Achene Blemish Syndrome: A New Disease of Sunflower in Israel

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ABSTRACT

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A new disease has affected sunflower (*Helianthus annuus*) achenes in Israel. Infection produces small, scattered lesions on the surface of the shell. Lesions are brown, black, or gray and some are surrounded by dark halos; they range in size from 0.5 to 2 mm and may be round, oval, elongate, or irregular. Infected crops are viewed by the industry as being of lower quality and therefore fetch a lower market price. Several fungi were isolated from infected achenes. The four steps of Koch's postulates were completed for *Alternaria alternata, Cladosporium* sp., and *Ulocladium atrum*. Artificial inoculation trials revealed that *A. alternata* induced a significantly higher disease incidence than the other two fungi and that inoculation with a mixture containing spores of all three fungi resulted in a higher disease incidence than that obtained by inoculation with each pathogen alone. Achenes were susceptible to infection only at the time of their development, but symptoms were only visible just before physiological maturity. A field trial demonstrated that western flower thrips (*Frankliniella occidentalis*) are associated with infection of the achenes. The effects of nine fungicides on disease severity were examined in two field trials in 1992. Although some fungicides significantly reduced disease severity, their effect was relatively minor and in general they were not highly effective.

The production area of sunflower (*Helianthus annuus* L.) in Israel increased from approximately 5,000 ha in the 1980s to approximately 20,000 ha in the early 1990s. Sunflowers are grown in Israel exclusively for human consumption, and achenes are mostly sold unpeeled. Achenes of the local confectionery cultivars differ in size, oil content, and taste from those grown for oil production or for animal feed. For example, a typical grade A achene of the local cultivar DY-3 is 2–2.5 cm long and weighs 150–200 mg. The size, weight, and appearance of the achenes determine the crop value, which in 1991 was $1,640 (U.S.) per ton (20).

During the 1991 harvest and at the time of yield processing, small scattered lesions were noticed on the surfaces of achene shells from some fields. The lesions varied (even on individual achenes) in color, size, and shape. They were brown, black, or gray and some were surrounded by a dark halo; they ranged in size from 0.5 to 2 mm and were round, oval, elongate, or irregular. The number of lesions varied substantially among achenes, and up to 30 lesions were observed on severely infected ones. Infected achenes did not differ in size or weight from uninfected ones, nor was seed taste altered. Nevertheless, infected crops were viewed by the industry as being of lower quality and received a market price of only $1,200 (U.S.) per ton.

The achene disease, referred to hereafter as achene blemish syndrome, was observed for the first time during the 1991 growing season. Sunflower fields in the central and southern growing regions of the country, but not those in the north, were affected. During the 1992 and 1993 seasons, achene blemish syndrome was observed in fields throughout the country. Disease severity varied substantially, even among fields in the same region.

The reduction in crop value imposed by achene blemish syndrome and the subsequent losses to growers promoted efforts to develop controls for the disease. Because the syndrome has not been described in other sunflower production areas in the world, we investigated the basic aspects of the disease. The objectives of this study were to: 1) identify the causal agent(s) of achene blemish syndrome, 2) examine factors affecting the infection process, and 3) investigate methods of disease control.

MATERIALS AND METHODS

Identification of the causal agents. Our initial hypothesis was that plant pathogens were associated with achene blemish syndrome. Accordingly, the four steps of Koch's postulates were followed. Approximately 100 mature achenes bearing typical lesions were treated with sodium hypochlorite (2% available chlorine) for 2 min and then washed twice in sterile distilled water. The pericarp was separated from the rest of the seed, and whole pericarps, pericarp sectors bearing typical symptoms, endosperm, or whole achenes were placed on potato-dextrose agar (PDA) amended with chloramphenicol (250 mg/L). Petri dishes were then placed in an incubator at 18–20 C and 12-hr photoperiod. Fungi growing on the PDA were isolated and subcultured on PDA several times until pure cultures were obtained. For visual observation of pathogens, achenes were shaken in distilled water containing 0.01% Tween 20 and the solutions were examined microscopically (×400). Achenes were also kept in moist conditions for 24 or 48 hr and then examined with the aid of stereoscopic binoculars (×200).

