Fusarium moniliforme as a Cause of Stem and Crown Rot of Asparagus and Its Association with Asparagus Decline

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ABSTRACT

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In addition to Fusarium oxysporum f. sp. asparagi, F. moniliforme was isolated from asparagus plants taken from production fields. F. moniliforme was isolated from stem and crown lesions of 12-yr-old plants in a declining field and infrequently from lesions on roots, crowns, and stems of plants from a 2-yr-old planting. Total root system collapse resulted when seedlings were inoculated with F. moniliforme, and typical reddish lesions with accompanying vascular discoloration developed on roots of seedlings inoculated with F. oxysporum f. sp. asparagi. F. moniliforme caused more

extensive crown rot and stem pith discoloration of asparagus plants in crown and stem pathogenicity tests than did *F. oxysporum* f. sp. asparagi. Based on isolation and pathogenicity studies, *F. moniliforme* is considered the pathogen of a separate disease of asparagus. Fusarium stem and crown rot is the proposed name for this disease. Both Fusarium stem and crown rot and Fusarium wilt and root rot caused by *F. oxysporum* f. sp. asparagi are associated with asparagus decline in New Jersey.

Additional key words: Asparagus officinalis L., etiology.

Fusarium wilt and root rot (2), crown rot complex (3), seedling blight (4), and decline and replant problem (5) are soilborne diseases of asparagus (Asparagus officinalis L.) in North America. Of all of these, decline and replant problems are the most poorly understood, and the cause has remained unknown. "Decline" has been described as the reduction in the profitable life of an asparagus planting (5). The "replant problem" is known only because of the difficulty in establishing new plantings where asparagus fields have

declined (5).

In California, Fusarium oxysporum (Schlecht.) emend. Snyd. & Hans. f. sp. asparagi Cohen was considered the most important pathogen associated with the decline and replant problem (5). Fusarium moniliforme (Sheld.) emend. Snyd. & Hans. was pathogenic but was isolated less frequently than F. oxysporum f. sp. asparagi and thus was not considered important in the asparagus decline and replant problem. Graham (4) in Canada isolated F. moniliforme from diseased seedlings and demonstrated that this fungus caused root tip necrosis but also concluded that it was not important in seedling blight. In contrast, Endo and Burkholder (3)

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considered *F. moniliforme* important as a cause of a brown, dry crown rot of asparagus. This work, together with a series of brief and recent reports from Washington (6), Massachusetts (1) and Michigan (7), have begun to implicate *F. moniliforme*, as well as *F.*

oxysporum f. sp. asparagi in asparagus decline.

Asparagus decline is believed to be a principal factor involved in the reduction of asparagus acreage in New Jersey from 32,500 acres in 1957 to 2,300 acres in 1977 (9). Isolations from asparagus plants of various ages revealed a predominance of *F. oxysporum* f. sp. asparagi, with cortical root lesions and typical discolored vascular tissue, in plants from 2-yr-old plantings, but *F. moniliforme* was the dominant Fusarium associated with crown and stem lesions in plants from a 12-yr-old planting. Because of the significant economic impact of the acreage reduction in New Jersey and because the exact cause of the reduced yields in production fields was unknown, this study was undertaken to determine (i) whether *F. moniliforme* is involved in the decline of asparagus in New Jersey and (ii) the relative pathogenicity of *F. moniliforme* compared with *F. oxysporum* f. sp. asparagi.

MATERIALS AND METHODS

Isolates of both F. moniliforme and F. oxysporum f. sp. asparagi were collected at monthly intervals for 1 yr from a 2-yr-old and a 12-yr-old planting and maintained on potato dextrose agar (PDA) slants at 4 C. All isolates were tested for pathogenicity by pouring a spore suspension of each isolate over asparagus seeds (cv. Mary Washington). After 6 wk in the greenhouse at 19-24 C, plants were uprooted and a disease severity index was determined following a system similar to that of Sherwood and Hagedorn (10). The more virulent isolates were tested in more extensive pathogenicity tests.

