Sensitivity of *Phytophthora capsici* on Vegetable Crops in Georgia to Mandipropamid, Dimethomorph, and Cyazofamid

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Abstract

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Phytophthora blight, caused by *Phytophthora capsici*, is a serious disease in vegetable production, and selective use of fungicides continues to be a significant component of disease management programs. The effect of three chemical compounds—mandipropamid, dimethomorph, and cyazofamid—on asexual stages of *P. capsici* collected from bell pepper and cucurbits in Georgia was assessed in this study. Forty isolates of *P. capsici* were determined to be sensitive to mandipropamid and dimethomorph based on mycelial growth, zoospore germination, and sporangial production. Concentrations that were 50% effective (EC_{50} values) of mandipropamid that inhibited mycelial growth, zoospore germination, and sporangial production of the isolates averaged 0.03, 5.70, and 0.02 μg/ml, respectively. EC_{50} values of dimethomorph in inhibiting mycelial growth, zoospore germination, and sporangial production averaged 0.24, 0.10, and 0.46 μg/ml, respectively. The majority of isolates were either resistant or intermediately sensitive to cyazofamid at 500 μg/ml or lower concentrations based on mycelial growth or sporangial production, although all the isolates were sensitive to this compound based on zoospore germination, with an average EC_{50} of 0.04 μg/ml. The results indicated that *P. capsici* populations in Georgia have not developed resistance to mandipropamid and dimethomorph whereas, for the majority of the isolates, certain asexual stages were resistant to cyazofamid.

Phytophthora blight, caused by the oomycete pathogen *Phytophthora capsici*, is a disease with serious effects on the production of cucurbits, pepper, eggplant, and a number of other important vegetable crops in the United States and worldwide (7,12,28,29). The pathogen may attack the roots and foliage of a plant and cause root rot, crown rot, seedling damping-off, leaf and stem blight, plant wilt, and fruit rot. The disease is more severe under wet and humid conditions that are common in the southeastern United States. In Georgia, Phytophthora blight occurs in both spring and fall growing seasons, causing significant yield loss, especially on pepper and cucurbit crops, including squash, zucchini, watermelon, cantaloupe, and cucumber.

The efficacy of current strategies for management of Phytophthora blight is limited. Cultural practices do not provide sufficient control of the disease and have not been generally adopted by commercial growers. Although considerable efforts have been directed to identify disease-resistant genetic sources (3,25), reliable resistance to *P. capsici* is not known to be available in commercial cultivars of many vegetable crops, especially cucurbits. Application of chemical fungicides has been the method commonly used in management of Phytophthora blight; however, *P. capsici* has a remarkable ability to develop resistance to single-site mode-of-action chemical fungicides. For instance, fungicides containing mefenoxam have been widely used for control of the disease but mefenoxam-resistant strains of *P. capsici* have developed in Georgia and several other states in the United States (2,6,11,13,15,20).

In recent years, fungicides in different chemical classes with new modes of action have emerged to target oomycete pathogens, including *Phytophthora* spp. Mandipropamid ($C_{23}H_{22}C_1NO_4$) and cyazofamid ($C_{13}H_{13}C_1N_4O_2S$) are among the new fungicides that belong to the mandelamide and cyanoimidazole classes, respectively. Mandipropamid inhibits lipids and membrane synthesis and cyazofamid causes respiratory inhibition at complex III in the mitochondria (18,24). Recent studies indicated that mandipropamid acted on the cell wall and inhibition of cellulose synthesis was the primary effect on *P. infestans* (1). Another new fungicide, Zampro (BASF Corp.), which contains two active ingredients (dimethomorph and ametoctradin), was recently developed for control of diseases caused by oomycetes (33). Dimethomorph is also the active ingredient of other fungicides, including Forum (BASF Corp.), that belongs to the mandelamide class and inhibits cell wall formation (18,19). Dimethomorph, mandipropamid, and cyazofamid are used by vegetable growers in Georgia for control of Phytophthora blight or other diseases caused by oomycetes. Determining the sensitivity of *P. capsici* to these compounds and monitoring resistance development in the pathogen populations are essential for effective disease management.

