Fusarium Fruit Rot of Pumpkin in Connecticut

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ABSTRACT

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In 1992, an unusual array of symptoms was found in Connecticut exclusively on cultivar Howden pumpkins (*Cucurbita pepo*). The lesions decreased marketability of the pumpkins and were categorized as a preharvest dry, hard rot (type 1) or a postharvest soft, sunken rot (type 2). In decreasing frequency of isolation, *Fusarium acuminatum, F. equiseti,* and *F. graminearum* were isolated from type 1 lesions, and *F. graminearum, F. equiseti, F. avenaceum,* and *F. solani* were recovered from type 2 lesions. When isolates of each species were wound-inoculated into mature pumpkins representing 13 cultivars of *C. pepo,* they all produced type 2 lesions except *F. acuminatum,* which produced type 1 lesions. The cultivar Atlantic Giant (*Cucurbita maxima*) was resistant to colonization by all *Fusarium* spp. tested. There was no effective inhibition of hyphal growth or spore germination of *F. acuminatum, F. equiseti,* and *F. graminearum* from eight fungicides tested in vitro.

In 1992, Connecticut growers noticed fruit lesions exclusively on cultivar Howden pumpkins *(Cucurbita pepo* L.), which reduced their marketability and storage life. The symptoms were different from those of black rot, caused by *Didymella bryoniae* (Auersw.) Rehm, in that there was no blackened halo surrounding the lesion, and there was no evidence of a gelatinous fluid oozing from the lesion. When the lesions were examined microscopically, macroconidia of *Fusarium* spp. were observed. Similar lesions were observed on pumpkin in Indiana in 1993 (K. Rane, *personal communication*).

In the United States, at least 10 *Fusarium* spp. have been reported to cause fruit rots on cucurbits (1,2,5), but there is only one report of a fruit rot on pumpkin, which was caused by *Fusarium equiseti* (Corda) Sacc. (4). Therefore, there is little information on the etiology and control of Fusarium fruit rot of pumpkin. The objectives of this paper were to describe the symptoms, to isolate and identify the *Fusarium* spp., to determine pathogenicity of isolates on mature pumpkin fruit and seedlings, and to assess the sensitivity of these *Fusarium* spp. to fungicides registered for use on pumpkins.

MATERIALS AND METHODS

Isolation and identification of *Fusarium* spp. Fourteen commercial pumpkin fields in Connecticut were scouted for symptoms of Fusarium fruit rot in September and October 1992. In these fields, pumpkin fruit rejected by the grower were examined. Commercial fields where symptomatic pumpkins were found

were replanted to pumpkins in 1993 and 1994 and scouted again during July through October. During these visits, the lesions were categorized into one of two types: those with no softening of rind (type 1) and those with an associated soft rot (type 2). Lesions were excised and examined under dissecting and compound microscopes for evidence of sporodochia and mycelium. The outer surface of the rind that surrounded lesions was removed with a sterile scalpel, surface-disinfested in 0.5% NaOCl for 1 min, rinsed, and placed on potato-dextrose agar (PDA) containing streptomycin sulfate (250 mg/liter), water agar containing streptomycin sulfate (250 mg/liter), or Komada's selective medium (7). Subsurface pieces of the rind tissue approximately 5×5 mm were cut and similarly treated. Plates were sealed and incubated on laboratory benches at ambient temperatures (18 to 25°C) for 7 days.

During these investigations, pumpkins with soft sunken lesions that contained macroconidia of *Fusarium* spp. were observed in Indiana (K. Rane, *personal communication*). An isolate of this *Fusarium* spp. and a photograph of the symptoms are included in this report.

Single spores of 25 isolates that included the Indiana isolate were subcultured on carnation-leaf agar (CLA) for identification (9). Clones of each type were sent to the Fusarium Research Center, Pennsylvania State University, for species confirmation by Paul Nelson.

