isolates collected from different hosts and geographic regions to measure the variability in virulence among

isolates, finding that virulence varied depending on the host from which it was isolated, the host on which it was inoculated, geographic origin, and isolate age. Tian and Babadoost (2004) identified basil, brassicas (cabbage, cauliflower, kale, kohlrabi, and mustard), corn, soybean, and parsley did not develop symptoms when inoculated with *P. capsici.* In contrast Krasnow and Hausbeck (2015) found that Brassica vegetables crops (cauliflower, red cabbage, broccoli, turnip, and radish) and Brassica cover crops (mustard, rape, and oilseed radish) were susceptible to three *P. capsici* isolates collected from Michigan.

Disease Cycle. *P. capsici* can reproduce sexually and asexually (Babadoost 2016; Erwin and Ribeiro 1996**;** Judelson and Blanco 2005)**.** Sexual reproduction is characterized by the heterothallic production of oospores (Polach and Webster 1972)**.** Oospores are formed when the opposite mating types (A1 and A2) come in close contact to form an antheridium (male gametangia) and an oogonium (female gametangia) via hormonal production **(**Ko, 1988; Uchida and Aragaki 1980)**.** Hemmes and Bartnicki-Garcia 91975) used electron microscopy to diagram the nine stages of oospore formation, including meiosis, plasmogamy, and karyogamy. Oospore formation is stimulated by phospholipids such as soybean lecithin. Ko (1985) compared oospore production of *Phytophthora cactorum*, *P. capsici*, *Pythium aphanidermatum*, and *Pythium texans* when media was amended with different phospholipids including soybean lecithin, beta-sitosterol, and cholesterol. Most oomycetes evaluated produced more oospores when media was amended with lecithin. Oospores are thick-walled and can survive unfavorable weather conditions for several years (Babadoost and Pavon, 2013; Bowers et al. 1990; Lamour and Hausbeck 2003). Oospores collected from infected plants in Illinois were able to survive up to 36 months in three different soil textures with no reduction in germination (Babadoost and Pavon 2013). Research performed in Michigan suggested that oospores can remain viable in soils

planted for 5 years with non-host crops and initiate a disease outbreak in the presence of a susceptible host in the sixth year (Lamour and Hausbeck 2003; Lamour and Hausbeck 2001). *P. capsici* oospore germination is asynchronous (Hord and Ristaino 1991) and requires a latent period of at least 30 days. After 30 days, oospores may germinate provided other requirements are met, including intermittent periods of darkness and light exposure, temperatures of 15℃ to 35℃ with an optimal temperature of 24 ℃ (Etxeberria et al. 2011; Hord and Ristaino 1991; Jiang et al. 1989; Seidl Johnson et al. 2015). Foster et al. (1983) evaluated different factors affecting the germination of *P. parasitica* oospores under laboratory conditions, concluding that oospore germination increases not only with culture age, but also when exposed to light during the maturity time. Up to 95% of oospores germinated when cultures were 56 days old and exposed to light, while only 44% of 56-day old cultures germinated when light was not provided. Förster et al. (1983) observed that the germination rate of *P. megasperma* increased when oospores were exposed to soil and root exudates, concluding that there is also a nutritional factor involved in the germination process. Oospores function in the disease cycle as primary inoculum and allow for increased genetic variability (Erwin and Ribeiro 1996; Lamour and Hausbeck 2003).

In Michigan, 14 farms with a history of *P. capsici* were sampled and both mating types were found with the majority of fields having a mating type ratio of 1:1 and reproducing sexually (Lamour and Hausbeck 2000). Lamour and Hausbeck (2001) studied the Michigan population of *P. capsici* from 1998 to 2000, finding that 75% of the isolates collected during 1998 and 1999 had a unique genotype, suggesting sexual outcrossing. They also found evidence of clonal reproduction in the same year; however, the population differed between years which may suggest that clonal lineages could have followed after sexual reproduction. Once oospores

germinate, they produce germ tubes and mycelia that infect the host and give rise to asexual sporangia, which act as secondary inoculum (Lamour et al. 2012). Sporangia are produced on sporangiophores (Erwin and Ribeiro 1996) and become dislodged in the presence of free water (Granke et al. 2009).

The production of sporangia requires the interaction of several factors including relative humidity, water potential, nutrients, sterols, aeration, light exposure, and culture age (Ribeiro 1983). Light exposure not only increases the production of sporangia but also affects shape and caducity (Azizollah et al. 1985). Nielsen et al. (2006) studied how the light/dark cycle impacts *P. capsici* sporulation in hydroponically grown pepper, concluding that 12-hours of light exposure and 12-hours of darkness increased sporangia production. Additionally, it was observed that the production of sporangia mostly occurs during the day. Constant light without dark exposure reduces sporangia production. Sporangia detach from the sporangiophore when in contact with water through capillary force (Granke et al. 2009) and are dispersed as water flows, especially during rainfall events (Schlub 1983; Granke et al. 2009).

Sporangia may germinate directly by forming a germ tube to initiate mycelia or may germinate indirectly by releasing 20-40 motile zoospores, significantly increasing the amount of inoculum available in the field (Erwin and Ribeiro 1996; Lamour et al. 2012; Ribeiro 1983). Temperature, nutrition, and water potential are the main factors leading to direct germination of sporangia (Ribeiro 1983). Bernhardt and Grogan (1982) observed that sporangia of *P. capsici* tended to germinate directly when water potential was near field capacity and indirect germination via zoospore occurred when in contact with free water. Indirect germination via zoospores is the most influential feature in a *P. capsici* epidemic since the population increases dramatically when free water is present, such as in flooding events (Erwin and Ribeiro, 1996).

Zoospores are uninucleate, motile structures that use chemotaxis to find their host and posteriorly encyst, producing a germ tube that initiates host penetration (Deacon and Donaldson 1993; Erwin and Ribeiro 1996). *P. capsici* completes its disease cycle within four days and rapid asexual reproduction (sporulation) occurs when temperatures range from 20°C to 30°C. Relative humidity ≥60% is required for sporulation; however, the optimal relative humidity varies according to isolate (Granke and Hausbeck 2010).

Symptoms and Signs. Phytophthora blight symptoms may include root rot, crown rot, fruit root, and foliar blight, and seedling damping-off (Erwin and Ribeiro 1996). In vegetable crops like pepper and cucurbits, root and crown infections are identified via brown to black discolored lesions. As the disease progresses, stems may become girdled leading to permanent wilting and plant death (Hausbeck and Lamour 2004). Fruit rot symptoms include dark, water-soaked lesions and sporangia reminiscent of "powdered sugar" on the surface of the fruit that develop 2 to 3-days after infection (Hausbeck and Lamour 2004).

Management of *Phytophthora capsici*

Cultural management. *P. capsici* can survive in the soil for long periods in the absence of a susceptible host, limiting the effectiveness of crop rotation as a single management strategy (Lamour and Hausbeck 2003). Exclusion, or preventing the spread of the pathogen to non-contaminated areas, is an important control strategy. Additional essential cultural control strategies include limiting infested soil movement via equipment and avoiding the dumping of diseased culls in production fields (Hausbeck and Lamour 2004). Gevens et al. (2006) determined that surface water from rivers, creeks, and ponds in Michigan may be contaminated with *P. capsici* and therefore should not be used for irrigation. Choosing well-drained sites,

planting on raised beds covered with plastic mulch, and establishing a non-host cover crop are recommended (Larkin et al. 1995; Ristaino et al. 1997; Ristaino and Johnston 1999).

Host resistance. Host resistance to *P. capsici* plays a crucial role in an integrated disease management program (Granke et al. 2012). Most cucurbits are susceptible to root, crown, and fruit rot, except cucumber which appears to tolerate root and crown infection but remains susceptible to fruit rot (Gevens et al. 2006; Hausbeck and Lamour 2004). In fruit of cucurbits crops, ontogenic (age-related) resistance has been identified (Ando et al. 2009). Fruit of cucumber, butternut and acorn squash, and pumpkin demonstrate resistance to *P. capsici* as fruits mature. In contrast, summer squash, zucchini, and melons remain susceptible (Ando et al. 2009). Squash cultivars within the *Cucurbita moschata* species develop age related resistance to fruit rot from 14 to 21 days post pollination (DPP). Lab studies demonstrated few, or no lesions developed after fruit were inoculated under laboratory conditions (Meyer and Hausbeck 2013; Alzohairy et al. 2021). In winter squash, fruit exocarp thickness and firmness increases as the fruit gets older, with most winter squash cultivars reaching their maximum fruit size at 20 to 24 DPP (Meyer and Hausbeck 2013; Krasnow and Hausbeck 2016). Krasnow and Hausbeck (2016) evaluated squash from different species for age-related resistance by inoculating wounded and unwounded fruits with *P. capsici* at 7, 14, 22 and 56 DPP. They observed that disease incidence and lesion size increased in all the wounded cultivars regardless of the time after pollination. In contrast, unwounded C. *pepo* (‘Diablo, ‘Autumn Delight’, ‘Table Ace’, and ‘Vegetable Spaghetti’) and *C. moschata* cultivars (‘Avalon’ and ‘Early Butternut’) were susceptible to *P. capsici* with moderate pathogen growth in 69 - 100% of the fruits at 7 DPP. The pathogen growth and the fruits infected (%) were reduced to light growth in 43 - 81% of the fruits and to water soaking in < 20% of the fruits at 22 DPP. They concluded that age-related resistance is a

phenomenon that develops in the fruit exocarp. *C. moschata* fruit was investigated to identify if age-related resistance was linked to the activity of chemical components in the exocarp (Alzohairy et al. 2021) or due to other changes in the exocarp such as physical barriers that could lead the fruit to become resistant (Alzohairy et al. 2020). Antimicrobial compounds were extracted from *C. moschata* cultivars at different DPP and evaluated for antifungal effect by testing them against *Cladosporium cucumerinun* (Alzohairy et al. 2021). Antimicrobial products from all the stages of fruit development could stop the growth of *C. cucumerinun* when cellulose or silica gel plates were sprayed with fruit extracts and posteriorly inoculated with spores of *C. cucumerinun*. Since antimicrobial compounds were present in the squash fruit regardless of fruit maturity, Alzohairy et al. (2021) concluded that it is unlikely that age-related resistance is driven by the activity of antimicrobial compounds.

Resistance to Phytophthora crown rot in peppers was investigated by Kimble and Grogan (1960) by screening more than 600 pepper lines, varieties, or plant introductions (PI) for resistance to *P. capsici*. They identified a tolerant variety (‘Jalapeño’) which expressed some level of resistance compared to the susceptible cultivars evaluated. They also reported that PI 187331, 123469, 201232, 188476, and 20123 were resistant, with up to 95% of plants showing resistance 30 days post inoculation. In commercial cultivars and breeding lines of pepper (*Capsicum annuum* L.), differing levels of resistance to *P. capsici* crown rot has been observed (Foster and Hausbeck 2010).

In cucurbits, particularly squash, crown rot resistance has been identified mostly in germplasm accessions and wild relatives but is difficult to find at the commercial level (Padley and Kabelka 2008; Chavez et al. 2011; Meyer and Hausbeck 2012). Despite the availability of germplasm accessions with resistance to Phytophthora crown rot in *Cucurbita pepo* and *C.*

moschata (Chavez et al. 2011; Kousik, et al. 2021; Padley and Kabelka 2008), incorporating this resistance into commercial cultivars has been difficult since resistance to Phytophthora crown root in butternut squash accessions of *C. moschata* is governed by the interaction of three independent, dominant genes (R1, R2, R3) (Padley et al. 2009).

Chemical management. The use of fungicides is an important component of an integrated disease management program for *P. capsici* (Granke et al. 2012). To optimize fungicide efficacy against *P. capsici,* it is essential to use the appropriate application method (Foster and Hausbeck 2010). Fungicides are commonly applied using foliar sprays to control diseases caused by *Phytophthora* spp., particularly *P. infestans* in tomato (Cohen et al. 1979). For *P. capsici,* Foster and Hausbeck (2010) observed that drench applications significantly reduced plant death of pepper compared to foliar applications. Similarly, drench applications were more effective than foliar applications at reducing crown and root rot on summer squash caused by *P. capsici* (Meyer and Hausbeck 2013; Yandoc-Ables et al. 2007).

Control of diseases caused by *Phytophthora* spp. improved after systemic fungicides were introduced in the 1970s, which included the carbamates, the isoxazoles, the cyanoacetamide oximes, the ethyl phosphonates, and the phenyl amides (Cohen and Coffey 1986**;** Schwinn and Urech 1986)**.** For many years, a phenyl amide (mefenoxam) was relied on to manage diseases caused by *Phytophthora* spp. (Bruck et al. 1980; Cohen et al. 1979; Ioannou and Grogan 1984). Mefenoxam inhibits the RNA synthesis of the pathogen by affecting RNA polymerase activity, which is a site-specific mode of action (Davidse et al. 1988). Thus, this fungicide is very effective for disease management but has a high risk of the pathogen developing resistance (Davidse et al. 1988; Granke et al. 2012). Following the introduction of the phenyl amide fungicides in 1979, pathogen resistance was noted in *P. infestans* in Europe by the

early 1980s. The mechanism leading to mefenoxam resistance is not completely understood, but it is thought that resistance is monogenic, driven by a semi-dominant gene that in field populations is selected for through repeated use of the fungicide (Lamour and Hausbeck 2000; Shattock 1988). In Michigan, resistance to mefenoxam by *P. capsici* was initially observed in 1997 (Lamour and Hausbeck 2000).

Fluopicolide, registered for the U.S. market in 2007 (Ojiambo et al. 2010)**,** induces redistribution of spectrin-like protein, which is a protein that is crucial for maintaining the stability and shape of the cell (Toquin et al. 2011). Hong Lu et al. (2011) developed a baseline sensitivity to fluopicolide using *P. capsici* isolates from Michigan. After generating mutants with sublethal doses, they reported that there is a high risk that the pathogen will develop resistance to fluopicolide since resistance is driven monogenically and controlled by a semi-dominant gene. It was observed in the same study that there was not a fitness cost for fluopicolide resistant mutants, which means that once resistance is observed in the field it could be persistent. Shortly after registration in the U.S., resistance to fluopicolide was observed in Michigan in a field trial of *Pseudoperonospora cubensis*, an oomycete pathogen closely related to *Phytophthora* spp. (Hausbeck and Linderman 2013; Thienes 2013). After observing a reduction in the efficacy of fluopicolide against *P. capsici* in fields in the state of Georgia, resistance was determined in 2018 and 2019 (Wang and Ji 2021). Since that time, resistance and intermediate resistance has also been reported in Tennesee (Siegenthaler and Hansen 2021), North Carolina, South Carolina, New York, and Michigan (Parada-Rojas and Quesada-Ocampo 2022).

The carboxylic acid amide (CAA) fungicides include dimethomorph (registered for use on cucurbits in 2002), and mandipropamid (registered in 2008). CAAs interfere with cellulose synthesis by targeting the cellulose synthase gene (CesA3). Blum et al. (2012) identified that the

CesA3 gene is highly conserved in oomycetes finding it present in 25 different species of oomycete within the orders Albuginales, Leptomitales, Peronosporales, Pythiales, Rhipidiales and Saprolegniales. A single point mutation at position 1105 of the CesA3 gene confers resistance to CAA fungicides in *Plasmopara viticola* (Blum et al. 2010; Gisi et al. 2019; Goldenhar and Hausbeck 2019; Keinath 2007). After investigating the difference in sensitivity to CAA among oomycetes, it was observed in laboratory-generated mutants of *P. capsici* that modification at position 1105 or position 1109 of the CesA3 could independently confer resistance to CAA fungicides (Blum et al. 2012). Another study identified a third mutation in the CesA3 gene, at position 1077, that conferred resistance to CAA fungicides, by exposing wild parents to sublethal doses of pyrimorph (another CAA fungicide), (Pang et al. 2013). In Michigan, resistance to CAA by *P. capsici* has not been detected in the field. However, CAA resistance of another oomycete pathogen (*P. cubensis*) was first observed in Michigan fields in 2010 (Hausbeck and Cortright 2012). Resistance in *P. cubensis* was found to be conferred by a mutation at position 1105 of the CesA3 gene, where the amino acid glycine was changed to valine or tryptophan, by Blum et al. (2011) that evaluated isolates collected from different geographic location.

Cross-resistance among the CAA fungicides were observed in the oomycetes *Plasmopara viticola* and *P. cubensis* so if the pathogen develops resistance to one of the CAA molecules it will be resistant to other CAA molecules; this phenomenon has not been determined for *P. capsici* (Blum et al. 2011; Gisi et al. 2007).