P. capsici is difficult to control in part because the pathogen produces different propagules that contribute to the disease development. In addition to mycelium that may initiate disease, the pathogen produces sporangia and zoospores for asexual reproduction under favorable environmental conditions. Both sporangia and zoospores are important in pathogen dissemination or initiating an infection, and mycelial growth is essential in all infection processes of the pathogen (12,28). Hence, chemical fungicides that inhibit sporangium formation, zoospore germination, or mycelial growth of the pathogen have the potential to reduce the disease. The objective of this study was to determine the effect of mandipropamid, dimethomorph, and cyazofamid on different asexual life stages of *P. capsici* isolates from vegetable crops in Georgia. Determining sensitivity and resistance of *P. capsici* populations to these chemicals will provide a useful guide for developing and implementing effective disease management programs involving these fungicides.

Materials and Methods

***P. capsici* isolates.** Isolates of *P. capsici* were collected during the 2011 growing season from symptomatic plant tissues, including root, stem, and fruit of bell pepper, watermelon, squash, cantaloupe, cucumber, and zucchini, from commercial production fields in several counties in Georgia. Plant tissues were surface disinfested with 70% ethanol, and a small piece from lesion margins was placed on pimaricin-ampicillin-rifampicin-pentachloronithrobenzene (PARP) semiselective medium (14). The plates were incu-

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bated at 25°C in the dark for 3 to 4 days and at room temperature under constant fluorescent light for an additional 2 to 3 days. Isolates on the plates were observed under a microscope and *P. capsici* was identified based on morphological characteristics (10,35). Putative *P. capsici* cultures were transferred to 5% V8 juice agar (50 ml of V8 juice, 1.0 g of $CaCO_3$, 16 g of agar, and 950 ml of distilled water). The plates were incubated at 25°C in the dark for 3 to 4 days and at room temperature under constant fluorescent light for an additional 2 to 3 days for preparation of single-zoospore isolates.

Single-zoospore isolation was completed by adding 10 ml of sterile distilled water (SDW) to the above-mentioned plates, which were chilled at 4°C for 20 min and then returned to room temperature (23 to 25°C) for 20 min to allow for zoospore release. Zoospore suspensions were collected in sterile 50-ml centrifuge tubes, vortexed for 1 min, and adjusted to 200 spores/ml by counting a droplet of the zoospore suspension with a hemacytometer. A volume of 50 µl of zoospore suspension was plated onto water agar plates with a glass spreader and the plates were incubated at 25°C in the dark for 3 to 4 h until zoospores germinated. One single germinated zoospore with some surrounding agar was transferred to PARP agar (three zoospores per isolate) using a flame-sterilized scalpel by observing under a stereo microscope in a sterile flow hood and incubated at 25°C in the dark for 2 to 3 days. A 7-mm-diameter plug removed from the edge of single colonies was placed on V8 agar and incubated at 25°C in the dark for 4 days. Pathogenicity of the isolates was determined by inoculation of squash plants with zoospores as described previously (34). Long-term storage of isolates was completed by putting a 7-mm mycelial plug of purified single-zoospore isolates in a 2-ml cryogenic vial containing one sterilized hemp seed and 1 ml of SDW, and the vials were incubated at 25°C for 2 weeks and stored at 15°C (7).

Identification by polymerase chain reaction analysis. Single-zoospore isolates were grown on V8 agar in the dark for 7 days. DNA was extracted as described earlier (34) and polymerase chain reaction (PCR) amplification was performed using *P. capsici*-specific primers (PC-1, 5′-GTCTTGTACCCTATCATGGCG-3′ and PC-2, 5′-CGCCACAGCAGGAAAAGCATT-3′) to verify the identity of the isolates (37). PCR was performed in 20 µl of reaction mixture containing 20 ng of DNA, 200 µM each dNTP, 0.5 µM each primer, 0.5 U of *Taq* DNA polymerase, and 1× PCR reaction buffer. The amplification was performed in a MyCycler thermal cycler (Bio-Rad Laboratories) using the following protocol: 94°C for 5 min; 94°C for 30 s, 65°C for 30 s, and 72°C for 30 s for 35 cycles; and a final extension step at 72°C for 7 min. PCR products were electrophoresed in 1.5% agarose gel containing ethidium bromide at 0.5 µg/ml in 0.5× Tris-borate-EDTA buffer. Gels were visualized using Molecular Imager Gel DOC XR+ system (Bio-Rad Laboratories).