Three kinds of inoculum were used for pathogenicity tests. Spore suspensions were produced by washing microconidia and macroconidia from plates of 7-day-old V8 agar cultures with sterile deionized water, filtering through cheesecloth, and adjusting to 2×10^6 spores per milliliter of water. Oat grain inoculum was produced by aseptically adding mycelium, microconidia, and macroconidia from 7-day-old PDA cultures to flasks containing autoclaved oat grains and by incubating for 30 days at 23 C. Toothpick inoculum was produced with toothpicks boiled in water, placed in potato dextrose broth, autoclaved, and infested with various selected isolates of F. moniliforme and F. oxysporum f. sp. asparagi.

In one root pathogenicity test, root systems of 4-wk-old asparagus seedlings (cvs. Rutgers Beacon and Mary Washington) were dipped in a spore suspension of *F. moniliforme* and *F. oxysporum* f. sp. asparagi and replanted into a steam-sterilized, 2:1 sand and soil mixture. The test was conducted in the greenhouse at 19-24 C. Plants were uprooted and evaluated as previously described 3 wk

after inoculation.

Oat grain inoculum was used to infest methyl-bromide fumigated soil at 0, 5.6, and 44.8×10^3 kg inoculum per hectare. The infested soil was used to test 5-wk-old asparagus seedlings against F. moniliforme and F. oxysporum f. sp. asparagi in the greenhouse at 17-23 C. Three weeks after inoculation, plants were uprooted and evaluated for the level of root lesions, as well as root collapse. In a stem pathogenicity test, an oat grain containing F. moniliforme, F. oxysporum f. sp. asparagi or an autoclaved, noninfested oat grain was inserted into a longitudinal slit in the stem, covered with petroleum jelly, and plants were placed in the greenhouse at 17-23 C. Three months after inoculation, stems and crowns were split longitudinally and the linear length of stem discoloration was determined.

In a crown pathogenicity test, toothpicks infested with F. moniliforme, F. oxysporum f. sp. asparagi or noninfested toothpicks were inserted into distal buds of 5-mo-old Rutgers Beacon asparagus crowns and placed in the greenhouse at 17–23 C. Three months after inoculation, crowns were split longitudinally and the percentage of internal discoloration was determined.

RESULTS

Association of Fusarium species with organ attacked. F. oxysporum f. sp. asparagi, if isolated, was found consistently in

discolored vascular root tissue and cortical root lesions and occasionally in crowns and stems. In contrast, *F. moniliforme*, if found, was in stem and crown lesions and rarely in root tissue. *F. moniliforme* was isolated infrequently from the 2-yr-old planting but was dominant in the 12-yr-old planting.

Disease severity indices for root, crown, and stem isolates of *F. oxysporum* f. sp. asparagi ranged from 41.0 to 92.0, 53.8 to 85.0, and 69.4 to 82.0, respectively. Of the *F. moniliforme* isolates from roots, crowns, and stems, the disease severity indices ranged from 64.0 to 69.0, 59.0 to 69.0, and 23.0 to 77.0, respectively.

Root pathogenicity test. Where roots were dipped in a spore suspension, foliage of inoculated plants became chlorotic and wilted 1–2 wk after inoculation. Isolates of both Fusarium spp. were pathogenic on both asparagus cultivars (Table 1). Both isolates of F. moniliforme were equally pathogenic, but the FO-4 isolate of F. oxysporum f. sp. asparagi was less pathogenic than the FO-14 isolate on Rutgers Beacon plants. Reddish lesions were present on root systems inoculated with isolates of F. oxysporum f. sp. asparagi and few roots had collapsed. All root systems of plants inoculated with isolates of F. moniliforme were completely collapsed at harvest, and no reddish lesions were present. Each Fusarium sp. tested was reisolated from correspondingly inoculated plants.