Oxathiapiprolin (OXTP), which was registered in 2015 (Goldenhar and Hausbeck 2019), is one of the newest fungicides available to manage *P. capsici*. It controls oomycetes by inhibiting the oxysterol binding protein, thus interrupting the movement of lipids between cell

membranes (Pasteris et al. 2016). OXTP effectively controls plant diseases caused by *Phytophthora, Peronospora,* and *Pseudoperonospora* spp., affecting all pathogen life stages including mycelial growth and spore (sporangia and zoospore) germination. However, a reduction in efficacy or inefficacy has been observed in some *Pythium* spp. in vitro (Miao et al. 2016). *P. capsici* mutants were generated via adaptation after exposing several wild isolates of *P. capsici* to OXTP in laboratory conditions. This assay mimicked the process whereby *P. capsici* would develop resistance in the field. The resistance observed in the mutants was conferred by a mutation in codon 769 of the oxysterol-binding protein, which is the targeting point of OXTP (Miao et al. 2016). Resistance to OXTP by *P. capsici* in the field was first observed in isolates collected in 2018 and 2019 from symptomatic vegetables from a commercial farm with a history of *P. capsici* problems in Tennessee. Isolates were categorized as moderately sensitive and resistant to OXTP in laboratory discriminatory assays (Siegenthaler and Hansen 2021). In Italy, a reduction in the efficacy of OXTP was observed in *P. viticola,* caused by the mutation in the oxysterol-binding protein gene (*PvORP1*)(Massi et al. 2023).

Ethaboxam is a thiazole carboxamide fungicide that disrupts microtubule assembly in oomyetes (Uchida et al. 2005). Ethaboxam, registered in 2017 (Goldenhar and Hausbeck 2019), showed pre-registration efficacy against *P. capsici* isolated from watermelon (Kousik et al. 2014). Soon after registration, the baseline sensitivity of *P. cubensis* to ethaboxam was evaluted by Thomas et al. (2018) using a multi-year collection of isolates from different geographic locations across the U.S. They found that all isolates collected from 14 states were sensitive to ethaboxam (Thomas et al. 2018). Noel et al. (2019) identified a mutation at position C239S of the β-tubulin gene that conferred insensitivity to ethaboxan for *Pythium* spp. isolated from corn and soybean. This mutation occurred in the absence of exposure to ethaboxan, as the fields did

not have history of exposure to the fungicide. However, resistance has not been reported in other oomycete pathogens. Field studies performed in the midwestern U.S. demonstrated that ethaboxam is an effective product against *P. capsici* to control Phytophthora blight on processing pumpkin (Babadoost et al. 2020, 2021; Seitz and Babadoost 2022; Babadoost and Salisu 2018, 2019). Ethaboxam is recommended for control of Phytophthora blight via soil, foliar, or drip application in rotation with other fungicides (Hausbeck et al. 2021).

Fumigation strategies. Fumigation is an effective strategy to manage soilborne diseases (McKeen 1954). Fumigants affect plants, pests, and pathogens and are classified as biocides (Schumann and D'Arcy 2012). Chloropicrin is one of the earliest fumigants and was used in World War I as a tear gas (Roark 1934). This product became available for managing soilborne pathogens in the United States in the 1920s (Roark 1934). In 1932, the fumigant methyl bromide was registered to control soilborne pathogens and weeds (NPIC 2000). When pre-plant fumigants (metam potasium alone or combined with chloropicrin) were tested in Michigan, plant death caused by *P. capsici* was reduced in tomato, eggplant, pepper, zucchini, winter squash, melon, and watermelon (Hausbeck et al. 2012). To maximize effectiveness, soil fumigants should be appplied to a well-aereated soil with 50-80% field moisture (Granke et al. 2013). In Michigan, soil fumigation is recommended to be applied in the fall when temperatures are appropiate (15 ℃ to 27 ℃) for best performance. The fumigation process requires proper soil moisture, soil conditions, and application depth (Armstrong and Whitehand 2005; Hausbeck et al. 2012).

Bio-fumigation uses glucosinolate compounds present in the tissue of plants in the Brassicaceae family to control soil-borne pests. Plant tissue is incorporated into the soil and glucosinolates from this tissue are then released into the soil and hydrolyzed into isothiocyanates

(Gimsing and Kirkegaard 2006). Biofumigant cover crops in the Brassicaceae family were evaluated for susceptibility *to P. capsici* by Krasnow and Hausbeck (2015). They found that *P. capsici* was able to reproduce in the roots of the cover crops, which acted as hosts, suggesting some cover crops used in bio fumigation may not provide an adequate control of *P. capsici.*

Integrated Disease Management. Integrated disease management uses several strategies to avoid or limit disease progression. Strategies are designed to slow pathogen dispersal and adaptability, to delay the pathogen developing resistance to fungicides, and contribute to the durability of host resistance (Mundt et al. 2002). A combination of grafting and soil amendments effectively limited incidence of Phytophthora blight on pepper grown under plastic tunnels in Italy (Gilardi et al. 2013). A Michigan study evaluated an integrated disease management strategy to limit Phytophthora blight of pepper under field and greenhouse conditions using host resistance combined with fungicides. Application of the best-performing fungicide onto susceptible pepper resulted in 25% plant death, whereas a combination of the same fungicide paired with a resistant cultivar limited plant death to 7% (Foster and Hausbeck 2010). Ristaino et al. (1997) investigated cultural practices aimed at reducing the dispersal of *P. capsici* and concluded that no-till planting after a wheat cover crop significantly reduced inoculum dispersion by reducing water splash, thus resulting in less disease.

Phytophthora infestans

P. infestans is an important pathogen of plants within the Solanaceae family, particularly potatoes and tomatoes (Forbes et al. 2013). Described as the causal agent of the late blight by Heinrich Anton de Bary in the second half of the 18th century, the pathogen devastated European potato production resulting in the Irish potato famine (Turner 2005). *P. infestans* was first reported in the U.S. in 1843 in New York. The same year Pennsylvania and Delaware reported

crop losses up to 50% of total potato production caused by *P. infestans* (Stevens 1933). The next year, New Jersey reported losses of 15%. In 1845, Indiana, Illinois, and Michigan reported crops infected by *P. infestans* (Stevens 1933). Furthermore, *P. infestans* is one of the most problematic diseases of tomato, capable of causing total crop loss in a susceptible, unprotected crop (Nowicki et al. 2012) .

Host Range. In addition to causing disease on potato and tomato, *P. infestans* has been reported to cause disease in ornamental crops such as petunia (*Petunia x hybrida*) and calibrachoa (*Calibrachoa x hybrida*) (Becktell et al. 2006). Solanaceous weeds may also harbor *P. infestans* (Lindqvist-Kreuze et al. 2020).

Disease Cycle. *P. infestans* can generate sexual and asexual spores (Nowicki et al. 2012). To produce sexual oospores, *P. infestans* requires two mating types (A1 and A2); the production of oospores under natural conditions is rare (Cohen et al. 1997; Flier et al. 2001). Studies based in the Netherlands reported that oospores produced naturally in potato and tomato fields may persist for up to 48 months (Drenth et al. 1995: Turkensteen et al. 2000). Fernández-Pavía et al. (2004) showed that naturally ocuring oospores are capable of surviving for up to two years in fallow fields in Mexico's central valley and served as the primary inoculum for potatoes planted in the third year, causing lower stem lesions 39-50 days after planting. In the U.S., *P. infestans* populations typically reproduce asexually, although there is evidence that sexual reproduction may occasionally occur (Goodwin et al. 1995).

Asynchonous germination of *P. infestans* oospores requires a period of dormancy (Erwin and Ribeiro 1996) and is influenced by light and temperature (Strömberg et al. 2001). Germination increases when oospores are incubated with soil extracts, suggesting that nutrients also influence germination (Strömberg et al. 2001). Studies under controlled conditions showed that oospore

production is influenced by water availability; a constant supply of free water, for at least one week is needed to induce oosporogenesis (Cohen et al. 1997).

Asexual reproduction, or the production of sporangia, occurs with a minimum of 2 hours of leaf wetness in conjunction with temperatures ranging from 18-21^{o}C (Becktell et al. 2005). The optimal temperature for sporangia germination ranges from 10 to 20^{o}C, with an increase in direct sporangia germination as the temperature approaches 20^{o}C. Indirect sporangia germination via zoospore release increases as the temperature declines to near 10^{o}C (Maziero et al. 2009). The optimal temperature for incubation and latent period ranges from 13-28°C, varying with the host (Becktell et al. 2005; Harrison and Lowe 1989; Maziero et al. 2009). Sporulation of *P. infestans* is optimal at temperatures of 15-22^{o}C, relative humidity greater than 80%, and the absence of light (Harrison and Lowe 1989; Maziero et al. 2009); Xiang and Judelson 2014). Sporangia are released via hygroscopic twisting as sporangiophores are exposed to a reduction in relative humidity (Hirst 1953) which commonly occurs between the hour 0800-1300 (Bashi et al. 1982).

Symptoms and Signs. Typically, foliar symptoms are similar across hosts and include dark green lesions that develop into water-soaked areas and become necrotic over time (Forbes et al. 2013; Henfling 1987). Stems and petioles may become infected, leading to plant death (Henfling 1987). In green and ripe tomatoes, lesions appear greasy, progressing rapidly to completely cover the fruit (Hausbeck 2016). Under favorable conditions, pathogen sporulation on potato foliage may appear along the margins of lesions. On tomato foliage, adaxial sporulation may have a purple hue (Hausbeck 2016; Henfling 1987).

Management of *Phytophthora infestans*

Cultural management. Oospores of *P. infestans* can persist in soil in the absence of a susceptible host (Drenth et al. 1995). However, there are no reports confirming that *P. infestans* overwinters via oospores in the U.S. (Fry and Goodwin 1997). The pathogen can survive via infected plant residue, volunteer plants, and potato cull piles (Boyd 1974; Easton 1982). In Michigan, Kirk (2003) found that the temperature within potato cull piles stabilizes above 0^{o}C, even when the surface temperature is lower, and concluded that this could allow *P. infestans* to overwinter in Michigan. Using certified seed and eliminating cull piles and volunteer plants are recommended to reduce initial inoculum (Kirk and Rosenzweig 2015; Lacy and Hammerschmidt 1995). Since the pathogen requires extended periods of leaf wetness, increasing plant spacing to open the canopy and reduce the duration of leaf wetness is also recommended (Struik 2010). Rotem et al. (1970) noted that overhead irrigation disperses the pathogen via water splash and extends the leaf wetness period creating conducive conditions for *P. infestans* infection. Other cultural practice recommendations include avoiding morning overhead irrigations to avoid extending the dew period or using drip irrigation (Lacy and Hammerschmidt 1995).

Host resistance. Qualitative host resistance to *P. infestans* is associated with a hypersensitive response (HR) of the plant. The resistance gene triggers an HR response after identifying pathogen elicitors during the infection process, causing plant cells to secrete active oxygen species which induces plant cell death (Bos et al. 2006; Vleeshouwers et al. 2000). The effectiveness of HR is dependent on how quickly the response occurs. The onset of HR may vary among plants, species, and cultivars (Benhamou 1996; Vleeshouwers et al. 2000).

Resistance genes against *P. infestans* were reported in the early 1950s after an evaluation of crosses between commercial potato (*Solanum tuberosum*) and wild potato (*S. demissum)*

(Mastenbroek 1952). Four resistance genes were identified based on the response of cultivars to *P. infestans* collected from different geographical regions (Black 1952; Mastenbroek 1952; Pristou and Gallegly 1956). In the 1960s, more resistance genes were introduced into commercial potato cultivars from *S. demissum*, totaling 11 resistance genes (R1-R11) (Malcolmson and Black 1966). Recently, new sources of resistance to *P. infestans* were identified from wild solanaceous species (*S. pinnatisectum* and *S. bolbocastanum*) that could be incorporated into commercial potato cultivars (Van der Vossen et al. 2003; Yang et al. 2017).

In tomatoes, resistance genes have also been identified in the wild tomato species *S. pimpinellifolium* and have been incorporated into commercial tomato cultivars (Foolad et al. 204). In Pennsylvania, Foolad et al. (2014) evaluated the efficacy of three of these resistance genes (Ph-1, Ph-2, and Ph-3) by studying different accessions of *S. pimpinellifolium* that carried one or more of each resistance gene. They observed that accessions carrying the Ph-1 resistance gene were the most susceptible to *P. infestans*, while accessions carrying resistance genes Ph-2 and Ph-3 showed an intermediate and high level of resistance, respectively. Overall, the highest level of resistance was observed in cultivars carrying both resistance genes Ph-2 and Ph-3. Other wild tomato species with resistance to *P. infestans* include *S. habrochaites* and *Lycopersicon pennellii* (Li et al. 2011; Smart et al. 2007).

Eleven tomato cultivars with Ph-2, Ph-3, Ph-2 and Ph-3 and heirloom were evaluated by (Seidl Johnson et al. 2014) for resistance to *P. infestans* clonal lineages US-22, US-23, and US-24 using a detached leaf assay at 5-, 7- and 9-days post infection. The heirloom cultivars ‘Matt Wild’ Cherry’, ‘Wapsipinicon Peach’, and ‘Pruden’s Purple’ reduced the size of the lesions when compared to the most susceptible cultivars. The cultivars containing Ph-2 or Ph-3 were not effective against the clonal lineage US-22; cultivars with Ph-2 or Ph-3 showed a reduction of the

size of the lesion when the plants were challenged with the clonal lineages US-23 and US-24. The cultivar 'Mountain Magic', which has the two resistant genes Ph-2 and Ph-3, showed a reduction of the lesion size after inoculation with any of the three clonal lineages tested.

Tomato cultivars (39) including those with late blight resistance genes Ph-1, Ph-2, Ph-3, and Ph-2 + Ph-3 and heirloom varieties were inoculated with *P. infestans* US-3 and evaluated under field conditions for two years in two New York locations (Hansen et al. 2014). Cultivars with Ph-2 + Ph-3 such as 'NC1CELBR', 'NC2CELBR', 'Legend' × 'NC1CELBR', 'Defiant PHR F1', 'Mountain Magic F1' and 'Mountain Merit'; and the heirloom cultivars 'Matt's Wild Cherry', 'Lemon Drop', and 'Mr. Stripey', were highly resistant.

Quantitative resistance has also been observed in wild cultivars of potato and tomato (Smart et al. 2007). Quantitative resistance is governed polygenically and is generally related to partial resistance; however, it is more durable than qualitative resistance (Schumann and D'Arcy 2012). Quantitative trait loci (QTLs) conferring quantitative resistance to *P. infestans* were identified on chromosome 6 of *Lycopersicon pennellii* (Smart et al. 2007).

Chemical management. The application of fungicides, including contact and systemic fungicides, is an effective strategy for managing *P. infestans* (Halterman and Gevens 2013). Contact fungicides provide a preventive barrier whereas systemic fungicides can be translocated to new tissue, providing curative action and protecting new tissue (Halterman and Gevens 2013). Protectant fungicides have been used preventively in the management of *P. infestans* for many years (Platt 1983). The protectant fungicides mancozeb and chlorothalonil, were evaluated in vitro and in vivo by Bruck et al. (1981) for their efficacy against *P. infestans*. They observed that these fungicides reduced sporangial germination and lesion expansion, concluding that weekly application of mancozeb and chlorothalonil could limit a late blight epidemic in potatoes. Kato et

al. (1997) evaluated the sensitivity of the United States and Canadian clonal lineages of *P. infestans* to mancozeb and chlorothalonil and found that all the clonal lineages evaluated (US-1, US-7, and US-8) were sensitive to both fungicides.

Mefenoxam has also been used to control *P. infestans*. Cohen et al. (1979) showed tomato plants treated with mefenoxam via foliar or drench applications at different concentrations showed a consistent reduction in sporulation, sporangial germination, and lesion expansion. However, mefenoxam has a single mode of action, which represents a high risk of the pathogen developing resistance. Resistance was noted under field conditions in the Netherlands in the 1980s (Davidse et al. 1981; Davidse et al. 1988).

In the United States, mefenoxam was the primary fungicide for *P. infestans* control in the 1970s while isolate US-1 was the predominant clonal lineage (Goodwin et al. 1994; Saville et al. 2015). However, the population of *P. infestans* in the United States and Canada changed in early the 1990s when clonal lineages US-7 and US-8 replaced US-1 as the predominant clonal lineages (Goodwin et al. 1998). After this population shift, resistance to mefenoxam caused epidemics in the United States and Canadian crops (Deahl et al. 1993; Goodwin et al. 1996). In 2009, US-22, US-23, and US-24 replaced US-7 and US-8 as the predominant clonal lineages (Fry et al. 2013); though, these clonal lineages were mostly sensitive to mefenoxam (Saville et al. 2015).

