ANNUAL REVIEWS

Annual Review of Phytopathology

Phytophthora capsici: Recent Progress on Fundamental Biology and Disease Management 100 Years After Its Description

L.M. Quesada-Ocampo,[1] C.H. Parada-Rojas,[1] Z. Hansen,[2] G. Vogel,[3] C. Smart,[3] M.K. Hausbeck,[4] R.M. Carmo,[5] E. Huitema,[5,6] R.P. Naegele,[7] C.S. Kousik,[8] P. Tandy,[2] and K. Lamour[2]

[1]Department of Entomology and Plant Pathology and NC Plant Sciences Initiative, North Carolina State University, Raleigh, North Carolina, USA; email: lmquesad@ncsu.edu

[2]Department of Entomology and Plant Pathology, University of Tennessee, Knoxville, Tennessee, USA

[3]School of Integrative Plant Science, Cornell University, Geneva, New York, USA

[4]Department of Plant, Soil, and Microbial Sciences, Michigan State University, East Lansing, Michigan, USA

[5]Division of Plant Sciences, University of Dundee, Dundee, United Kingdom

[6]James Hutton Institute, Invergowrie, Dundee, United Kingdom

[7]Sugarbeet and Bean Research Unit, USDA, ARS, East Lansing, Michigan, USA

[8]US Vegetable Laboratory, USDA, ARS, Charleston, South Carolina, USA

Annu. Rev. Phytopathol. 2023. 61:185–208

First published as a Review in Advance on May 31, 2023

The *Annual Review of Phytopathology* is online at phyto.annualreviews.org

https://doi.org/10.1146/annurev-phyto-021622-103801

Keywords

oomycete, plant pathogen, vegetables, disease, fungicide resistance

Abstract

Phytophthora capsici is a destructive oomycete pathogen of vegetable, ornamental, and tropical crops. First described by L.H. Leonian in 1922 as a pathogen of pepper in New Mexico, USA, *P. capsici* is now widespread in temperate and tropical countries alike. *Phytophthora capsici* is notorious for its capability to evade disease management strategies. High genetic diversity allows *P. capsici* populations to overcome fungicides and host resistance, the formation of oospores results in long-term persistence in soils, zoospore differentiation in the presence of water increases epidemic potential, and a

broad host range maximizes economic losses and limits the effectiveness of crop rotation. The severity of disease caused by *P. capsici* and management challenges have led to numerous research efforts in the past 100 years. Here, we discuss recent findings regarding the biology, genetic diversity, disease management, fungicide resistance, host resistance, genomics, and effector biology of *P. capsici*.

INTRODUCTION

Phytophthora capsici is one of the most destructive and economically important oomycete pathogens ever described (59). It affects an unusually broad range of hosts, ranging from vegetables to tropical fruits. *P. capsici* can survive in soil for several years and lead to disease outbreaks occurring year after year if left unmanaged. Its high genetic variability allows *P. capsici* to rapidly adapt to adverse conditions, limiting the efficacy of fungicides and resistant varieties as disease control strategies. Since its description in 1922 by L. H. Leonian as the causal agent of a blight on pepper plants in the United States (New Mexico), *P. capsici* has become a widespread plant pathogen in the United States and many other countries (81). In this article, we build on advances reported in other reviews (41, 44, 77, 105, 126, 127) and expand on new findings that have advanced our knowledge of the biology and management of *P. capsici*.

PATHOGEN BIOLOGY, DISTRIBUTION, AND HOST RANGE

P. capsici belongs to the genus *Phytophthora*, which falls within the Kingdom Stramenopila, Phylum Oomycota, Class Oomycetes, Order Peronosporales, Family Pythiaceae (77). Studies have revealed a heterothallic pathogen with a polycyclic disease cycle (**Figure 1**) (41, 44, 105). Asexual reproduction results in the production of flagellated zoospores that emerge from water-exposed papillate sporangia, whereas sexual reproduction involves mating types A1 and A2, which produce amphigynous oospores when they come into contact with each other. Zoospore affinity toward plant roots renders *P. capsici* a difficult pathogen to control without efforts to prevent within- and between-field water movement (42). In the presence of water, 20 to 40 zoospores per sporangium can deploy and reach the plant root system (32, 105). Flooding allows for movement of propagules down rows and heavy rain or irrigation splash facilitates transport of the inoculum to aboveground plant parts, including fruits (42). When zoospores contact the plant's surface, they encyst and germinate, forming germ tubes. The germ tube enters the host directly with the aid of macerating enzymes or through natural openings (4). Hyphae grow inside the plant tissue and form haustoria to gain nutrients from the host cells (77). Oospores, which are key survival structures, overwinter and can remain dormant in soil for more than 5 years (9). Oospores develop inside infected stems or fruits of host plants. As plant tissue rots, the oospores remain in the soil, germinating when conditions are favorable. Fluctuation between low and high soil moisture combined with temperatures between 16°C and 32°C promotes oospore germination (111). Recombination during sexual reproduction contributes to genetic diversity and can generate new genotypes with improved fitness such as fungicide resistance or increased virulence (16, 108, 138).

A recent review updated the range of hosts that *P. capsici* can infect (105) (**Table 1**). Strawberry (*Fragaria* × *ananassa* Dutch) and tangerine (*Citrus reticulata*) (10, 22) have also been described as hosts. The most economically significant hosts include members of very diverse plant families: Solanaceae (pepper, eggplant, petunia, and tomato), Cucurbitaceae (cucumber, watermelon, squash, cantaloupe, and pumpkin), Malvaceae (cotton, okra, fig, and cacao), and Fabaceae (lima bean, pea, lupine, and alfalfa) [41, 105; see the US National Fungus Collections (**https://nt.ars-grin.gov**)]. Although *P. capsici* attacks many crops, symptoms vary from crown and root rots that