Chemical compounds. Mandipropamid and dimethomorph (technical grade) were purchased from Sigma-Aldrich, and Ridomil Gold (45.3% a.i. mefenoxam) was obtained from Syngenta Crop Protection. Cyazofamid (technical grade) and Ranman (34.5% a.i. cyazofamid) were provided by ISK Biosciences Corp.

Sensitivity of *P. capsici* populations to mefenoxam. In total, 154 *P. capsici* isolates collected in 2011 were tested for sensitivity to mefenoxam in this study. Isolates were grown on V8 juice agar at 25°C in the dark for 5 days. An agar plug (7 mm in diameter) taken from the edge of the colony was placed at the center of a V8 juice agar plate amended with mefenoxam at a final concentration of 0, 10, and 100 µg/ml. Ridomil Gold (a.i. mefenoxam) dissolved in SDW was used to amend the agar medium. Triplicate plates were used for each concentration and were incubated at 25°C in the dark. Colony diameter was measured in two perpendicular directions 4 days after incubation and averaged for analysis. The diameter of the agar plug was subtracted from the total colony diameter for calculating actual diameter of colony. The relative growth rate of *P. capsici* on mefenoxam-amended and nonamended control plates was used to determine sensitivity to the fungicide, where sensitive = <30% of the control (i.e., colony diameter on mefenoxam-amended plates was less than 30% of colony diameter on nonamended control plates), intermediate sensitive = 30 to 90% of the control, and resistant = more than 90% of the control (20). The experiments were conducted twice under similar conditions.

Sensitivity of mycelial growth to mandipropamid, dimethomorph, and cyazofamid. In all, 40 *P. capsici* isolates (20 sensitive, 10 resistant, and 10 intermediately sensitive to mefenoxam) were selected from the 154 isolates collected in 2011 and isolates previously collected in Georgia (13). The isolates were grown on V8 juice agar at 25°C in the dark for 5 days. An agar plug (7 mm in diameter) taken from the edge of the colony was placed at the center of a V8 juice agar plate amended with one of the compounds at different concentrations. The final concentrations in the agar were 0, 0.005, 0.01, 0.05, 0.1, 0.5, and 1 µg/ml for mandipropamid; 0, 0.1, 0.25, 0.5, 1, 2.5, and 5 µg/ml for dimethomorph, and 0, 0.1, 1, 10, 100, 500, and 1,000 µg/ml for cyazofamid. Technical grade of mandipropamid and dimethomorph dissolved in acetone was used to amend the agar medium. Agar plates amended with the same concentration of acetone were used as a control. In preliminary experiments, technical grade of cyazofamid dissolved in acetone was used, with final concentrations of cyazofamid in the agar of 0 to 100 µg/ml. In subsequent experiments, concentrations of cyazofamid were increased to 0 to 1,000 µg/ml and Ranman (a.i. cyazofamid) dissolved in SDW was used. Triplicate plates were used for each concentration of the compounds, and the plates were incubated at 25°C in the dark. Two perpendicular colony diameters were measured per plate 4 days after incubation, and colony diameter was calculated for each concentration using the mean of the two perpendicular colony diameters as described above. The experiments were conducted twice under similar conditions. Data from the two experiments were pooled, and concentrations that were 50% effective (EC_{50} values) were calculated by fitting linear regression lines of probit-transformed inhibition data against the $\log_{10}$-transformed fungicide concentration according to Stein and Kirk (30).

Sensitivity of zoospore germination to mandipropamid, dimethomorph, and cyazofamid. The 40 *P. capsici* isolates were grown on V8 juice agar plates wrapped with parafilm at 25°C in the dark for 4 days. The plates, with parafilm removed, were then incubated at room temperature under constant fluorescent light for 2 days. Zoospore suspensions were prepared as described above and a volume of 50 µl of zoospore suspension was plated using a glass spreader on clarified V8 agar plates amended with mandipropamid, dimethomorph, or cyazofamid. Technical grade of chemicals was used at 0, 0.01, 0.025, 0.05, 0.1, and 0.5 µg/ml for cyazofamid and similar concentrations for mandipropamid and dimethomorph, as in the mycelial growth study. The experiments were repeated twice, with triplicate plates used for each concentration of the products in each experiment. The plates were incubated at 25°C for 4 h, and germinated and nongerminated zoospores were counted for 100 zoospores on each plate under a stereo microscope at ×100 magnification. Data from two experiments were pooled and EC_{50} values were calculated as described above.