Fungi isolated by the above process were inoculated into sunflower. We first examined the effect of inoculating detached, mature, noninfected achenes. Achenes were placed in 20 × 30 cm plastic trays (50 achenes per tray) and sprayed to runoff with a spore suspension (10^5 spores per milliliter) of each isolated fungus. Achenes sprayed with distilled water served as controls. After inoculation, the plastic trays were placed in an incubator at 18–20 C and 12-hr photoperiod under moist conditions, and the achenes were inspected periodically over 21 days. These inoculations did not result in disease development. We therefore proceeded to inoculate achenes on plants at various stages of development. Seeds of the sunflower cultivar DY-3 were planted in 2-L pots (two seeds per pot) filled with sandy loam soil. Plants were grown in an unheated greenhouse and were irrigated and fertilized as necessary. After flowering, heads were pollinated daily with a small paint brush, since self-incompatibility is common in *H. annuus*. When achenes were at the stage of initiation of color change, heads were sprayed to runoff with a spore suspension (10^5 spores per milliliter) of each test fungus and then covered with plastic bags for 48 hr to maintain humid conditions. Heads sprayed with distilled water and covered with plastic bags served as controls. There were four pots (replicates) for each tested fungus. Following inoculation, achenes were inspected periodically for symptoms up to maturity and then were collected for further examination.

Symptoms of artificially inoculated achenes were compared visually with those of naturally infected ones. Fungi were then reisolated from the artificially inoculated achenes by the procedure described above and compared microscopically with the original (×400).

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Factors affecting the infection process. Following identification of potential causal agents of the disease, factors affecting the infection process were studied. The growth stages at which infection occurred and possible interactions among the three fungi associated with the syndrome were examined in greenhouse trials. Plants were grown in pots as described above, and a factorial experiment in a randomized complete block design was conducted. The two main factors were growth stage at the time of inoculation (five levels) and fungi (four levels). Achenes were inoculated at the following growth stages: 1) 3–5 days after flowering, 2) white achenes, 3) initiation of achene color change, 4) advanced stages of achene color change, and 5) after physiological maturity. Treatments for the second main factor were spore suspensions of each of three test fungi, sprayed at a concentration of 3×10^4 spores per milliliter, and a spore suspension containing a mixture of the three test fungi, sprayed at a concentration of 10^4 spores per milliliter each (i.e., a total of 3×10^4 spores per milliliter). Sunflower heads sprayed to runoff with distilled water served as controls. Each treatment was replicated three times (two plants per replicate). Following inoculation, heads were covered with plastic bags for 48 hr. Achenes were collected 2 wk after reaching maturity, and the number exhibiting the syndrome was recorded. Disease incidence was calculated as the percentage of diseased achenes out of the total number of achenes per head. The trial was repeated once, but because overall trends were similar, results of only one trial are presented.

Achenes sampled from commercial fields at different growth stages were used to determine whether the fungi causing the disease had penetrated to the inner pericarp layer or had contaminated only its outer surface. Achenes were surface-sterilized with an aqueous solution of sodium hypochlorite (2% available chlorine) for 2 min and then washed twice in sterile distilled water and placed on PDA in petri dishes. For comparison, achenes were planted on PDA without surface sterilization. Samples (20–50 achenes each) were taken at the following growth stages: white achenes, initiation of achene color change, advanced stages of achene color change, and after physiological maturity.

In the field, disk florets are attached to the developing achenes up to maturity. The role of these florets in the infection process was examined on plants grown in the greenhouse. Heads with florets attached or removed by hand were inoculated at the stage of initiation of achene color change. A spore suspension containing a mixture of three test fungi, at a concentration of 10^4 spores per milliliter each (i.e., a total of 3×10^4 spores per milliliter), was sprayed to runoff by means of a hand sprayer. Following inoculation, heads were covered with plastic bags for 48 hr. There were four pots (replicates), two plants per pot, for each treatment. Achenes were collected after reaching maturity, and the number exhibiting the syndrome was recorded.

