Chapter 4.

Control of Seed Transmission of *Cylindrocladium parasiticum* in Peanut with Seed Treatment Fungicides

ABSTRACT

VA 98R and NC-V 11 seed with speckled testae was treated with fungicide and assayed on a selective medium to determine the viability of *Cylindrocladium parasiticum*. The fungus was isolated from untreated speckled seed of VA 98R and NC-V 11 at frequencies of 78 and 90 %, respectively. Seed treatment with captan + pcnb + carboxin, fludioxonil, captan, trifloxystrobin, and thiophanate methyl led to the greatest reduction in recovery of the pathogen. Tebuconazole and an experimental compound, LS 176, did not reduce pathogen recovery but did prevent mycelial growth on the seed testae. Speckled seed of NC 7, VA-C 92R, VA 98R, and NC-V 11 was treated with fungicide and planted in two greenhouse and three field trials in Suffolk, Virginia in 1999 and 2000. Plants became infected with *C. parasiticum* after treated and untreated speckled seed was planted in steam-treated soil in the greenhouse. In one trial, all fungicides except thiophanate methyl and thiabendazole significantly reduced the number of symptomatic plants compared to untreated seed. Seed treatment with fludioxonil, tebuconazole, and LS 176 significantly reduced taproot colonization by *C. parasiticum* compared to the untreated check. In a second greenhouse trial, only fludioxonil provided significant suppression of disease. Seed transmission in the field depended on the level of viable *C. parasiticum* present in speckled seed. *C. parasiticum* was recovered from speckled seed of NC-V 11 at a rate of 75 % and the effects of fungicides on seed transmission of the fungus were evident. Treatment with fludioxonil, thiram, and tebuconazole significantly lowered CBR incidence compared to the untreated check and captan + pcnb + carboxin. Treatment with thiram significantly reduced taproot colonization compared to all treatments except fludioxonil. Based on the present study, seed treatment with thiram and fludioxonil may offer the best protection against seed transmission of *C. parasiticum*. Combinations of fungicides are currently being evaluated for increased activity against the seedborne pathogen.

INTRODUCTION

Cylindrocladium black rot (CBR) of peanut (*Arachis hypogaea* L.) is a disease of economic importance to growers in Virginia where the disease is widespread. CBR caused an estimated yield loss of 6.7 million dollars for the state in 2000 (9). The causal fungus, *Cylindrocladium parasiticum*, Wingfield and Alfenas (teleomorph=*Calonectria ilicicola* Boedijn and Reitsma), overwinters in soil and plant debris as multicellular, thick-walled microsclerotia (1, 2, 16). During the growing season, root exudates from peanut are thought to stimulate the germination of microsclerotia and subsequent taproot infection is favored when soil temperatures are 20 to 25°C and soil moisture is near field capacity (7, 11). Taproot colonization by *C. parasiticum* results in decay and eventually plant death (1, 3).

In addition to taproots, the fungus can also infect hypocotyls, pegs, pods, and seed (1). A speckled testae is a symptom of seed infection by *C. parasiticum* (15). In some instances, asymptomatic seed may also carry the pathogen but at very low frequencies. The incidence of *C. parasiticum* in speckled seed has been documented and survival of the fungus in speckled seed during winter storage makes them likely agents for the dissemination of *C. parasiticum* (14, 15, Chapter 2).

Greenhouse as well as field trials in Virginia and North Carolina have provided evidence for transmission of *C. parasiticum* after planting speckled seed of some seed lots (4, 5, 6, 15, Chapter 3). Plants grown from speckled seed planted in steam-treated soil in the greenhouse became infected by *C. parasiticum*. In the field, seed transmission was inconsistent among seed lots and its occurrence was associated with the level of viable inoculum of *C. parasiticum* in speckled seed at the time of planting. Differences in temperature and duration of winter seed storage among seed lots as well as physiological differences among cultivars may contribute to the variation in pathogen survival in speckled seed. When speckled seed with a high recovery rate of the pathogen was planted in field plots, CBR incidence was correlated to the number of speckled seeds planted.