Pathogenicity tests on mature fruits. Mature pumpkins of the cultivars Aspen, Baby Bear, Baby Pam, Howden, New England Pie, Oz, Progold 500, Spirit, Spooky, Tom Fox, Trick or Treat, Wizard, Young's Beauty, and the *Cucurbita maxima* Duch. cultivar Atlantic Giant were grown at the Lockwood farm in Hamden, Connecticut, in 1993 and in 1994. Healthy

pumpkins exhibiting no symptoms of fruit rot were rinsed in 0.5% NaOCl followed by tap water and placed on greenhouse benches for 5 to 7 days.

Two inoculation tests were conducted that required wounding. Inoculation tests were first performed with 25 isolates of *Fusarium* spp. on Howden pumpkin fruit. The first method involved injecting 0.5 ml of a spore suspension (10^4 to 10^5 conidia per ml) into a hole made in the rind using a 1-cm-diameter cork borer. The excised tissue was replaced, wounds were covered with clear adhesive tape, and the fruits were set on greenhouse benches. Inoculated fruits were evaluated 10 to 14 days later. The conidia from each *Fusarium* spp. were grown on CLA and contained mostly, if not exclusively, macroconidia. Each isolate was injected into three different pumpkins. There were three injections per pumpkin, and the experiment was repeated once.

In the second method, Howden pumpkin fruits were inoculated by placing PDA agar plugs colonized by a representative isolate of *Fusarium acuminatum* Ellis & Everh. (PFa1), *F. equiseti* (PFe7), or *F. graminearum* Schwabe (PFg4) into holes cut with cork borers (1 cm diameter) and then treated as before. Each isolate was used three times. There were three inoculations on each pumpkin fruit, and the experiment was repeated two times with Howden pumpkins.

In two separate experiments, the PFa1, PFe7, and PFg4 isolates were inoculated using the second method into 14 mature pumpkins listed before. All of the pathogenicity tests included inoculations using distilled water or sterile agar plugs and with isolates of *Didymella bryoniae* for comparison.

Pathogenicity tests on seedlings. Pathogenicity tests with the representative isolates of each *Fusarium* spp. also were conducted once on seedlings of Howden pumpkins. Seeds were germinated in 10-cm plastic pots filled with commercial potting mix. When seedlings developed their first true leaves, conidial suspensions (10^7 conidia/100 ml) of the PFa1, PFe7, and PFg4 plus *F. avenaceum* (Fr.:Fr.) Sacc. (PFav18), *F. solani* (Mart.) Sacc. (PFs3), *F. proliferatum* (T. Matsushima) Nirenberg (PFp1), and *F. oxysporum* Schlechtend:Fr. (PFo1) were poured around the plants. There were three replicate pots with three seedlings per pot. After 3 weeks, seedlings were evaluated for disease, and the fresh and dry weights of the foliar portions were recorded.

Fungicide assays. Eight fungicides were tested for their efficacy in inhibiting the radial growth and sporulation of PFa1, PFe7, and PFg4. The fungicides tested were benomyl (Benlate 50 DF), chlorothalonil (Bravo 720), copper hydroxide (Kocide 101), mancozeb (Manzate 200), thiophanate (Topsin 85M), triadimefon (Bayleton 50 WP), zineb (Zineb 75 WP), and metalaxyl plus chlorothalonil (Ridomil/Bravo 81 W). Metalaxyl plus chlorothalonil was applied according to the chlorothalonil concentration. Inhibition of spore germination was tested using a paper disk assay described below, and the effects on radial growth were examined on fungicide-amended agar.

The paper disk assay was prepared as follows. Conidia were washed from 8-day-old cultures that were grown on CLA, and suspensions of 1×10^5 to 3×10^5 conidia per ml were prepared. Petri plates (10 cm diameter) of PDA were seeded with the conidia by dipping sterile cotton applicators into the spore suspension and gently rubbing them onto the agar surface three times. A preliminary test using 0, 10, 100, 1,000, and 10,000 µg a.i./ml revealed that 1,000 µg a.i./ml was the optimum concentration for testing spore sensitivity in the paper disk assay. Sterile Whatman no. 1 paper disks (0.75 cm) were dipped into agitated suspensions of the formulated products diluted to 1,000 µg a.i./ml and then placed onto the agar. Plates were incubated at 25°C, and the zones of inhibition were recorded after 2 days. The zones of inhibition were comprised of the distance from the edge of the paper disk to areas where 50% of the spores were germinating. There were three disks per plate and three plates per fungicide–isolate combination, and the experiment was repeated once.