Dimethomorph (registered in the United States in 1998) was first described in 1988 in the United Kingdom and controls oomycete pathogens by inhibiting cellulose synthase, which interferes with hyphal morphology and zoospore encystment and germination (Cohen et al. 1995; Kuhn et al. 1991). Dimethomorph effectively controlled a *P. infestans* population with resistance to metalaxyl, especially when sprayed preventively (Cohen et al. 1995; Stein and Kirk 2003).

However, resistance is possible through a point mutation (G1105V) which was identified by generating mutants in vitro with reduced sensitivity to dimethomorph and other CAA fungicides (Blum et al. 2010). A different CAA product, iprovalicarb, was tested in China against field populations of *P. infestans* and it was concluded that there is a low risk of developing resistance to CAA fungicides due to a fitness penalty (Chen et al. 2018). Similar conclusions were derived from a different study determining the base line for fluomorph, which is an analogous structure of dimethomorph (Yuan et al. 2006).

Ametoctradin (QoI fungicides) affects mitochondrial respiration in oomycetes by inhibiting the formation, release, and germination of zoospores (Merk et al. 2011). Ametoctradin was evaluated in Germany against *P. viticola* isolates with the G143A mutation, which corresponds to resistance to Quinone outside Inhibitor (QoI) fungicides and showed a reduction in disease incidence and no cross resistance (Merk et al. 2011). In the United States, ametoctradin was registered in 2012 under the trade name Zampro, which is a pre-mix of ametoctradin with dimethomorph (EPA 2023). In trials performed under greenhouse conditions, Zampro effectively limited lesions caused by *P. capsici* on peppers (Matheron and Porchas 2014). In multi-year Michigan field studies, Zampro effectively controlled *P. cubensis*. However, in the same studies, *P. cubensis* was insensitive to dimethomorph applied alone, suggesting ametoctradin is the active ingredient controlling *P. cubensis* (Goldenhar and Hausbeck 2019). Resistance to ametoctradin has been observed in low frequencies in *P. viticola* isolates collected in France, which was demonstrated to be conferred by the point mutation S34L in the cytochrome b gene (Fontaine et al. 2019). A group in China demonstrated *Phytophthora litchii* resistance to ametoctradin could be conferred by the point mutations S33L and D228N in the cytochrome b gene by generating mutants in vitro (Gao et al. 2022). These *P. litchi* mutants

appeared to have similar fitness as the wild type, suggesting little or no fitness cost for mutations and thus a moderate resistance risk for this fungicide (Gao et al. 2022).

Cyazofamid affects mitochondrial respiration, affecting complex III in the mitochondria by binding to the Q_i redox site (Mitani et al. 2001), which impacts all life stages of oomycetes (Mitani et al. 2002). Mitani et al. (2002) evaluated the efficacy of cyazofamid against *P. infestans*, discovering that preventive applications effectively limited disease ; however, curative effects were not observed (Mitani et al. 2002). In the U.S., cyazofamid was registered under the commercial name of Ranman 400 SC in 2004 (EPA 2023). In the United States, cyazofamid has continued to provide effective control of *P. cubensis* (Goldenhar and Hausbeck 2019) and *Pythium irregulare* (Linderman et al. 2008). However, resistance has been reported in *P. capsici* (Kousik and Keinath 2008; Siegenthaler and Hansen 2021).

Propamocarb hydrochloride was registered in the U.S. in 1984 (EPA 1995). The mode of action of propamocarb hydrochloride is not well understood, although a study with *P. nicotianae* isolated from geranium documented successful control of most growing stages; mycelia growth stage was not affected by the fungicide (Hu et al. 2007). Samoucha and Cohen (1990), studied the toxicity of propamocarb hydrochloride on *P. infestans* isolates collected from Israel, concluding that propamocarb hydrochloride has a low toxicity of the pathogen because it didn't limit spore germination and establishment. However, propamocarb hydrochloride prevented young mycelia growth in the leaves, suggesting that the fungicide is effective when applied preventively. A tank mix of propamocarb hydrochloride and mancozeb in a 1:1 ratio more effectively controlled *P. infestans* than either fungicide applied alone (Samoucha and Cohen 1990), suggesting that using protectant and systemic fungicides in combination may have a synergistic effect for *P. infestans* control (Samoucha and Cohen 1986).

LITERATURE CITED

Alzohairy, S. A., Hammerschmidt, R., and Hausbeck, M. K. (2020). Changes in winter squash fruit exocarp structure associated with age-related resistance to *Phytophthora capsici*. Phytopathology 110:447–455.

Alzohairy, S. A., Hammerschmidt, R., and Hausbeck, M. K. (2021). Antifungal activity in winter squash fruit peel in relation to age related resistance to *Phytophthora capsici.* PMPP 114:101603.

Ando, K., Hammar, S., and Grumet, R. (2009). Age-related resistance of diverse cucurbit fruit to infection by *Phytophthora capsici*. J. Am. Soc. Hortic. Sci. 134:176-182.

Armstrong, J., and Whitehand, L. (2005). Effects of methyl bromide concentration, fumigation time, and fumigation temperature on Mediterranean and oriental fruit fly (Diptera: Tephritidae) egg and larval survival. J. Econ. Entomol. 98:1116-25.

Azizollah, B., Zadeh, A. L. I., and Tsao, P. H. (1985). Effect of light on sporangium formation, morphology, ontogeny, and caducity of *Phytophthora capsici* and '*P. palmivora*' MF4 isolates from black pepper and other hosts. Trans. Br. Mycol. Soc.85:47–69.

Babadoost, M. (2016). Phytophthora blight of cucumber. The Plant Health Instructor. https://doi.org/10.1094/PHI-I-2005-0429-01

Babadoost, M., and Pavon, C. (2013). Survival of Oospores of *Phytophthora capsici* in Soil. Plant Dis. 97:1478–1483.

Babadoost, M., and Salisu, S. (2018). Efficacy of selected fungicides for control of Phytophthora blight in processing pumpkin, 2017. PDMR 12:V104.

Babadoost, M., and Salisu, S. (2019). Effectiveness of selected fungicide for control of Phytophthora blight in processing pumpkin, 2018. PDMR 13:V030.

Babadoost, M., Salisu, S., and Khanal, S. (2020). Effectiveness of selected fungicide for control of Phytophthora blight in processing pumpkin, 2019. PDMR 14:V00665.

Babadoost, M., Sulley, S., and Acheampong, F. (2021). Effectiveness of selected fungicide for control of Phytophthora blight in processing pumpkin, 2020. PDMR 15:V083.

Bashi, E., Ben-Joseph, Y., and Rotem, J. (1982). Inoculum Potential of *Phytophthora infestans* and the Development of Potato Late Blight epidemics. Phytopathology 72:1043–1047.

Becktell, M. C., Daughtrey, M. L., and Fry, W. E. (2005). Temperature and Leaf Wetness Requirements for Pathogen Establishment, Incubation Period, and Sporulation of *Phytophthora infestans* on *Petunia* × *hybrida*. Plant Dis. 89: 975–979.

Becktell, M. C., Smart, C. D., Haney, C. H., and Fry, W. E. (2006). Host-pathogen interactions between *Phytophthora infestans* and the solanaceous hosts *Calibrachoa x hybridus*, *Petunia x hybrida*, and *Nicotiana benthamiana*. Plant Dis. 90: 24–32.

Benhamou, N. 1996. Elicitor-induced plant defense pathways. Trends Plant Sci. 1:233–240.

Bernhardt, E. A., and Grogan, R. G. (1982). Effect of Soil Matric Potential on the Formation and Indirect Germination of Sporangia of *Phytophthora parasitica, P. capsici,* and *P. cryptogea*. Phytopathology 72:507-511.

Black, W. (1952). A genetic basis for the classification of Phytophthora infestans. Proc. Roy. Soc. Edin, Section B: Biological Sciences 65: 36-51.

Blum, M., Boehler, M., Randall, E., Young, V., Csukai, M., Kraus, S., Moulin, F., Scalliet, G., Avrova, A. O., Whisson, S. C., and Fonne-Pfister, R. (2010). Mandipropamid targets the cellulose synthase-like PiCesA3 to inhibit cell wall biosynthesis in the oomycete plant pathogen, *Phytophthora infestans*. Mol. Plant Pathol. 11: 227–243.

Blum, M., Gamper, H. A., Waldner, M., Sierotzki, H., and Gisi, U. (2012). The cellulose synthase 3 (CesA3) gene of oomycetes: structure, phylogeny, and influence on sensitivity to carboxylic acid amide (CAA) fungicides. Fungal Biol. 116: 529–542.

Blum, M., Waldner, M., Olaya, G., Cohen, Y., Gisi, U., and Sierotzki, H. (2011). Resistance mechanism to carboxylic acid amide fungicides in the cucurbit downy mildew pathogen *Pseudoperonospora cubensis*. Pest Manag. Sci. 67: 1211–1214.

Bos, J. B. I., Kannegati, T. D., Young, C., Cakir, C., Huitema, E., Win, J., Armstrong, M. R., Birch, P. R. J., and Kamoun, S. (2006). The C-terminal half of *Phytophthora infestans* RXLR effector AVR3a is sufficient to trigger R3a-mediated hypersensitivity and suppress INF1-induced cell death in *Nicotiana benthamiana.* Plant J. 48:165–176.

Boyd, A. (1974). Sources of Potato Blight (*Phytophthora infestans*) in the East of Scotland. Plant Pathol. 23:30-36.

Bowers, J. H., Papavizas, G. C., and Johnston, S. A. (1990) Effect of soil temperature and soil-water matric potential on survival of *Phytophthora capsici* in natural soil. Plant Dis. 70:771-777.

Brown, J. G., and Evans, M. M. (1933). A Phytophthora Rot of Watermelon. University of Arizona. College of Agriculture. Technical Bulletin No. 51:45–65.

Bruck, R. I., Fry, W. E., and Apple, A. E. (1980). Effect of Metalaxyl, and Acylalanine Fungicide, on Developmental Stages of *Phytophthora infestans*. Plant Dis. 70: 597–601.

Bruck, R. I., Fry, W. E., Apple, A. E., and Mundt, C. C. (1981). Effect of Protectant Fungicides on the Developmental Stages of *Phytophthora infestans* in Potato Foliage. Phytopathology 71:164–166.

Chavez, D. J., Kabelka, E. A., and Chaparro, J. X. (2011). Screening of *Cucurbita moschata* Duchesne germplasm for crown rot resistance to Floridian isolates of *Phytophthora capsici* Leonian. HortScience 46:536–540.

Chen, F., Zhou, Q., Qin, C., Li, Y., and Zhan, J. (2018). Low evolutionary risk of iprovalicarb resistance in Phytophthora infestans. Pestic. Biochem. Phys. 152:76–83.

Cohen, Y., Baider, A., and Cohen, B. (1995). Dimethomorph Activity Against Oomycete Fungal Plant Pathogens. Phytopathology 85:1500-1506.

Cohen, Y., and Coffey, M. D. (1986). Systemic fungicides and the control of oomycete. Annu. Rev. Phytopathology 24:311–338.

Cohen, Y., Farkash, S., Reshit, Z., and Baider, A. (1997). Oospore Production of *Phytophthora infestans* in Potato and Tomato Leaves. Phytopathology 87: 191–196.

Cohen, Y., Reuveni, M., and Eyal, H. (1979). The systemic antifungal activity of Ridomil against *Phytophthora infestans* on tomato plants. Phytopathology 69:645–649.

Davidse, L., Looijen, D., Turkensteen, L. J., and Van der Wal, D. (1981). Occurrence of metalaxyl-resistant strains of *Phytophthora infestans* in Dutch potato fields. Neth. J. Pl. Path. 87:65-68.

Davidse, L. C., Gerritsma, O. C. M., Deler, J., Pie, K., and Velthuis, G. C. (1988). Antifungal modes of action of metalaxyl, cyprofuram, benalaxyl and oxadixyl in phenylamide-sensitive and phenylamide- resistant strains of *Phytophthora megasperrna* f. sp. *medicaginis* and *Phytophthora infestans.* Crop Prot. 7:347–355.

Deacon, J. W., and Donaldson, S. P. (1993). Molecular recognition in the homing responses of zoosporic fungi, with special reference to Pythium and Phytophthora. Mycol. Res. 97: 1153–1171.

Deahl, K. L., DeMuth, S. P., Pelter, G., and Ormrod, D. J. (1993). First report of resistance of Phytophthora infestans to metalaxyl in eastern Washington and southwestern British Columbia. Plant Dis. 77:428-429.

Drenth, A., Janssen, E. M., and Govers, F. (1995). Formation and survival of oospores of *Phytophthora infestans* under natural conditions. Plant Pathol. 44:86–94.

Easton, G. D. (1982). Late Blight of potatoes and prediction of epidemics in arid central Washington state. Plant Dis. 66:452–455.

Environmental Protection Agency (EPA). (1995). Propamocarb Hydrochloride. R.E.D. Facts. Retrieved 8 May 2023 from https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/fs_PC-119301_1-Sep-95.pdf.

Environmental Protection Agency (EPA). (2004). Application for extension of exclusive use period for cyazofamid. Retrieved 8 May 2023 from https://www.epa.gov/sites/default/files/2014-04/documents/cyazofamid-petition-sep4.pdf.

Environmental Protection Agency (EPA). (2019). Registration Review Label Mitigation for Dimethomorph. Retrieved 8 May 2023 from https://www3.epa.gov/pesticides/chem_search/ppls/007969-00302-20190930.pdf.

Erwin, D., and Ribeiro, O. (1996). Phytophthora Diseases Worldwide. American Phytopathological Society Press, St. Paul, MN.

Etxeberria, A., sorkunde, M., and Larregla, S. (2011) Thermal inactivation of *Phytophthora capsici* oospores. Rev Iberoam Micol. 28:83–90

Fernández-Pavía, S. P., Grünwald, N. J., Díaz-Valasis, M., Cadena-Hinojosa, M., and Fry, W. E. (2004). Soilborne oospores of *Phytophthora infestans* in central Mexico survive winter fallow and infect potato plants in the field. Plant Dis. 88:29–33.

Flier, W. G., Grünwald, N. J., Fry, W. E., and Turkensteen, L. J. (2001). Formation, production, and viability of oospores of *Phytophthora infestans* from potato and *Solanum demissum* in the Toluca Valley, central Mexico. Mycol. Res. 105:998–1006.

Fontaine, S., Remuson, F., Caddoux, L., and Barrès, B. (2019). Investigation of the sensitivity of *Plasmopara viticola* to amisulbrom and ametoctradin in French vineyards using bioassays and molecular tools. Pest Manag. Sci.75:2115-2123.

Foolad, M. R., Sullemberger, M. T., Ohlson, E. W., and Gugino, B. k. (2014). Response of accessions within tomato wild species, *Solanum pimpinellifolium* to late blight. Plant Breed. 133:401–411.

Forbes, G. A., Morales, J. G., Restrepo, S., Perez, W., Gamboa, S., Ruiz, R., Cedeño, L., Fermin, G., Andreu, A. B., Acuña, I., and Oliva, R. (2013). Phytophthora infestans and Phytophthora andina on Solanaceous Host in South America. In: Lamour, K. (ed). Phytophthora: A global perspective (p. 48-58). CABI international.

Förster, H., Ribeiro, O., and Erwin, D. C. (1983). Factors affecting oospores germination of *Phytophthora megasperma* f.sp. *midicaginis.* Phytopathology 73, 882–448.

Foster, J. M., and Hausbeck, M. K. (2010). Managing Phytophthora crown and root rot in bell pepper using fungicides and host resistant. Plant Dis. 94:697–702.

Fry, W. E., and Goodwin, S. B. (1997). Re-emergence of potato and tomato Late Blight in the United States. Plant Dis. 81:1349–1357.

Fry, W. E., McGrath, M. T., Seaman, A., Zitter, T. A., McLeod, A., Danies, G., Small, I. M., Myers, K., Everts, K., Gevens, A. J., Gugino, B. K., Johnson, S. B., Judelson, H., Ristaino, J., Roberts, P., Secor, G., Seebold, K., Snover-Clift, K., Wyenandt, A. Smart, C. D. (2013). The 2009 Late Blight pandemic in the eastern United States – Causes and Results. Plant Dis. *97*:296–306.