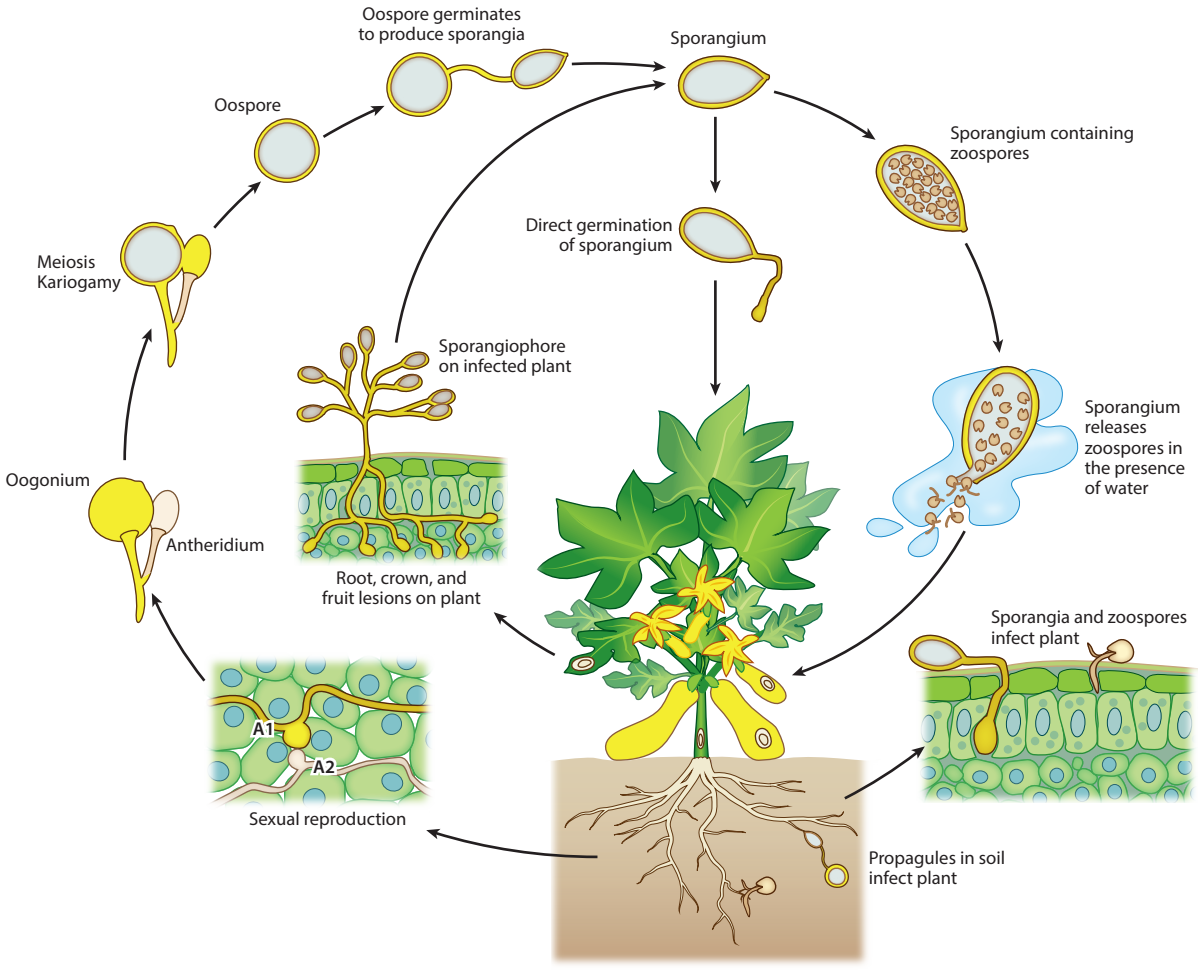

Figure 1

Phytophthora capsici disease cycle on summer squash. The two mating types of *P. capsici* can come into contact in the field, form reproductive structures, undergo sexual reproduction, and form an oospore. The oospore persists in soil or plant debris and germinates, producing mycelia and generating sporangiophores and sporangia. Sporangia are dispersed via water and can directly germinate or differentiate into many zoospores in the presence of water. Sporangia or zoospores can germinate and form appressoria and a germ tube that enters the plant through natural openings, wounds, or directly with the help of enzymes. Mycelia can grow between plant cells, and haustoria are formed to obtain nutrients from plant cells. Mycelia generate new sporangiophores and sporangia that become exposed on the plant tissue surface through natural openings or necrotic tissue. Exposed sporangia can then be dispersed to continue the infection cycle.

lead to wilting and plant death to fruit rots (105). Crown and root rots advance first in water-soaked regions that become necrotic (**Figure 2*b***). Fruit symptoms begin with water-soaked lesions and progress until *P. capsici* mycelia appear on the fruit surface as white, powdery growth (**Figure 2*c*,*d***).

P. capsici tolerates a wide range of environmental conditions from tropical to temperate regions (**Figure 3**). Its worldwide distribution is broad despite *P. capsici*'s inability to disperse aerially. Flooding and human movement are thought to facilitate the long-distance dispersal to other continents (116). *P. capsici* has been described as a vegetable pathogen throughout North America, most of South and Central America, and parts of Africa, Europe, Asia, and Australia (105).

Table 1 Known hosts for *Phytophthora capsici* and their distribution

Host	Distribution
Aloaceae	
Aloe sp. (aloe)	USA
Apiaceae	
Daucus carota (carrot)	USA
Araceae	
Philodendron scandens (heart leaf philodendron)	Argentina, USA
Anthurium andraeanum (flamingo lily)	
Asteraceae	
Carthamus tinctorius (safflower)	USA
Cosmos sp. (cosmos)	
Annonaceae	
Annona squamosa (custard apple)	Australia
Apocynaceae	
Mandevilla sp. (rocktrumpet)	Australia
Brassicaceae	
Brassica oleracea (cauliflower)	USA
Raphanus sativus (radish)	
Brassica rapa (turnip)	
Caryophyllaceae	
Dianthus barbatus (carnation)	USA
Cactaceae	
Opuntia ficus-indica (Indian fig)	USA
Chenopodiaceae	
Spinacia oleracea (spinach)	USA
Beta vulgaris (sugar beet, red beet, Swiss chard)	
Caryophyllaceae	
Gyposophila paniculata (baby's breath)	Taiwan
Caricaceae	
Carica papaya (papaya)	Trinidad
Cucurbitaceae	
Cucurbita pepo var. *medullosa* (pumpkin)	Albania, Canada, France, Italy, Korea, Mexico, Norway, Spain, USA
Cucumis melo (melon, cantaloupe)	
Momordica charantia (bitter gourd)	
Luffa cylindrica (sponge gourd)	
Bryonia dioica (red bryony)	
Cucurbita moschata (squash, pumpkin)	
Citrullus lanatus (watermelon)	
Cucurbita pepo (zucchini)	
Cucumis sativus (cucumber)	
Euphorbiaceae	
Hevea brasiliensis (rubber tree)	Brazil
Ebenaceae	
Diospyros kaki (persimmon)	Brazil

(*Continued*)

Table 1 (*Continued*)

Host	Distribution
Ericaceae	
Enkianthus quinqueflorus (Chinese New Year flower)	USA
Fabaceae	
Lupinus polyphyllus (lupine)	Brazil, Italy, USA
Medicago sativa (alfalfa)	
Vicia faba (broadbean)	
Pisum sativum (pea)	
Phaseolus lunatus (lima bean)	
Phaseolus vulgaris (snap bean)	
Geraniaceae	
Geranium carolinianum (Carolina geranium)	USA
Lauraceae	
Persea americana (avocado)	USA
Liliaceae	
Allium cepa (onion)	USA
Linaceae	
Linum sp. (flax)	USA
Malvaceae	
Gossypium hirsutum (cotton)	China, Cuba, France, India, Pakistan, USA
Abelmoschus esculentus (okra)	
Ficus carica (fig)	
Theobroma cacao (cacao)	
Albutilon theophrasti (velvet leaf)	
Orchidaceae	
Dendrobium candidum (orchid)	China, French Polynesia
Vanilla planifolia (vanilla)	
Portulacaceae	
Portulaca oleracea (common purslane)	USA
Piperaceae	
Piper nigrum (black pepper)	Ethiopia, Indonesia, Malaysia, Philippines, Vietnam
Piper betle (betel)	
Proteaceae	
Leucospermum (pincushion flower)	USA
Macadamia sp. (macadamia)	
Pinaceae	
Abies fraseri (Fraser fir)	USA
Rosaceae	
Malus pumila (apple)	Brazil, Mexico, USA
Crataegus oxyacantha (hawthorne)	
Fragaria × *ananassa* Dutch (strawberry)	
Rutaceae	
Citrus spp. (citrus)	USA
Citrus reticulata (tangerine)	