The possibility of interaction between the pathogens associated with achene blemish syndrome and western flower thrips (*Frankliniella occidentalis* (Pergande)) was examined in the field in 1993. Plants of cv. DY-3 were tagged with colored strips on 18 May, at the flowering stage. Individual heads were sprayed to runoff with the fungicide tebuconazole (Folicur, 0.05%) and the insecticide methiocarb (Mesurol, 0.1%) by means of a hand sprayer. Treatments were as follows: untreated control; tebuconazole or methiocarb applied four times on 25 May at growth stage 2 (white achenes), 31 May at growth stage 3 (initiation of achene color change), 7 June at growth stage 4 (advanced stages of achene color change), and 14 June at growth stage 5 (after physiological maturity); tebuconazole and methiocarb applied concurrently four times on the above dates; tebuconazole applied once on each of the above dates; and methiocarb applied once on each of the above dates. The experiment was laid out in a completely randomized design, and each treatment was replicated six times. After reaching maturity, individual heads were collected and threshed manually. A sample of 100 achenes was arbitrarily selected from the harvest of each head. In each achene, disease severity was determined visually, on both sides separately, and ranked according to a scale of 0–3 in which 0 = uninfected and 3 = severely infected (Fig. 1). A disease index (*DI*) representing the severity of achene blemish syndrome was calculated according to the proportion of achenes (P_i) in each of the four categories (S_i) as:

$$DI = \sum_{i=0}^{3} (P_i * S_i).$$

Thus, severe infection is denoted by a high *DI* value (with a maximum of 3) and mild infection by a low *DI* value (with a minimum of 0).

Suppression of achene blemish syndrome. The effects of various fungicides on the severity of achene blemish syndrome were examined in 1992 in two field trials. The trials were conducted primarily to evaluate fungicide efficacy on *Puccinia helianthi* Schwein., the causal agent of sunflower rust, and *Rhizopus* sp., the causal agent of sunflower head rot. Samples taken from harvests of plots treated with the different fungicides were used to evaluate the influence of the fungicides on the severity of achene blemish syndrome.

The trials were conducted in the northern Negev (trial 1) and Lakhish (trial 2) regions of Israel. The cv. DY-3 was sown in the last week of March 1992; plants were spaced 0.5 m apart within rows and 1 m between rows. The crop was irrigated via a drip irrigation system and was grown according to the cultural practices recommended for sunflowers in these regions, but fungicides and insecticides were not applied. The experiments were laid out in a randomized block design with four replicates. The size of each experimental plot was 6×12 m. Each trial consisted of the following 10 fungicidal treatments:

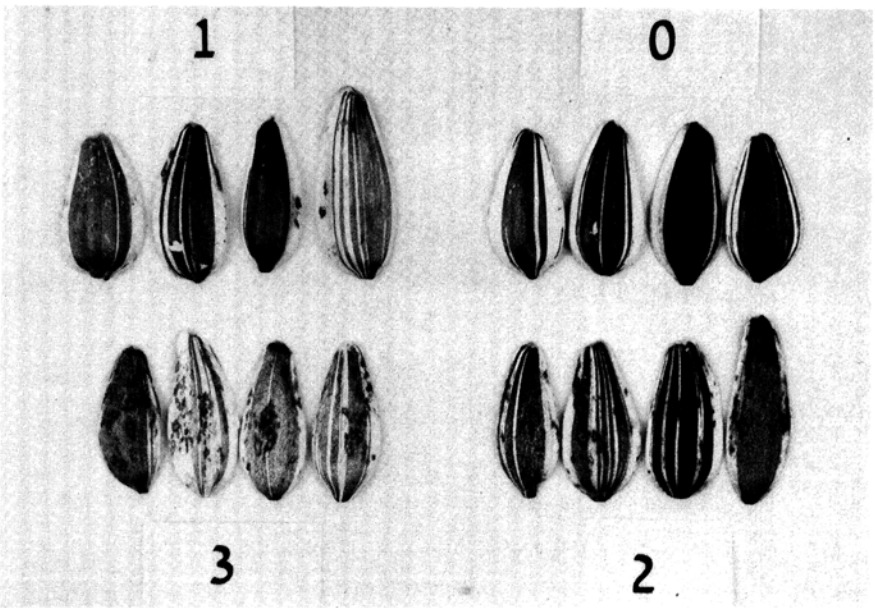

Fig. 1. A 0–3 rating scale for evaluation of the severity of achene blemish syndrome of sunflower in which 0 = uninfected achene amd 3 = severely infected achene.