Sensitivity of sporangium formation to mandipropamid, dimethomorph, and cyazofamid. The 40 isolates were grown on V8 agar plates wrapped with parafilm at 25°C for 4 days in the dark. Three 10-mm-diameter plugs removed from the edge of an actively growing culture were placed with the mycelium side facing up in a petri dish containing SDW amended with the compounds at different concentrations. Mandipropamid, dimethomorph, and cyazofamid were used at concentrations similar to those in the mycelial growth study. Technical grade of mandipropamid and dimethomorph dissolved in acetone and Ranman dissolved in SDW were used in the studies. Three petri dishes were used for each concentration of the products and the petri dishes, not wrapped with parafilm, were incubated at room temperature (23 to 25°C) under continuous light. After 48 h, solutions in the petri dishes were removed and agar plugs were stained and fixed with acid fuchsin in 85% lactic acid (16,21). The number of sporangia on two microscopic fields of each agar plug was counted under a stereo microscope at ×100 magnification. The experiment

was repeated one more time under the same conditions, and EC_{50} values were calculated for each isolate as described previously. Suppression of sporangium formation was also calculated by dividing the number of sporangia produced in fungicide-amended dishes by sporangia produced in nonamended control dishes (sensitive: <30% of the control; intermediate sensitive: 30 to 90% of the control; resistance: >90% of the control).

Results

Confirmation of *P. capsici*. A collection of putative isolates of *P. capsici* was obtained from several vegetable crops in 2011. In all, 154 isolates were identified as *P. capsici* based on morphological characteristics and PCR analysis using *P. capsici*-specific primers PC-1 and PC-2 that produced a fragment of 560 bp. All the isolates caused disease on squash plants when inoculated with zoospore suspensions under greenhouse conditions.

Sensitivity to mefenoxam. Of the 154 isolates tested, the majority (70.8%) were sensitive to mefenoxam at 100 and 10 µg/ml while 11.0 and 18.2% of the isolates were resistant and intermediately sensitive, respectively.

Sensitivity of mycelial growth, zoospore germination, and sporangium formation to mandipropamid. All 40 isolates were sensitive to mandipropamid based on mycelial growth, zoospore germination, and sporangial production. The minimum inhibitory concentration (MIC) that completely suppressed mycelial growth of the isolates was 0.5 to 1 µg/ml. EC_{50} values of mandipropamid in inhibiting mycelial growth of the isolates were 0.01 to 0.05 µg/ml, with an average of 0.03 ± 0.01 (standard deviation) µg/ml (Fig. 1A). The MIC that completely inhibited zoospore germination of the isolates was 1 to >5 µg/ml. EC_{50} values of mandipropamid in suppressing zoospore germination of the isolates were 0.72 to 10.3 µg/ml, with an average of 5.7 ± 2.3 µg/ml (Fig. 1B). The MIC that completely inhibited sporangium formation of the isolates was 0.5 to 1 µg/ml. EC_{50} values of mandipropamid in suppressing sporangium formation of the isolates were 0.001 to 0.05 µg/ml, with an average of 0.02 ± 0.01 µg/ml (Fig. 1C). Suppression of mycelial growth, zoospore germination, and sporangial production were not correlated with one another (i.e., suppression of mycelial growth of an isolate at a higher level did not mean suppression of zoospore germination or sporangial production of the isolate at a higher level; *data not shown*).

Sensitivity of mycelial growth, zoospore germination, and sporangium formation to dimethomorph. All 40 isolates were sensitive to dimethomorph based on mycelial growth, zoospore germination, and sporangial production. The MIC that completely suppressed mycelial growth of the *P. capsici* isolates was 1 to 5 µg/ml. EC_{50} values of dimethomorph in inhibiting mycelial growth of the isolates were 0.13 to 0.34 µg/ml, with an average of 0.24 ± 0.07 µg/ml (Fig. 2A). The MIC that completely inhibited zoospore