When asparagus seedlings were transplanted into Fusarium-infested soil, vigor ratings taken 3 days afer transplanting varied and depended on the inoculum rate and the Fusarium sp. (Table 2). At the lower inoculum density, both F. moniliforme and F. oxysporum f. sp. asparagi caused slight reduction in vigor with both cultivars, but at the higher rate F. moniliforme caused greater reduction in vigor on both cultivars than did F. oxysporum f. sp.

TABLE 1. Vigor ratings and disease severity indices for two isolates of Fusarium oxysporum f. sp. asparagi and two isolates of F. moniliforme on asparagus cultivars Rutgers Beacon and Mary Washington

Pathogen	Rutgers Beacon		Mary Washington		
	Vigorx	DSI ^y	Vigor	DSI	
F. oxysporum					
FO-4	2.5 ab^2	70.8 b	3.2 abc	89.6 cd	
FO-14	6.6 cd	87.5 c	5.6 bcd	95.8 cde	
F. moniliforme					
FM-7	8.3 d	97.9 de	5.2 bcd	100.0 e	
FM-9	6.6 cd	100.0 e	4.8 bcd	100.0 e	
Control	1.0 a	0.0 a	1.0 a	0.0 a	

^{*}Rated 1-10 (excellent-poor).

yDisease severity index, 1-100 (no symptoms-dead).

TABLE 2. Disease expression in Rutgers Beacon and Mary Washington asparagus cultivars inoculated with two rates of Fusarium oxysporum f. sp. asparagi-infested and F. moniliforme-infested oat grains

Cultivar-Pathogen- Inoculum Density (kg/ha × 10 ³)		Vigor ^x	Mortality (%)	Root lesion ^y	Root collapse ^y
Rutgers Beacon		101100000 110		400000000000000000000000000000000000000	200 747
F. oxysporum	(5.6)	$1.0 a^{z}$	0.0 a	6.0 d	1.5 a
	(44.8)	5.5 c	41.7 bc	4.9 cd	6.0 b
F. moniliforme	(5.6)	2.0 ab	41.7 bc	3.0 abc	6.1 b
	(44.8)	9.2 d	100.0 d	1.7 a	10.0 c
Control		1.2 a	0.0 a	1.0 a	1.0 a
Mary Washington					
F. oxysporum	(5.6)	2.5 ab	8.3 ab	6.0 d	2.7 a
	(44.8)	6.0 c	66.6 cd	4.7 bcd	8.0 bc
F. moniliforme	(5.6)	3.7 b	66.6 cd	2.6 abc	7.0 b
	(44.8)	9.0 d	100.0 d	2.5 ab	10.0 c
Control		1.0 a	0.0 a	1.0 a	1.0 a

^{*}Rated 1-10 (excellent-poor).

^zNumbers in a column with a letter in common are not significantly different (P = 0.05).

y₁₋₁₀ (none-severe).

Numbers in a column with a letter in common are not signicantly different (P = 0.05).

asparagi. At the lower inoculum level, F. oxysporum f. sp. asparagi did not cause an increase in mortality compared with the control, whereas F. moniliforme caused an increase in mortality. Inoculation with both Fusarium spp. resulted in higher mortality than in the control at the higher inoculum rate, and F. moniliforme resulted in greater mortality than did F. oxysporum f. sp. asparagi on both cultivars.

As in the previous test, two distinct symptoms were observed on roots of plants growing in Fusarium-infested soil. Reddish lesions accompanied by vascular discoloration were produced on fibrous and fleshy roots of some root systems, whereas other root systems were completely collapsed. On Rutgers Beacon, F. oxysporum f. sp. asparagi infections resulted in a greater amount of reddish lesions at both inoculum rates than did both F. moniliforme and the control. The same was true for the lower inoculum density of F. oxysporum f. sp. asparagi on Mary Washington.

F. moniliforme infections resulted in more severe root collapse at both inoculum levels than occurred in controls, whereas F. oxysporum f. sp. asparagi was able to cause a root collapse at the higher inoculum level only. Each Fusarium sp. tested was reiso-

lated from correspondingly inoculated plants.