Gao, X., Hu, S., Liu, Z., Zhu, H., Yang, J., Han, Q., Fu, Y., Miao, J., Gu, B., and Liu, X. (2022). Analysis of resistance risk and resistance-related point mutations in *Cyt b* of QioI fungicide ametoctradin in *Phytophthora litchii*. Pest Manag. Sci. 78:2921–2930.

Gevens, A. J., Donahoo, R. S., Lamour, K. H., and Hausbeck, M. K. (2006). Characterization of *Phytophthora capsici* from Michigan surface irrigation water. Phytopathology 97:421–428.

Gevens, A. J., Donahoo, R. S., Lamour, K. H., Hausbeck M. K. 2008. Characterization of Phytophthora capsici causing foliar and pod blight of snap bean in Michigan. Plant Dis. 92:201-209.

Gilardi, G., Baudino, M., Moizio, M., Pugliese, M., Garibaldi, A., and Gullino, M. L. (2013). Integrated management of *Phytophthora capsici* on bell pepper by combining grafting and compost treatment. Crop Prot. 53:13–19.

Gimsing, A. L., and Kirkegaard, J. A. (2006). Glycosylate and isothiocyanate concentration in soil following incorporation of Brassica biofumigants. Soil Biol. Biochem. 38:2255–2264.

Gisi, U., Lamberth, C., Mehl, A., Seitz, T., and Blum, M. (2019). Carboxylic Acid Amide (CAA) Fungicides. In P. Jeschke M. Witschel, W. Krämer and U Schirmer (Eds.), *Modern Crop Protection Compounds* (pp. 845–869). Wiley-VCH Verlag GmbH & Co. KGaA.

Gisi, U., Waldner, M., Kraus, N., Dubuis, P. H., and Sierotzki, H. (2007). Inheritance of resistance to carboxylic acid amide (CAA) fungicides in *Plasmopara viticola*. Plant Pathol. 56:199–208.

Goldenhar, K. E., and Hausbeck, M. K. (2019). Fungicides for control of Downy Mildew on pickling cucumber in Michigan. Plant Health Prog. 20:165–169.

Goodwin, S. B., Cohen, B. A., and Fry, W. E. (1994). Panglobal distribution of a single clonal lineage of the Irish potato famine fungus. PNAS. 91:11591–11595.

Goodwin, S. B., Smart, C. D., Sandrock, R. W., Deahl, K. L., Punja, Z. K., and Fry, W. E. (1998). Genetic change within populations of *Phytophthora infestans* in the United States and Canada during 1994 to 1996: Role of migration and recombination. Phytopathology 88:939–949.

Goodwin, S. B., Sujkowski, L. S., Dyer, A. T., Fry, B. A., and Fry, W. E. (1995). Direct detection of gene flow and probable sexual reproduction of *Phythophthora infestans* in Northern North America. Phytopathology 85:473–479.

Goodwin, Stephen. B., Sujkowski, Ludwik. S., and Fry, William. E. (1996). Widespread distribution and probable origin of resistance to metalaxyl in clonal genotypes of *Phytophthora infestans* in the United States and western Canada. Phytopathology 86:793–800.

Ganke, L. L., and Hausbeck, M. K. (2010) Effects of Temperature, Humidity, and Wounding on Development of Phytophthora Rot of Cucumber fruit. Plant Dis. 94:1417-1424.

Granke, L. L., Quesada-Ocampo, L., Lamour, K., and Hausbeck, M. K. (2012). Advances in Research on *Phytophthora capsici* on Vegetable Crops in The United States. Plant Dis. 96:1588–1600.

Granke, L. L., Windstam, S. T., Hoch, H. C., Smart, C. D., and Hausbeck, M. K. (2009). Dispersal and Movement Mechanisms of *Phytophthora capsici* Sporangia. Phytopathology 99:1258–1264.

Granke, L., Quesada-Ocampo, L., and Hausbeck, M. (2013). *Phytophthora capsici* in the Eastern USA. In K. H. Lamour (Ed.), Phytophthora A Global Perspective (p. 244). CABI international.

Ho, H. (2018). The Taxonomy and Biology of Phytophthora and Pythium. J. Bacteriol. Mycol Open Access *6*:40–45.

Halterman, D., and Gevens, A. J. (2013). Phytophthora infestans in the USA. In K. H. Lamour. (Ed.), Phytophthora: A Global Perspective (p. 244). CABI international.

Harrison, J. G., and Lowe, R. (1989). Effects of humidity and air speed on sporulation of *Phytophthora infestans* on potato leaves. Plant Pathol. 38:585–591.

Hausbeck, M. K., and Cortright, B. D. (2012). Evaluation of fungicides and application methods for control of downy mildew of cucumber, 2010. PDMR. 6:V136.

Hausbeck, M. K., Enzenbacher, T. B., Cortright, B. D., and Linderman, S. D. (2012). Phytophthora, films and fumigation (p. 1). Michigan State University Extension. Retrieved 15 April 2021 from https://www.canr.msu.edu/news/phytophthora_films_and_fumigants.

Hausbeck, M. K., Higgins, D., and Engfehr, C. (2021). *Managing Phytophthora on Winter Squash and Pumpkin*. Michigan State University Extension. Retrieved 14 December 2022 from https://veggies.msu.edu/wp-content/uploads/2022/02/FS-winter-squash-pumpkin-Phytophthora-updated-2021-Dec-protected.pdf.

Hausbeck, M. K., and Lamour, K. 2004. *Phytophthora capsici* on vegetables crops: research progress and management challenges. Plant Dis. 88:292–1301.

Hausbeck, M. K., and Lamour, K. H. (2004). *Phytophthora capsici* on Vegetable Crops: Research Progress and Management Challenges. Plant Dis. 88:1292–1303.

Hausbeck, M. K., and Linderman, S. D. (2013). Evaluation of fungicides for control of downy mildew of cucumber, 2013. PDMR. *8*:V304.

Hemmes, D. E., and Bartnicki-Garcia, S. (1975). Electron microscopy of gametangial interaction and oospore development in *Phytophthora capsici*. Arch. Microbiol. 103:91–112.

Henfling, J. W. (1987). *Late Blight* of Potato (p. 25). International Potato Center. Technical Information Bulletin 4.

Hirst, J. M. (1953). Changes in Atmospheric Spore Content: Diurnal Periodicity and The Effects of Weather. Trans. Br. Mycol. Soc. 36:375–393.

Hong Lu, X., Hausbeck, M. K., Li Liu, X., and Hao, J. J. (2011). Wild Type Sensitivity and Mutation Analysis for Resistance Risk to Fluopicolide in *Phytophthora capsici*. Plant Dis. 95:1535–1541.

Hord, M. J., and Ristaino, J. B. (1991). Effects of Physical and Chemical Factors on Germination of Oospores of *Phytophthora capsici* in vitro. Phytopathology 81:1541–1546.

Hu, J., Hong, C., Stromberg, E. L., and Moorman, G. W. (2007). Effects of Propamocarb Hydrochloride on Mycelial Growth, Sporulation, and Infection by *Phytophthora nicotianae* Isolates from Virginia Nurseries. Plant Dis. 91:414–420.

Hulvey, J., Telle, S., Nigrelli, L., Lamour, K., and Thines, M. (2010). Salisapiliaceae - a new family of oomycetes from marsh grass litter of Southeastern North America. Pers.: Mol. Phylogeny Evol. Fungi. 25:109–116.

Ioannou, N., and Grogan, R. G. (1984). Control of Phytophthora root rot of processing tomato with ethanol and methalaxyl. Plant Dis. 68:429-435.

Jiang, J., Stephenson, L. W., Erwin, D. C., and Leary, J. (1989). Nuclear changes in Phytophthora during oospore maturation and germination. Mycol. Res. 92:463–469.

Judelson, H. S., and Blanco, F. A. (2005). The spores of Phytophthora: Weapons of the plant destroyer. Nat. Rev. Microbiol. 3:47–58.

Kato, M., Mizubuti, E. S. G., Goodwin, S. B., and Fry, W. E. (1997). Sensitivity to Protectant Fungicides and Pathogenic Fitness of Clonal Lineages of Phytophthora infestans in the United States. Phytopathology 87:973–978.

Keinath, A. P. (2007). Sensitivity of Populations of *Phytophthora capsici* from South Carolina to Mefenoxam, Dimethomorph, Zoxamide, and Cymoxanil. Plant Dis. 91:743–748.

Kimble, K. A., and Grogan, R. G. (1960). Resistance to Phytophthora root rot in pepper. Plant Dis. Rep. 44:872–873.

Kirk, W. W. (2003). Thermal Properties of Overwintered Piles of Cull Potatoes. *Amer J of* Potato Res. 80:145–149.

Kirk, W., and Rosenzweig, N. (2015). Potato late blight recommendations for 2015. Michigan State University Extension. Retrieved 8 May 2023 from https://www.canr.msu.edu/news/potato_late_blight_recommendations#:~:text=Nighttime%20temperatures%20of%2050%20to,late%20blight%20infection%20and%20development.

Knott, J. E., and Lorenz, O. A. (1950). Vegetable Production. Advances in Agronomy *2*:113–155.

Ko, W. H. (1985). Stimulation of Sexual Reproduction of *Phytophthora cactorum* by Phospholipids. Microbiology 131:2591–2594.

Ko, W. (1988). Hormonal Heterothallism and Homothallism in Phytophthora. Ann. Rev. Phytopathol. 26:57–73.

Kousik, C. S., and Keinath, A. P. (2008). First Report of Insensitivity to Cyazofamid Among Isolates of *Phytophthora capsici* from the Southeastern United States. Plant Dis. *92*:979–979.

Kousik, C. S., Ikerd, J. L., and Harrison, H. F. (2014). Development of Pre- and Postharvest Phytophthora Fruit Rot on Watermelons Treated with Fungicides in the Field. Plant Heal. Prog. *15*:145–150.

Kousik, C. S., Vogel, G., Ikerd, J. L., Mandal, M. K., Mazourek, M., Smart, C. D., and Turechek, W. W. (2021). New sources of resistance in winter squash (*Cucurbita moschata*) to Phytophthora Crown Rot and their relationship to cultivated squash. Plant Health Prog. 22:323–331.

Krasnow, C. S., and Hausbeck, M. K. (2015). Pathogenicity of Phytophthora capsici to Brassica Vegetables Crops and Biofumigation Cover Crops (*Brassica spp*). Plant Dis. 99:1721–1726.

Krasnow, C. S., and Hausbeck, M. K. (2016) Evaluation of winter squash and pumpkin cultivars for age-related resistance to *Phytophthora capsici* fruit rot. HortScience 51:1251–1255.

Kuhn, P. J., Pitt, D., Lee, S. A., Wakley, G., and Sheppard, A. N. (1991). Effects of dimethomorph on the morphology and ultrastructure of Phytophthora. Mycol. Res. 95:333–340.

Lacy, M. L., and Hammerschmidt, R. (1995). *Best Management Practices for Potatoes. Diseases of Potato: Late Blight* (p. 4). Michigan State University Extension. Retrieved 14 September 2022 from https://www.canr.msu.edu/uploads/files/e1802.pdf.

Lamour, K. H., and Hausbeck, M. K. (2000). Mefenoxam Insensitivity and the Sexual Stage of *Phytophthora capsici* in Michigan Cucurbit Field. Phytopathology 90:396–400.

Lamour, K. H., and Hausbeck, M. K. (2001). The Dynamics of Mefenoxam Insensitivity in a Recombining Population of *Phytophthora capsici* Characterized with Amplified Fragment Length Polymorphism Markers. Phytopathology 91:553–557.

Lamour, K. H., and Hausbeck, M. K. (2001). Investigating the Spatiotemporal Genetic Structure of *Phytophthora capsici* in Michigan. Phytopathology 91:973–980.

Lamour, K. H., and Hausbeck, M. K. (2003). Effect of Crop Rotation on the Survival of *Phytophthora capsici* in Michigan. Plant Dis. 87:841–845.

Lamour, K. H., Stam, R., Jupe, J., and Huitema, E. (2012). The oomycete broad-host-range pathogen *Phytophthora capsici*. Mol. Plant Pathol. 13:329–337.

Larkin, R. P., Gumpertz, M. L., and Ristaino, J. B. (1995) Geostatistical Analysis of Phytophthora Epidemic Development in Commercial Bell Pepper Fields. Phytopathology 85:191-203.

Leonian, L. H. (1922). Stem and fruit blight of pepper caused by *Phytophthora capsici* species nov. Phytopathology 12:401–408.

Li, J., Liu, L., Bai, Y., Finkers, R., Wang, F., Du, Y., Yang, Y., Xie, B., Visser, R. G. F., and van Heusden, A. W. (2011). Identification and mapping of quantitative resistance to late blight (Phytophthora infestans) in *Solanum habrochaites* LA1777. Euphytica 179:427–438.

Linderman, R. G., Davis, E. A., and Masters, C. J. (2008). Efficacy of Chemical and Biological Agents to Suppress *Fusarium* and *Pythium* Damping-Off of Container-Grown Douglas-fir Seedlings. Plant Heal. Prog. 9:1-10.

Lindqvist-Kreuze, H., Gamboa, S., Izarra, M., Pérez, W., Correa, M. Y., Astete, A., Särkinen, T., Cueva, M., and Gonzáles, P. (2020). Population structure and host range of the potato late blight pathogen *Phytophthora infestans* in Peru spanning two decades. Plant Pathol. 69:334–346.

Malcolmson, J. F., and Black, W. (1966). New R genes in Solanum demissum lindl. And their complementary races of *Phytophthora infestans* (Mont.) de bary. Euphytica 15:199–203.

Massi, F., Torriani, S. F., Waldner-Zulauf, M., Bianco, P. A., Coatti, M., Borsa, P., Borghi, L., and Toffolatti, S. L. (2023). Characterization of Italian *Plasmopara viticola* populations for resistance to oxathiapiprolin. Pest Manag. Sci. 79:1243–1250.

Mastenbroek, C. (1952). Investigations Into the Inheritance of The Immunity from *Phytophthora infestans* De B. of *Solanum Demissum* Lindl. Euphytica 1:187–198.

Matheron, M. E., and Porchas, M. (2014). Effectiveness of 14 Fungicides for Suppressing Lesions Caused by *Phytophthora capsici* on Inoculated Stems of Chile Pepper Seedlings. Plant Heal. Prog. 15:166–171.

Maziero, J. M. N., Maffia, L. A., and Mizubuti, E. S. G. (2009). Effects of temperature on events in the infection cycle of two clonal lineages of Phytophthora infestans causing late blight on tomato and potato in Brazil. Plant Dis. 93: 459–466.

McKeen, C. D. (1954). Methyl Bromide as A Soil Fumigant for Controlling Soil-Borne Pathogens and Certain Other Organisms in Vegetable Seedbeds. Canad. J. Bot. 32:101–115.

Merk, M., Gold, R. E., Schiffer, H., Levy, T., Frechen, T., and Saramago, J. (2011). Initium®: A New Innovative Fungicide of a New Chemical Class for The Control of Late Blight and Downy Mildew Diseases. Acta Hortic. 917:143–148.

Meyer, M. D., and Hausbeck, M. K. (2012). Using cultural practices and cultivar resistance to manage Phytophthora crown rot on summer squash. HortScience 47:1080–1084.

Meyer, M. D., and Hausbeck, M. K. (2013). Using Soil-Applied Fungicides to Manage Phytophthora Crown and Root Rot on Summer Squash. Plant Dis. 97:107–112.

Meyer, M. D., and Hausbeck, M. K. (2013). Age-related resistance to Phytophthora fruit rot in 'Dickenson Field' processing pumpkin and 'Golden Delicious' winter squash fruit. Plant Dis. 97:446–452.

Miao, J., Cai, M., Dong, X., Liu, L., Lin, D., Zhang, C., Pang, Z., and Liu, X. (2016). Resistance Assessment for Oxathiapiprolin in *Phytophthora capsici* and the Detection of a Point Mutation (G769W) in PcORP1 that Confers Resistance. Front. Microbiol. *7*:615.

Miao, J., Dong, X., Lin, D., Wang, Q., Liu, P., Chen, F., Du, Y., and Liu, X. (2016). Activity of the novel fungicide oxathiapiprolin against plant-pathogenic oomycetes. Pest Manag. Sci. 72:1572–1577.

Mitani, S., Araki, S., Takii, Y., Ohshima, T., Matsuo, N., and Miyoshi, H. (2001). The Biochemical Mode of Action of the Novel Selective Fungicide Cyazofamid: Specific Inhibition of Mitochondrial Complex III in *Phythium spinosum*. Pestic. Biochem. Phys. 71:107–115.