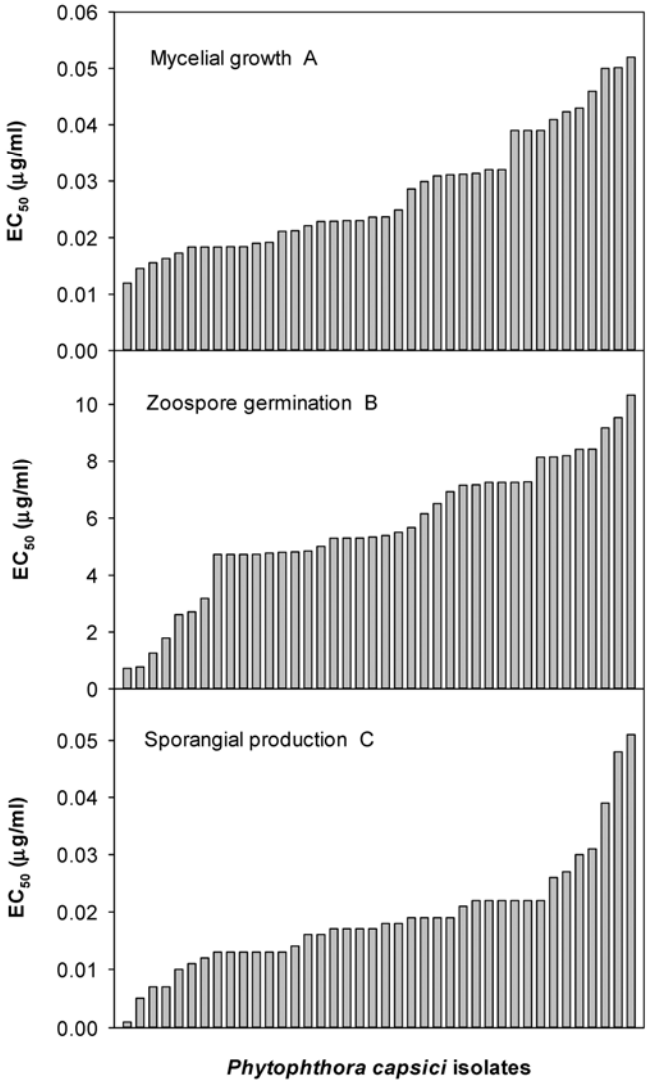

Fig. 1. Concentrations that were 50% effective (EC_{50} values) of mandipropamid for inhibiting **A,** mycelial growth; **B,** zoospore germination; and **C,** sporangial production of *Phytophthora capsici* isolates.