Six fungicides were selected from the paper disk assay, suspended in sterile water, and amended into cool molten PDA (48 to 50°C) to yield concentrations of 0, 10, 100, and 1,000 µg/ml agar. The molten agar was agitated and dispensed into 10-cm-diameter petri plates. The solidified plates were seeded in the center with an agar plug (4 mm diameter) colonized by one of the three aforementioned *Fusarium* spp. These plugs were removed from the outer margins of actively growing cultures on PDA. Radial growth was measured on each plate every 2 days until the fungal growth on the control plates had covered approximately 50% of the distance across the plate. The effective concentration required to inhibit the radial growth by 50% (EC_{50}) was determined from regression plots of growth versus log fungicide concentration that were significant at $P \le 0.05$ (11). Any predicted EC_{50} values for fungicides that were greater than 1,000 µg/ml were termed invalid.

RESULTS

Symptoms and isolation. Three pumpkin fields out of the 14 scouted in 1992 had fruit rot on only the cultivar Howden. One field in 1993 had approximately 60% loss in the number of pumpkins due to Fusarium fruit rot, while the other two fields had between 10 and 30% loss due to the disease. There was no obvious pattern that would suggest why pumpkins in these fields developed Fusarium fruit rot. Other cultivars, including Wizard, Progold 500, Spooky, and Oz, that were grown in and among the affected Howden pumpkins were asymptomatic.

There was considerable variation in Fusarium fruit rot symptoms, but lesions were categorized into type 1 and type 2. Type 1 lesions were commonly observed on immature green fruit in the field and appeared as dry, hard, circular to oval lesions (0.5 to 2.0 cm diameter) with depressed dry, corky centers (0.2 to 0.5 cm deep) with or without green halos (Fig. 1, Fig. 2). Orange sporodochia frequently appeared in the center of the lesion. The discolored areas penetrated 0.3 to 0.5 cm into the rind. Type 1 lesions did not expand as the fruit ripened, and pumpkins with type 1 lesions could retain their integrity at greenhouse temperatures for months. The *Fusarium* spp. isolated from type 1 lesions and listed in decreasing order of occurrence were *F. acuminatum,*
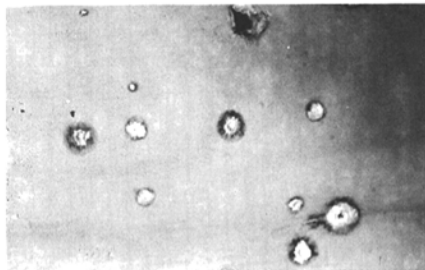

Fig. 1. Preharvest type 1 lesions of Fusarium fruit rot showing the darkened halo.

Fig. 2. Pumpkin fruit covered with the preharvest type 1 lesions of Fusarium fruit rot.

Table 1. Classification of *Fusarium* spp. recovered from two types of lesions observed on cultivar Howden pumpkins

| Species | Total (%)[x] | Recovery from lesion types[w] | | Pathogenic[y] |
		Type 1	Type 2	
F. acuminatum	28	Common	Never isolated	Yes
F. avenaceum	4	Rare	Rare	Yes
F. equiseti	28	Common	Common	Yes
F. graminearum	24	Common	Common	Yes
F. oxysporum	4	Never isolated	Rare	No
F. proliferatum	4	Never isolated	Rare	No
F. solani (CT)	4	Rare	Never isolated	No
F. solani (IN)	4	...	Common[z]	Yes

[w] Lesion types refer to field symptoms: type 1 = preharvest dry, hard rot with circular to oval lesions (0.5 to 2.0 cm diameter) with a depressed dry, corky center (0.2 to 0.5 cm deep) (Fig. 1, Fig. 2); type 2 = soft, sunken lesions with or without water-soaked appearance that may or may not have aerial mycelium (Fig. 3A-C).

[x] Total of 25 isolates of *Fusarium* spp. that were identified to species.

