

Vine-decline of melons caused by *Monosporascus cannonballus* in Arizona: Epidemiology and Cultivar Susceptibility

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INTRODUCTION AND HISTORICAL PERSPECTIVE

A destructive disorder of muskmelons, characterized by the sudden (commonly within two weeks of harvest) and generally uniform collapse of entire fields has plagued the industry for over 40 years. Common names of the disorder include collapse, vine decline, quick decline, and sudden wilt. The disorder is particularly severe in the warmer climatic production regions of the United States (Arizona, California, and Texas). Grower concerns regarding this disorder increased dramatically since 1985. Multiple as well as consecutive cropping of fields to melons have been associated with the increase in the prevalence and severity of the disorder.

In Arizona, foliar symptoms associated with this disorder are a general yellowing/browning of the oldest crown leaves which progress outward toward the tips of individual vines. Occasionally, wilting of individual vines occurs. Structural roots of affected plants, particularly those exhibiting early foliar symptoms, appear healthy and no root rot or internal discoloration of the vascular system is usually evident at this time.

In the United States, the cause of the disorder was not identified until recently. In 1990 researchers in Texas (May, Mertely et al, *Plant Disease* 75:1133-1137) and Arizona (September, Stanghellini and Rasmussen, unpublished), independently, discovered and attributed the cause of the disorder to a root-infecting fungus known as *Monosporascus cannonballus*. This fungus was first discovered in Arizona by Troutman and Matejka (*Phytopathology* 60:1317) on decayed roots of cantaloupe in 1970 and the fungus was named in 1974 (Pollack and Uecker, *Mycologia* 66:346-349). A review of the literature, subsequent to identification of the pathogen, indicated that the fungus had been associated with a collapse of melons in Japan (*Trans. Mycol. Soc. Japan* 20:312-316) in 1979 and Spain (*Bol. San. Veg. Plagas* 17:133-163) in 1991. A similar disease of muskmelon and watermelon, attributed to a different species of the fungus (*M. eutypoides*) was reported in Israel (*Phytopathology* 73:1223-1226) in 1983.

In 1992 we initiated extensive field studies on the epidemiology of this destructive and currently uncontrolled disease. The objectives of this paper are to summarize a portion of our research which was conducted over the past 3 years (1992-1994). Specifically, data regarding the epidemiology and the susceptibility/tolerance of melon cultivars to the fungus are presented.

MATERIAL AND METHODS

Unless otherwise specified, all studies were conducted in commercial melon fields (located in Harquahala and Aguila) which have a known history of disease caused by *M. cannonballus*. Soil populations of the fungus (i.e., ascospores) were enumerated via an extraction method developed by the authors (*Phytopathology* 82:1115). The onset of root infection, development of symptoms (both root and foliar), and the rate of disease progression was monitored weekly over several cropping seasons. Soil temperatures at the 10 cm depth were recorded. Two trials (1993 and 1994) were conducted to identify melon genotypes with promising levels of field tolerance. In 1993, twenty-four melon cultivars were seeded, on August 8, on drip-irrigated beds 40 inches between bed centers.

Cultivars were replicated four times in a randomized block design. Each replicate consisted of 18 feet of bed. In 1994, twenty-eight melon cultivars and breeding lines were seeded, on June 13, on furrow irrigated beds 80 inches between bed centers. Cultivars were replicated four times in a randomized block design and each replicate consisted of 40 feet of bed. The severity of vine decline caused by *M. cannonballus* in each replicate was assessed visually using the following rating system: 0 = all plants dead; 1 = severe vine decline and most fruits exposed; 2 = moderate vine decline and some fruits exposed; 3 = slight vine decline and few fruits exposed, and 4 = no vine decline and no fruits exposed. In 1993 disease severity ratings were made on November 10 and in 1994 they were made on September 9. All fertilization, irrigation, pest control, and cultural practices were performed by the commercial grower.

RESULTS AND DISCUSSION

Distribution of the pathogen in soil. Results showed that the fungus was uniformly distributed, both horizontally and vertically, in commercial fields which had a known history of the disease (Table 1A & Fig. 1). Additionally, the fungus was shown to be uniformly distributed, both vertically and horizontally, in the native desert habitat adjacent to cultivated fields (Table 1B). These results provide an explanation for the uniform distribution of the disease in commercial melon fields: that is – the fungus is uniformly distributed in soil and it is indigenous in Arizona.