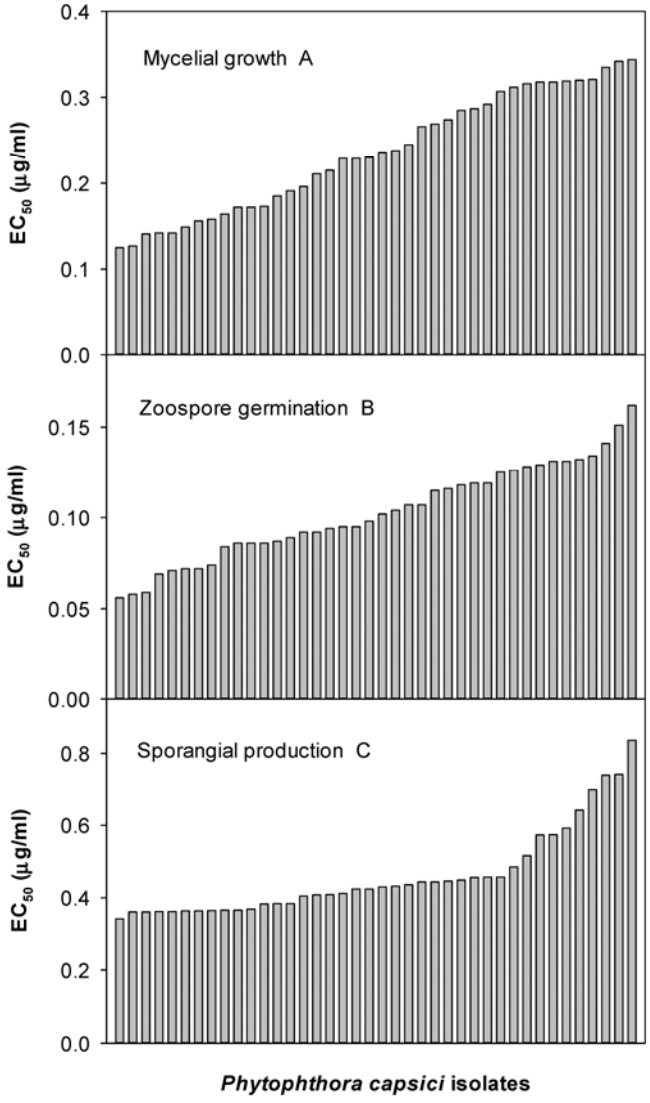

Fig. 2. Concentrations that were 50% effective (EC_{50} values) of dimethomorph for inhibiting **A,** mycelial growth; **B,** zoospore germination; and **C,** sporangial production of *Phytophthora capsici* isolates.

germination of the isolates was 0.5 to >1 µg/ml. EC_{50} values of dimethomorph in suppressing zoospore germination of the isolates were 0.06 to 0.16 µg/ml, with an average of 0.1 ± 0.03 µg/ml (Fig. 2B). The MIC that completely inhibited sporangium formation of the isolates was 2.5 to 5 µg/ml. EC_{50} values of dimethomorph in suppressing sporangium formation were 0.34 to 0.84 µg/ml, with an average of 0.46 ± 0.12 µg/ml (Fig. 2C). Suppression of mycelial growth, zoospore germination, and sporangial production were not correlated with one another (*data not shown*).

Sensitivity of mycelial growth, zoospore germination, and sporangium formation to cyazofamid. All 40 isolates tested were either intermediately sensitive or resistant to cyazofamid at 100 µg/ml based on mycelial growth. When evaluated at higher concentrations, all of the isolates were intermediately sensitive to the compound at 500 µg/ml, and 27.5 and 72.5% of the isolates were sensitive and intermediately sensitive to 1,000 µg/ml, respectively (Fig. 3A). The majority of isolates were resistant or intermediately sensitive to cyazofamid at concentrations of 100 µg/ml or lower based on sporangium formation, with 7.5, 82.5, and 10% being sensitive, intermediately sensitive, and resistant to 100 µg/ml (Fig. 3B). All the isolates were either sensitive or intermediately sensitive to cyazofamid at 500 or 1,000 µg/ml for sporangium formation. The isolates were sensitive to cyazofamid based on zoospore germination. EC_{50} values of cyazofamid in suppressing zoospore germination of the isolates were 0.007 to 0.08 µg/ml, with an average of 0.04 ± 0.02 µg/ml (Fig. 4).

Discussion

The active ingredients of three recently available fungicides for management of Phytophthora blight were evaluated in this study. Mycelial growth, zoospore germination, and sporangial production of *P. capsici* from vegetable crops in Georgia were suppressed by mandipropamid and dimethomorph but cyazofamid was ineffective in suppression of some asexual stages of the pathogen. These results provide useful information for understanding the effects of the chemical compounds on the pathogen's life cycle that is crucial for effective disease suppression.

Mandipropamid is a new carboxylic acid amide (CAA) fungicide that has been demonstrated to be effective in reduction of diseases caused by oomycete pathogens, including Phytophthora blight on vegetables (8,9,23). Little is known about the activity of this compound on different development stages of *P. capsici.* In the present study, the 40 isolates of *P. capsici* from different vegetable crops in Georgia were sensitive to mandipropamid with mean EC_{50} values of 0.03, 5.7, and 0.02 µg/ml for suppression of mycelial growth, zoospore germination, and sporangial production, respectively. It appeared that mandipropamid inhibited mycelial growth and sporangial production of *P. capsici* at lower concentrations compared with suppression of zoospore germination. Cohen et al. (5) evaluated the effect of mandipropamid on mycelial growth of *P. infestans*, causal agent of potato late blight. All isolates tested were sensitive to this compound, with EC_{50} values of 0.02 to 2.98 µg/ml. In a study to determine baseline sensitivity of *Peronophythora litchi*, which causes downy blight on litchi, mandipropamid strongly inhibited mycelial growth, sporangial production, and zoospore germination of the pathogen with mean EC_{50} values of 0.0048, 0.0032, and 0.0023 µg/ml, respectively (32). The effectiveness of mandipropamid in suppressing multiple asexual stages of *Phytophthora capsici* and other oomycete pathogens makes it a good choice in developing integrated fungicide programs for managing Phytophthora blight and diseases caused by oomycetes.

