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Evaluation of *Asparagus* Species for resistance to *Fusarium oxysporum* f. sp. *asparagi* and *F. moniliforme*

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Abstract. "Asparagus decline" decreases production and kills *Asparagus officinalis* L. The principal pathogens involved in the decline are considered to be *Fusarium oxysporum* f. sp. *asparagi* Cohen and Heald (FOA) and *F. moniliforme* (Sheld.) emend. Synder and Hans. (FM). Three- to four-month-old plants of *A. officinalis* and three other asparagus species were inoculated in the greenhouse and evaluated for resistance to these *Fusarium* spp. Of the 90 *A. officinalis* accessions evaluated, two all-male cultivars, Lucullus 234 and 328, received the lowest disease ratings to FOA and FM. *Asparagus densiflorus* 'Sprengeri' and 'Myersii' received the lowest disease ratings of the other asparagus species tested. Of the total 95 germplasm entries evaluated, 39% were more resistant than the susceptible control 'UC 157', 44% were rated similar in susceptibility, and 17% were more susceptible. Accessions responded similarly to both *Fusarium* spp.

Asparagus is a perennial vegetable crop that should be productive for 20 years or more with average yields of 2800 kg·ha⁻¹. In 1986, the average yield in Michigan was only 1344 kg·ha⁻¹ (Bernthal and Karsan,

1986). Fields in Michigan are being removed after 8 to 15 years due to small spear size and sparse stands (Takatori and Souther, 1978). Crown loss may be as high as 50% in the first 5 years. Asparagus decline also occurs in other major production areas of the United States (Grogan and Kimble, 1959; Herner and Vest, 1974) and overseas (Cassini, 1981; Molot and Simone, 1965; Van Bakel and Kerstens, 1970). It has also been demonstrated that replanting fields previously affected by the decline syndrome often results in asparagus failure (Putnam and Lacy, 1977).

FOA and FM are found in all asparagus fields and are considered to be the primary pathogens involved in asparagus decline (Cohen and Heald, 1941; Endo and Burkholder, 1971; Grogan and Kimble, 1959; Johnston, et al., 1979; Molot and Simone

1965; Van Bakel and Kerstens, 1970). Both organisms are often isolated from the same plant (Endo and Burkholder, 1971; Johnston, et al., 1979). Asparagus plants affected by either pathogen show similar symptoms in the field: crown and stem discoloration; yellowed, stunted ferns that may die at various stages of elongation; reddish-brown root lesions; and rotting of the roots (Cohen and Heald, 1941; Endo and Burkholder, 1971). *Fusarium* wilt and root rot is the common name for the disease caused by FOA, whereas stem and crown rot refers to FM (Endo and Burkholder, 1971; Johnston, et al., 1979).

Methods of controlling these two diseases are limited due to the lack of efficacious chemicals (Lacy, 1979), limited long-term effectiveness of fumigation due to the perennial nature of the crop (Lacy, 1979), and the rapid colonization of young asparagus plants by *Fusarium* spp. in the field (Damicone and Manning, 1985). To date, the most successful control strategy for *Fusarium* wilt in other vegetable crops has been the development of resistant cultivars (Mace, et al., 1981). Resistant cultivars could allow asparagus growers to avoid the expensive management practice of finding new land for replantings, and lower productivity losses from deteriorating fields. Therefore, we evaluated *A. officinalis* and three other asparagus species (Table 1) to identify possible sources of resistance to FOA and FM.

Ninety-five cultivars and breeding lines of the four asparagus species were evaluated in the greenhouse. Plants were grown from seed, or micropropagated by crown divisions or lateral meristem cultures. Germplasm sources included: a) seed companies: George J. Ball (Chicago, Ill), De Giorgi (Council Bluff, Iowa), Royal Sluis (Netherlands), Univ. of California-Davis Foundation Seed Service; b) breeding programs: the Dept. of Scientific and Industrial Research (New Zealand), Michigan State Univ., (East Lansing), Rutgers Univ. (New Brunswick, N.J.), plant in-

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roduction accessions (Geneva, N.Y.) from Afghanistan, Denmark, England, France, Holland, Spain, Turkey, Federal Republic of Germany; c) selected crowns surviving in deteriorated Michigan asparagus fields.