Mitani, S., Araki, S., Yamaguchi, T., Takii, Y., Ohshima, T., and Matsuo, N. (2002). Biological properties of the novel fungicide cyazofamid against *Phytophthora infestans* on tomato and *Pseudoperonospora cubensis* on cucumber. Pest Manag. Sci. 58:139–145.

Mundt, C. C., Cowger, C., and Garrett, K. A. (2002). Relevance of integrated disease management to resistance durability. Euphytica 124:245–252.

Nielsen, C. J., Ferrin, D. M., Stanghellini, M. E., Nielsen, C. J., Ferrin, D. M., and Stanghellini, M. E. (2006). Cyclic production of sporangia and zoospores by Phytophthora capsici on pepper roots in hydroponic culture. Can. J. Plant. Pathol. 28:461-466.

Noel, Z. A., Sang, H., Roth, M. G., and Chilvers, M. I. (2019). Convergent Evolution of C239S Mutation in *Pythium* spp. β-Tubulin Coincides with Inherent Insensitivity to Ethaboxam and Implications for Other Peronosporalean Oomycetes. Phytopathology 109:2087–2095.

Nowicki, M., Foolad, M. R., Nowakowska, M., and Kozik, E. U. (2012). Potato and Tomato Late Blight Caused by *Phytophthora infestans*: An Overview of Pathology and Resistance Breeding. Plant Dis. 96:4–17.

NPIC. (2000). *Methyl Bromide* (p. 6). Oregon State University. http://npic.orst.edu/factsheets/MBgen.pdf

Ojiambo, P. S., Paul, P. A., and Holmes, G. J. (2010). A Quantitative Review of Fungicide Efficacy for Managing Downy Mildew in Cucurbits. Phytopathology 100:1066–1076.

Padley Jr, L. D., and Kabelka, E. A. (2008). Evaluation of Cucurbita pepo Accessions for Crown Rot Resistance to Isolates of Phytophthora capsici. HortScience 43:1996–1999.

Padley Jr, L. D., Kabelka, E. A., and Roberts, P. D. (2009). Inheritance of Resistance to Crown Rot Caused by Phytophthora capsici in Cucurbita. HortScience 44:211–213.

Pang, Z., Shao, J., Chen, L., Lu, X., Hu, J., Qin, Z., and Liu, X. (2013). Resistance to the Novel Fungicide Pyrimorph in *Phytophthora capsici*: Risk Assessment and Detection of Point Mutations in CesA3 That Confer Resistance. PLoS ONE *8*:e56513.

Parada-Rojas, C. H., and Quesada-Ocampo, L. M. (2022). *Phytophthora capsici* Populations Are Structured by Host, Geography, and Fluopicolide Sensitivity. Phytopathology 112:1559–1567.

Pasteris, R. J., Hanagan, M. A., Bisaha, J. J., Finkelstein, B. L., Hoffman, L. E., Gregory, V., Andreassi, J. L., Sweigard, J. A., Klyashchitsky, B. A., Henry, Y. T., and Berger, R. A. (2016). Discovery of oxathiapiprolin, a new oomycete fungicide that targets an oxysterol binding protein. Bioorg. Med. Chem. 24:354–361.

Platt, H. W. (1983). Effects of metalaxyl, mancozeb, and chlorothalonil on blight, yield, and tuber rot of potato. Can. J. of Plant Pathol. 5:38–42.

Pristou, R., and Gallegly, M. (1956). Differential reaction of potato hosts to foreign and domestic potato physiologic races of Phytophthora infestans. American Potato Journal 33:287-294

Polach, F. J., and Webster, R. K. (1972). Identification of Strains and Inheritance of Pathogenicity in *Phytophthora capsici*. Phytopathology 62:20-26.

Quesada-Ocampo, L. M., Fulbright, D. W., and Hausbeck, M. K. 2009. Susceptibility of Fraser Fir to *Phytophthora capsici*. Plant Dis. 93:135-141.

Quesada-Ocampo, L., Granke, L. L., Mercier, M. R., Olsen, J., and Hausbeck, M. K. (2011). Investigating the Genetic Structure of Phytophthora capsici Populations. Phytopathology 101: 1061–1073.

Ribeiro, O. (1983). Physiology of Asexual Sporulation and Spore Germination in Phytophthora. In Erwin, D.C., S. Bartnicki-Garcia, and P. H. Tsao (Eds.), *Phytophthora its Biology, Taxonomy, Ecology, and Pathology.* (2nd ed., p. 392). APS PRESS.

Ristaino, J. B., Parra, G., and Campbell, C. L. (1997). Suppression of Phytophthora Blight in Bell Pepper by a No-Till Wheat Cover Crop. Phytopathology 87:242–249.

Ristaino, J. B., and Johnston, S. A. (1999). Ecologically Based Approaches to Management of Phytophthora Blight on Bell Pepper. Plant Dis. 83:1080–1089.

Roark, R. C. (1934). A Bibliography of Chloropicrin, 1848-1932 (R. Roak, Ed.; p. 88). U.S. Department of Agriculture.

Rotem, J., Palti, J., and Lomas, J. (1970). Effects of Sprinkler Irrigation at Various Times of the Day on Development of Potato Late Blight. Phytopathology 60:839–843.

Samoucha, Y., and Cohen, Y. (1986). Efficacy of Systemic and Contact Fungicides Mixtures in Controlling Late Blight in Potatoes. Phytopathology 76:855–859.

Samoucha, Y., and Cohen, Y. (1990). Toxicity of propamocarb to the late blight fungus on potato. Phytoparasitica 18:27-40.

Saville, A., Graham, K., Grünwald, N. J., Myers, K., Fry, W. E., and Ristaino, J. B. (2015). Fungicide Sensitivity of U.S. Genotypes of *Phytophthora infestans* to Six Oomycete-Targeted Compounds. Plant Dis. 99:659–666.

Schlub, R. L. (1983) Epidemiology of *Phytophthora capsici* on bell pepper. J. Agric. Sci. Camb. 100:7-11

Schumann, G. L., and D'Arcy, C. J. (2012). Essential Plant Pathology (G. L. Schumann and C. J. D'Arcy, Eds.; Second Edi). APS PRESS St. Paul, MN.

Schwinn, F. J., and Urech, P. A. (1986). Progress in the Chemical Control of Diseases Caused by Oomycete. In M. B. Green and D. A. Spilker (Eds.), *Fungicide Chemistry, Advances and Practical Applications* (ASC Sypons, pp. 89–106). American Chemical Society.

Seidl Johnson, A. C., Jordan, S. A., and Gevens, A. J. (2015). Efficacy of organic and conventional fungicides and impact of application timing on control of tomato late blight caused by US-22, US-23, and US-24 isolates of *Phytophthora infestans.* Plant Dis. 99:641–647.

Seitz, H., and Babadoost, M. (2022). Effectiveness of selected fungicides for control of Phytophthora blight in processing pumpkin, 2022. PDMR 17:V022.

Shattock, R. C. (1988). Studies on the inheritance of resistance to metalaxyl in *Phytophthora infestans*. Plant Pathol. 37:4–11.

Siegenthaler, T. B., and Hansen, Z. R. (2021). Sensitivity of *Phytophthora capsici* from Tennessee to Mefenoxam, Fluopicolide, Oxathiapiprolin, Dimethomorph, Mandipropamid, and Cyazofamid. Plant Dis. 105:3000–3007.

Smart, C. D., Tanksley, S. D., Mayton, H., and Fry, W. E. (2007). Resistance to *Phytophthora infestans* in Lycopersicon pennellii. Plant Dis. 91:1045–1049.

Stein, J. M., and Kirk, W. W. (2003). Variations in the Sensitivity of *Phytophthora infestans* Isolates from Different Genetic Backgrounds to Dimethomorph. Plant Dis. 87*:*1283–1289.

Stevens, N. E. (1933). The dark ages in Plant Pathology in America:1830-1870. J. Wash. Acad. Sci. 23:435–446.

Strömberg, A., Boström, U., and Hallenberg, N. (2001). Oospore Germination and Formation by the Late Blight Pathogen *Phytophthora infestans* in vitro and under Field Conditions. J. Phytopathol. 149:659–664.

Struik, P. C. (2010). Can Physiology Help Us to Combat Late Blight in Potato? Potato Research 53: 277–287.

Thienes, M. (2013). Taxonomy and Phylogeny of Phytophthora and related Oomycetes. In K. Lamour (Ed.), *Phytophthora: A Global Perspective* (CABI plant, p. 243).

Thomas, A., Neufeld, K. N., Seebold, K. W., Braun, C. A., Schwarz, M. R., and Ojiambo, P. S. (2018). Resistance to Fluopicolide and Propamocarb and Baseline Sensitivity to Ethaboxam Among Isolates of *Pseudoperonospora cubensis* From the Eastern United States. Plant Dis. 102*:*1619–1626.

Tian, D., and Babadoost, M. (2004). Host range of *Phytophthora capsici* from pumpkin and pathogenicity of isolates. Plant Dis. 88*:*485–489.

Tompkins, C. M., and Tucker, C. M. (1937). Phytophthora rot on honeydew melon. J. Agric. Res. 54:933–944.

Toquin, V., Latorse, M. P., and Beffa, R. (2011). Fluopicolide: A New Anti-Oomycetes Fungicide? In P. W. Krämer, U. Schirmer, M. Jeschke and M. Witschel, (Eds.), Modern Crop Protection Compounds (Second Edi, p. 1498). Wiley-VCH Verlag GmbH & Co. KGaA.

Turkensteen, L. J., Flier, W. G., Wanningen, R., and Mulder, A. (2000). Production, survival, and infectivity of oospores of *Phytophthora infestans.* Plant Pathol. 49:688–696.

Turner, R. S. (2005). After the famine: Plant pathology, *Phytophthora infestans*, and the late blight of potatoes, 1845–1960. HSNS. 35:341–370.

Uchida, J. Y., and Araki, M. (1980) Chemical stimulation of oospore formation in *Phytophthora capsici*. Mycologia 6:1103-1108.

Uchida, M., Roberson, R. W., Chun, S.-J., and Kim, D. S. (2005). In vivo effects of the fungicide ethaboxam on microtubule integrity in *Phytophthora infestans.* Pest Manag. Sci. 61:787–792.

USDA (2019). Vegetable 2019 summary Department of Agriculture, National Agriculture Statistics Service. Retrieved September 2019 from http://nass.usda.gov.

Van der Vossen, E., Sikkema, A., Lintel Kekkert, B., Gros, J., Stevens, P., Muskens, M., Wouters, D., Pereira, A., Stiekema, W., and Allefs, S. (2003). An ancient R gene from the wild potato species *Solanum bulbocastanum* confers broad-spectrum resistance to *Phytophthora infestans* in cultivated potato and tomato. TPJ. 36: 867-882

Vleeshouwers, V. G. A. A., van Dooijeweert, W., Govers, F., Kamoun, S., and Colon, L. T. (2000). The hypersensitive response is associated with host and nonhost resistance to *Phytophthora infestans*. Planta 210:853–865.

Wang, L., and Ji, P. (2021). Fitness and Competitive Ability of Field Isolates of *Phytophthora capsici* Resistant or Sensitive to Fluopicolide. Plant Dis. 105:873–878.

Welbaum, G. E. (2015). Vegetable Production and Practices (p. 453). CABI.

Wiant, J. S., and Tucker, C. M. (1940). A Rot of Winter Queen Watermelons Caused by *Phytophthora capsici*. J. Agric. Res. 60:73–88.

Wilhelm, S., and Koch, E. E. (1956). Verticillium Wilt Controlled. Cal. Agric. 10 (5):3-14

Xiang, Q., and Judelson, H. S. (2014). Myb Transcription Factors and Light Regulate Sporulation in the Oomycete *Phytophthora infestans.* PLoS ONE. 9:e92086.

Yandoc-Ables, C. B., Rosskopf, E. N., and Lamb, E. M. (2007). Management of Phytophthora Crown Rot in Pumpkin and Zucchini Seedlings with Phosphonates. Plant Dis. 91:1651–1656.

Yang, L., Wang, D., Xu, Y., Zhao, H., Wang, L., Cao, X., Chen, Y., and Chen, Q. (2017). A New Resistance Gene against Potato Late Blight Originating from *Solanum pinnatisectum* Located on Potato Chromosome 7. Front. Plant Sci. *8*: 1729.

Yuan, S. K., Liu, X. L., Si, N. G., Dong, J., Gu, B. G., and Jiang, H. (2006). Sensitivity of *Phytophthora infestans* to flumorph: in vitro determination of baseline sensitivity and the risk of resistance. Plant Pathol. 55:258–263.

CHAPTER 2. COMMERCIAL HARD SQUASH CULTIVARS EXHIBIT DIFFERENCES IN SUSCEPTIBILITY TO PHYTOPHTHORA CROWN ROT

Source

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Summary

The Michigan vegetable industry is threatened by the oomycete plant pathogen, *Phytophthora capsici*, which causes crown and fruit rot in a wide range of hosts. *P. capsici*-susceptible hosts represent 67% of the total vegetable production in Michigan and include crops in the Cucurbitaceae, Fabaceae, and Solanaceae families. The Michigan squash processing industry faces significant challenges in limiting disease caused by *P. capsici.* Due to tight profit margins preventive cultural strategies including raised plant beds covered with plastic mulch and the use of drip irrigation are not practical. Fungicide applications are costly and adequate coverage can be difficult due to a dense plant canopy. Also, processors require that fruit meet specific standards for color, water, and sugar content. This research sought to identify commercially available hard squash cultivars with resistance to *P. capsici* crown rot in order to improve disease management. Twelve commercial cultivars of hard squash representing *Cucurbita moschata* and *Cucurbita maxima* were included in an inoculated field study and evaluated for crown rot resistance for two years, fruit rot resistance was evaluated for one year. Additionaly, fruit characteristics relevant to processing, including mesocarp soluble solids, percent dry matter, and average fruit weight were evaluated for two field seasons. Results indicated that *C. moschata* cultivars were less susceptible to crown and fruit rot than *C. maxima.* We also identified that among the *C. maxima* cultivars, ‘Thunder’ and ‘Autumn Cup’ developed age-related resistance to fruit rot; ‘Thunder’ was resistant to fruit rot 40 days after pollination. Fruit characteristic data indicate that the *C. maxima* cultivars Thunder and Autumn Cup and the *C. moschata* cultivar Ultra have the fruit quality characteristics required by the processing industry. In summary, results of this research could improve the integrated management of *P. capsici* in hard squash thus reducing the risk of growing this crop in Michigan.

CHAPTER 3. HOST RESISTANCE IN COMMERCIAL TOMATO CULTIVARS TO MANAGE *PHYTOPHTHORA INFESTANS*

Abstract

Late blight (LB), incited by *Phytophthora infestans*, is a devastating disease of tomato. In 2018 and 2019, 12 and 23 tomato cultivars, respectively, were tested under growth chamber, greenhouse, and field conditions. Plants were inoculated with an isolate of *P. infestans* clonal lineage US-23. In the growth-chamber study, the lowest disease severity at the final assessment (<20%) was observed in 'Matt's Wild Cherry' and 'Tomato Stellar', with significantly less disease than all other cultivars. The rAUDPC data indicated these cultivars were significantly less susceptible than all other cultivars except for 'Mountain Magic' which was similar to 'Matt's Wild Cherry'. In greenhouse experiment 1, 'Mountain Magic', 'Tomato Stellar', and 'Mountain Merit' had the least amount of foliar disease severity (0 – 8.0%) for each observation date. For the field experiment, eleven of the cultivars included in the study had foliar disease severity <5% on the final observation date. According to the rAUDPC data, 'Iron Lady', and 'Defiant' had the lowest disease severity but were similar to 'Lemon Drop'. 'Lemon Drop', 'Cherry Bomb', and 'Fantastico' were similar to each other; 'Plum Regal' was similar to 'Cherry Bomb' and 'Fantastico'. In greenhouse experiment 2, 'Iron Lady', and 'Defiant' had the lowest disease severity but were similar to 'Lemon Drop', according to the rAUDPC data. Disease control in tomatoes grown for both organic and conventional markets could be advanced by using tomato cultivars with resistance to *P. infestans*.

Introduction

Tomato (*Solanum lycopersicum*) is one of the most widely consumed vegetables in the world. In 2018, global tomato production was estimated at 182 million metric tons with China, India, Turkey, and the United States accounting for 89% of the world's production (FAO, 2019). The United States produced about 14 million metric tons of tomatoes in 2018; Michigan growers contributed 137,500 metric tons (USDA 2019).