Root infection, symptom development, and disease progression. Results are summarized in Table 2. The onset of root infection and the rate of disease progression, which varied between the Spring, Summer and Fall cropping seasons, was related to soil temperatures. Early root infection and rapid symptom development were associated with soil temperatures greater than 25 C at the 10 cm soil depth (Fig. 2).

Relative to the Fall planted crop (which had the most rapid development of symptoms), root infection, in the absence of any visible symptoms, occurs ca. 24 days after planting. Root lesions were first observed ca. 35-40 days after planting and root symptoms, observable only after washing of the roots, consisted of small, relatively inconspicuous crusty, tan-colored non-girdling root lesions, measuring 2-4 mm in length, on small (2-4 mm in diameter) feeder roots. Obvious root rot and the presence of perithecia of the fungus on some necrotic feeder roots were first observed ca. 60 days after planting and coincided with the first observation of vine decline. Depending upon soil temperature, rapid vine decline and root lesion expansion, which can include structural roots, occurs within the next 10-20 days.

The soil depth at which lesions are found is related to the type of irrigation (Fig.3). In furrow irrigated fields, ca. 80% of the lesions (which correspond to the initial sites of root infection by the fungus) occur on roots excavated from the 11 to 25 cm soil depth. This is in contrast to the location of lesions on roots in drip or drip-mulched irrigated fields. Most of the lesions in the latter fields occur on feeder roots excavated from the 0 to 10 cm soil depth. These results indicate that, in addition to soil temperature, high soil moisture levels are an important environmental factor influencing root colonization by this fungus. Lack of moisture in the upper layers of soil in furrow irrigated fields apparently restricts colonization by the fungus. Providing sufficient soil moisture is present, however, the fungus can attack roots at all soil depths. The distribution of lesions throughout the soil profile supports data previously presented on the uniform vertical distribution of the fungus in the soil.

Foliar symptoms, which occur ca. 50 days after planting, consisted of a yellowing and necrosis of the oldest crown leaves which progressed outward to the tip of vines. Over the past 3 years we have also observed a foliar symptom which is consistently associated with the disorder: a V-shaped necrotic sector which extends from the base to the outer margin of the leaf blade. A similar, if not identical, foliar symptom was observed and recorded in descriptions of a disease of melons referred to as "crown blight" in the mid to late 1960's.

Cultivar susceptibility/tolerance to *Monosporascus cannonballus*. Results of the two field trials, presented in Table 4 and 5, indicate that tolerance to the fungus exists in certain melon cultivars. Further studies on field performance of promising cultivars and breeding lines are warranted.

ACKNOWLEDGEMENTS

Appreciation is expressed to all the seed companies for supplying seed for the field trials, to personnel of Martori Farms for providing the sites for field testing, and financial support from concerned growers.

Table 1. Vertical distribution of ascospores of *Monosporascus cannonballus* in (A) commercial melon fields and (B) native desert soils.

(A)

ASCOSPORES/GRAM SOIL

SOIL DEPTH (cm)	FIELD A (DRIP)	FIELD B (DRIP)	FIELD C (MULCH + DRIP)	FIELD D (FURROW)
0-5	1.75	1.40	1.60	2.40
6-10	1.25	1.15	1.70	1.90
11-15	1.30	1.35	1.95	2.00
16-20	1.60	1.65	2.05	2.00
21-25	<u>1.65</u>	<u>1.90</u>	<u>2.35</u>	<u>1.55</u>
MEAN/SD	1.51+ /-0.22	1.49+ /-0.29	1.93+ /-0.30	1.97+ /-0.30

(B)