1) untreated control; 2) hexaconazole (Anvil, 0.05 kg a.i./ha); 3) maneb (Manebgan, 2.0 kg a.i./ha, only in trial 1); 4) iprodione (Rovral, 0.5 kg a.i./ha); 5) fenbuconazole (Indar, 0.05 kg a.i./ha); 6) promoconazole (Granit, 0.1 kg a.i./ha); 7) cyproconazole (Atemi, 0.05 kg a.i./ha); 8) tebuconazole (Folicur, 0.125 kg a.i./ha); 9) difenoconazole (Score, 0.187 kg a.i./ha; in trial 2, also at 0.125 kg a.i./ha); and 10) oxine-copper (Quinolate 400, 0.8 kg a.i./ha). Two sprays were applied in trial 1, on 31 May at 100% flowering and on 11 June at the white achenes stage. Three sprays were applied in trial 2, on 25 May at 25% flowering, on 1 June at 100% flowering, and on 10 June at the white achenes stage. Sprays were applied via a motorized back sprayer in 240 L/ha of water with cone-jet ×6 nuzzles at a pressure of 275 kPa.

During the first week of September, seeds were harvested by means of a commercial combine and samples (about 0.5 kg each) were taken from the harvest of each experimental plot. Two subsamples of 100 achenes each were arbitrarily selected from the harvest of each plot, and the severity of achene blemish syndrome was assessed as described above. Data were subjected to statistical analysis; whenever the F values were significant at $P < 0.05$, treatments were compared according to Fisher's protected LSD test.

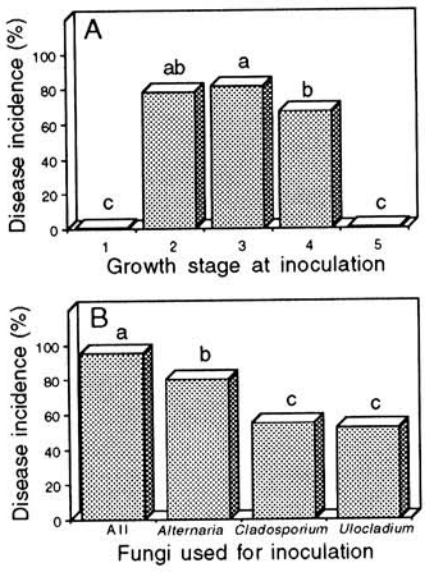

Fig. 2. Effects of **(A)** the host growth stage at inoculation (1 = 3–5 days after flowering, 2 = white achenes, 3 = initiation of achene color change, 4 = advanced stages of achene color change, and 5 = after physiological maturity) and **(B)** the fungi used for inoculation (*Alternaria alternata, Cladosporium* sp., *Ulocladium atrum*, or a combination of all three) on the incidence of achene blemish syndrome. Bars followed by the same letters do not differ significantly ($P = 0.05$), as determined by Fisher's protected LSD test.

RESULTS AND DISCUSSION

Identification of the causal agents. Several fungi were isolated from the infected achenes. The predominant ones were *Alternaria alternata* (Fr.:Fr.) Keissl., *Cladosporium* sp., and *Rhizopus* sp. Less common was *Ulocladium atrum* G. Preuss (identified by Y. Ben-Ze'ev, Ministry of Agriculture, Bet-Dagan, Israel). *Aspergillus* sp. was isolated in few cases. Urediospores and teliospores of *P. helianthi* were very common on seed. Koch's postulates were performed for the first four fungi. We assumed that *Aspergillus* sp. and *P. helianthi* were not associated with achene blemish syndrome because *Aspergillus* was isolated only very rarely and *P. helianthi* is an obligate parasite incapable of infecting a nonphotosynthesizing host tissue such as an achene. In addition, rust, caused by *P. helianthi*, was very common in many fields across the country where achene blemish syndrome was not observed. *A. alternata, Rhizopus* sp., *Aspergillus* sp., and *P. helianthi* have all been reported to colonize sunflower achenes in Israel (13) and elsewhere (3,16,19).

Of the fungi tested in artificial inoculations, *Rhizopus* did not induce the typical achene blemish syndrome. Sunflower heads inoculated with *Rhizopus* spores developed soft rot, typical of disease caused by that pathogen. Consequently, it was concluded that *Rhizopus* was not related to achene blemish syndrome. On the other hand, achenes from heads inoculated with *A. alternata, Cladosporium* sp., or *U. atrum* exhibited symptoms resembling those observed originally in the field. Moreover, all three fungi were reisolated from artificially inoculated achenes. The completion of Koch's postulates for these three fungi confirms their involvement in achene blemish syndrome.