Effective control measures are needed to prevent transmission of *C. parasiticum* in seed. Several strategies have been proposed to prevent disease spread due to seed transmission of *C. parasiticum*. A three year rotation with non leguminous crops as well as soil fumigation with metam sodium two weeks prior to planting can reduce populations of *C. parasiticum* in seed production fields (8, 10, 12). Soil fumigation is currently practiced in approximately 75 % of

Virginia's peanut acreage. The establishment of thresholds for CBR incidence in fields where seed peanuts are produced may reduce the number of infected seeds in commercial seed lots. However, plants with no apparent aboveground disease symptoms but with infected pods and seed belowground may go unnoticed during field inspections. Increasing the already present efforts of shellers to remove speckled seed from commercial seed during routine electronic sorting procedures has been recognized as another means to reduce seed transmission of the fungus. Finally, the application of seed treatment fungicides is yet another strategy for the prevention of seed transmission of *C. parasiticum*. Chemical treatment of all commercial seed may provide the additional protection necessary for speckled seed that escape other management practices. The application of seed treatment fungicides has an advantage that it is already a well established practice in the Virginia seed industry. The most effective strategy for prevention of seed transmission of CBR will likely include the integration of several practices.

Previous research on the role of seed treatment fungicides in the eradication of *C. parasiticum* from seed has led to conflicting results. In early laboratory studies, Porter et al. were unable to isolate the pathogen from seed after treatment with dicloran + captan, carboxin + captan + dicloran, captan + maneb + pentachloronitrobenzene (pcnb) + etridiazole, thiram + pcnb, and carboxin + thiram + dicloran (13, 14). These studies were conducted before the symptom of speckled testae now associated with seed infection by *C. parasiticum* had been thoroughly described (15). More recently, Randall-Schadel demonstrated seed transmission of *C. parasiticum* in a field with no history of peanut cropping after planting speckled seed treated with either captan + carboxin + dicloran (Gus 3-Way, Gustafson), or captan + pcnb + carboxin (Vitavax PC, Gustafson) (15). Greenhouse and field trials in Virginia have confirmed that transmission of *C. parasiticum* can occur when planting speckled seed treated with captan + pcnb + carboxin, the commercial standard seed treatment fungicide of the industry (Chapter 3).

The goal of the present study was to identify fungicides with increased activity against *C. parasiticum* compared to the commercial standard. Seed treatment fungicides were evaluated for their ability to eradicate *C. parasiticum* from speckled seed in the laboratory and prevent seed transmission of the pathogen in the greenhouse and field.

MATERIALS AND METHODS

Seed collection. Seed utilized in all laboratory, greenhouse, and field trials was collected from seed lots in Virginia from the 1998 and 1999 growing season. Speckled seed was

handpicked from seed lots at commercial shelling plants after standard shelling, sizing, and sorting procedures but before fungicide treatment. After collection, seed was packaged in polyethylene bags and stored in an incubator at 15°C. In all experiments, only seed from the previous year's crop was utilized.

Laboratory and greenhouse trials. Fungicides representing a wide range of chemistry were evaluated in the laboratory and greenhouse for activity against *C. parasiticum* in speckled seed and control of seed transmission of the pathogen. Speckled seed of VA 98R and NC-V 11 was separated into 80 g batches and treated with the following fungicides: captan (Captan 400, Gustafson), trifloxystrobin (Flint, Novartis), fludioxonil (Maxim 4FS, Novartis), azoxystrobin (Abound 2.08F, Zeneca), tebuconazole (Raxil 2.6F, Gustafson), thiophanate methyl (Topsin M 70W, Elf Atochem), thiabendazole (Mertect 340F, Novartis), difenoconazole + mefenoxam (Dividend XL, Novartis), LS 176 (Gustafson), and captan + pentachloronitrobenzene (pcnb) + carboxin (Vitavax PC, Gustafson). Tables in the results provide a list of fungicide rates (g a.i./kg seed) tested in laboratory, greenhouse, and field trials. The rate of the experimental compound, LS 176, was expressed as the weight of formulated product/kg seed. Fungicides were applied to seed while rotating in a Gustafson seed treater for laboratory use. Liquid and wettable formulations were diluted with water and applied to seed as a mist at a spray volume of 7 ml/kg seed using an airbrush. Captan + pcnb + carboxin was applied to seed as a dust treatment. Ethephon dust at 1.25 g/kg seed was applied after treatment of seed with fungicide in order to break seed dormancy in greenhouse trials.