Plants grown from seed. Seeds were germinated on water agar [0.6% (w/v), Difco, Detroit, Mich.] or on moistened sterile Whatman no. 1 filter paper in 100 × 15 mm sterile petri plates for 7–10 days at 22 ± 2C under a 12-hr photoperiod (Molot and Simone, 1965). Individual 14-day-old seedlings were transferred into 72-cell flats in the greenhouse and transplanted singly at 12 weeks into 10-cm clay pots containing synthetic media (Baccto, Michigan Peat Co., Houston, Texas) (1 vermiculite : 1 peat : 1 perlite) and grown for an additional 12–16 weeks. Plants were fertilized twice (Peters 20N–20P–20K, 200 ppm) after transplanting.

Micropropagated plants. Crowns and lateral meristems were micropropagated using modifications of the protocol devised by Chin (1982). Medium was dispensed as 30 or 50 ml into either 125- or 250-ml Erlenmeyer flasks, respectively, and autoclaved for 20 min at 138 kPa. Plantlets were maintained on this medium until a moderate root system with a minimum of three primary roots and some secondary roots had formed. Subsequently, the plantlets were removed and the agar gently rinsed from the roots in running tap water. The plantlets were individually planted into 7.5-cm clay pots (0.68 kg) containing the synthetic media described above, amended with polypropylene pellets [3:1, (v/v)] (Norchem, Des Plaines, Ill.). After 7 to 10 days under an intermittent mist system, plantlets were transferred to a greenhouse, re-potted into 10-cm clay pots (0.9 kg), fertilized as previously described, and allowed to grow for a minimum of 8 weeks.

Inoculation. To prepare inoculum, 200 g of millet seed were combined with 100 ml of distilled water in 1-liter flasks and autoclaved for 1 hr on two consecutive days. Fungal cultures were prepared by inoculating potato dextrose agar [PDA, 3.9% (w/v), Difco] plates with virulent Michigan isolates (FOA-10 and FM-12) (from the collection of M. Lacy, Michigan State Univ.), which were maintained in soil (Nelson et al., 1983). After a 7- to 10-day incubation period at 24C, a 10-mm-diameter piece of either FOA, FM, or uncolonized PDA was placed aseptically into individual flasks of sterile millet. After 14 days incubation at 24C, the colonized millet was removed from the flasks, placed in separate sterile paper bags, and air-dried for 7 days. The bags were rotated daily to promote rapid drying. The prepared millet inoculum was then incorporated into a 2:1 (v/v) mixture of the synthetic medium described above and sand at the rate of 8 g millet to 1140 g soil-sand per treatment. Subsequently, asparagus plants were individually transplanted into 12.5-cm clay pots (1.14 kg) of this inoculated planting medium. Fresh inoculum was prepared for each of the 34 experiments.

Table 1. Comparisons of asparagus accessions in greenhouse evaluations for resistance to *Fusarium oxysporum* f. sp. *asparagi* and *F. moniliforme* using *Fusarium*-colonized millet inoculation and *Asparagus officinalis* 'UC157' as a susceptible control.