ASCOSPORES/GRAM SOIL

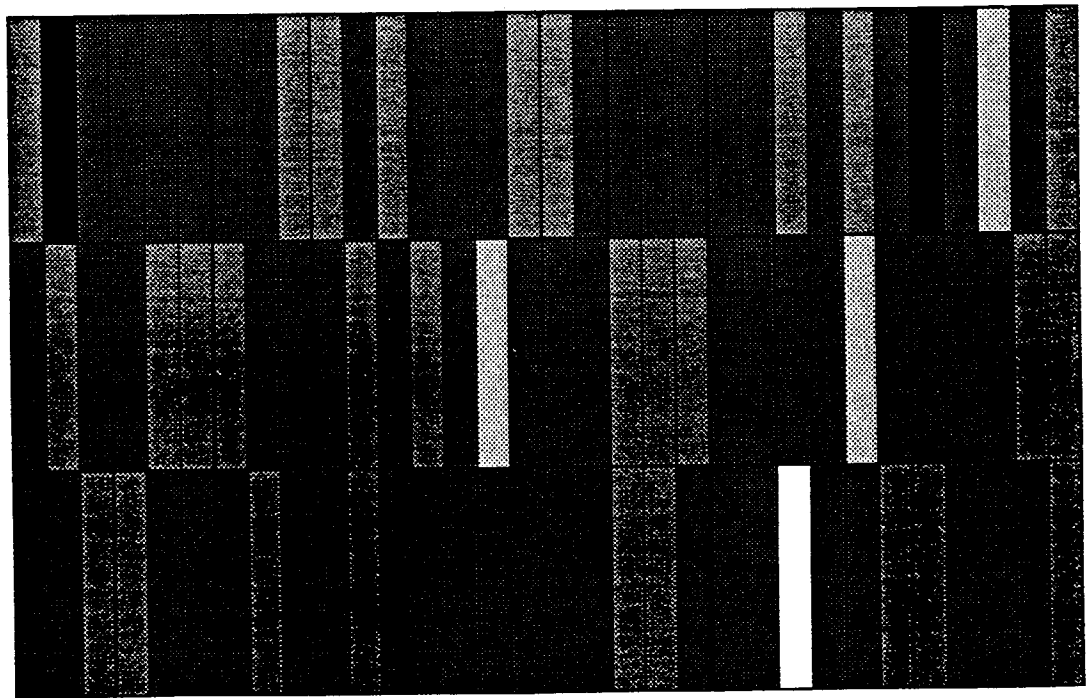
SOIL DEPTH (cm)	DESERT SITE A	DESERT SITE B
0-5	1.75	1.55
6-10	0.80	1.30
11-15	1.45	0.85
16-20	1.15	1.00
21-25	<u>1.70</u>	<u>1.40</u>
MEAN/SD	1.37+ /-0.40	1.22+ /-0.29

Table 2. Disease progression in commercial melon fields.

DAYS FROM PLANTING TO:

VARIETY	SEASON	PLANTING DATE	FIRST OBSERVATION OF:			
			Root Infection (No Symptoms)	Root Lesions	Foliar Symptoms	Perithecia on Roots (Vine Decline)
Caravelle	Spring	Jan 94	65	107	145	160
Caravelle	Spring	Feb 94	47	89	115	122
Caravelle	Summer	June 94	30	49	64	81
Caravelle	Fall	Aug 93	25	40	54	59
Gold Mark (Martori)	Fall	Aug 94	24	35	47	61

Figure 1. Horizontal distribution of ascospores of *Monosporascus cannonballus* in a commercial field.



Field Mean (96 samples) = 1.85

- = 1/g
- = 1.1 - 2/g
- = 2.1 - 3/g
- = 3.1 - 4/g
- = 4.1 - 5/g

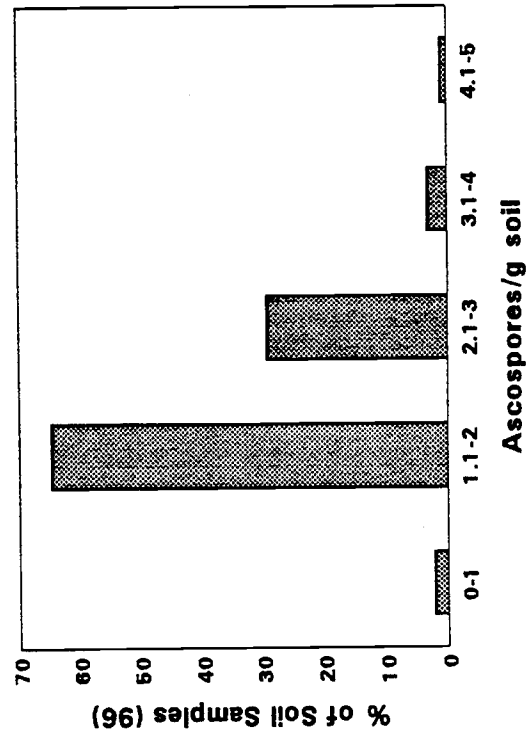


Figure 2. Soil temperature at the 10 cm depth in a commercial melon field.