Dimethomorph is another CAA fungicide that showed activities in suppression of various *Phytophthora* spp. (4,15,21,27,30,31). EC_{50} values of dimethomorph in suppression of mycelial growth, zoospore germination, and sporangial production of the 40 *P. capsici* isolates in this study averaged 0.24, 0.10, and 0.46 µg/ml, respectively. In previous studies, sensitivity of 11 *P. infestans* isolates to dimethomorph was examined (30), which indicated that mycelial growth and zoospore germination were sensitive with EC_{50} values for most isolates <0.2 µg/ml, and EC_{50} values were 0.04 to 0.44 µg/ml with a mean of 0.22 µg/ml for sporangial production. A study by Cohen et al. (4) indicated that dimethomorph inhibited mycelial growth of *P. infestans* isolates by 90% at 0.3 µg/ml and zoospore germination was completely inhibited at 0.06 µg/ml. In studies on *P. capsici* at several geographic locations, different asexual stages of the pathogen were sensitive to dimethomorph. Assessment of 90 *P. capsici* isolates in China indicated that dimethomorph inhibited mycelial growth with EC_{50} values of 0.12 to 0.20 µg/ml (31). An isolate of *P. capsici* from chili pepper in Arizona was sensitive to dimethomorph with EC_{50} values from <0.1 to 3.9 µg/ml for suppression of mycelial growth, sporangium formation, and zoospore germination (21). The effect of dimethomorph on mycelial growth and zoospore germination of 61 and 30 *P. capsici* isolates, respectively, in South Carolina was investigated (15), which showed that dimethomorph inhibited mycelial growth with a mean EC_{50} of 0.19 µg/ml and zoospore germination with a mean EC_{50} of 0.07 µg/ml. The results in our study with *P. capsici* isolates in Georgia were in agreement with the above-mentioned studies on this pathogen. Dimethomorph is an active ingredient in several fungicides targeting oomycete pathogens and has been used in the

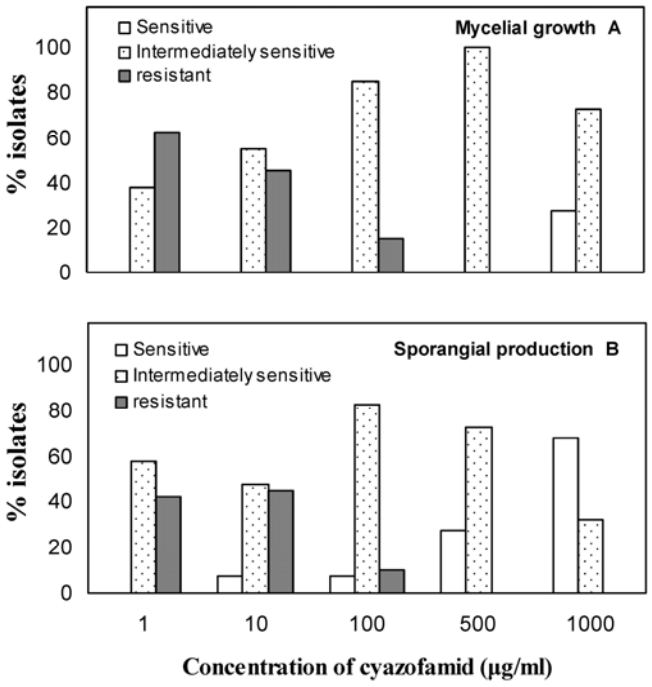

Fig. 3. Percentage of *Phytophthora capsici* isolates that were resistant, intermediately sensitive, and sensitive to different concentrations of cyazofamid. **A,** Based on mycelial growth and **B,** based on sporangial production.