(*Continued*)

Table 1 (*Continued*)

Host	Distribution
Solanaceae	
Capsicum annuum (pepper, bell pepper, red chili, chili pepper)	Afghanistan, Albania, Algeria, Bhutan, Bosnia, Brazil, Bulgaria, Burkina Faso, Canada, China, Colombia, Costa Rica, Côte D'Ivoire, Croatia, Cyprus, Ecuador, Egypt, El Salvador, Ethiopia, France, Greece, Guatemala, Herzegovina, Hungary, India, Indonesia, Iran, Iraq, Italy, Japan, Kazakhstan, Laos, Lebanon, Libya, Macedonia, Malta, Monaco, Montenegro, Morocco, Nepal, Nigeria, Pakistan, Peru, Philippines, Puerto Rico, Romania, Russia, Serbia, Slovenia, South Africa, Spain, Syria, Taiwan, Thailand, Tunisia, Turkey, USA, Uzbekistan
Capsicum pubescens (pepper)	
Capsicum baccatum (pepper)	
Capsicum chinense (pepper)	
Capsicum frutescens (pepper)	
Solanum lycopersicum (tomato)	
Solanum melongena (eggplant)	
Calibrachoa × *hybrida* (million bells)	
Nicotiana sp. (tobacco)	
Petunia sp. (petunia)	
Nierembergia scoparia (cup flower)	
Datura stramonium (jimson weed)	
Solanum americanum (American black nightshade)	
Solanum carolinense (Carolina horsenettle)	
Solanum marginatum (purple African nightshade)	
Solanum nigrum (black nightshade)	

Modified from the lists of hosts by Granke et al. (41) and Parada-Rojas et al. (105).

POPULATION GENETICS, PATHOGEN DIVERSITY, AND EVOLUTION

Phytophthora species are classified into 10 phylogenetic clades based on multiple loci. *P. capsici* is placed in clade 2b, along with several poorly characterized species, including the closely related *Phytophthora tropicalis*, which causes disease on a wide range of woody, tropical species (145). There has been debate on whether *P. tropicalis* and *P. capsici* are truly distinct species or belong to the same species complex (15). Although *P. capsici* and *P. tropicalis* are genetically differentiated, some isolates of the two are interfertile in laboratory crosses and may overlap in morphology and virulence (27). Evidence of greater genetic diversity in *P. tropicalis* compared to *P. capsici* supports the hypothesis that *P. tropicalis* represents the ancestral population from which *P. capsici* radiated (15, 116).

Although *P. capsici* is less genetically diverse than *P. tropicalis*, sequencing data from global isolate collections show that it contains a tremendous amount of genetic diversity, featuring on average one single nucleotide polymorphism (SNP) every 35–45 base pairs and a nucleotide diversity (π) of 0.012 (76, 116). Isolates may also vary dramatically in terms of chromosomal copy number, a phenomenon that is increasingly noticed from next-generation sequencing data sets in population genomic studies. Although most isolates of *P. capsici* are diploid, as are oomycetes in general, many polyploid isolates have been discovered that possess three or more sets of chromosomes (138). Other isolates show evidence of aneuploidy, possessing an unbalanced set of chromosomes, or perhaps even copy number variation within individual chromosomes (12, 138). To date, the phenotypic consequences of polyploidy and aneuploidy in *P. capsici* remain unknown.

Recent studies have begun to associate molecular markers in *P. capsici* with important phenotypic traits. Approaches using experimental, biparental populations localized the mating type–determining region to a locus on scaffold 4 of the reference genome (16, 76). Genotypic differences between A1 and A2 isolates support the hypothesis that mating type is inherited analogously to sex in an XY system, where A1 isolates are homozygous (i.e., XX) and A2 isolates heterozygous (i.e., XY) at this locus (16, 138).

Traits may also be mapped in *P. capsici* via genome-wide association studies (GWAS), a method whose resolution is determined by the length of linkage disequilibrium (LD) blocks across the

Figure 2

Typical Phytophthora blight, fruit, and root-rot symptoms on vegetables. (*a*) Summer squash leaf necrotic lesion. (*b*) Wilting, crown rot, and leaf necrosis on bell pepper plants. (*c*) Heavily infected watermelon and (*d*) pumpkin fruits exhibiting water-soaking, rot, characteristic white powder-like sporangia, and mycelium on the fruit surface.

genome. In a population of predominantly New York isolates, LD decayed rapidly, to $r^2 < 0.10$ by ~12 kb, which implies a high resolution for GWAS and indicates that the species has experienced large historical population sizes and frequent recombination through sexual reproduction (138). A GWAS in this population validated the location of the mating-type region and discovered a novel locus associated with mefenoxam resistance on scaffold 62 of the reference genome (138).

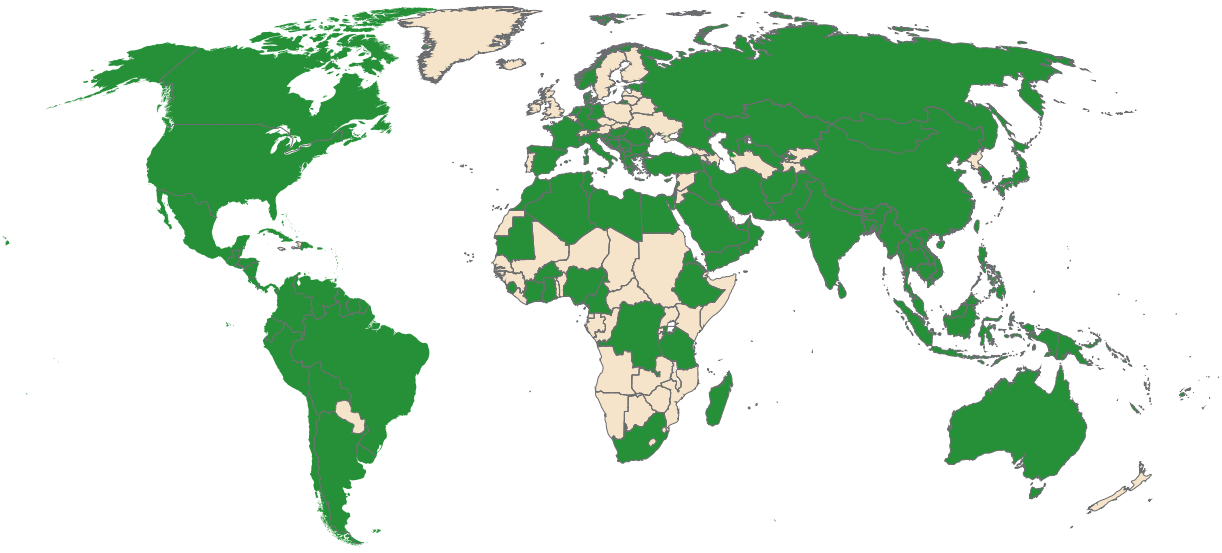

Figure 3

Worldwide distribution of *Phytophthora capsici* from 1918 to 2022. Countries with reports are shown in green and include observations on field- and greenhouse-grown vegetable and ornamental hosts.