The oomycete, *Phytophthora infestans*, is an important pathogen of plants within the Solanaceae family, particularly potatoes and tomatoes (Forbes et al. 2013). Described as the causal agent of late blight by Heinrich Anton de Bary in the second half of the 18th century, the pathogen devastated European potato production resulting in the Irish potato famine (Turner 2005). *P. infestans* was first reported in the United States in 1843 in New York. The same year Pennsylvania and Delaware reported crop losses from *P. infestans* of up to 50% of total potato production with New Jersey reporting losses of 15% the following year (Stevens 1933). In 1845, Indiana, Illinois, and Michigan also reported crops infected by *P. infestans* (Stevens 1933).

P. infestans is an important disease of tomato, capable of causing total loss in a susceptible, unprotected crop (Nowicki et al. 2012). The pathogen infects all above-ground tissue of the tomato plant with symptoms appearing as dark greasy lesions on the leaves, stems, and fruit. Under conducive conditions including temperatures of 15 to 22 ℃ and relative humidity greater than 80%, sporangial production may occur on infected tissue (Fry et al. 2013). *P. infestans* has not been reported to overwinter in the United Stated as oospores but can survive in infected potato plant residue, volunteers, and cull piles (Easton 1982; Fry and Goodwin 1997). Disease management strategies include reducing primary inoculum by using certified potato seed and eliminating potato cull piles and volunteers (Kirk and Rosenzweig 2015; Lacy and

Hammerschmidt 1995). Secondary inoculum can be reduced through increased plant spacing and limiting overhead irrigation to reduce leaf wetness (Rotem et al. 1970; Struik 2010).

Fungicides are the primary means for managing late blight on tomatoes (Seidl Johnson et al. 2015). Systemic and contact fungicides are available for conventional production (Midwest production guide) but options are limited for organic growers (Halterman and Gevens 2013). Fungicide efficacy may vary according to the clonal lineage of *P. infestans* (Seidl Johnson et al. 2015). Systemic fungicides applied post infection as foliar or drench applications reduce sporangial production and germination, and lesion expansion (Cohen et al. 1979). Since most systemic fungicides have a single mode of action, there is a high risk of resistance developing in the pathogen (Cohen et al. 1979; Davidse et al. 1981, 1988). Alternating or tank mixing fungicides with different modes of action increases disease control while reducing the risk of pathogen resistance (Quesada-Ocampo et al. 2021; Samoucha and Cohen 1986).

Combining fungicides with host resistance improves late blight management while reducing cost, labor, and fungicide inputs (Nærstad et al. 2007). Choosing resistant cultivars is recommended to manage the disease in organic and conventional production systems (Ghorbani et al. 2004). Resistance genes (*Ph-1, Ph-2, and Ph-3*) obtained from wild tomatoes confer resistance to different *P. infestans* clonal lineages and have been incorporated into commercial cultivars (Foolad et al. 2008). Since *P. infestans* has high genetic diversity (Nowicki et al. 2012), the pathogen may quickly overcome single-gene resistance; incorporating more than one *Ph*-gene in a cultivar confers durable resistance (Goodwin et al. 1995; Nowicki et al. 2012). Tomato cultivars with single resistance genes *Ph-1* have been overcome by the most predominant clonal lineages of *P. infestans* in the United States, clonal lineage US-22, US-23, and US-24. In contrast, tomato cultivars with single *Ph-2* or *Ph-3* resistant gene expressed a moderate or higher

level of resistance against *P. infestans* US-23; although, their resistance is weaker against *P. infestans* US-22 compared to US-23. However, tomato cultivars with *Ph-2 + Ph-3* have provided protection against *P. infestans* clonal lineage US-22, US-23, and US-24 (Hansen et al. 2014; Seidl Johnson et al. 2014).

The objective of our study was to evaluate commercial tomato cultivars for resistance to *P. infestans* clonal lineage US-23 under greenhouse, growth chamber, and field conditions.

Material and Methods

Planting Material. Twenty-eight tomato cultivars were included (Table 1)**.** Seed for all experiments was planted into 72-cell trays containing a soilless peat mixture (Suremix Michigan Grower Products, Inc. Galesburg, MI) and grown for four weeks at the plant science research greenhouse of Michigan State University (MSU) in East Lansing, MI. Five days after germination, plants were sprayed with a 5-ppm solution of a growth regulator (Uniconazole-P, Valent U.S. A. Corporation, San Ramon, Ca). After four weeks, the plants were transplanted to 2.5 L plastic pots containing a soilless peat mixture (Suremix, Michigan Grower Products Inc, Galesburg, MI) and grown for two weeks (growth chamber and greenhouse trials). For the field experiment, four-week-old seedlings were removed from the greenhouse and maintained outside for fourteen days to acclimate before planting.

Table 1. Tomato cultivars, evaluated for resistance to *Phytophthora infestans* clonal lineage US-23, in 2018 and 2019.

Cultivar	Year evaluated	Resistance gene	Fruit Type	Source
Better Boy	2018, 2019	N/A	Slicer/Processing	W. Atlee Burpee & Co[o]
Mountain Merit	2018, 2019	Ph-2 and Ph-3	Slicer	Bejo seeds Inc[p]
Defiant	2018, 2019	Ph-2 and Ph-3	Slicer	Johnny's Selected Seeds[q]
Tomato Stellar	2018, 2019	Ph-2 and Ph-3	Slicer	Pan American Seed Co[r]
Mountain Magic	2018, 2019	Ph-2 and Ph-3	Cocktail	Bejo seeds Inc
Cherry Bomb	2018, 2019	N/A	Cherry	Johnny's Selected Seeds
Amish Paste	2018	N/A	Processing	Johnny's Selected Seeds
Early Girl	2018	N/A	Slicer	Johnny's Selected Seeds
Mr. Stripey	2018	N/A	Slicer	Harris Seeds Company[s]
Damsel	2018	N/A	Slicer	Harris Seeds Company.
Rugged Boy	2018	N/A	Slicer	HPS Seed Company[t]
Matt's Wild Cherry	2018	Ph-3	Cherry	Johnny's Selected Seeds
Iron Lady	2019	Ph-2 and Ph-3	Processing	Nursery Compan[u]
Heatmaster	2019	N/A	Processing	Holmes Seed Co[v]
Grand Marshall	2019	N/A	Processing	Sakata Seed America[w]
Plum Regal	2019	Ph-3	Roma	Bejo seeds Inc.
Little Napoli	2019	N/A	Roma	Pan American Seed Co
Monica Roma	2019	N/A	Roma	Sakata Seed America.
Little Sicily	2019	N/A	Slicer	Pan American Seed Co
Better Bush Hybrid	2019	N/A	Slicer	Tomato Growers Supply Company[x]
Fantastico	2019	N/A	Grape tomato	Park Seed Company[y]
Black Cherry	2019	N/A	Cherry	W. Atlee Burpee & Co.
Chocolate Sprinkles	2019	N/A	Cherry	Pan American Seed Co.
Husky Cherry Red Hybrid	2019	N/A	Cherry	Tomato Growers supply Company
Lemon Drop	2019	N/A	Cherry	Park Seed Company
Sakura	2019	N/A	Cherry	Johnny's Selected Seeds
SunSugar	2019	N/A	Cherry	Seminis[z]
Yellow Pear	2019	N/A	Cherry	W. Atlee Burpee & Co

[o]12844 Creek Rd, Fannettsburg, PA 17221
[p] 1972 Silver Spur Pl, Oceano, CA 93445

Table 1 (cont'd)
[q] 955 Benton Ave, Winslow, ME 04901
[r] 622 Town Rd, West Chicago, IL 60185
[s] 355 Paul Rd, Rochester, NY 14624
[t] 334 W. Stroud St., Ste 1, Randolph, WI 53956
[u]Greendale, IN 47025
[v] 2125 46th St NW, Canton, OH 44709
[w]18095 Serene Dr, Morgan Hill, CA 95037
[x] 12165 Metro Pkwy #14, Fort Myers, FL 33966
[y]3507 Cokesbury Rd Hodges, SC 29653
[z]2700 Camino Del Sol Oxnard, CA 93030

Preparation of Inoculum. A Michigan isolate of US-23 *P. infestans* (mating type A1, sensitive to mefenoxam) was obtained from Dr. Noah Rosenzweig's MSU culture collection. Mycelial plugs from actively growing cultures were sub-cultured onto pea agar (120 g frozen peas, 0.05 g B-sitosterol, 20.0 g sucrose, and 16.0 g agar) for 15 days in darkness at room temperature (20 - 22 ℃) as performed by Goodwin et al. (1994). Sporangial suspensions were prepared for each trial by adding sterile water to an actively growing culture and scraping the surface of the agar with an L-shape cell spreader (VWR, Radnor, PA, U.S.) (Sharma et al. 2010). The sporangial concentration was adjusted to 1.0 x 10^5 sporangia/ml for the growth chamber and greenhouse experiments and 1.0 x 10^4 sporangia/ml for field experiments using a hemocytometer. The sporangial suspension was incubated at 4 ^oC for 2 hours to stimulate zoospore release before inoculating each trial (Sharma et al. 2010).

Evaluation of Tomato Cultivars Under Growth Chamber Conditions. This experiment was conducted in 2018 in growth chambers located on the campus of MSU in East Lansing, MI. Plants previously transplanted into 2.5 L pots were placed inside a wire basket (0.3 m x 0.3 m) manufactured using poultry netting (20 gauge, 0.02 m mesh size, 3.0 m, Grainger, Chicago, IL) and covered with a translucent plastic bag (0.5 m x 0.1 m x 0.7 m, Westrock, Ravena, OH).

Plants were inoculated on 2 July with a 1.0 x 10^5 sporangia/ml suspension of *P. infestans* isolate US-23 using a manual sprayer (473.2 ml round plastic spray bottle with graduations, Impact Products LLC, Toledo, OH) until runoff (Abreu et al. 2008) and incubated in a growth chamber (20^{o}C, 16-hour photoperiod, 97% relative humidity) for 7 days, plant were assessed on 9, 12 and 16 July. Immediately after inoculation, the plants were enclosed in translucent bags containing 200 ml of water to increase relative humidity. Bags were partially closed using a rubber band to allow gas exchange and placed in the growth chamber. The plants were arranged in a completely randomized design (CRD) with four replicates; a replicate consisted of a single tomato plant.

Evaluation of Tomato Cultivars Under Field Conditions. Field experiments were conducted during the summer of 2018 (field experiment 1) and 2019 (field experiment 2) to evaluate 12 and 22 tomato cultivars, respectively, for resistance to *P. infestans* isolate US-23 (Table 1). In both years, the experiments were conducted in a plot located at the MSU Plant Pathology Farm in Lansing, MI. A Capac loam soil site, previously planted to cucumber, was treated prior to planting with glyphosate (Roundup PowerMax at 2.34 liters/ha, Monsanto Company, St. Louis, MO) for weed control. The plot was fertilized pre-planting with nitrogen (114 kg/ha), potassium (205 kg/ha), sulfur (28.4 kg/ha), and boron (2.3 kg/ha) each year. Six-week-old seedlings were transplanted 0.3 m apart into 0.15 m raised beds covered with black polyethylene plastic (0.15 m x 0.6 m) with rows spaced 2.4 m apart on 15 June (field experiment 1) and 20 June (field experiment 2). For plot irrigation, a single drip tape (2.47 LPM/30.5 m) was installed under the plastic mulch. Plants were fertilized weekly (Jack's Professional® 20-20-20 water-soluble fertilizer, JR Peters Inc, Allentown, PA) at 9 kg/ha through the drip tape. One application of Lambda-cyhalothrin (Warrior II with Zeon Technology Insecticide at 0.14 L/ha, Syngenta

Corporation, Greensboro, NC) was made to control tomato hornworm (*Manduca quinquemaculat*). The plot was hand-weeded as needed. Treatments were arranged in a randomized complete block design with four replicates, a replicate consisted of 12 plants in a single 6.1-m row with a 1.3-m buffer between treatments within a row. Each year, the experiments were inoculated with inoculum prepared as previously described using a 1.0×10^4 sporangia/ml suspension of *P. infestans* isolate US-23 on 15, 28 August, and 5 September (field expeiment 1), and 28 August (field experiment 2). In both years, the inoculation was performed using a manual backpack sprayer (Stihl SG 20, Waiblingen, Germany) equipped with a hollow cone nozzle (Stihl 408BA015KN, Waiblingen, Germany) and calibrated to spray approximately 15 ml of inoculum per plant (Abreu et al. 2008).

Cultivar Evaluation Under Greenhouse Conditions. In 2019, 22 cultivars were evaluated in two trials at the MSU Plant Science Greenhouse in East Lansing, MI. The temperature was set at 26 ℃ with a 16 hr photoperiod. Eight cultivars were evaluated from 1 to 13 May (greenhouse experiment 1) and 14 cultivars were evaluated from 16 May to 3 June (greenhouse experiment 2). Plants previously transplanted into 2.5 L pots were placed inside a wire basket following the procedure described for the growth chamber experiment. Plants were inoculated with approximately 15 ml of a 1.0×10^5 sporangia/ml suspension of *P. infestans* isolate US-23 using a manual sprayer (473.2 ml round plastic spray bottle with graduations, Lansing, MI, U.S.) until runoff on 1 May (greenhouse experiment 1) and 16 May (greenhouse experiment 2) (Abreu et al. 2008). Immediately after inoculation, plants were enclosed in translucent bags as previously described for the growth chamber experiments. In both experiments, the cultivars were arranged in a CRD with ten replications, where an experimental unit consisted of a single plant.

Data Collection and Statistical Analysis. Foliar disease was evaluated for all experiments (growth chamber, field, and greenhouse) by visually assessing disease severity as the percentage of foliar blight for each experimental unit (single plant for the growth chamber and greenhouse experiments and 20 plants in a single plot replicate for field experiments) following James (1971). For the growth chamber evaluation, foliar disease severity (%) was visually assessed three times in one week (which spanned the time from disease onset to death of the susceptible plants) using a 0-100% scale, where 0= healthy plant and 100= dead plant. For the field experiments, single evaluations of foliage with symptoms (%) of late blight were taken for field experiment 1. For field experiment 2, the foliage (%) with LB symptoms was visually assessed, beginning seven days after inoculation, and continuing every 7-days and ending 48 days after inoculation. The greenhouse experiments were visually assessed as described for the growth chamber experiments.

The relative area under the disease progress curve (rAUDPC) was calculated for all trials using the formula ((AUDPC)/N)) as proposed by Fry (1978), where the standard area under disease progress curve (AUDPC) (Shaner and Finney 1977) was divided by the number of days (N) from the inoculation date to the final rating date.

Foliar disease severity at the final rating date and rAUDPC for all experiments was analyzed using SAS 9.4 (SAS Institute, Cary, NC) using PROC GLIMMIX. The normality and homogeneity of variance assumptions for each data set were investigated by observing the residuals plots, residuals versus predicted means plots, percentage of residual distribution histogram, and Levene's test ($P<0.05$). Log transformation and unequal variance models were used when necessary to meet ANOVA assumptions of normality and equality of variances.

Fisher's protected least significant difference LSD test was used for cultivar pairwise comparisons ($P<0.05$).

Results

Evaluation of Tomato Cultivars Under Growth Chamber Conditions. Initial late blight symptoms included water-soaked lesions (Forbes et al. 2013) and were observed seven days after inoculation. At the first assessment on 9 July, seven days after inoculation, foliar disease severity ranged from 7.5% ('Tomate Stellar') to 94.5% ('Amish Paste') (Table 2).

'Better Boy', 'Mr. Stripey', 'Early Girl', and 'Amish Paste' were the most susceptible cultivars at the last disease evaluation (16 July) with greater than 90% foliar disease severity and the highest rAUDPC values. 'Damsel', 'Rugged Boy', 'Mountain Merit' and 'Cherry Bomb' exhibited less disease severity (60.0 - 73.8%) compared to the most susceptible cultivars. Although these cultivars were similar to each other at the final assessment for foliar disease severity, according to rAUDPC data, 'Cherry Bomb' was less susceptible than 'Damsel' and 'Rugged Boy'; 'Damsel', 'Rugged. Boy', and 'Mountain Merit' were similar to each other. 'Defiant' and 'Mountain Magic' (36.3 - 60.0 % foliar disease severity) were among the least susceptible cultivars at the final evaluation and according to the rAUDPC. The lowest disease severity at the final assessment (<20%) was observed in 'Matt's Wild Cherry' and 'Tomato Stellar', with significantly less disease than all other cultivars. The rAUDPC data indicated these cultivars were significantly less susceptible than all other cultivars except for 'Mountain Magic' which was similar to 'Matt's Wild Cherry'.