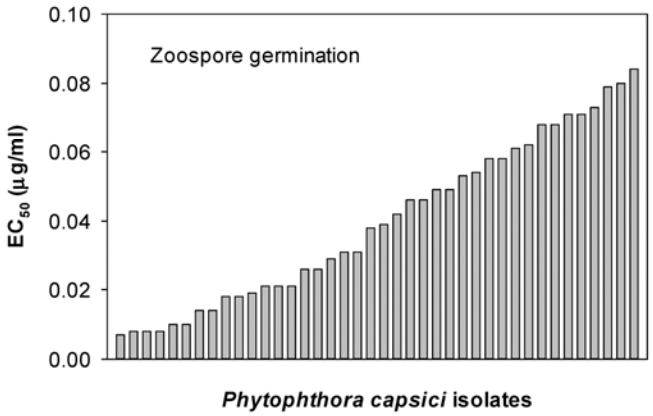

Fig. 4. Concentrations that were 50% effective (EC_{50} values) of cyazofamid for inhibiting zoospore germination of *Phytophthora capsici* isolates.

past decade. It is noteworthy that no *P. capsici* isolate was found to be resistant to dimethomorph or mandipropamid in this study. Continuous monitoring of potential development of resistance in *P. capsici* populations will help ensure effective disease control by these compounds.

Resistance to mefenoxam has been found in *P. capsici* in different areas in the United States (2,6,11,13,15,20). Café-Filho and Ristaino (2) reported that 63% of *P. capsici* isolates from squash and pepper fields in North Carolina were resistant to mefenoxam. The percentage of isolates resistant to mefenoxam in Georgia was relatively lower. Of the 154 *P. capsici* isolates evaluated in this study, 70.8% were sensitive to mefenoxam at 100 µg/ml and 29.2% of the isolates were either resistant or intermediately sensitive. Although the majority of isolates in Georgia was found to be sensitive to mefenoxam, the presence of resistant populations challenges the usefulness of the compound. Studies to determine whether shifts of sensitive population of *P. capsici* toward resistant population may occur along with further mefenoxam usage are warranted.

Cyazofamid is a newer fungicide that showed activity in controlling *P. capsici* and other oomycetes in recent years (8,22,23,26). Limited studies have been conducted to determine the effect of cyazofamid on different developmental stages of *Phytophthora* spp. The effect of cyazofamid on various asexual stages of 40 *P. capsici* isolates collected from different vegetable crops in Georgia was evaluated in this study. All isolates were resistant or only intermediately sensitive to cyazofamid at concentrations of 100 µg/ml or lower, based on mycelial growth. In addition, the majority of the isolates were resistant or intermediately sensitive to cyazofamid at concentrations of 500 µg/ml or lower, based on sporangial production. However, the compound was effective in suppressing zoospore germination, with an average EC_{50} value of 0.04 µg/ml. Earlier studies indicated that EC_{50} values of cyazofamid for inhibiting mycelial growth of five *P. infestans* isolates were between 0.008 and 0.03 µg/ml, and 0.2 µg/ml for suppressing mycelial growth of a *P. sojae* isolate (24). A study on *P. capsici* isolates indicated that some isolates were resistant to this compound and EC_{50} values for suppressing mycelial growth were 3.8 to 535 µg/ml (17). Ranman (a.i. cyazofamid) was registered for control of vegetable diseases caused by oomycetes in recent years. Because cyazofamid appeared to be ineffective in suppressing some asexual stages in the life cycle of *P. capsici* in Georgia, the usefulness of this compound in control of this pathogen deserves further investigation. In considering that cyazofamid was very active in inhibiting zoospore germination, developing programs integrating this compound with other fungicides less effective in suppressing zoospore germination may improve disease control efficacy.

In summary, mandipropamid and dimethomorph were effective in suppression of *P. capsici* at all stages of pathogen development evaluated in the study. Mandipropamid inhibited mycelial growth and sporangial production at lower concentrations than dimethomorph, while dimethomorph inhibited zoospore germination at lower concentrations than mandipropamid. Cyazofamid was effective in suppressing zoospore germination but not effective in suppression of mycelial growth and sporangial production. It is notable that the *P. capsici* populations studied in Georgia were not resistant to mandipropamid and dimethomorph. For the majority of isolates, certain asexual stages were resistant to cyazofamid. It was reported that some *P. infestans* isolates developed resistance to dimethomorph (36), and cross-resistance may occur in this fungicide group. Hence, there is always a chance of resistance occurring, and studies to monitor resistance development in *P. capsici* populations need to be pursued. Effective strategies for managing Phytophthora blight are desperately needed due to the economic importance of the vegetable crops and destructive nature of this disease in production. It may be beneficial to develop disease management programs integrating effective fungicides consisting of different modes of action and other established practices to enhance and sustain disease suppression capacity.

Acknowledgments

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