In addition, four loci were identified that were associated with virulence on pepper, three of which colocalized with plausible candidate genes (137, 139). GWAS appear to be a promising strategy for further elucidating the genetic control of economically relevant traits in *P. capsici*.

P. capsici population structure has been characterized extensively at the local and regional scale in the United States, be it at the level of a single site (75, 115), county (39), or state (17, 31, 73, 74, 132). Several patterns of genetic variation are generally consistent among *P. capsici* populations in the United States. Populations typically consist of clonally derived and genotypically unique isolates that are restricted to individual fields or years (31, 74, 75, 132, 138). This is consistent with the lack of long-distance air dispersal in *P. capsici*, the overwintering of inoculum as oospores, and the role that asexual reproduction plays in disease spread within a growing season. *P. capsici* populations typically feature a combination of A1 and A2 isolates, and oospores are directly observed in diseased tissue in the field (31, 73, 132, 138). Frequent sexual reproduction maintains genetic variability in the population so that no single clonal lineage dominates in the long-term. Sexual reproduction also allows for advantageous mutations arising in different individuals to potentially recombine into a single genotype. Oospores, which germinate asynchronously and may remain viable in the soil for years (16), serve as a bank of genetic diversity from previous seasons. Genetically diverse oospores can effectively buffer the effects of selection against any alleles with a seasonably variable fitness effect (e.g., any hypothetical alleles conferring poor adaptability to a particular host crop that may be rotated to a different species in a subsequent year).

In the United States, populations from distinct field sites are typically genetically differentiated. This suggests that movement of inoculum is infrequent and that populations evolve in relative isolation once a set of founder isolates colonize a particular location via an event such as a flood or human-mediated introduction (17, 31, 74, 132, 138, 141). This model of restricted gene flow between isolated subpopulations is supported by time-series studies of individual populations: Both natural and experimentally founded studies show that although allele frequencies at certain loci may change over time, genome-wide differentiation is largely unobserved (16, 30, 75).

Characterization of the *P. capsici* population structure at a larger scale has revealed few consistent patterns. Across the United States, isolates differentiate by state in some cases, but not all, and there are examples of isolates from geographically distant states showing greater genetic similarity than isolates from different sites within a single state (106, 108, 115, 138). Little is known about either the historical migratory pathways of *P. capsici* in the United States or the current-day mechanism and frequency of long-distance inoculum transport, although current efforts to densely genotype isolates from across the country may shed light on these areas.

As in the United States, isolates of *P. capsici* in South Africa, central Mexico, northern China, and Hainan Island, China, are genotypically diverse and of both mating types, suggesting that sexual reproduction takes place in local pathogen populations (18, 47, 93). In other regions, however, including Peru, Argentina, Taiwan, northern Mexico, and much of mainland China, widespread clonal lineages of *P. capsici* are distributed over large geographic areas and persist for multiple years (12, 18, 40, 47, 52). It is unclear what genetic or environmental factors contribute to the differences in reproductive strategy and population structure between regions worldwide. Some tempting hypotheses for the persistence of asexual lineages in these regions, such as their featuring milder winters or pathogen populations that are able to form chlamydospores, are not supported by evidence from those locations (48, 52).

DISEASE MANAGEMENT

Vegetable producers seek to lessen the risk posed by *P. capsici* through integrating strategies, including water management, cultural practices, host resistance, and application of fungicides. The efficacy of a standard 3-to-4-year rotation to nonhost crops as a stand-alone tool is limited because *P. capsici*'s primary inoculum survives for long periods in the soil in the absence of a host (9, 75). Excluding *P. capsici* from noninfested production sites is important because once the pathogen is established, complete remediation of the site is unlikely with currently available strategies. Working an infested site last, cleaning field equipment afterward, and disposing of diseased cull fruit off-site are fundamental exclusionary practices. Using contaminated surface water for irrigation is considered an important means of pathogen dissemination (38, 50) and, as a result, well water is recommended. Isolates resistant to the fungicide mefenoxam were common in surface water sources from some Michigan regions (38).

Integral to managing *P. capsici* is the sowing of seeds or transplanting seedlings into raised, crowned beds covered with polyethylene mulch to avoid excess soil moisture around plant crowns and prevent infested soil from splashing onto aboveground plant tissues (123). Using trickle irrigation with plastic mulch facilitates application of fungicides directly to the crown and root system. Crown-directed applications and soil drenches of fungicides improve control of crown and root rot of cucurbits and pepper; however, not all fungicides are labeled for soil application (34, 94). The use of raised plant beds as a management tactic has limited success when producing cucurbit crops whose vining habit results in the fruit developing in the furrows between the raised plant beds, thus coming into direct contact with infested soil and water (61). Also, when rain events occur resulting in water levels that exceed the height of the raised plant bed, the benefit of this cultural practice is negated, and plant death is likely. Growers of some cucurbits (e.g., cucumbers and hard squash), tomatoes, and peppers for the processing market are limited by mechanical harvesting and a narrow profit margin. They typically use a traditional flat plant bed growing system with overhead irrigation and foliar fungicide sprays. Growers rely on genetic resistance where available and reduced plant populations to assure good spray coverage.

Soil fumigation can reduce soil inoculum, but its use is being replaced by more economical and sustainable control strategies, including biofumigant crops. However, planting mustard may not reduce soil populations of *P. capsici* (69). Although vegetable crops in the Brassicaceae family have

not been considered hosts of *P. capsici*, the pathogen reduced the fresh weight of all *Brassica* spp. tested and killed *B. juncea* 'Pacific Gold' plants. Other novel strategies have included testing the ability of potassium phosphite, calcium oxide, and a water suspension from *Trichoderma* sp. TW2-enriched compost to activate systemic acquired resistance in pepper to limit foliar blight (14).

Seed treatment with fungicides or biorationals may reduce damping off, thereby increasing plant stands and yield (126). Using fungicides belonging to different FRAC (Fungicide Resistance Action Committee) groups is recommended to delay the development of pathogen resistance. At the time that *P. capsici* isolates were found to be resistant to the key fungicide mefenoxam (73, 110) effective alternatives were largely unavailable. Today, several fungicides are registered and considered effective against oomycetes, including *P. capsici*. Matheron & Porchas (91) determined that different fungicides may differ in their control based on inoculum type, inoculation timing following treatment, and fungicide application site. Jackson et al. (55) found that mandipropamid and dimethomorph effectively suppressed all stages of *P. capsici* development. Controlled greenhouse and laboratory studies indicated that the fungicides including ametoctradin + dimethomorph and fluopicolide were the most effective in limiting lesions on inoculated chili pepper seedling stems when multiple parameters were assessed (91). Qu et al. (113) conducted greenhouse studies to illustrate the systemic properties of the fungicide oxathiapiprolin in pepper plants. When applied as a drench, oxathiapiprolin was detected in the root within 3 days and in the stem and first true leaf within 6 days. When a higher rate was used, the product was found in the foliage twice as quickly (113). Kousik et al. (63) determined that fruit rot of watermelon in the field was reduced with weekly applications of oxathiapiprolin, ethaboxam, ametoctradin + dimethomorph, dimethomorph, potassium phosphite + copper hydroxide, mandipropamid alternated with potassium phosphite + copper hydroxide, and rotations of mandipropamid with fluopicolide and acibenzolar-S-methyl with mandipropamid. When fungicide rotation programs for watermelon fruit rot were tested in field plots across three southeastern US states, alternating oxathiapiprolin with ametoctradin + dimethomorph was effective across three locations. The following programs were also effective: rotations of acibenzolar-S-methyl followed by cyazofamid + mefenoxam, fluopicolide, ethaboxam, and oxathiapiprolin, or a rotation of mandipropamid alternated with fluopicolide (62).