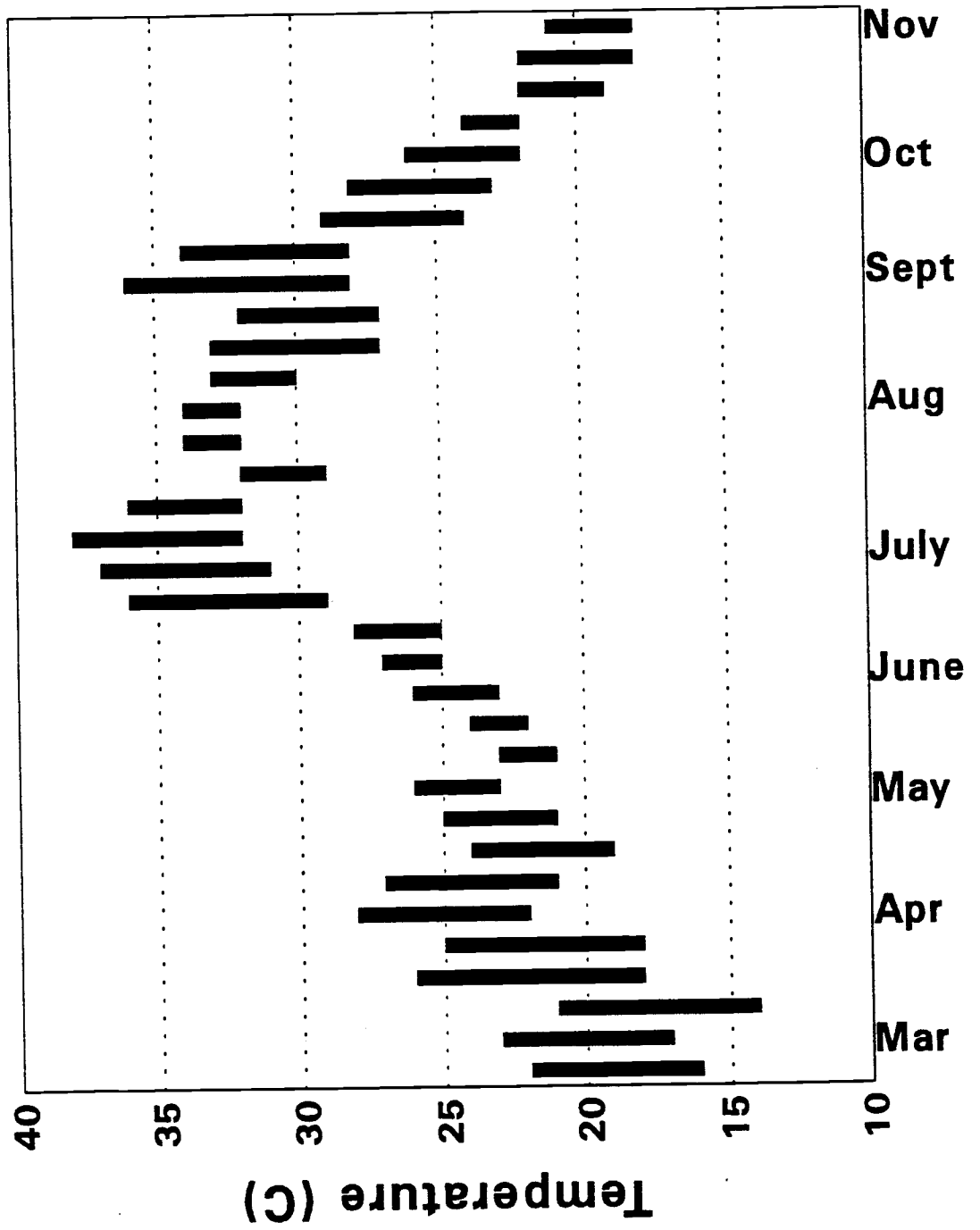


Figure 3. Influence of irrigation/mulching on vertical distribution of *Monosporascus cannonballus* lesions on melon roots in commercial melon fields.