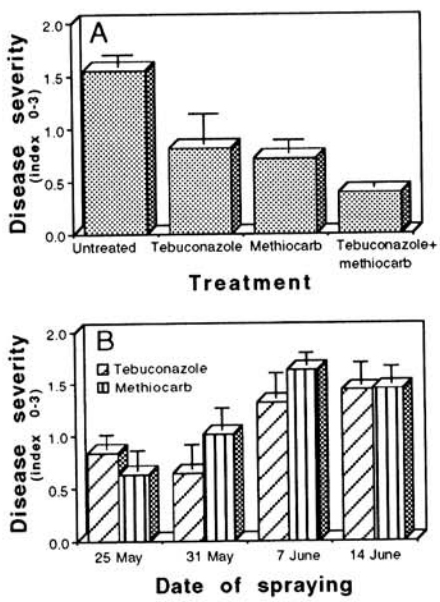

Fig. 3. Effects of the fungicide tebuconazole and insecticide methiocarb on the severity of achene blemish syndrome in a field trial. Pesticides were applied **(A)** four times and **(B)** once only on 25 May at growth stage 2 (white achenes), 31 May at growth stage 3 (initiation of achene color change), 7 June at growth stage 4 (advanced stages of achene color change), and 14 June at growth stage 5 (after physiological maturity). Disease severity was rated on a 0–3 scale in which 0 = uninfected achene and 3 = severely infected achene. Bars indicate the standard error.

Factors affecting the infection process. Sunflower heads were inoculated at five different growth stages with individual and combined spore suspensions of *A. alternata, Cladosporium* sp. or *U. atrum*. Since the interaction between the two main factors examined in these trials—the fungi used for inoculation and the growth stage at inoculation—was not significant ($P = 0.05$), only effects of the main factors are presented (i.e., averages for all levels of each main factor). Inoculation of achenes at flowering and physiological maturity did not result in any symptom development. On the other hand, a high incidence of symptom development was achieved when achenes were inoculated at various stages of their development (Fig. 2A). Nevertheless, even in achenes inoculated at early stages, symptoms were only visible just before physiological maturity.

The three fungi differed slightly with respect to infectivity. *A. alternata* induced a significantly higher disease incidence than did either of the other two fungi. The incidence of infection resulting from inoculation with a mixture containing spores of the three fungi (at a concentration similar to that of each fungus inoculated separately) was significantly higher than that resulting from inoculation with each fungus alone (Fig. 2B). This may suggest that interaction among the three fungi promotes their infectivity, possibly through a synergistic effect. Coexistence of these fungi has been reported in other crops. For example, they induce sooty mold of peaches (12), seed and seedling death of verbena (22), and discoloration of rice seed (6).

The microscopic structure of the sunflower seed, a cypsela, was described by Singh et al (21) and Vaughan (25). It is a type of achene consisting of an outer pericarp, an inner pericarp together with the testa, one or two layers of endosperm covered with a thick cuticle, and a spatulate embryo with a dome-shaped stem apex. Achenes sampled from the field at different growth stages were used to determine whether and when the fungi causing achene blemish syndrome penetrate into the inner pericarp. Surface sterilization prevented the growth of fungi on agar medium for all achenes sampled at the growth stages of white achenes and initiation of achene color change, indicating that they had infested only the outer layer of the peri-

carp. Microscopic observation revealed that mycelia had infected and decomposed the achene hairs. Similarly, Hadas (12) reported that *A. alternata, Ulocladium* sp., and *Cladosporium* sp., the causal agents of sooty mold in peaches, did not penetrate the peel tissue but became attached to the fruit hairs and decomposed them by enzyme action. Whenever achenes were sampled from the field at later growth stages (i.e., at advanced stages of achene color change or after physiological maturity), surface sterilization did not prevent the growth of fungi (mainly *A. alternata* and *Cladosporium* sp.) on agar medium, indicating that they had colonized the inner pericarp as well. Similarly, previous studies indicated that *A. alternata* was isolated from the inner pericarp of mature sunflower achenes (13,16,21).

Sunflower plants grown in the greenhouse were inoculated with a spore suspension of *A. alternata, Cladosporium* sp., and *U. atrum*. Inoculation was performed on heads with disk florets attached and on heads from which florets were removed. The incidence of infection was 61% in the former and 78% in the latter (differences were significant, $P < 0.05$). In another test, heads sampled from the field at the anthesis stage were kept in moist conditions for 72 hr. After that time, hyphae and conidia of *A. alternata* and *Cladosporium* sp. were observed on the disk florets. Since these heads were not inoculated artificially, the findings indicate that these fungi exist naturally in that vicinity. Access of fungi to developing seeds and fruits via the flowers has been reported in many pathosystems. Examples include *Diplodia viticola* Desmaz. in grapes (23), *Alternaria* sp. in tomato (26) and sweet pepper (14), and *Botrytis cinerea* Pers.: Fr. in strawberry, raspberry (15), and cucumber (10).