[y] Pathogenic means the isolate caused a lesion on mature pumpkin fruit and was reisolated from the inoculated fruit.

[z] Isolates of *F. solani* were provided by K. Rane, Purdue University, and were observed only once in the summer of 1993.

F. graminearum, *F. equiseti*, and *F. avenaceum* (Table 1). *F. acuminatum* was recovered more frequently from the subepidermal tissue than were the other *Fusarium* spp., while *F. equiseti* was found more often sporulating on the surface of type 1 lesions than were the other *Fusarium* spp.

Type 2 lesions were found more often on fruit after harvest (Fig 3A–C), were less well defined, and could be associated with aerial mycelium. They were commonly found on fruits that were affected with the type 1 symptoms (Fig. 3A). Pumpkins exhibiting these lesions would more often collapse. The *Fusarium* spp. isolated from type 2 lesions and listed in decreasing order of occurrence were *F. graminearum*, *F. equiseti*, and *F. avenaceum* (Table 1). On one occasion in 1993, isolates of *F. solani*, *F. proliferatum*, and *F. oxysporum* were recovered from pumpkins on the verge of collapse, but they were not isolated in 1994, nor was *F. avenaceum* isolated in 1994. The isolate that was retrieved from symptomatic mature pumpkins from Indiana was identified as *F. solani*, and although the symptoms appeared slightly different from those in Connecticut, they were characterized as type 2 lesions (Fig. 3C). Of the 25 isolates that were identified to species in this study, seven were *F. acuminatum*, seven were *F. equiseti*, six were *F. graminearum*, two were *F. solani* (one from Connecticut and one from Indiana), and one each were *F. avenaceum*, *F. oxysporum*, and *F. proliferatum* (Table 1).

Pathogenicity tests with mature fruit. Both methods of inoculation were effective in reproducing lesions from the *Fusarium* spp. Species of *F. acuminatum*, *F. graminearum*, *F. avenaceum*, and *F. equiseti*, and the *F. solani* culture from Indiana all expanded from the original wound and produced lesions. The fungi were readily reisolated from the lesions. *F. acuminatum* commonly caused a type 1 lesion that expanded approximately 0 to 1.0 cm over a period of 4 weeks. Inoculations with *F. graminearum*, *F. equiseti*, and *F. avenaceum* produced the type 2 lesion. Lesions caused by *F. equiseti* frequently produced a water-soaked appearance, which was one of the field symptoms (Fig 3B). The lesions caused by *F. equiseti* expanded approximately 2 to 5 cm over 4 weeks, as opposed to those caused by *F. graminearum* or *F. avenaceum*, which expanded 1 to 4 cm. *F. graminearum* tended to produce lesions with aerial mycelium more than *F. equiseti* or *F. avenaceum*. When aerial mycelium was present on *F. equiseti*-inoculated lesions, it appeared as zonal rings filled with macroconidia. The Indiana isolate of *F. solani* produced soft, sunken lesions with dark, sporulating centers, which were very similar to the symptoms observed in the field (Fig 3C). The Connecticut isolate of

Fig. 3. Postharvest symptoms of type 2 lesions of Fusarium fruit rot on cultivar Howden pumpkins in Connecticut (**A** and **B**) and Indiana (**C**). (**A**) Lesions with profuse aerial mycelium on a fruit that also has type 1 lesions. (**B**) Soft, sunken, water-soaked lesion without aerial mycelium. (**C**) Soft, sunken lesion with aerial mycelium and sporulation (courtesy K. Rane).

F. solani was not pathogenic in these tests. Pumpkins inoculated with sterile water or sterile PDA plugs did not produce any lesions, and pumpkins that were inoculated with *D. bryoniae* resulted in typical concentric black halos that surrounded lesions with gelatinous ooze. The isolates of *F. oxysporum*, *F. proliferatum*, and *F. solani* from Connecticut did not produce any lesions when they were inoculated into Howden pumpkins. However, in a few tests, these species began to colonize the perimeter of the wound as the fruit began to age and collapse.