Treated speckled seed of VA 98R and NC-V 11 was stored for about three weeks in polyethylene bags at 15°C and then assayed on a medium selective for *C. parasiticum*, consisting of potato broth from 200 g potatoes, 20 g dextrose, 17 g agar, and 1 liter distilled water. The medium was autoclaved for 10 min at 120°C and 103 kPa. After cooling to 55°C, the medium was amended with chlortetracycline (100 mg), chloramphenicol (100 mg), pcnb (52.5 mg), thiabendazole (2.3 mg), and dicloran (2 mg), poured into 10 x 2.5 cm plastic petri plates and allowed to congeal. For each cultivar, 50 treated and untreated seeds were each placed in a single layer in small plastic dishes. Distilled water was added to dishes until the water level reached the top of the seed layer and dishes were covered with moist towels in order to promote the imbibition of fungicides. After soaking for 1 hour, seed with intact testae was rinsed with water and cut latitudinally (each cotyledon cut in half). Both cut ends were then placed in

contact with the isolation medium. Five seeds were assayed per petri plate and a total of 50 seeds were assayed per treatment. Plates were incubated at room temperature and observed for growth of *C. parasiticum* for 14 days. The number of seeds positive for *C. parasiticum* was counted and recovery percentage of the pathogen was calculated.

Greenhouse trials were conducted at the Tidewater Agricultural Research and Extension Center in Suffolk, Virginia and at Virginia Tech in Blacksburg. Treated and untreated speckled seed was planted in a complete block design with five replications. A soil mixture containing 2:2:1 parts steamed soil, peat moss, and vermiculite was utilized at the Suffolk location and a 2:1 mixture of commercial potting soil and sand was utilized at Blacksburg. *Rhizobium* inoculant (Nitro-fix, Trace Chemicals LLC) was added to potting mixtures before seed was planted. Seed was planted at a depth of 3.8 cm (radicle down) in 15.2-cm pots. Five seeds were planted per pot and each pot was a replication. Plants were watered as needed to maintain adequate soil moisture and the maximum and minimum air temperatures were recorded daily. Thrips were controlled through foliar applications of acephate as needed.

The number of emerged seedlings was counted at ca. 24 days after planting and the emergence percentage was calculated for each replication. Each week, plants were visually rated for aboveground disease symptoms using a zero to three scale (0=healthy, 1=stunted, 2=wilted, 3=dead). Non-emerged seedlings were given a rating of three. Plants were removed from soil approximately 11 weeks after planting and root systems were rinsed until free of debris. Each taproot was evaluated for severity of decay using a zero to five scale (0=healthy, 5=completely decayed). Non-emerged seedlings were given a rating of five. Taproots were assayed for the presence of *C. parasiticum*. For each root, five thin sections were excised, disinfested in a 0.526 % sodium hypochlorite solution for two min, and placed on the previously described medium. Petri plates were incubated at room temperature and observed for growth of *C. parasiticum* for 14 days. Non-emerged seedlings and plants without taproots were evaluated as positive for *C. parasiticum*. Fresh weight of total plant tissue and root tissue were recorded.

Field trials. Three field trials were conducted at the Tidewater Agricultural Research and Extension Center. Seed treated with fungicide was planted in randomized complete blocks with four replications and plots consisted of two rows either 7.6 or 9.2-m in length. In 1999, seed mixtures containing normal and speckled seed of VA-C 92R (360 g speckled and 540 g normal) were planted. In 2000, speckled seed of NC 7 was utilized in one field trial. In a second

trial, speckled seed of VA 98R and NC-V 11 was planted in a split-plot design. Main plots were treatments and subplots were cultivars.