Table 2. Foliar disease severity (%) and relative area under the disease progress curve (rAUDPC) of tomato cultivars inoculated with *Phytophthora infestans* clonal lineage US-23 on 2 July and incubated under growth chamber conditions in 2018.

	Foliar disease severity (%)			
Cultivars	**July-9**	**July-12**	**July-16**	**rAUDPC[y]**
Amish Paste	94.5 a[z]	95.8 a	97.3 a	47.9 a
Early Girl	92.5 a	95.0 a	96.0 a	47.9 a
Better Boy	83.3 ab	88.8 a	90.8 a	44.1 a
Mr. Stripey	70.0 a-c	85.3 a	91.5 a	41.9 a
Damsel	58.8 b-d	67.5 b	73.8 b	33.1 b
Rugged Boy	60.0 b-d	62.5 b	71.3 b	32.2 b
Mountain Merit (*Ph*-2 + *Ph*-3)	46.7 dc	63.3 b	70.0 b	30.1 bc
Cherry Bomb	40.0 d	47.5 c	60.0 b	24.7 c
Defiant (*Ph*-2 + *Ph*-3)	18.8 e	35.0 cd	37.5 c	16.1 d
Mountain Magic (*Ph*-2 + *Ph*-3)	18.8 e	27.5 de	36.3 c	14.1 ed
Matt's Wild Cherry	12.5 ef	17.5 f	18.8 d	8.4 ef
Tomato Stellar (*Ph*-2 + *Ph*-3)	7.5 f	13.3 ef	15.0 d	6.3 f
P-value	<.0001	<.0001	<.0001	<.0001

[Y] rAUDPC Calculated using three observations of disease severity (%) divided by 14 days (time from inoculation to the last rating date).
[z]Column means with a letter in common are not statistically different (Fisher's protected least significant difference LSD; P=0.05).

Evaluation of Tomato Cultivars Under Field Conditions. Field experiment 1 (2018) was inoculated on 15, 18 Aug and 6 Sept; disease symptoms were observed on 18 September and severity assessed. Differences among the tomato cultivars evaluated were not statistically significant (P=0.2613) (Table 6).

In field experiment 2, initial disease symptoms were observed on 28 August, 35 days after inoculation (Table 3). At the first observation date of 2 October, only four cultivars developed late blight severity greater than 2%; two cultivars ('Black Cherry' and 'Chocolate Sprinkles') developed disease severity ranging from 20.3 to 23.8% which was significantly more than all other cultivars. 'Monica Roma' and 'Grand Marshall' developed 2.5% foliar disease severity which was significantly greater than the cultivars displaying negligible (0 to 0.2%)

disease severity symptoms. On the second observation date of 6 October, overall disease severity remained low for most cultivars. Disease severity increased > 4% from the first observation period for ‘Husky Cherry’, ‘Monica Roma’, and ‘Grand Marshall’. ‘Black Cherry’ and ‘Chocolate Sprinkles’ had a significantly higher disease severity than all other cultivars on this observation date. For the third assessment, foliar disease severity remained relatively static with disease increase (>4%) observed for ‘Monica Roma’. On the final observation date of 13 October, late blight symptoms were observed on each cultivar. Eleven of the cultivars included in the study had foliar disease severity <5%. An intermediate level of foliar disease severity (11.0 – 24.8%) was observed for eight cultivars. ‘Grand Marshall’, ‘Black Cherry’, and ‘Chocolate Sprinkles’ had significantly more foliar disease severity (29.5% - 43.8%) than most other cultivars on the last assessment date. According to the rAUDPC data, only ‘Black Cherry’ and ‘Chocolate Sprinkles’ were significantly more susceptible to late blight than all other cultivars included in this study. All other cultivars were similar in their disease susceptibility.

Table 3. Foliar disease severity (%) and relative area under the disease progress curve (rAUDPC) of tomato cultivars inoculated with *Phytophthora infestans* clonal lineage US-23 on 28 August for field experiment 2 in 2019.

	Foliar disease severity (%)				
Cultivars	**Oct-2**	**Oct -6**	**Oct-7**	**Oct-13**	**rAUDPC[y]**
Cherry Bomb	0.0 c[z]	0.0 e	0.1 e	1.5 f	0.02 b
Iron Lady (*Ph*-2 + *Ph*-3)	0.0 c	0.0 e	0.0 e	1.5 f	0.04 b
Lemon Drop	0.0 c	0.0 e	0.0 e	1.5 f	0.04 b
Mountain Magic (*Ph*-2 + *Ph*-3)	0.0 c	0.0 e	0.0 e	1.5 f	0.04 b
Mountain Merit (*Ph*-2 + *Ph*-3)	0.0 c	0.0 e	0.0 e	1.5 f	0.04 b
Tomato Stellar (*Ph*-2 + *Ph*-3)	0.0 c	0.0 e	0.0 e	1.5 f	0.04 b
Defiant (*Ph*-2 + *Ph*-3)	0.0 c	0.0 e	0.0 e	1.5 f	0.05 b
Yellow Pear	0.0 c	0.0 e	0.0 e	1.7 f	0.1 b
Plum Regal	0.0 c	0.1 de	0.3 e	3.5 ef	0.2 b
Fantastico	0.0 c	0.0 e	0.1 e	4.3 de	0.3 b
Little Sicily	0.0 c	0.3 de	0.3 de	3.5 ef	0.3 b
Little Napoli	0.0 c	0.0 e	1.4 c-e	12.8 c-f	0.9 b
Better Bush	0.1 c	0.8 c-e	1.5 c-e	12.8 c-f	1.0 b
Heat Master	0.2 c	1.5 c-e	0.8 de	17.5 b-d	1.3 b
Sun Sugar	0.1 c	1.5 c-e	1.5 c-e	11.0 c-f	1.4 b
Sakura	0.0 c	0.0 e	3.9 c-e	15.5 b-e	1.5 b
Better Boy	0.1 c	2.5 c-e	5.3 c-e	21.5 bc	1.9 b
Husky Cherry	0.1 c	4.8 b-d	6.3 b-d	20.3 b-d	2.0 b
Monica Roma	2.5 b	8.9 bc	15.1 bc	24.8 b-e	3.4 b
Grand Marshall	2.5 b	13.3 b	17.0 ab	43.8 a	4.9 b
Black Cherry	20.3 a	20.5 a	21.8 a	29.5 ab	13.7 a
Chocolate Sprinkles	23.8 a	24.5 a	24.5 a	30.0 ab	15.3 a
P-value	<.0001	<.0001	<.0001	<.0001	<.0001

[Y] rAUDPC Calculated using nine observations of disease severity (%) divided by 48 days.
[z]Column means with a letter in common are not statistically different (Fisher's protected least significant difference LSD; P=0.05).

Evaluation of Tomato Cultivars Under Greenhouse Conditions. In greenhouse experiment 1, 'Mountain Magic', 'Tomato Stellar', and 'Mountain Merit' had the least amount of foliar disease severity (0 – 8.0%) for each observation date (Table 4). On the first observation date of 7 May, 'Monica Roma' developed an intermediate amount of late blight disease (27.5%) which was not significantly different from 'Sakura', 'Better Boy', and 'Husky Cherry' but was significantly less

diseased than ‘Better Bush’ (42.0%). On the second and third assessment dates, ‘Sakura’, ‘Monica Roma’, ‘Better Boy’, ‘Husky Cherry’, and ‘Better Bush’ had a similar level of foliar disease severity (57.5 – 80.0%). According to the rAUDPC data, ‘Mountain Magic’, ‘Tomato Stellar’, and ‘Mountain Merit’ were the least susceptible cultivars among those included in this trial.

Table 4. Foliar disease severity (%) and relative area under the disease progress curve (rAUDPC) of tomato cultivars inoculated with *Phytophthora infestans* clonal lineage US-23 on 1 May for greenhouse experiment 1 in 2019.

	Foliar disease severity (%)			
Tomato cultivars	**May-7**	**May-9**	**May-13**	**rAUDPC[w]**
Mountain Magic (Ph-2 + Ph-3)	0.0 c[z]	0.0 b	0.0 b	0.0 b
Tomato Stellar (Ph-2 + Ph-3)	0.5 c	0.5 b	0.5 b	0.2 b
Mountain Merit (Ph-2 + Ph-3)	4.5 c	6.5 b	8.0 b	3.3 b
Sakura	33.5 ab	62.0 a	70.5 a	25.4 a
Monica Roma	27.5 b	57.5 a	78.5 a	27.5 a
Better Boy	37.0 ab	66.0 a	68.9 a	28.7 a
Husky Cherry	36.0 ab	63.5 a	80.0 a	29.7 a
Better Bush	42.0 a	67.0 a	75.5 a	30.3 a
P-value	<0.0001	<0.0001	<0.0001	<0.0001

[y]rAUDPC calculated using three observations of disease severity (%) divided by 12 days.
[z]Column means with a letter in common are not statistically different (Fisher’s protected least significant difference LSD; P=0.05).

In greenhouse experiment 2, the first disease severity assessment on 20 May indicated that ‘Defiant’, ‘Iron Lady’, ‘Lemon Drop’, and ‘Cherry Bomb’ were either not diseased or had low levels (≤6.3%) of foliar blight and were significantly different than nearly all other cultivars tested (Table 5). Several cultivars including ‘Fantastico’, ‘Plum Regal’, ‘Little Sicily’, ‘Little Napoli’, and ‘Chocolate Sprinkles’ developed an intermediate level of disease symptoms ranging from 12.5 – 19.5%; except for Fantastico, these cultivars were similar to ‘Heat Master’, ‘Black

Cherry', and 'Grand Marshall'. 'Sun Sugar' and 'Yellow Pear' were significantly more diseased (29.0-29.5%) than all other cultivars except for 'Heat Master', 'Black Cherry', and 'Grand Marshall'. For the second foliar disease severity rating (23 May), all cultivars developed disease although severity remained low (0.1 – 13.6%) for 'Defiant', 'Iron Lady', 'Lemon Drop', and 'Cherry Bomb'. The cultivars with an intermediate level of foliar disease severity (22.8 – 35.5%) included 'Fantastico', 'Plum Regal', 'Little Sicily', 'Little Napoli', and 'Heat Master'. 'Sun Sugar' and 'Yellow Pear' had the greatest disease severity (50.5 – 52.0%) but were similar to 'Black Cherry' and 'Grand Marshall'.

On the third and fourth assessments (29 May and 3 June), disease severity increased for nearly all cultivars. 'Defiant' and 'Iron Lady' were significantly less diseased than all other cultivars for both assessment dates. On 29 May, 'Lemon Drop', 'Cherry Bomb', and 'Fantastico' were similar and had less disease severity than most of the cultivars included in the study. A group of cultivars that were similar with an increased level of disease included 'Plum Regal', 'Little Sicily', 'Little Napoli', 'Chocolate Sprinkles', 'Heat Master', and 'Black Cherry'. In contrast, on 3 June, 'Lemon Drop', 'Cherry Bomb', 'Fantastico', and 'Plum Regal' were similar in their foliar disease severity (19.6 - 33.5%); 'Little Sicily' and 'Little Napoli' were similar to 'Plum Regal'. A group with a similar level of increased foliar disease severity (45.0 – 58.0%) included 'Little Napoli', 'Chocolate Sprinkles', 'Heat Master', and 'Black Cherry'. 'Grand Marshall' with 68% foliar disease severity was similar to 'Black Cherry' (58%) and 'Sun Sugar' (81.5%). For the 29 May and 3 June assessments, the most susceptible cultivars were 'Grand Marshall', 'Sun Sugar', and 'Yellow Pear' with 'Yellow Pear' having a greater level of foliar disease severity than 'Grand Marshall'.

According to the rAUDPC data, ‘Iron Lady’, and ‘Defiant’ had the lowest disease severity but were similar to ‘Lemon Drop’. ‘Lemon Drop’, ‘Cherry Bomb’, and ‘Fantastico’ were similar to each other; ‘Plum Regal’ was similar to ‘Cherry Bomb’ and ‘Fantastico’. A group of cultivars with a similar level of intermediate disease included ‘Plum Regal’, ‘Little Sicily’, ‘Little Napoli’, ‘Chocolate Sprinkles’, and ‘Heat Master’; ‘Black Cherry’ was similar to these cultivars with the exception of ‘Plum Regal’. The most susceptible cultivars based on rAUDPC data included ‘Grand Marshall’, ‘Sun Sugar’, and ‘Yellow Pear’; ‘Yellow Pear’ had a significantly higher rAUDPC value than ‘Grand Marshall’.

Table 5. Foliar disease severity (%) and relative area under the disease progress curve (rAUDPC) of tomato cultivars inoculated with *Phytophthora infestans* clonal lineage US-23 on 16 May for greenhouse experiment 2 in 2019.

	Foliar disease severity (%)				
Tomato cultivars	**May-20**	**May-23**	**May-29**	**June-3**	**rAUDPC[y]**
Defiant	0.0 e[z]	0.1 f	0.7 g	2.1 i	0.5 i
Iron Lady	0.0 e	1.5 ef	1.5 g	2.0 i	1.1 i
Lemon Drop	3.1 e	7.6 ef	17.5 f	19.6 h	9.7 hi
Cherry Bomb	6.3 ed	13.6 de	19.0 f	23.5 h	12.3 gh
Fantastico	12.5 dc	22.8 cd	29.0 ef	27.5 gh	18.3 f-h
Plum Regal	14.6 b-d	30.5 bc	36.0 de	33.5 f-h	23.2 e-g
Little Sicily	17.0 bc	30.5 bc	41.0 de	41.0 e-g	25.8 d-f
Little Napoli	19.5 bc	31.8 bc	41.5 de	45.0 de	27.0 de
Chocolate Sprinkles	17.0 bc	29.5 bc	44.5 de	52.0 de	27.9 de
Heat Master	21.1 ab	35.5 b	44.5 c-e	45.0 de	28.9 de
Black Cherry	23.0 ab	41.0 ab	50.5 dc	58.0 cd	33.8 cd
Grand Marshall	22.5 ab	43.0 ab	58.0 bc	68.0 bc	37.7 bc
Sun Sugar	29.0 a	50.5 a	69.5 ab	81.5 ab	45.1 ab
Yellow Pear	29.5 a	52.0 a	77.5 a	89.0 a	48.8 a
P-value	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

[y] rAUDPC calculated using four observations of disease severity (%) divided by 18 days.
[z]Column means with a letter in common are not statistically different (Fisher's protected least significant difference LSD; P=0.05).

Discussion

Genetic resistance is one of the most effective disease management strategies for late blight control (Nowicki et al. 2012) but this strategy alone may not be sufficient (Fry 2008). Growers using fungicides and/or biopesticide products may benefit from an integrated approach (Seidl Johnson et al. 2014; Seidl Johnson et al. 2015). In the United States, *P. infestans* has several clonal lineages originating from mitotic recombination, mutation, or migration. Pathogenicity differences among

hosts may result from this diversity as some clonal lineages affect tomato, potato, or both. Genetic diversity may allow *P. infestans* to rapidly develop resistance to fungicides and overcome host resistance (Danies et al. 2013; Goodwin et al. 1995; Malcolmson 1969).

In Pennsylvania, Foolad et al. (2014) evaluated the efficacy of resistance genes *Ph-1*, *Ph-2*, and *Ph-3* by studying different accessions of *S. pimpinellifolium* carrying one or more of the resistance genes. They observed that accessions carrying the *Ph-1* resistance gene were the most susceptible to *P. infestans*, while accessions carrying resistance genes *Ph-2* and *Ph-3* showed an intermediate and high level of resistance, respectively. Other wild tomato species with resistance to *P. infestans* include *S. habrochaites* and *Lycopersicon pennellii* (Li et al. 2011; Smart et al. 2007). Overall, the highest level of resistance was observed in cultivars carrying both resistance genes *Ph-2 and Ph-3* due to resistance to multiple clonal lineages of *P. infestans* (Sanchez-Perez et al. 2017).

The resistance genes *Ph-2* and *Ph-3* present in wild relatives of tomato including *Solanum pimpinellifolium* have been integrated into commercial tomato cultivars (Foolad et al. 2014; Kim and Mutschler 2006). However, when one of these genes was incorporated alone into a tomato cultivar, there was a significant but not complete disease reduction (Foolad et al. 2008; Foolad et al. 2014). In our research, we have demonstrated that tomato cultivars with the resistance genes *Ph-2* and *Ph-3* (Table 1) effectively limit *P. infestans* US-23, the dominant clonal lineage in Michigan. ‘Mountain Magic’ and ‘Tomato Stellar’ were consistently the least susceptible cultivars in all experiments (growth chamber, greenhouse, and field). ‘Mountain Merit’, ‘Defiant’, ‘Iron Lady’, and ‘Cherry Bomb’ exhibited an intermediate level of late blight resistance similar to results of Hansen et al. (2014) and Seidl Johnson et al. (2014).