Entry ^y	Source ^x	Mean disease rating ^z		
		FOA ^w	FM ^y	UC 157 ^u
<i>More resistant than control</i>				
P.I. 207461	AF	2.3	2.2	4.5
<i>A. densiflorus</i> Myersii	BS	1.2	1.3	3.7
<i>A. densiflorus</i> Sprengeri	BS	1.2	1.8	3.2
<i>A. setaceus</i> Nanus	DG	2.3	2.2	3.2
P.I. 277828	EN	2.5	1.8	3.3
P.I. 277826	HO	4.5	3.2	4.5
J5	HO	3.2	3.8	4.7
Joite	HO	3.3	4.0	4.3
83 DKF1	MI	2.8	3.0	3.9
83 DKM3	MI	2.8	3.3	3.6
83 GLF1	MI	2.7	2.8	3.5
B-25	MSU	2.5	3.3	3.8
C-7	MSU	2.5	3.0	3.9
C-11	MSU	2.2	2.7	3.9
D-5	MSU	2.7	2.5	3.5
E-2	MSU	3.2	2.8	3.5
C-7-3	MSU	2.7	3.3	3.6
D-23-2	MSU	4.0	3.0	3.8
3247/83	NY	3.3	3.8	4.3
CRD 84006	NZ	2.8	3.0	3.8
CRD 84008	NZ	2.3	3.7	3.8
CRD 84010	NZ	2.7	2.0	3.8
CRD 84013	NZ	2.8	3.7	3.8
CRD 84075	NZ	2.8	2.8	3.8
CRD 84078	NZ	3.0	4.0	4.7
CRD 84079	NZ	2.7	3.3	4.7
CRD 84081	NZ	3.7	4.2	4.3
CRD 84085	NZ	2.3	2.8	4.7
Backlim	RS	2.8	2.8	3.8
277C × 22-8	RU	2.2	2.8	3.1
277E × 22-8	RU	2.7	2.7	3.1
291B × 22-8	RU	1.8	2.7	3.1
362M × 14-15	RU	3.2	3.0	3.7
382B × 309K-1	RU	3.5	3.0	3.8
Lucullus 55	WG	2.3	2.7	4.5
Lucullus 234	WG	1.5	1.5	3.3
Lucullus 328	WG	2.0	2.0	3.3
<i>As susceptible as control</i>				
<i>A. setaceus</i>	BS	3.2	2.7	3.2
<i>A. acutifolius</i>	DG	2.2	2.7	2.2
P.I. 262166	FR	2.2	2.7	1.9
DeJeerew	HO	3.5	4.1	3.5
Michigan Select	MI	3.2	2.7	2.6
Sodus 1968	MI	3.2	4.2	3.1
83 GLF2	MI	2.7	2.2	2.6
83 TGM1	MI	3.3	3.7	2.9
D-23	MSU	2.5	2.8	2.9
E-8	MSU	2.8	2.8	3.1
E-3-2	MSU	4.0	2.8	2.8
E-8-2	MSU	3.7	3.3	3.6
GRNHS	MSU	3.2	3.7	2.9
H78-10	MSU	2.5	3.0	2.1
CRD 84003	NZ	2.8	3.5	3.4
CRD 84004	NZ	3.0	4.5	3.4
CRD 84005	NZ	3.0	4.3	3.4
CRD 84009	NZ	3.7	4.2	3.8
CRD 84011	NZ	3.3	4.7	3.7
CRD 84082	NZ	2.8	4.0	3.8
Franklim	RS	4.2	3.7	3.8
D2 × 14-15	RU	2.3	2.7	2.4
53 × 14-15	RU	2.5	3.2	2.7
277C × 14-15	RU	3.5	2.7	2.7
277E × 14-15	RU	3.3	5.0	3.7
291B × 14-15	RU	2.8	4.3	3.7
61 × 24-15	RU	2.5	2.5	2.4
Greenwich	RU	4.3	3.5	3.6
Jersey Knight	RU	4.2	4.5	3.6
61 × 22-8	RU	3.3	3.7	3.6
382B × 22-8	RU	2.3	2.3	2.4
D2 × 22-8	RU	3.2	4.7	3.8
D2 × 309K-1	RU	2.7	4.2	3.1
G27 × 309K-1	RU	3.3	4.5	3.1

Table 1 continued

Entry ^y	Source ^x	Mean disease rating ^z		
		FOA ^w	FM ^v	UC 157 ^a
53 x 309K-1	RU	3.2	4.3	3.1
61 x 309K-1	RU	3.0	3.2	2.4
291B x 309K-1	RU	2.2	3.3	2.4
362M x 309K-1	RU	3.2	3.5	3.8
P.I. 174056	TU	3.2	3.5	2.8
Lucullus mr 3	WG	3.0	3.7	2.8
Lucullus wr 3	WG	3.5	2.8	2.8
Lucullus 327	WG	3.8	3.8	3.8
<i>More susceptible than control</i>				
P.I. 277822	DE	2.5	2.7	1.7
P.I. 277827	HO	2.5	2.8	1.9
83 DKM2	MI	4.0	3.8	2.9
83 KBM1	MI	3.7	3.5	2.9
C-3	MSU	3.7	3.3	2.8
E-3	MSU	3.2	4.0	2.4
MKLS3	MSU	2.7	3.5	2.1
2422/83	NY	4.0	4.5	3.7
Boonlim	RS	3.0	3.0	2.6
Jersey Giant	RU	2.6	3.0	2.1
277E x 309K-1	RU	3.3	4.0	2.4
G27 x 14-15	RU	3.3	3.3	2.7
P.I. 262900	SP	2.5	2.7	1.9
P.I. 169010	TU	3.2	2.3	1.7
P.I. 169013	TU	2.7	2.3	1.7
SM 20A7	WG	4.0	3.8	2.2

^aDisease ratings based on scale of 1–5, where: 1 = healthy plant, no evidence of disease (highly resistant); 2 = few root lesions and/or rotted roots with no reduction in shoots or root numbers and no vascular discoloration in the roots, crowns, or stems (resistant); 3 = moderate number of root lesions and/or rotted roots with slight reduction in the number of shoots and roots and slight vascular discoloration (moderately susceptible); 4 = many root lesions present with much reduction in the number of shoots and roots, pronounced vascular discoloration, and roots slightly to moderately flaccid (susceptible); 5 = all roots flaccid or dead (highly susceptible).