The following fungicide treatments were tested: captan (Captan 400, Gustafson), thiram (42-S Thiram, Gustafson), pcnb (RTU PCNB, Gustafson), trifloxystrobin (Flint, Novartis), fludioxonil (Maxim 4FS, Novartis), fludioxonil + mefenoxam (Maxim 4FS + Apron XL, Novartis), azoxystrobin (Abound 2.08F, Zeneca), tebuconazole (Raxil 2.6F, Gustafson), tebuconazole + captan + pcnb + carboxin, difenoconazole + mefenoxam (Dividend XL, Novartis), and captan + pcnb + carboxin (Vitavax PC, Gustafson). All rates were expressed in g ai/kg seed. Experimental compounds, LS 193 and LS 193 + LS 176, were applied to seed at 2.5 g product/kg seed. Liquid and wettable formulations were diluted with water and applied as a mist using an airbrush at a spray volume of 7.8 ml/kg seed. The fungicide sprays were applied while seed tumbled in a rotating drum of a Gustafson seed treater. In 1999, two fungicides were applied as in-furrow treatments. Tebuconazole, 227 g/ha (Folicur 3.6F, Bayer) and azoxystrobin, 153 g/ha (Quadris 2.08F, Zeneca) were applied to the seed furrow through microtubes at planting. Seed treated with captan + pcnb + carboxin was planted in the treated furrow.

All field plots were treated with metam sodium at 36 kg ai/ha (Metam 42 %, UCB Chemicals Corp.) for the destruction of soilborne inoculum of *C. parasiticum*. The fumigant was applied as a row treatment 20 cm below the soil surface using a coulter with a trailing shank, and rows were shaped to form beds (61 cm wide x 10 cm high). Several days after the first application, metam sodium at 36 kg ai/ha was applied to all plots a second time to ensure even application of the chemical. Both applications of metam sodium were made at least two weeks prior to planting. Seed was planted in the center of beds using KMC planters. The seeding rate was about 10 to 13 seed/m and placement was about 5 cm deep. Thereafter, standard practices for peanut production in Virginia were followed.

Plant populations were recorded at *ca*. 2 and 4 weeks after planting. Plots were scouted for CBR incidence every 2 weeks and the number of symptomatic and/or dead plants per plot was recorded. AUDPC (area under the disease progress curve) was calculated in some trials to allow comparison of treatments over an entire season. Field plots were dug in October. Immediately after digging, 25 taproots were randomly selected from each plot and assayed on the selective medium for the detection of *C. parasiticum*. In 2000, the potato, dextrose, and agar components of the medium were replaced with 39 g of potato dextrose agar (DIFCO). One

section from each root was excised, disinfested, and assayed as previously described. Petri dishes were incubated at room temperature and the percentage of taproots colonized by *C. parasiticum* per plot was recorded. Pods were harvested in mid to late October with a two-row Amadas machine modified for plot research. Whole pods were dried with forced air and supplemental heat as recommended in the region until seed showed about 8 to 10 % moisture content. Pod yield was standardized for 7 % moisture content.

Laboratory assay of seed planted in field trials. The recovery rate for *C. parasiticum* in untreated speckled seed was determined for all seed utilized in field trials. For each cultivar/seed lot planted, 50 speckled seeds were assayed on the seed isolation medium in June. Seed was rinsed for 1 min under running water, cut latitudinally, and surface disinfested in a 0.26 % sodium hypochlorite solution for 1 min. The cut ends were placed in contact with the medium and five seeds were assayed per petri plate. Petri plates were incubated at room temperature and observed for growth of *C. parasiticum* for 14 days.

Statistical analysis. Analyses were performed using SAS (17). Treatment means were separated using Duncan's new multiple range test (P=0.05).