All of the pumpkin cultivars that belong to *C. pepo* reacted similarly to inoculation in the greenhouse with the PFa1, PFe7, and PFg4 isolates. The cultivar Atlantic Giant (*C. maxima*), however, was resistant to colonization in these tests.

Pathogenicity tests with seedlings. Conidial suspensions of the test *Fusarium* spp. did not produce any visual root or leaf differences on the Howden seedlings when they were compared to seedlings exposed to distilled water. Fresh and dry weights did not differ from seedlings exposed to distilled water (data not shown).

Fungicide assays. Of the eight fungicides tested, mancozeb and chlorothalonil were the most inhibitory to spore germination in the paper disk assay, while benomyl restricted the radial growth of the three *Fusarium* spp. more than the other fungicides (Table 2). Chlorothalonil was equal to mancozeb in toxicity to the spores of *F. acuminatum*, but mancozeb was more toxic to the other two species. *F. acuminatum* was more sensitive to the fungicides than were *F. equiseti* or *F. graminearum*. Copper hydroxide, thiophanate, and triadimefon were not toxic at these concentrations. Surprisingly, *F. acuminatum* grew faster when cultured on agar containing 100 or 1,000 µg of copper hydroxide per ml of agar than on nonamended agar.

DISCUSSION

The pumpkin fruit rot symptoms were classified as dry, hard preharvest lesions (type 1) and soft postharvest lesions (type 2). Of the seven *Fusarium* spp. isolated, *F. acuminatum*, *F. equiseti*, and *F. graminearum* were the most common ones found. *F. equiseti* and *F. graminearum* were found on type 1 and type 2 lesions, whereas *F. acuminatum* was only recovered from type 1. On mature pumpkins in the greenhouse, *F. acuminatum* produced type 1 lesions, while *F. equiseti*, *F. graminearum*, *F. avenaceum*, and *F. solani* all produced slightly different type 2 lesions. It is possible that type 1 lesions may develop into type 2 lesions, but this was not observed in the current study. Given the close association between *F. acuminatum* and the type 1 lesion in the field, there may be two distinct symptoms types. However, Bruton (1) and Johnson (6) both cautioned that the symptom expression on the fruit of cucurbits could be strongly influenced by time of infection and environmental conditions. For example, the relative humidity is likely very important in determining whether aerial mycelium is present.

Losses from this disease have not been reported previously in the northeastern United States. Although the damage was limited to Howden pumpkins, the impact could be great because Howden is one of the most widely grown cultivars. Other popular cultivars may also be susceptible under different conditions. The potential destructiveness of Fusarium fruit rot has been evident in muskmelon (*Cucumis melo* L.), where an alarming 30% of the sampled fruit shipments to New York City during 1972 to 1984 had Fusarium rot (3).

F. acuminatum and *F. graminearum* have not been reported on pumpkins, but *F. equiseti* was implicated with pumpkin losses in Arkansas (4). In Australia, *F. acuminatum* and *F. equiseti* were implicated in a disease of butternut (*Cucurbita moschata* (Duch.) Duch. ex Poir.) (6). In that report (6), *F. equiseti* caused zonal lesions on immature and mature fruit, whereas *F. acuminatum* attacked only immature fruit. This description supports the current observations on pumpkins. These two species, along with several others *Fusarium* spp., also cause a dry, spongy fruit rot of cantaloupes (1,2). Bruton (1) reported that two symptom types on cantaloupe could be differentiated by the coloration of the diseased tissue. *F. acuminatum* caused distinct reddish or purplish lesions on cantaloupe, whereas other *Fusarium* spp., including *F. equiseti*, caused an internal brown discoloration. No differences in tissue coloration were noted on pumpkins in the current report. Correll et al. (4) reported that *F. equiseti* caused soft, sunken areas on pumpkins, which was similar to the type 2 symptom observed in this study. Since *F. equiseti* was isolated from both type 1 and type 2 lesions, it may also produce different symptoms under field conditions when environmental conditions vary. *F. graminearum* was also isolated from lesions in both symptom types, and this fungus may also have variable field reactions depending on the time of infection and the environment. It is also likely that other species may be implicated in Fusarium fruit rot of pumpkin, as has been the pattern with other cucurbits (1,2,5).