^yUnless otherwise indicated, all listed accessions are *A. officinalis*.

^xAF = Afghanistan; BS = George J. Ball Seed Co.; DG = De Giorgi Seed Co.; DE = Denmark; EN = England; FR = France; HO = Holland; MI = Michigan deteriorated asparagus fields; MSU = Michigan State Univ.; NY = Plant Introduction Station, Geneva, N.Y.; NZ = Dept. of Scientific and Industrial Research, New Zealand; RS = Royal Sluis Seed Co.; RU = Rutgers Univ.; SP = Spain; TU = Turkey; WG = Federal Republic of Germany.

^wMean disease rating from six replications of entry inoculated with *Fusarium oxysporum* f. sp. *asparagi*.

^vMean disease rating from six replications of entry inoculated with *F. moniliforme*.

^zCombined mean disease rating for the 12 replications (six each) of the susceptible control 'UC 157' inoculated with *F. oxysporum* or *F. moniliforme*.

Evaluation. The plants were removed from the pots and the soil was gently removed from the roots after 8 weeks. Individual plants were washed in tap water and visually rated for the severity of FOA or FM infection on a scale of 1–5, where: 1 = healthy plant, no evidence of disease (highly resistant); 2 = few root lesions and/or rotted roots with no reduction in shoot or root numbers and no vascular discoloration in the roots, crowns, or stems (resistant); 3 = moderate number of root lesions and/or rotted roots with slight reduction in the number of shoots and roots and slight vascular discoloration (moderately susceptible); 4 = many root lesions present with much reduction in the number of shoots and roots, pronounced vascular discoloration, and roots slightly to moderately flaccid (susceptible); 5 = all roots flaccid or dead (highly susceptible).

The collection of asparagus germplasm was screened against FOA and FM in a series of 34 greenhouse experiments during 18 months. The number of asparagus entries varied (minimum of four) among experiments; however, an internal control ('UC 157') was included in each. Three treatments were applied to each entry. Each treatment consisted

of a six-plant replicate that was inoculated with a millet inoculum of either FOA, FM, or sterile uninoculated millet. Each experiment was designed as a factorial experiment using the two *Fusarium* spp. as one factor and the germplasm entries as the other. All experiments were arranged on greenhouse benches in a randomized complete block design under a natural photoperiod at 25–30°C. Data were subjected to analysis of variance.

The F ratio for interaction of *Fusarium* inoculum × germplasm entry was not significant in any experiment; thus, the entries responded similarly to both *Fusarium* spp. Therefore, the main effect for entry (a mean of two *Fusarium* spp. × six replications) was used to compare all entries to the susceptible internal control 'UC 157' using an LSD mean separation. The entries were separated into three classes: those more resistant, as susceptible as, or more susceptible than 'UC 157'.

The results indicated that two *A. officinalis* genotypes, the all-male Lucullus cultivars 234 and 328, received the lowest disease ratings to FOA and FM. Thirty-two additional *A. officinalis* accessions were rated more

resistant than 'UC 157', 40 accessions were rated as susceptible as 'UC 157', and 16 were rated more susceptible than 'UC 157'.

A. densiflorus (Kunth) Jessop 'Sprengeri' and 'Myersii' were found more resistant than any other asparagus cultivar or species tested. The immunity of 'Sprengeri' to FOA was reported earlier by Lewis and Shoemaker (1964). *A. setaceus* (Kunth) Jessop 'Nanus' was another asparagus species that was rated more resistant than the susceptible cultivar UC 157. *A. acutifolius* L. and *A. setaceus* (Kunth) Jessop were both rated as susceptible as 'UC 157'.

Field research is needed on those lines identified as potential sources of resistance to *Fusarium* spp. Sexual incompatibility between *A. officinalis* and 'Sprengeri' (and possibly 'Myersii') must be overcome in order to transfer fusarium resistance. One possibility is somatic fusion, which has been used to introduce disease resistance and other economically valuable traits into some crop species (Larkin and Scrowcroft, 1981).