RESULTS

Laboratory assay of fungicide-treated seed. *C. parasiticum* was isolated from 78 % of untreated speckled seed of VA 98R (Fig. 5A). All fungicides significantly reduced pathogen recovery compared to untreated seed. The greatest reductions resulted when seed was treated with captan + pcnb + carboxin or the low and high rates of captan, trifloxystrobin, fludioxonil, or thiophanate methyl. Pathogen recovery for these treatments was reduced to 6, 12, 4, 24, 24, 8, 0, 28, and 14 %, respectively. In NC-V 11, *C. parasiticum* was recovered from 90 % of untreated speckled seed (Fig. 5B). Again, seed treatment with captan + pcnb + carboxin, captan, trifloxystrobin, fludioxonil, and thiophanate methyl led to the lowest recovery rates of the pathogen from speckled seed. Fludioxonil at 0.1 g/kg seed was the only fungicide that significantly lowered pathogen recovery compared to captan + pcnb + carboxin. *C. parasiticum* was isolated from seed treated with the high rate of fludioxonil at a frequency of 34 %. Fungal colonies growing from seed treated with fludioxonil were exceptionally small and did not exhibit the cottony mycelial growth typical of *C. parasiticum*. Seed treatment with LS 176 and tebuconazole did not result in significant reductions in pathogen recovery but did prevent mycelial growth on the seed testae.

Greenhouse trials. Only 52 % of the untreated speckled seed of VA 98R produced seedlings by 19 days after planting (Table 6). Seed treatment with captan + pcnb + carboxin, captan (0.94 g), LS 176 (0.22 g), fludioxonil (0.05 g), azoxystrobin (0.33 g), and tebuconazole (0.05 and 0.1 g/kg seed) led to the greatest increase in seedling emergence. These fungicides increased seedling emergence to 88, 96, 88, 84, 92, 84, and 100 %, respectively. Treatment with thiophanate methyl and thiabendazole reduced the number of emerged plants below that of the untreated check.

At 74 days after planting, disease indices for aboveground symptoms were highest for untreated seed and seed treated with thiophanate methyl or thiabendazole (Table 6). All other treatments resulted in fewer diseased plants. Captan + pcnb + carboxin, LS176, tebuconazole, the low rates of captan and fludioxonil, and the high rate of trifloxystrobin provided the greatest level of disease control. Seed treatment with LS 176, fludioxonil, and the high rate of tebuconazole were also shown to significantly lower the severity of taproot decay compared to the untreated check. Root assays confirmed that these fungicides indeed provided the best protection against taproot colonization by *C. parasiticum*. Recovery percentage of the pathogen in plants grown from untreated seed was 80 %. Upon treatment with LS 176, fludioxonil, and tebuconazole, recovery rates were ≤36 %. The fungus was not isolated from any of the 18 surviving plants from the fludioxonil (0.1 g/kg seed) treatment. All 25 plants from the tebuconazole (0.1 g/kg seed) treatment remained healthy throughout the study and the pathogen was not isolated from any of these plants. Fresh weight of root and total plant tissue was significantly increased when seed was treated with captan + pcnb + carboxin, captan, LS 176, or the high rate of tebuconazole.

In NC-V 11, 84 % of the untreated speckled seed germinated and seed treated with fungicide showed similar germination levels (Table 7). Thrips populations in the greenhouse caused leaf distortion and stunted growth in many plants. Symptoms typical of tomato spotted wilt virus (TSWV) were observed on many plants. Aboveground symptoms of CBR were minimal in plants grown from untreated seed and no treatment was significantly different from the untreated check. Seed treatment with fludioxonil (0.05 g/kg seed) significantly lowered the severity of taproot decay compared to the untreated check and *C. parasiticum* was recovered from taproots at rates of ≥80 % in all treatments except fludioxonil. Taproot colonization was reduced to 52 and 48 % in plants grown from seed treated with the low and high rate of

fludioxonil. No fungicide significantly increased the fresh weight of total plant tissue, but the high rate of fludioxonil did lead to a significant increase in fresh weight of root tissue.