Several lines of evidence accumulated during the 1992 growing season suggest that the western flower thrips may be associated with achene blemish syndrome. This insect was first observed in Israel in 1988 and since then has infested an increasing number of crops, causing severe losses (2,18). It invaded sunflower fields for the first time in the 1991 season. The thrips are attracted to yellow flowers, and relatively high populations may develop on sunflower heads. It was noticed that large populations of thrips occurred in fields severely infected with achene blemish syndrome. The possible insect involvement in achene blemish syndrome was examined in the field during the 1993 season. The fungicide tebuconazole or the insecticide methiocarb, applied four times during achene development, significantly reduced the severity of achene blemish syndrome relative to that in untreated achenes. Moreover, when heads were treated with both pesticides, the severity of disease was reduced even further (Fig. 3A). In other treatments during this trial, tebuconazole or methiocarb was applied only once, on each of four dates. The severity of achene blemish syndrome was reduced only when the pesticide was applied at early stages of achene development (Fig. 3B). The numbers of thrips in treated heads were not monitored during the growing season so as not to disturb the natural development of the achenes. However, thrips populations in adjacent heads were relatively high.

The western flower thrips feed by rasping and sucking, thereby wounding the pericarp of young achenes. The wounding may provide an ideal point of entry for fungi. Another possibility is that the insect transfers fungal spores while moving, thus enhancing disease dispersal. Whatever the mechanism for infection, it is evident that the prevalence of the insect promoted the severity of achene blemish syndrome. Association of thrips with plant pathogens (viruses, bacteria, and fungi) has been reported in other crops as well (1,4,5,7–9,11,17,24).

Suppression of achene blemish syndrome. The effects of several fungicides on the severity of achene blemish syndrome were examined in two field trials in 1992. In both trials, disease severity in untreated plots was moderate. Some of the fungicides significantly reduced achene blemish syndrome. For example, difenoconazole reduced disease severity by 39% in trial 1 and by 17% in trial 2. However, this cannot be considered a substantial reduction, and in general no fungicide was highly effective (Table 1). The disappointing field results were expected, for several reasons. Soon after flowering, sunflower heads bend and face down. Pesticides, which are usually applied to field crops by means of a boom located above the crop, do not penetrate the canopy in a way that allows them good access to the developing achenes. Thus, the amount of fungicide reaching the target (i.e., the developing achenes) was relatively low. However, when pesticides were directed onto the target, as was done in the thrips study in 1993 (by means of a hand sprayer), the severity of achene blemish syndrome was reduced by 50% (fungicide or insecticide) and 75% (fungicide and insecticide) (Fig. 3A). Another reason for the low efficacy of the fungicides is that they were not applied with an insecticide and therefore did not affect the population of western flower thrips. On the basis of these results, and in view of the difficulty of suppressing both the pathogens and thrips, pesticides do not offer an adequate solution to achene blemish syndrome. Further studies should be conducted to find a potent and cost-effective method for reducing this disease.

Table 1. Effects of various fungicides on the severity of achene blemish syndrome of sunflower in two field trials in 1992

Fungicide (kg a.i./ha)	Trial 1	Trial 2
Untreated	1.18 a[z]	1.81 a
Hexaconazole (0.05)	1.21 a	1.79 ab
Maneb (2.0)	1.19 a	...
Iprodione (0.5)	1.14 a	1.56 ab
Fenbuconazole (0.05)	0.96 ab	1.58 ab
Promoconazole (0.1)	0.85 ab	1.82 a
Cyproconazole (0.05)	0.81 b	1.76 ab
Tebuconazole (0.125)	0.74 b	1.60 ab
Difenoconazole (0.125)	...	1.68 ab
Difenoconazole (0.187)	0.72 b	1.51 b
Oxine-copper (0.8)	0.70 b	1.55 ab

[z] Severity is expressed in terms of a 0–3 rating scale in which 0 = uninfected achene and 3 = severely infected achene (see Figure 1). Numbers in columns followed by the same letters do not differ significantly ($P = 0.05$), as determined by Fisher's protected LSD test.

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