During periods of favorable environmental conditions, susceptible crops may succumb to *P. capsici* despite aggressive fungicide programs (61). Thus, mitigation efforts have focused on assessing crop susceptibility to the pathogen. To mitigate crown rot of pepper, Foster & Hausbeck (34) combined a resistant cultivar and fungicides in field and greenhouse trials. Ontogenic or age-related resistance (ARR) can also be used to limit fruit rot in hard squash and time fungicide application to coincide with the fruit's most susceptible stage. For example, Meyer & Hausbeck (95) determined that fruit rot susceptibility decreased with fruit age in 'Dickenson Field' processing pumpkin (*Cucurbita moschata*), whereas 'Golden Delicious' winter squash (*Cucurbita maxima*) remained susceptible even at fruit maturity. Michigan growers used these results to exclude 'Golden Delicious' from processor contracts in favor of 'Dickenson Field.'

FUNGICIDE RESISTANCE

The closely related fungicides metalaxyl and mefenoxam have been used to manage oomycete pathogens since the 1970s. In 1998, Parra & Ristaino (109) published the first report of mefenoxam insensitivity in field isolates of *P. capsici*. In that study, 161 isolates of *P. capsici* were collected from bell pepper fields in North Carolina and New Jersey. They found that 34% of the isolates were sensitive, 9% were intermediate, and 57% were insensitive. Since then, numerous additional reports of insensitivity to mefenoxam have been published (31, 60, 73, 108, 131).

Since the widespread adoption of mefenoxam as a management tool for *P. capsici* and subsequent reports of resistance, numerous additional fungicides have been registered for use against the disease in vegetables. Currently, there are at least 13 conventional fungicide active ingredients belonging to 10 FRAC groups recommended for management of *P. capsici* in commercial vegetables. Those are ametoctradin (FRAC group 45), cyazofamid (FRAC group 21), cymoxanil (FRAC group 27), dimethomorph and mandipropamid (FRAC group 40), ethaboxam (FRAC group 22), famoxadone and fenamidone (FRAC group 11), fluazinam (FRAC group 29), fluopicolide (FRAC group 43), metalaxyl and mefenoxam (FRAC group 4), and oxathiapiprolin (FRAC group 49) (36). Several of these products are sold as premixed products containing two active ingredients as a precautionary measure to delay resistance development.

Because *P. capsici* is known to readily develop resistance to single-site mode-of-action fungicides, researchers have explored the sensitivity of field isolates of *P. capsici* to several of these fungicide active ingredients. In 2008, Kousik & Keinath (67) were the first to report insensitivity to cyazofamid in the southeastern United States. They found a wide range of EC_{50} values among 28 field isolates when tested in vitro, ranging from <1 to >100 mg/L. Similar results were found when a subset of the sensitive and resistant isolates was tested by inoculating cucumber and watermelon fruit and applying the recommended field rate of cyazofamid. Additional reports of cyazofamid resistance have since been made in Georgia and Tennessee (55, 131).

Fluopicolide has been examined in several studies for resistance in *P. capsici*. In 2010, Jackson et al. (54) investigated the sensitivity of 25 *P. capsici* isolates to fluopicolide in vitro and in field trials. They found that all isolates were sensitive when tested using in vitro spore germination assays and found a high level of disease suppression in field trials. Similarly, Matheron & Porchas (92) found significant *P. capsici* suppression in chili peppers in greenhouse and field trials following applications of fluopicolide. Recently, resistance to fluopicolide has been reported in studies from Tennessee, Georgia, and North Carolina. In Tennessee, 47% of the 184 isolates tested were found to be resistant to fluopicolide in vitro (131), and in Georgia 43% of the 174 tested isolates were resistant (140). In a study by Wang & Ji (140), the relative fitness of fluopicolide-sensitive and fluopicolide-resistant isolates was also compared. No fitness costs associated with resistance were observed, indicating the potential for fluopicolide resistance to be persistent in populations. In the North Carolina study, 15% of the isolates were resistant to fluopicolide (108).

Oxathiapiprolin is a relatively new product for managing diseases caused by oomycetes. In 2016, Miao et al. (96) induced oxathiapiprolin resistance in twelve mutant strains of *P. capsici* derived from two sensitive parent isolates. They found that mutants derived from one of the parents showed no fitness cost associated with the fungicide resistance mutation, whereas those derived from the other parent lost the ability to produce zoospores. Their study demonstrated the potential for stable fungicide resistance to this active ingredient to develop. In 2021, reduced sensitivity to oxathiapiprolin was reported in Tennessee. Of the 184 isolates tested in vitro, one was considered resistant and 15 showed reduced sensitivity (131).

In the same study, Siegenthaler & Hansen (131) found no resistance to the carboxylic acid amide (CAA) fungicides dimethomorph and mandipropamid. In 2010, Lu et al. (87) did not find resistance in field isolates to the CAA fungicide iprovalicarb. However, they were able to generate resistant mutants in vitro and demonstrated that the resistance was stable after 10 transfers and was not associated with fitness costs. In 2013, similar results were observed for the CAA fungicide pyrimorph. Again, resistant isolates were generated in vitro, and the authors found no fitness cost associated with resistance after 10 transfers of the isolates. In both studies, cross-resistance was observed among all other CAA fungicides tested, including flumorph, dimethomorph, and mandipropamid (104).

To date, resistance to at least six FRAC groups in *P. capsici* has been reported (FRAC group 4, phenylamides; FRAC group 11, quinone outside inhibitors; FRAC group 21, quinone inside inhibitors; FRAC group 43, benzamides; FRAC group 49, oxysterol binding protein homolog inhibitors; FRAC group 40, carboxylic acid amides). Cross-resistance to several active ingredients within these groups is common (87, 88, 104). Additionally, individual *P. capsici* isolates with resistance to multiple active ingredients belonging to different FRAC groups have been observed (130, 131). The relatively large number of fungicide active ingredients and modes of action for managing *P. capsici* should slow the evolution of fungicide resistance in field populations. Growers should continue to rotate fungicide modes of action and implement cultural controls to reduce disease pressure with the goal of delaying the onset of fungicide resistance.