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Nature and Inheritance of Compact Plant Habit in *Cuphea leptopoda* Hemsley

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Abstract. Plants with a compact growth habit have been identified in populations of *Cuphea leptopoda* Hemsley. The primary morphological effect has been shown to be conditioned by a single recessive gene, designated *si* for shortened internodes. Short internodes on compact plants result in reduced length of the main stem, reduced number and length of both primary and secondary branches, and reduced flowering resulting from a decrease in number of flowers per node on the main stem and branches. The compact growth habit and more concentrated flowering of the shortened-internode plants may have horticultural and agronomic value in new interspecific hybrids being developed.

Several cuphea species are used on a limited scale in landscape plantings as tender annuals and perennials in the southern part of the United States (Thompson, 1984). Some of our recently synthesized interspecific hybrids are being evaluated as new bedding and pot plants because of their attractive flowers and plant growth characteristics (Ray et al., 1988; Thompson, 1986; Thompson et al., 1987). In addition, cuphea species are being domesticated and developed to serve as a new industrial oilseed crop for the production of lauric and other medium-chain fatty acids (Hirsinger and Knowles, 1984; Thompson, 1984, 1985; Thompson and Kleiman, 1988). Major constraints to successful commercialization of cuphea for either horticultural or agronomic purposes are indeterminate growth and flowering, excessive seed shattering, seed dormancy, and viscid glandular hairs on flowers, stems, and leaves. Various breeding, genetic, and cultural methods are being employed to remove these constraints. We are using interspecific hybridization to develop enhanced germplasm and new cultivars (Ray et al., 1988; Thompson, 1984).

During the ongoing germplasm evaluation, plants with a compact growth habit were observed within populations of three accessions of *Cuphea leptopoda* Hemsley [A0029

(Graham 602), A0065 (Graham 713), and A0072 (Graham 720)] (Fig. 1). These accessions have chromosome numbers of $N = 10$ and produce predominately capric acid

(C10:0) in the seed oil (Hirsinger and Knowles, 1984; Ray et al., 1988; Thompson and Kleiman, 1988). *C. leptopoda* plants usually exhibit a high level of seed dormancy, are normally highly cross-pollinated by insects, and set few seeds in the greenhouse. The objective of this study was to characterize the nature of the growth and flowering habit of the compact plants in comparison with normal plants, and to possibly determine the mode of inheritance.

Seeds from accession number A0072 were germinated on moistened filter paper in petri dishes on 13 Mar. 1985, transplanted into seed flats in a greenhouse on 18 Mar., and moved into pots with volume of 400 cm³ on 5 Apr. 1985. On 17 May 1985, 65 days after planting, 17 of the 38 plants obtained were classified as having the compact growth habit. We measured node number, internode length, total height of the main stem, and recorded the number and length of primary and secondary branches and the node from which they arose. Flower numbers on main stem nodes and on primary and secondary branches were counted and recorded. Means, standard errors, and coefficients of variation were calculated for each measurement within each

Table 1. Comparison of internode length and total plant height of normal and compact (shortened internode) *Cuphea leptopoda* plants 65 days after planting.

Internode number	Internode length (mm)		
	Normal (n = 21)	Compact (n = 17)	Difference (N - C)
0 ^z	20.5	13.5	7.0
1	8.6	7.1	1.5
2	15.0	9.1	5.9
3	23.8	14.4	9.4
4	37.6	23.5	14.1
5	44.3	29.7	14.6
6	45.5	28.8	16.7
7	37.6	26.5	11.1
8	27.9	19.4	8.5
9	21.7	13.8	7.9
10	17.6	9.1	8.5
11	14.0	8.2	5.8
12	11.2	6.8	4.4
13	7.9	4.1	3.8
14	5.0	2.4	2.6
15	2.1	0.9	1.2
16	1.2	0.0	1.2
Internode length (\bar{x})	21.3	13.6	7.7
SE	0.7	0.3	0.9
CV (%)	15.2	9.1	---
t (df = 36)	---	---	7.98**
Total plant height (\bar{x})	341.5	217.3	124.2
SE	11.0	8.7	14.6
CV (%)	14.8	16.5	---
t (df = 36)	---	---	8.53**

^zInternode number 0 indicates the stem from the soil line to the cotyledonary node.

**Differences between means of two unequal sized groups (21 normal vs. 7 compact plants) are significantly different from zero at the 1% level.

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