It was interesting that symptoms were only observed on Howden pumpkins when other cultivars in the same affected areas remained asymptomatic. Since seedlings were not affected by these *Fusarium* spp., and wounding was necessary for infection, there may be histological differences in the formation of the fruit rind that confer resistance to fruit rot. Lester (8) reported that shelf life and perishability of muskmelon was correlated with the thickness of hyphodermal tissue of the fruit. A relation between pumpkin rind thickness and/or cuticle formation and susceptibility to *Fusarium* spp. may also exist. Cracks in the rind and/or cuticle following rapid fruit growth may create natural wounds, and this may occur more frequently in Howden pumpkins than in other cultivars. Experi-

Table 2. Sensitivity of selected fungicides to *Fusarium* spp. associated with pre- and postharvest decay of pumpkin fruit

| Fungicide[x] | *F. acuminatum* | | *F. equiseti* | | *F. graminearum* | |
	Spore inhib.[v] (ZOI, mm)	Radial growth[w] (EC$_{50}$)	Spore inhib.[v] (ZOI, mm)	Radial growth[w] (EC$_{50}$)	Spore inhib.[v] (ZOI, mm)	Radial growth[w] (EC$_{50}$)
Benomyl	0.8 a	19.5	0.1 a	89.1	1.8 ab	158.5
Chlorothalonil	3.2 b	53.7	2.3 ab	ND[y]	1.1 ab	1,001.0
Cu hydroxide	0.0 a	ND	0.0 a	ND	0.0 a	ND
Mancozeb	3.3 b	426.6	4.0 b	ND	4.4 b	ND
Metalaxyl + chlorothalonil	2.1 ab	...[z]	1.9 ab	...	1.3 ab	...
Thiophanate	0.0 a	ND	0.0 a	ND	0.0 a	ND
Triadimefon	0.0 a	...	0.0 a	...	0.0 a	...
Zineb	2.0 ab	ND	1.1 ab	ND	0.5 a	ND

[v] ZOI = Zone of spore inhibition after 24 h around paper disks containing an active fungicide concentration of 1,000 µg/ml; values followed by differing letters are significantly different by Tukey test at (*P* = 0.05).

[w] Radial growth of hyphae across fungicide-amended potato-dextrose agar 25°C in the dark; the EC$_{50}$ values were computed from linear regression plots of growth versus concentration after 8 days for *F. acuminatum* and 4 days for *F. equiseti* and *F. graminearum* when fungal growth on control plates had covered approximately 50% of the distance across the plate; no statistics can be computed on these values.

[x] Fungicides applied as formulated product. Benomyl = Benlate 50 DF, chlorothalonil = Bravo 720, copper hydroxide = Kocide 101, mancozeb = Manzate 200, metalaxyl plus chlorothalonil = Ridomil Bravo 81 W (was applied according to the chlorothalonil concentration), thiophanate = Topsin 85M, triadimefon = Bayleton 50 WP, zineb = Zineb 75 WP.

[y] ND = not determined because the regression plot of growth versus fungicide concentration was not significant at *P* = 0.05, or the EC$_{50}$ value was computed to be greater than the highest fungicide concentration (1,000 µg/ml agar) tested.

[z] Radial growth studies were not conducted on these fungicides.

ments by the author are underway to determine whether Fusarium fruit rot symptoms can result from applying conidial suspensions to young developing pumpkins.

Only a few fungicides had in vitro activity against the *Fusarium* spp. Although these laboratory assays may not reflect the efficacy of each fungicide under field conditions, many of the these fungicides were in use in fields that had symptomatic fruit. Chemical suppression of this disease may not be an effective strategy. More importantly, the major obstacle to fungicide control in the field may be obtaining sufficient coverage of the fruit. Studies designed to suppress *F. semitectum* Sacc. on harvested muskmelons with benzimidazoles concluded that the fruit needed to be dipped in concentrations of 1,000 µg/ml to reduce Fusarium rot (10). Since fruit dips are not practical in pumpkin culture, more information on the etiology of Fusarium fruit of pumpkin is needed to design control strategies.

ACKNOWLEDGMENTS

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