ADVANCES IN DEVELOPING RESISTANT CUCURBITACEOUS AND SOLANACEOUS CROPS

Management of *P. capsici* using host resistance is challenging because of the high genetic variability of the pathogen and multigenic nature of resistance. Depending on the host and source, resistance can be tissue, age, or isolate specific and varies in durability (37, 125). This complexity is a major limitation for breeding sustainable host resistance and has historically hindered the development of resistant commercial-quality cultivars. Sources of resistance have been identified for major crops like peppers, watermelon, and cucumber but are not always easily bred into cultivars with commercial standards. Molecular markers based on quantitative trait loci (QTLs) or major genes help to facilitate resistance breeding but are not always available. Studies in cucumber, pumpkin, and squash have all demonstrated ontogenic resistance or ARR (8, 37, 70). However, this resistance has yet to be successfully bred into a commercial cultivar, and no molecular markers have been developed. In pepper, mapping populations and diversity panels have been used to show inheritance of isolate-specific and broad-spectrum resistance loci to Phytophthora root rot (11). Some of these loci have been incorporated into commercially available cultivars. Breeding for *P. capsici* resistance in pepper has made notable advancements compared to many other susceptible, economically important crops.

Resistance to *Phytophthora capsici* in Cucurbits

For most of the *Cucurbita* species (*C. moschata* and *Cucurbita pepo*), crown rot has been the primary area of breeding efforts. Several sources of resistance have been identified (19, 68, 78), and germplasm resources have been developed (68). Similarly, sources for crown rot resistance have been identified for melon (*Cucumis melo*), and breeding lines have been developed (28). Studies to determine inheritance of resistance to crown rot in squash (*C. moschata*) indicated that resistance was conferred by three dominant genes (103). Three QTLs associated with resistance to crown rot in squash were later confirmed (120), and several linked markers were developed for marker-assisted selection (MAS). In summer squash (*C. pepo*), a combined bulked segregant analysis (BSA)-seq and linkage mapping approach identified six QTLs associated with crown rot resistance (139). In this study, BSA-seq of 13,000 F2 seedlings identified five genomic regions linked to resistance, whereas linkage mapping using an F2:3 population suggested a four-QTL model (139). Such studies point to the complexity of host–pathogen interactions involving *P. capsici* and the potentially numerous genes required in conferring resistance. Regardless, both studies concluded that MAS could be a suitable approach for improving crown rot resistance in squash.

Phytophthora fruit rot on cucurbits can be a pre- or postharvest problem, and if the disease occurs early during fruit set it could potentially result in total yield loss (64). Although *P. capsici* has been known since the 1940s to cause fruit rot in cucurbits (143), the active search for resistance in

any of the cucurbit crops began about six decades later (37). So far, sources of resistance to fruit rot have been identified in cucumber, watermelon, and squash (8, 37, 66, 95).

In cucumber and *Cucurbita* species, resistance to fruit rot is related to age of the developing fruit (ARR). Early searches for resistance identified several sources of ARR to fruit rot of cucumber (37). Young fruit were found to be susceptible and developed resistance about 10 to 12 days post pollination (dpp). Cucumber fruit peels collected 16 dpp displayed resistance, suggesting that the fruit surface played a role in resistance (7). The exocarp from 16-dpp fruit had thicker cuticles compared to 8-dpp fruit. Methanolic extracts from 16-dpp cucumber fruit peels also inhibited growth of *P. capsici*, suggesting a combination of physical and chemical components involved in ARR (7). A significant increase in terpenoid glycosides was found to be associated with ARR based on metabolomic analysis of cucumber fruit peels (90). The same study also identified 80 genes that were upregulated in 16-dpp resistant fruit compared to fruit from a susceptible cultivar that did not display ARR (90). Because it is well known that serious losses can occur when young fruit get infected, Colle et al. (25) evaluated 1,076 cucumber plant introductions (PI) and identified several sources of resistance. One of these sources of resistance (PI 109483) to fruit rot was developed into a breeding line (43).

Similar to cucumbers, ARR has been observed in *Cucurbita* species (70, 95). As described above, several sources of crown rot resistance have been identified in *Cucurbita* species; unfortunately, crown rot resistance did not correlate with fruit rot resistance (71). Evaluation of more than 20 highly crown rot–resistant *C. moschata* lines (65) for young fruit rot also indicated no correlation between resistance to crown rot and fruit rot (C.S. Kousik, unpublished results). A significant correlation between increases in cuticle and epidermal thickness in *C. moschata* fruit exhibiting ARR was observed, indicating the presence of structural barriers to infection as the fruit matured (4). Methanolic extracts of fruit peel from *C. moschata* fruit at different ages induced antifungal activity, but unlike in cucumber this activity did not correlate with ARR (5).

Sources of resistance to fruit rot have also been identified in watermelon (66), and resistant germplasm with broad resistance to isolates of *P. capsici* (65) has been developed (63). Unlike ARR observed in cucumber and squash, the fruit of the resistant watermelon germplasm was resistant at all stages of development (64). These resistant germplasm lines belong to egusi-type watermelon (*Citrullus mucosospermus*) and can be easily crossed with cultivated watermelon (*Citrullus lanatus*). Similar to the challenges faced in breeding peppers, cucumbers, and squashes for resistance to *P. capsici*, introgressing resistance into cultivated watermelon from the resistant germplasm lines has been challenging because of the large differences in desirable horticultural traits and the complex genetics of resistance.

Resistance to *Phytophthora capsici* in Solanaceae

Solanaceae is a diverse plant family that includes susceptible hosts such as eggplant, pepper, tomato, and some ornamentals (99, 114, 118). *Phytophthora* resistance research and breeding advancements have primarily been done in pepper (*Capsicum annuum*). Breeding efforts have focused on root rot, and partial resistance has been integrated into select commercial cultivars (35, 107). Crossing barriers exist among *Capsicum* spp. and can impede the introduction of favorable alleles depending on the donor and recipient species.

Capsicum spp. with resistance to *P. capsici* have been found in Mexico, Africa, and Asia through controlled environment trials testing regional isolates (97, 100, 119, 122). Although many of these studies verified known sources of resistance, additional sources with potential novel alleles have also been found through targeted germplasm collection and evaluation. Diversity studies using low-resolution markers (e.g., simple sequence repeats) identified population structure and genetic

diversity associated with resistance (101, 119). In one such study, 39 sources of resistance from Ethiopia and India were identified, five of which were estimated to have novel diverse resistance loci (119). Associating population and regional genetic diversity with disease resistance has also been done to a lesser extent in eggplant and tomato (98, 117).

Disease resistance to *P. capsici* in the Solanaceae has broad-spectrum, isolate-specific, and tissue-specific components (102, 125). More than 45 physiological races have been identified, and more are expected as regional screenings continue (13). In Taiwan, a screening of 24 isolates identified 24 new physiological races with varying levels of virulence using a panel of pepper differentials (13). The complexity of the pathogen is mirrored in the host's response: Epistatic, additive, and dominant (resistance and resistance inhibitor) gene interactions with high broad sense heritability all play a role depending on the host source and isolate.