Field trials. In 1999, *C. parasiticum* was recovered from speckled and normal seed of VA-C 92R at 2 and 0 %, respectively. In 2000 trials, the pathogen was recovered from speckled seed of NC 7, VA 98R, and NC-V 11 at rates of 14, 17, and 75 %, respectively.

1999. All fungicide treatments except tebuconazole significantly increased seedling emergence of VA-C 92R (Table 8). The highest plant populations resulted when seed was treated with captan + pcnb + carboxin, LS 193 + LS176, and fludioxonil + mefenoxam. CBR incidence was very low in all treatments throughout the season and on 5 October only four symptomatic plants were present in plots planted with untreated seed (Table 8). Root assays detected only low incidence of *C. parasiticum* in taproots from all treatments (Table 8). All fungicide treatments increased pod yield compared to the untreated check (Table 8).

2000. In NC 7, seed treatment with captan + pcnb + carboxin significantly increased plant stands compared to the untreated check and resulted in the highest plant populations (Table 9). Early stand counts revealed that both rates of tebuconazole (0.1, 0.2 g/kg seed) led to significantly lower plant populations compared to all other treatments. By four weeks after planting, plant stands were still significantly lower for seed treated with the high rate of tebuconazole. A significant reduction in row width was observed on 7 July in plots planted with seed treated with the high rate of tebuconazole compared to all other treatments. Disease levels were comparable in plots of untreated and treated seed up through 31 August. On 26 September, CBR was lowest in plots planted with seed treated with tebuconazole (Table 9). No fungicide was shown to be more effective than another in preventing taproot colonization by *C. parasiticum* and differences in pod yield were insignificant among treatments (Table 9).

In the second field trial, none of the treatments significantly increased emergence of plants compared to the untreated check in VA 98R (Table 10). Plots planted with untreated seed and seed treated with captan + pcnb + carboxin, fludioxonil, or azoxystrobin had the highest plant populations. In NC-V 11, treatment with captan + pcnb + carboxin, thiram, pcnb, or fludioxonil resulted in a significant increase of seedling emergence compared to the untreated check. For both cultivars, seed treatment with tebuconazole (0.05, 0.1 g/kg seed) delayed emergence and stunted plant growth. By four weeks after planting, plant stands for seed treated with the low rate of tebuconazole increased to levels comparable with other treatments but

populations remained among the lowest for seed treated with the high rate of tebuconazole. Row width measurements on 7 July revealed that treatment with tebuconazole resulted in a significant reduction in plant growth compared to the untreated check (Table 10).

Statistical analyses of disease incidence throughout the growing season revealed AUDPC levels were not significantly different among treatments in VA 98R (Fig. 6A). Disease levels were lowest when seed was treated with thiram, pcnb, fludioxonil, tebuconazole, or the low rate of trifloxystrobin or difenoconazole + mefenoxam. Taproot colonization was lowest in plants grown from seed treated with pcnb or trifloxystrobin although these treatments were not significantly different from the untreated check (Fig. 7A). Similar to disease incidence and taproot colonization in VA 98R, differences in pod yield among treatments were not significant but the greatest pod yields resulted when seed was treated with captan + pcnb + carboxin (Fig. 8A).

In NC-V 11, differences in AUDPC levels were significant among treatments (Fig. 6B). Thiram, fludioxonil, tebuconazole, and the low rate of trifloxystrobin or difenoconazole + mefenoxam provided significant disease suppression throughout the season. Only thiram, fludioxonil, and tebuconazole performed significantly better than captan + pcnb + carboxin. Root assays confirmed that thiram and fludioxonil provided the best protection against taproot colonization by *C. parasiticum* (Fig. 7B). The percentage of taproots colonized when speckled seed was not treated (76 %) was reduced to 35, 38, 53, and 45 % when seed was treated with thiram at 0.94 and 1.88 g/kg and fludioxonil at rates of 0.05 and 0.1 g/kg seed, respectively. Seed treatment with thiram and fludioxonil also resulted in the highest yields (Fig. 8B).