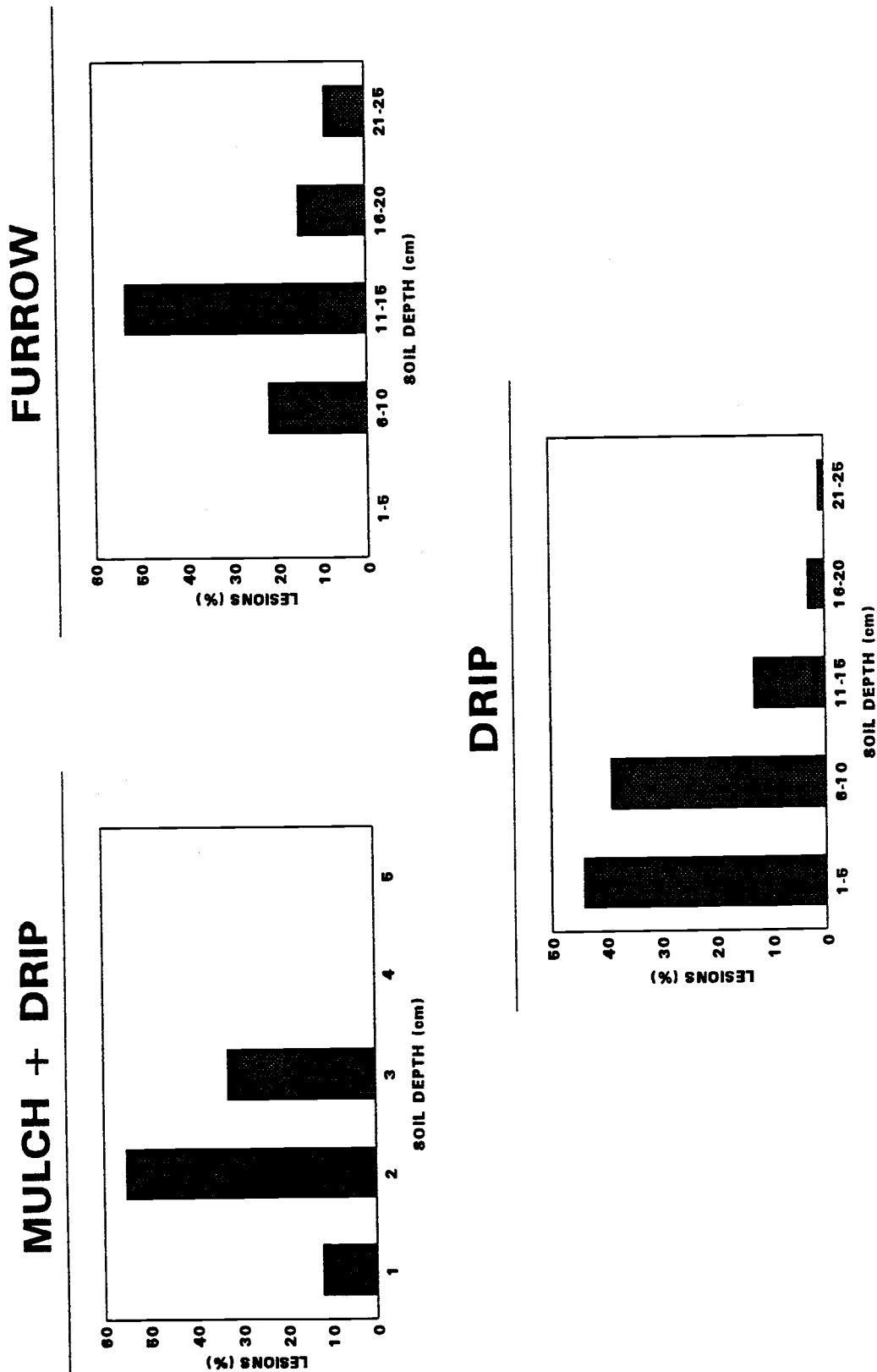
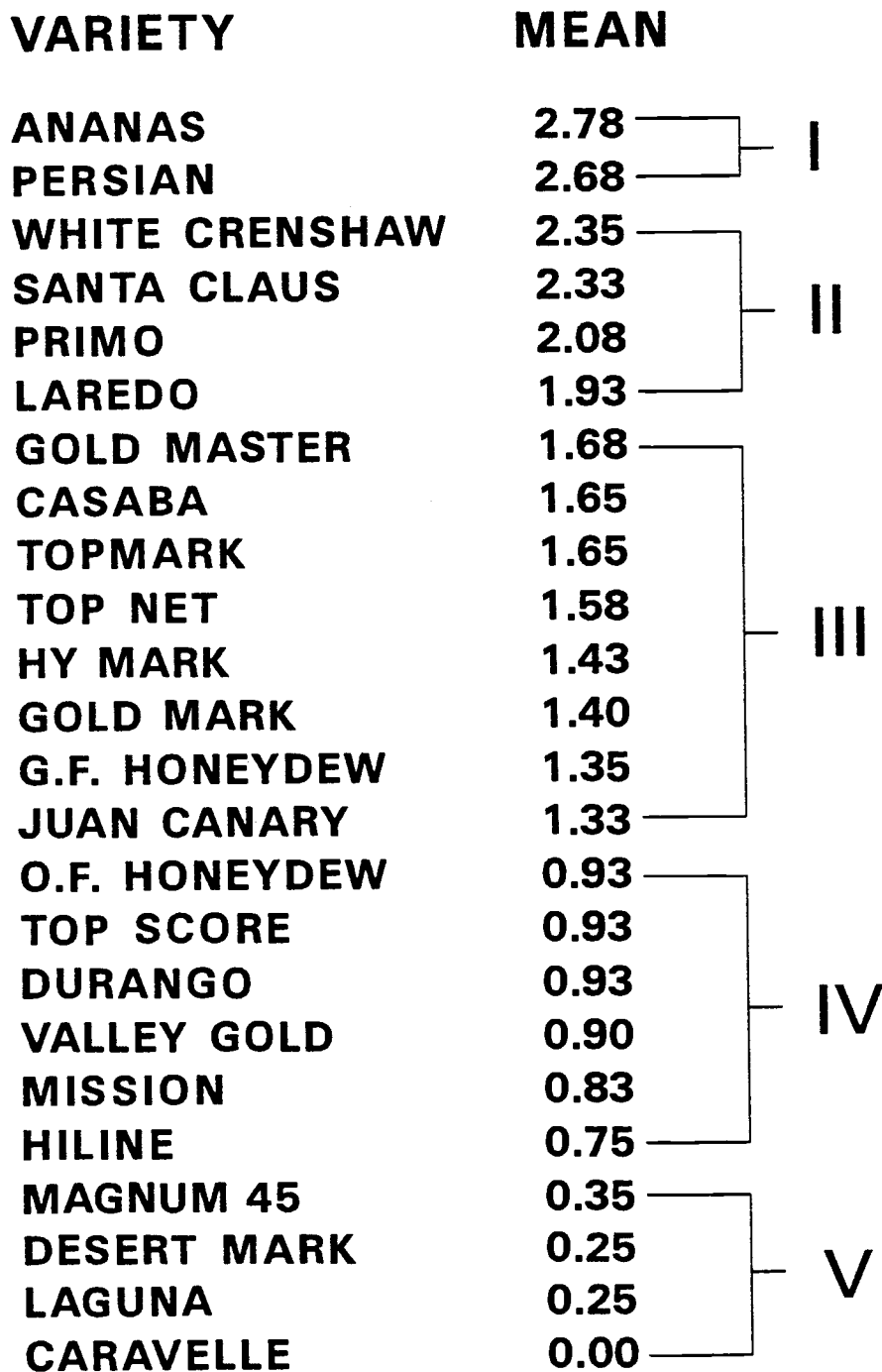


Figure 4. Disease rating for melons grown in a commercial field naturally infested with *Monosporascus cannonballus*, 1993.



Pooled Standard Deviation = 0.485

Figure 5. Disease rating for melons grown in a commercial field naturally infested with *Monosporascus cannonballus*, 1994

Cultivar	Mean Rating	Group
XPH 6244	3.50	I
Solid Gold	2.61	
WM 2426	2.54	II
NVH 898	2.38	
WM 2402	2.25	
KXPM 111	2.21	
Gold Rush	2.11	
Primo	1.95	
XPH 6242	1.94	
XPH 6245	1.93	
KXPM 137	1.73	
Mission	1.73	
Caravelle	1.71	III
Gold Mark	1.70	
Desert Mark	1.53	
Valley Gold	1.44	
Laredo	1.40	
Veracruz	1.25	
WM 21028	1.24	
Challenger	1.05	
Durango	1.04	IV
XPH 6240	1.01	
HMX 9584	1.01	
Top Mark	0.99	
XPH 6112	0.91	
Laguna	0.87	
PMR 45	0.79	
Cruiser	0.71	

Pooled Standard Deviation = 0.499