A landrace from Mexico, Criollo de Morelos 334 (CM334), is a robust source of broad-spectrum resistance used extensively in breeding programs, genetic mapping populations, and gene expression studies. CM334 has a single locus for broad-spectrum resistance, in addition to isolate-specific QTLs (144). Additional sources have been used, but many exhibit only partial or isolate-specific resistance (122). Race-specific resistance alleles can be used to protect major resistance genes by improving their longevity but are insufficient on their own. GWAS and QTL studies using panels of peppers have found SNPs associated with isolate-specific or broad-spectrum resistance on each of the 12 chromosomes (24, 124).

A major locus on chromosome 5 for broad-spectrum resistance to root and fruit rot has been identified through genetic mapping and GWAS in mapping populations and diversity panels (24, 98). This large region (6.2 to 139.2 Mbp) was subdivided into three smaller QTLs (129). Putative resistance genes have been found in these QTL regions, including leucine-rich repeat (LRR) receptors and other receptor kinases (121, 129). One interval, P5.1, contained 23 candidate LRR genes, of which three showed differential expression under *P. capsici* infection (85).

In addition to targeted QTL regions, general receptor classes and gene families known to be involved in plant development, growth, and stress response have shown differential expression and regulation during *P. capsici* infection of pepper (29). Tissue-specific activation of ethylene response factors (ERFs) was associated with improved resistance to *P. capsici* and silencing of select ERFs weakened the defense response (56). In an RNAseq experiment, the WRKY family of transcription factors had enriched expression in both resistant and susceptible peppers (23). Expression of two WRKY genes, in particular, *CaWRKY08-4* and *CaWRKY01-10*, increased upon infection, and silencing them reduced resistance in CM334 (23). Another pepper transcription factor, *CanTF*, affected resistance to *P. capsici* as well as resistance to abiotic stresses like cold and drought (45). Chitin-binding proteins, pathogenesis-related proteins related to plant defense, have also shown differential tissue-specific expression during *P. capsici* infection (2). When silenced, one of these chitin-binding proteins (CaChiIV1) resulted in greater host susceptibility to *P. capsici* and was later shown to affect heat-stress resistance (2, 3). Calcium-binding receptors also play a role in *P. capsici* defense response with silencing and overexpression experiments impacting resistance (147). Although changes in expression have consistently been identified across studies, how or whether these genes contribute to *P. capsici* resistance is often unknown.

THE GENOME OF *PHYTOPHTHORA CAPSICI*

The first reference genome produced for *P. capsici* was based on a combination of approaches that began with a series of in vitro inbreeding crosses to reduce the heterozygosity of this highly polymorphic organism (76). Attempts at close inbreeding (crosses between siblings) were thwarted due to apomixis, a process where normally formed sexual oospores produce only a parental genotype

(53). A milder strategy was employed using a highly fecund cross between isolates from Michigan (LT51) and Tennessee (LT263), and subsequent backcrosses to LT263 (to produce moderate inbreeding) and LT1534 were selected from the progeny of the third backcross for whole-genome sequencing (WGS) using traditional expression sequence tags, cloning vectors, and the short-read technology of Illumina (76). In addition, a set of progeny from the initial F1 cross was sequenced using a genotype-by-sequencing approach and the SNPs were used to produce a first linkage map. The linkage map arranged the majority of the 917 scaffolds assembled after sequencing into 18 linkage groups.

The WGS proved insightful regarding the gene content (e.g., effectors) and also provided a first glimpse of large-scale copy neutral loss of heterozygosity (LOH) within individual genomes. LOH refers to the loss of one allele of a gene in a diploid organism, making that locus appear homozygous. When tabulating the genomic changes across all the oospore progeny, LOH was shown to impact more than 30% of the genome. Since that initial genome sequencing effort, multiple long-read assemblies of *P. capsici* have become available and are proving valuable in documenting diversity within *P. capsici* genomes (80, 133). These efforts resulted in 76-Mb (514 scaffolds, N50 of 6 Mb) and 94-Mb (782 scaffolds, N50 of 485 kb) genome assemblies, a significant improvement from the original 64-Mb assembly (917 scaffolds, N50 of 706 kb).

Phenotypic screening of the oospore progeny used in the F1 cross above showed that LOH may play an important role in changes in pathogenicity, virulence, drug resistance, and switches in mating type. Oospore progeny with the highest incidences of LOH displayed the most significant changes (e.g., total loss of pathogenicity), and only the A2 mating type, which requires heterozygosity, was not stable. LOH was previously documented during TILLING (targeting induced local lesions in genomes) reverse-genetic experiments, a process in which inducing point mutations and screening for mutations within specific genes are employed (51, 72). LOH was also observed within the context of population genetic studies, where members of clonal lineages often displayed minor changes in genotypes (e.g., a single Aa locus switching to AA or aa within the context of a clonal lineage) (47).

Further work to explore the mitotic plasticity of *P. capsici* was conducted by genomic and phenotypic analyses of single zoospore progeny and isolates with visible sectoring while growing on standard media (49). Sectoring is where the normal phenotype (e.g., fluffy or appressed) of the growing mycelium has changed in just a portion of a mycelial colony. The results further illuminated unique trends in heterozygosity within individual genomes, which go beyond the relatively extreme situation of LOH. It was shown that the heterozygous allele frequencies for SNP markers within a single genome can be stably rearranged extensively during the process of mitosis. More than 600 field and single zoospore isolates of *P. capsici* were tested for inheritance of mating type and sensitivity to mefenoxam, and a subset was resequenced to determine putative chromosome copy number and visualize heterozygous allele frequencies across the genome (49).

Interestingly, the A2 mating type was highly unstable with only 26% of 241 A2 isolates remaining A2, whereas the A1 mating type was stable over time. Isolates intermediately resistant to mefenoxam (known to be a codominantly inherited trait) produced fully resistant single-spore progeny, whereas the sensitive isolates remained fully sensitive. Genome resequencing of single zoospore isolates revealed extreme diversity, a phenomenon dubbed dynamic extreme aneuploidy (DEA) (49). DEA was characterized by aneuploidy ranging from 2N to 3N and stable heterozygous allele frequencies during asexual reproduction. The genome of *P. capsici* is highly plastic with many changes occurring (and being held stable) within the context of asexual reproduction. The implications are wide-ranging and may help explain the rapid evolution of populations to diverse control methods, such as A2 mating types switching to A1 and intermediately resistant isolates becoming fully resistant to single-site-of-action fungicides (e.g., metalaxyl/mefenoxam) (49).

EFFECTOR BIOLOGY

The defense response against microbes comprises two layers of induced immune responses, known as pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) (57). PTI acts through the recognition of microbial/pathogen-associated molecular patterns (MAMPs/PAMPs) or signals generated by cell disintegration, namely damage-associated molecular patterns (DAMPs) (26). Perception leads to an active immune response that restricts microbe invasion and colonization. Thus, successful pathogens must limit perception or suppress immune responses. Effectors are delivered inside host tissues, where they suppress signal perception and transduction events that normally lead to PTI. Their activity results in effector-triggered susceptibility (ETS), a common feature among pathogens and pest species. To counter ETS, plants have evolved another rapid and robust immune response, activating effector-triggered immunity (ETI) mechanisms or host-specific resistance (1). PTI, ETS, and ETI all create selective pressures on host and pathogen, driving a coevolutionary arms race (136). However, we do not understand how host–microbe coevolution operates in pathogens with a broad host range that suppress immunity in multiple and distinct plant families.