Crown pathogenicity test. The percentage of crown discoloration of Rutgers Beacon crowns inoculated with F. moniliforme, F. oxysporum f. sp. asparagi, and the control were 68.8, 46.3, and 36.3, respectively. F. moniliforme produced significantly more crown discoloration than did F. oxysporum f. sp. asparagi and the control, according to Duncan's multiple range test (P = 0.05). F. moniliforme was reisolated from discolored portions of internal crown tissue inoculated with F. moniliforme and from 75% of crowns in the control, whereas F. oxysporum f. sp. asparagi was reisolated only from discolored portions of internal crown tissue inoculated with F. oxysporum f. sp. asparagi.

Stem pathogenicity test. Linear lengths of stem pith discoloration of Rutgers Beacon asparagus stems inoculated with F. moniliforme, F. oxysporum f. sp. asparagi, and the control were 8.9 cm, 4.5 cm, and 1.0 cm, respectively. The length of pith discoloration was significantly greater in stems inoculated with F. moniliforme than in those inoculated with F. oxysporum f. sp. asparagi or noninoculated stems, according to Duncan's multiple range test (P = 0.05). Although stems inoculated with F. oxysporum f. sp. asparagi did not have significantly greater discoloration of the pith than the control, all replicates exhibited pith and vascular discoloration, and only one replicate of control plants exhibited pith discoloration. In 50% of F. moniliforme-inoculated stems, stem pith discoloration extended into the crown, and early stages of crown rot were initiated. No crown discoloration occurred in plants inoculated with F. oxysporum f. sp. asparagi or in control plants. Each Fusarium sp. tested was reisolated from correspondingly inoculated plants, and no Fusarium sp. was isolated from the control.

DISCUSSION

In addition to the isolation of F. moniliforme from older asparagus plants exhibiting crown rot symptoms as previously reported (3), this species was recovered frequently from stem lesions of plants in declining plantings. The (i) frequent isolation of F. moniliforme from crown and stem lesions of plants in older plantings, (ii) extensive crown rot that developed in plants inoculated with F. moniliforme, (iii) basipetal progression of F. moniliforme from stem infection foci into crown tissue, and (iv) initiation of a

crown rot from stem inoculations suggest that F. moniliforme is able to colonize senescing asparagus crown tissue more readily than F. oxysporum f. sp. asparagi. If this is so, it would account for the predominant isolation of F. moniliforme from the older asparagus planting in this study.

Both F. oxysporum f. sp. asparagi (5,8) and F. moniliforme (3) have been reported as surface contaminants on seed. Since these fungi were capable of killing seedlings in pathogenicity tests in this study, damping-off of asparagus seedlings probably would result from seedborne inoculum of both species. This would result in a reduction of asparagus stands in crown nurseries or, more importantly, in direct-seeded production fields.

The reduced vigor of the transplants within 3 days of transplanting into Fusarium-infested soil suggests that a toxin was responsible for the phytotoxicity. Further, the toxin was most likely produced during spore germination and soil colonization, since plants exhibited foliar chlorosis and necrosis within several days of transplanting.

Root lesion ratings were inversely correlated with root system collapse and seedling mortality. This suggests that root system collapse interferes with typical root lesion development and would explain the differences in symptom expression of plants naturally infected with F. moniliforme and F. oxysporum f. sp. asparagi.

The results of this study indicate that F. oxysporum f. sp. asparagi is an important pathogen associated with asparagus decline in New Jersey, as it is in other states having asparagus decline (2,5). However, the association of F. moniliforme in asparagus decline is more important than it appears from accounts in the literature (4,5). Because F. moniliforme was the predominant species isolated from an older planting and caused extensive stem and crown rot in pathogenicity tests, it should be considered the pathogen of a separate disease of asparagus. The proposed name for this disease is Fusarium stem and crown rot.

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