DISCUSSION

Captan + pcnb + carboxin (Vitavax PC) is the standard seed treatment fungicide of the peanut industry in Virginia. Shellers apply the fungicide as a dust treatment to seed using seed treaters. The fungicide has both protectant and systemic activity and is registered for control of seed rot and damping off diseases caused by *Rhizoctonia solani*, *Rhizopus* spp., and *Aspergillus* spp. in peanut (18).

In the present study, seed treatment with captan + pcnb + carboxin significantly reduced the recovery of *C. parasiticum* from speckled seed compared to untreated seed in laboratory assays of seed. This treatment also resulted in some of the highest plant populations in both greenhouse and field trials. However, in agreement with previous studies, the seed treatment did

not provide adequate protection against seed transmission of *C. parasiticum* from speckled seed in the greenhouse or field. Laboratory assays of treated seed revealed additional fungicides with activity against *C. parasiticum*. Captan, fludioxonil, trifloxystrobin, and thiophanate methyl significantly reduced pathogen recovery in speckled seed of VA 98R and NC-V 11. Although treatment with LS 176 and tebuconazole did not reduce pathogen recovery, these fungicides showed activity against the pathogen and prevented mycelial growth on the testae of seed.

As observed in previous trials, seed transmission was inconsistent in occurrence and depended upon the incidence of *C. parasiticum* in speckled seed at the time of planting. In the present study, the pathogen was isolated from speckled seed for greenhouse trials at high rates (78 and 90 %). Transmission of the pathogen in seed planted in steam-treated soil, and the effects of seed treatment fungicides on disease development were apparent. In seed planted in field trials, pathogen recovery ranged from 2 to 75 % from speckled seed of various seed lots and cultivars. Levels of disease after planting in the field varied according to the level of seed infection at planting. After planting speckled seed with a low incidence of *C. parasiticum*, no fungicide outperformed the captan + pcnb + carboxin standard in terms of protecting against seedborne and/or soilborne diseases, or producing a high plant population and yield. However, several seed treatment fungicides were found to be superior in performance compared to the industry standard in the prevention of seed transmission of *C. parasiticum* after planting speckled seed with a high incidence of the pathogen.

Seed treatment with fludioxonil, tebuconazole, thiram, and LS 176 resulted in the greatest level of protection against transmission of *C. parasiticum* from speckled seed planted in the greenhouse and/or field. Although tebuconazole showed activity against *C. parasiticum*, treatment with this fungicide delayed emergence and stunted plant growth. LS 176 is an experimental compound and its composition remains confidential. At present, seed treatment with fludioxonil or thiram may offer the best protection against seed transmission of *C. parasiticum*. Both fungicides are registered seed treatments for use on peanut. Thiram was not tested in the laboratory or greenhouse, but a field test revealed seed treatment with this fungicide resulted in significant disease suppression compared to all other treatments except fludioxonil. Thiram significantly reduced CBR incidence and taproot colonization compared to the standard of captan + pcnb + carboxin. Fludioxonil consistently performed better than the industry

standard in suppressing seed transmission of *C. parasiticum* in laboratory, greenhouse, and field trials. This fungicide also resulted in some of the highest plant populations and pod yields.

The mechanism by which fungicides protect against seed transmission of *C. parasiticum* is not known. Thiram is a contact, protective, organic sulfur fungicide. Fludioxonil is a protective, phenylpyrrole compound with long residual activity (18). Fungicides may act by penetrating the seed coat and eradicating the pathogen directly from seed. Compounds that become systemic within the plant may offer additional protection against taproot infection by *C. parasiticum* as plants mature.

In order to provide increased protection against seed transmission of *C. parasiticum*, the addition of one or more fungicides to the standard captan + pcnb + carboxin mixture is necessary. Now that several fungicides have been identified with increased activity against the pathogen, ongoing research is aimed at evaluating fungicide mixtures.

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