Six cultivars were evaluated under growth chamber, greenhouse, and field conditions for two years (Table 6), while the other cultivars were evaluated under some, but not all conditions. Of the six cultivars evaluated under all conditions both years, we observed that while cultivars with one or more resistance genes had little or no disease in the field and greenhouse, they developed moderate levels of disease when evaluated under growth chamber conditions. The growth chamber conditions were 20 ^{o}C, 16 hours photoperiod, and 97% relative humidity to favor pathogen sporulation, germination, and penetrations (Harrison and Lowe 1989; Maziero et al. 2009; Xiang and Judelson 2014), which increased disease pressure. The defense mechanism of tomato cultivars with the *Ph-3 gene* at three different plant stages (3, 6 and 9 leaves), includes a reduction in disease incidence as the plants aged ≥9 leaves, suggesting age-related resistance (Rashad et al. 2015). The plants used for the growth chamber experiments had fewer than 9 leaves and under the conducive conditions in the growth chamber, disease was observed even when the cultivars had the resistance genes.

Combining host resistance with fungicides for late blight control in potato was tested by Nærstad et al. (2007), and included cultivars with different levels of resistance. They observed that even the most resistant cultivars developed disease symptoms when weather conditions were conducive for disease and fungicides were not applied. However, the performance of the resistant cultivars was improved when paired with fungicides. Perla and Hausbeck (2019) paired ‘Better Boy’, ‘Damsel’, ‘Mr. Stripey’, and ‘Mountain Merit’ with biopesticide fungicides to control foliar diseases of tomato. Late blight development was limited but occurred when cultivars were not treated with fungicides; little to no disease developed when fungicides were applied.

A survey performed by Hoagland et al. (2015) identified late blight as one of the top ten most problematic diseases among conventional and organic tomato growers in the Midwest. We confirmed that commercial cultivars with one or two resistance genes can limit late blight symptoms without fungicide applications. However, host resistance should be utilized with an integrated disease management approach to prevent late blight when weather conditions are conducive to disease.

Table 6. Summary of the foliar disease (%) at the last rating day of the tomato cultivars evaluated under different conditions for resistance to *Phytophthora infestans* US-23 in 2018 and 2019.

	2018 trials		2019 trials		
Cultivars	**Field**	**Growth chamber**	**Field**	**GH-1**	**GH-2**
Better Boy	3.0	90.8 a	21.5 ab	68.9 ab	-
Mountain Magic	0.0	36.3 cd	1.5 c	0.0 c	-
Mountain Merit	0.0	70.0 ab	1.5 c	8.0 bc	-
Tomato Stellar	0.0	15.0 d	1.5 c	0.5 c	-
Defiant	0.0	37.5 cd	1.5 c	-	2.1 h
Cherry Bomb	0.0	60.0 bc	1.5 c	-	23.5 f-h
Matt's Wild Cherry	0.0	18.8 d	-	-	-
Mr. Stripey	0.0	91.5 a	-	-	-
Amish Paste	0.0	97.3 a	-	-	-
Rugged Boy	0.0	71.3 ab	-	-	-
Damsel	5.0	73.8 ab	-	-	-
Early Girl	13.0	96.0 a	-	-	-
Husky Cherry	-	-	20.3 a-c	80.0 a	-
Monica Roma	-	-	24.8 a-c	78.5 a	-
Better Bush	-	-	12.8 a-c	75.5 a	-
Sakura	-	-	15.5 a-c	70.5 a	-
Lemon Drop	-	-	1.5 c	-	19.6 gh
Iron Lady	-	-	1.5 c	-	2.0 h
Yellow Pear	-	-	1.7 bc	-	89.0 a
Plum Regal	-	-	3.5 bc	-	33.5 d-g
Little Sicily	-	-	3.5 bc	-	41.0 d-g
Fantastico	-	-	4.3 bc	-	27.5 e-g

Table 6 (cont'd)

Sun Sugar	-	-	11.0	a-c	-	81.5	ab
Little Napoli	-	-	12.8	a-c	-	45.0	c-f
Heat Master	-	-	17.5	a-c	-	45.0	c-f
Black Cherry	-	-	29.5	a	-	58.0	b-d
Chocolate Sprinkles	-	-	30.0	a	-	52.0	c-e
Grand Marshall	-	-	43.8	a	-	68.0	a-c
P-value	0.2613	<.0001	<0.0001		<0.0001	<0.0001	

LITERATURE CITED

Abreu, F., Henriques da Silva, D. J., Damião Cruz, C., and Gomide Mizubuti, E. S. 2008. Inheritance of resistance to *Phytophthora infestans* (Peronosporales, Pythiaceae) in a new source of resistance in tomato (*Solanum sp.* (formerly Lycopersicon sp.), Solanales, Solanaceae). Genet. Mol. Biol. *31*: 493–497.

Cohen, Y., Reuveni, M., and Eyal, H. 1979. The Systemic Antifungal Activity of Ridomil against *Phytophthora infestans* on Tomato Plants. Phytopathology 69: 645–649.

Danies, G., Small, I. M., Myers, K., Childers, R., and Fry, W. E. 2013. Phenotypic Characterization of Recent Clonal Lineages of *Phytophthora infestans* in the United States. Plant Dis. 97:873–881.

Davidse, L. C., Looijen, D., Turkensteen, L. J., and Van Der Wal, D. 1981. Occurrence of metalaxyl-resistant strains of *Phytophthora infestans* in Dutch potato fields. Neth. J. Plant. Path. 87: 65–68.

Davidse, L. C., Gerritsma, O. C. M., Deler, J., Pie, K., and Velthuis, G. C. M. 1988. Antifungal modes of action of metalaxyl, cyprofuram, benalaxyl and oxadixyl in phenylamide-sensitive and phenylamide- resistant strains of *Phytophthora megasperma* f. sp. *medicaginis* and *Phytophthora infestans*. Crop Prot.7:347–355.

Easton, G. D. 1982. Late Blight of Potatoes and Prediction of Epidemics in Arid central Washington State. Plant Dis. 66: 452–455.

FAO. 2019. Food and Agriculture Organization of the United Nations. Available online*:* Https://www.Fao.Org/Faostat/En/#home.

Foolad, M. R., Merk, H. L., and Ashrafi, H. 2008. Genetics, Genomics and Breeding of Late Blight and Early Blight Resistance in Tomato. Crit. Rev. Plant Sci. *27*: 75–107.

Foolad, M. R., Sullemberger, M. T., Ohlson, E. W., and Gugino, B. K. 2014. Response of accessions within tomato wild species, *Solanum pimpinellifolium* to late blight. Plant Breed. *133*: 401–411.

Forbes, G. A., Morales, J. G., Restrepo, S., Perez, W., Gamboa, S., Ruiz, R., Cedeño, L., Fermin, G., Andreu, A. B., Acuña, I., and Oliva, R. 2013. *Phytophthora infestans* and *Phytophthora andina* on Solanaceous Host in South America. In K. Lamour (Ed.), *Phytophthora: A Global Perspective* (CABI plant, p. 244). CAB international.

Fry, W. E. 1978. Quantification of General Resistance of Potato Cultivars and Fungicide Effects for Integrated Control of Potato Late Blight. Phytopathology 68: 1650–1655.

Fry, W. E., and Goodwin, S. B. 1997. Re-emergence of Potato and tomato Late Blight in the United States. Plant Dis. 81: 1349–1357.

Fry, W. E. 2008. Phytophthora infestans: The plant (and R gene) destroyer. Mol. Plant Pathol.9: 385–402.

Fry, W. E., McGrath, M. T., Seaman., A., Zitter, T. A., McLeod, A., Danies, G., Small, I. M., Myers, K., Everts, K., Gevens, A. J., Gugino, B. K., Johnson, S. B., Judelson, H., Ristaino, J., Roberts, P., Secor, G., Seebold, Jr. K., Snover-Clift, K., Wyenandt, A., Grünwald, N. J., Smart, C. D. 2013. The 2009 Late Blight Pandemic in the Eastern United States – Causes and Results. Plant Dis. 97:296-306

Ghorbani, R., Wilcockson, S. J., Giotis, C., and Leifert, C. 2004. Potato late blight management in organic agriculture. In *Outlooks on Pest Management* (Vol. 15, Issue 4, pp. 176–180).

Goodwin, S. B., Cohen, B. A., Deahl, K., and Fry, W. E. 1994. Migration from Northern Mexico as the Probable Cause of Recent Genetic Changes in Population of Phytophthora infestans in the United States and Canada. Phytopathology 84:553–558.

Goodwin, S. B., Sujkowski, S. L., and Fry, W. E. 1995. Rapid Evolution of Pathogenicity Within Clonal Lineages of the Potato Late Blight Disease Fungus. Phytopathology 85: 669.

Halterman, D., and Gevens, A. J. 2013. Phytophthora infestans in the USA. In L. Kurt (Ed.), *Phytophthora: A Global Perspective* (CABI plant, p. 244). CABI.

Hansen, Z. R., Small, I. M., Mutschler, M., Fry, W. E., and Smart, C. D. 2014. Differential susceptibility of 39 tomato varieties to *Phytophthora infestans* clonal lineage US-23. Plant Dis. 98:1666–1670.

Harrison, J. G., and Lowe, R. 1989. Effects of humidity and air speed on sporulation of *Phytophthora infestans* on potato leaves. Plant Pathol. 38: 585–591.

Hoagland, L., Navazio, J., Zystro, J., Kaplan, I., Vargas, J. G., and Gibson, K. 2015. Key Traits and Promising Germplasm for an Organic Participatory Tomato Breeding Program in the U.S. Midwest. HortScience 50:1301–1308.

James, W. C. 1971. An Illustrated Series of Assessment Keys for Plant Diseases, Their Preparation and Usage. Can. Plant Dis. Surv. 51: 39–65.

Kim, M. J., and Mutschler, M. A. 2006. Characterization of Late Blight Resistance Derived from Solanum pimpinellifolium L3708 against Multiple Isolates of the Pathogen Phytophthora infestans. J. Amer. Soc. Hort. Sci. 131: 637–645.

Kirk, W. W., and Rosenzweig, N. 2015. *Potato late blight recommendations for 2015*. Extension Newsletters. https://www.canr.msu.edu/news/potato_late_blight_recommendations

Lacy, M. L., and Hammerschmidt, R. 1995. Best Management Practices for Potatoes. *Diseases of Potato: Late Blight* (p. 4). Michigan State University Extension.

Li, J., Liu, L., Bai, Y., Finkers, R., Wang, F., Du, Y., Yang, Y., Xie, B., Visser, R. G. F., and van Heusden, A. W. (2011). Identification and mapping of quantitative resistance to late blight (Phytophthora infestans) in *Solanum habrochaites* LA1777. Euphytica 179:427–438.

Malcolmson, J. F. 1969. Races of Phytophthora infestans occurring in Great Britain. TBMS. *53*: 417-423.

Maziero, J. M. N., Maffia, L. A., and Mizubuti, E. S. G. 2009. Effects of Temperature on Events in the Infection Cycle of Two Clonal Lineages of Phytophthora infestans Causing Late Blight on Tomato and Potato in Brazil. Plant Dis. *93*:459–466.

Nærstad, R., Hermansen, A., and Bjor, T. 2007. Exploiting host resistance to reduce the use of fungicides to control potato late blight. Plant Pathol. *56*:156-166.

Nowicki, M., Foolad, M. R., Nowakowska, M., and Kozik, E. U. 2012. Potato and tomato late blight caused by phytophthora infestans: An overview of pathology and resistance breeding. Plant Dis. 96: 4–17.

Perla, D. E., and Hausbeck, M. K. 2019. Evaluation of cultivars and organic fungicides for management of late blight, bacterial spot, and early blight on tomato, 2018. PDMR 13: V134.

Quesada-Ocampo, L. M., Meadows, I., and Gorny, A. 2021. Disease control for commercial vegetables. *North Carolina Agricultural Chemicals Manual*.

Rashad, S., Shah, A., Li, T., Haijuan, C., Lei, L., Zheng, Z., Li, J., and Yongchen, D. U. 2015. Horticultural Plant Journal Age-Related Resistance and the Defense Signaling Pathway of Ph-3 Gene Against *Phytophthora infestans* in Tomatoes. Hortic. Plant J. 1:70–76.

Rotem, J., Palti, J., and Lomas, J. 1970. Effects of Sprinkler Irrigation at Various Times of the Day on Development of Potato Late Blight. Phytopathology 60:839–843.

Samoucha, Y., and Cohen, Y. 1986. Efficacy of Systemic and Contact Fungicides Mixtures in Controlling Late Blight in Potatoes. Phytopathology 76: 855–859.

Sanchez-Perez, A., Halterman, D., Jordan, S., Chen, Y., and Gevens, A. J. 2017. RB and Ph resistance genes in potato and tomato minimize risk for oospore production in the presence of mating pairs of *Phytophthora infestans*. Eur. J. Plant Pathol. 149: 853–864.

Seidl Johnson, A. C., Jordan, S. A., and Gevens, A. J. 2014. Novel resistance in heirloom tomatoes and effectiveness of resistance in hybrids to *Phytophthora infestans* US-22, US-23, and US-24 clonal lineages. Plant Dis. 98: 761–765.

Seidl Johnson, A. C., Jordan, S. A., and Gevens, A. J. 2015. Efficacy of organic and conventional fungicides and impact of application timing on control of tomato late blight caused by US-22, US-23, and US-24 isolates of *Phytophthora infestans*. Plant Dis. 99: 641–647.

Shaner, G., and Finney, R. E. 1977. The Effect of Nitrogen Fertilization on the Expression of Slow-Mildewing Resistance in Knox Wheat. Phytopathology 67: 1051–1056.

Sharma, K., Butz, A. F., and Finckh, M. R. 2010. Effects of host and pathogen genotypes on inducibility of resistance in tomato (*Solanum lycopersicum*) to *Phytophthora infestans*. Plant Pathol. 59: 1062–1071.

Smart, C. D., Tanksley, S. D., Mayton, H., and Fry, W. E. (2007). Resistance to *Phytophthora infestans* in Lycopersicon pennellii. Plant Dis. 91:1045–1049.

Stevens, N. E. 1933. The dark ages in plant pathology in America:1830-1870. J. Wash. Acad. Sci. *23*: 435–446.

Struik, P. C. 2010. Can Physiology Help Us to Combat Late Blight in Potato? Potato Research *53*:277–287.

Turner, R. S. (2005). After the famine: Plant pathology, *Phytophthora infestans*, and the late blight of potatoes, 1845–1960. HSNS. 35:341–370.

USDA 2019. *Vegetable 2019 summary.* Department of Agriculture, National Agriculture Statistics Service. Online: Http://Nass.Usda.Gov.

Xiang, Q., and Judelson, H. S. 2014. Myb transcription factors and light regulate sporulation in the oomycete *Phytophthora infestans*. *PLoS ONE 9:4* e92086.

FUTURE WORK

Michigan processing hard squash growers are limited to using reduced disease management strategies in their production systems due to low-profit margins. Therefore, using cultivars with genetic resistance is an important option to reduce the negative impact caused by *Phytophthora capsici.* We identified commercial cultivars of hard squash with resistance to *P. capsici* that also express desirable quality characteristics for the processing industry. However, the genetic diversity of *P. capsici* indicates that these cultivars may need to be evaluated using a wider range of isolates collected from different growing regions in the state. Phytophthora crown root resistance was observed in some of the cultivars evaluated. Investigating the mechanism governing resistance could provide insights to determine whether the plants are resistant throughout development or if resistance is age-related. This information could inform growers as to the susceptible period. Perhaps the susceptible cultivars could be induced to express resistance by using resistance inducers such as salicylic acid. We observed that *C. moschata* cultivars developed age-related resistance and investigating fungicides that could be used during the early stages of the fruit development would be helpful. Additionally, we identified tomato cultivars with resistance to *P. infestans* that could be used for conventional or organic tomato production; availability of effective biorational products is limited. A multi-pronged treatment program is needed to limit *P. infestans*. Therefore, additional research is necessary to identify a range of plant protection products that could be paired with resistant cultivars to improve the management of *P. infestans* in tomatoes.