P. capsici effectors target a diverse range of host proteins, making this pathogen a facile model to study effector biology (77). Genome-wide surveys and functional studies have identified members of all known effector classes in *P. capsici*. The *P. capsici* effector repertoire includes NLPs (Nep1-like proteins), CRNs (crinkling- and necrosis-inducing proteins), CBMs (carbohydrate-binding module family proteins), SCRs (small cysteine-rich proteins), and the RXLR (Arg-x-Leu-Arg) effectors (the most studied family of effectors) (20, 58, 134, 146, 148).

The NLP effector family is present in prokaryote and eukaryote pathogens but has expanded considerably within the oomycetes. The N-terminal section of NLPs features a secretion signal peptide and the NPP1 (necrosis-inducing Phytophthora protein 1) domain, where the hemi-conserved motif can be found (112). The number of conserved cysteine residues in the motif was used to classify NLP proteins into type 1, 2, or 3, but only proteins from type 1 are found in oomycetes (112). Studies investigating *P. capsici* NLPs demonstrated that four members induce cell death in Solanaceous hosts, including tomato, *Nicotiana benthamiana*, and pepper, but the mechanism of cell death induction and its role in virulence have not been described (20).

Another necrosis-inducing protein family is the CRN. This name comes from a leaf-crinkling phenotype caused by the expression of *P. infestans* CRN protein in plants (135). The N-terminal region of CRN proteins carries a conserved LFLAK motif that seems to specify translocation (128). Meanwhile, CRN C-termini are more diverse and have been implicated in virulence. Several CRN effectors have now been characterized in *P. capsici* (134), revealing that some localize to the host nucleus, where they alter immune responses (89) and change chromatin configuration (134).

Pathogens can also instigate cell death through enzymatic degradation of plant cell walls. CBM proteins could play a key role as 87 families have been defined in the carbohydrate-active enzymes (CAZy) database (86). Among these, the carbohydrate-binding module family 1 protein (CBM1) features a cellulose-binding domain and is predominant in fungi and oomycetes (79). In fungi, most CBP (CBM1-containing proteins) possess one CBM1 domain coupled to a catalytic domain, whereas oomycetes contain multiple CBM1s without a described catalytic domain (79). Recently, *Pc*CBP3 (CBM1-containing protein 3) was shown to cause BAK1-dependent cell death in *N. benthamiana*. Given that secretion and the N-terminal region of *Pc*CBP3 were required for function, members of this family are now considered apoplastic effectors with roles in infection (146).

The SCR proteins are small secretory proteins that are thought to be similar to Avr proteins from plant-pathogenic fungi because they share a range of features with these proteins. Such features are their short length, high cysteine abundance, and high expression during infection

(142). In *P. capsici*, *Pc*SCR82 was identified as a virulence factor that also triggers host immune responses (148). The basis of this seemingly dual functionality needs to be further characterized along with other yet uncharacterized *Pc*SCR proteins. These studies should shed light on effector functions and more importantly, the role of cell death in virulence of hemi-biotrophic pathogens such as *P. capsici*.

In addition to the apoplastic and (cytoplasmic) CRN effector family, *P. capsici* carries another family of effectors. The RXLR effector family shares a conserved N-terminal RXLR motif required for translocation to the host cytosol, fused to C-terminal effector domains (6). Computational analyses have now identified large RXLR repertoires in *Phytophthora* (58, 76), and functional studies have firmly established their roles in establishing ETS. Detailed functional studies now unveil the mechanistic basis of susceptibility, immunity, and virulence.

P. capsici has at least 346 putative RXLR-encoding genes that are expressed during in vitro growth and infection and colonization of tomato (58). Some of these effectors directly suppress host immunity by perturbing main regulators such as nonexpressor of pathogenesis-related genes 1 (NPR1) (83), binding partner 1 of accelerated cell death 11 (BPA1) and BPA1-like proteins (82), and enhanced disease susceptibility1 (EDS1) (84). Others were shown to interact with other immunity-related proteins, such as the host protein phosphatase 2A (PP2Aa) (33) and an endoplasmic reticulum–localized protein (FKBP15–2) (21).

Besides the identification of host targets in Solanaceous hosts, recent work has also identified targets in the nonhost *Arabidopsis*. RXLR effector *Pc*PSR2 (Phytophthora suppressor of RNA silencing 2) inhibits secondary siRNA biogenesis in *Arabidopsis* to promote infection (46). However, a few of these *P. capsici* RXLR effectors were characterized in the interaction with *Arabidopsis* (21, 46, 82), where their targets were identified. Whether effector–target interactions, as identified in *Arabidopsis*, are conserved in susceptible hosts such as pepper, tomato, and within the cucurbits is an important question. This question is particularly important if we wish to grasp the molecular basis of *P. capsici*'s broad host range.

In short, although researchers have made meaningful progress toward understanding *P. capsici* effector biology, the sheer number of effectors means that most remain uncharacterized. Future efforts aimed at broadening our knowledge will yield more insight into *P. capsici* biology. In addition, our understanding of *P. capsici* effectors and their regulation during infection remains unexplored. For instance, the identification of both host signals and understanding how *P. capsici* uses these (divergent) signals to regulate effector gene expression could lead to effective pathogen control strategies.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

AUTHOR CONTRIBUTIONS

L.M. Quesada-Ocampo outlined, assembled, and edited the full manuscript; rewrote sections as needed for missing information, accuracy, grammar, flow, and compliance with word limits; generated figures, legends, tables, abstract, and formatted references; and reviewed and approved the submitted version of the manuscript. L.M. Quesada-Ocampo and C.H. Parada-Rojas wrote the section titled Pathogen Biology, Distribution, and Host Range; generated figures, legends, tables, and formatted references; and reviewed and approved the submitted version of the manuscript. Z. Hansen wrote the section titled Fungicide Resistance; provided some photos for figures; and

reviewed and approved the submitted version of the manuscript. M.K. Hausbeck wrote the section titled Disease Management and reviewed and approved the submitted version of the manuscript. E. Huitema and R.M. Carmo wrote the section titled Effector Biology and reviewed and approved the submitted version of the manuscript. C.S. Kousik and R.P. Naegele wrote the sections titled Resistance to *Phytophthora capsici* in Cucurbits and Resistance to *Phytophthora capsici* in Solanaceae, respectively, in the section titled Advances in Developing Resistant Cucurbitaceous and Solanaceous Crops and reviewed and approved the submitted version of the manuscript. K. Lamour and P. Tandy wrote the section titled The Genome of *Phytophthora capsici* and reviewed and approved the submitted version of the manuscript. C. Smart and G. Vogel wrote the section titled Population Genetics, Pathogen Diversity, and Evolution and reviewed and approved the submitted version of the manuscript.

ACKNOWLEDGMENTS

The authors thank all the members of the Quesada lab for their valuable help. This work was supported by the United States Department of Agriculture (USDA) (project number NC02890), the New York State Department of Agriculture and Markets (grant number C00237GG), and the USDA National Institute of Food and Agriculture Specialty Crop Research Initiative (2020-51181-32139).

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