Occurrence and Control of Anthracnose of Almond in Israel

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ABSTRACT

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Anthracnose of almond (*Prunus amygdalus*) was found for the first time in Israel in March 1977. Isolates obtained from infected fruits were identified as *Colletotrichum gloeosporioides*. The pathogen infected young almond fruits during the spring and early summer. Although *C. gloeosporioides* did not penetrate the twigs, it caused wilting and early drop of leaves, resulting in bare twigs and shoots. Laboratory and field tests showed that protective fungicidal treatment with captafol, captan, and folpet reduced disease severity.

Additional key words: *Gloeosporium amygdalinum*

In Israel, almond (*Prunus amygdalus*) is grown mainly in two regions with annual rainfall of about 400 mm. Nevertheless, on the average, there are 15 rainy days from mid-February to the end of April. Fruit set takes place in various almond cultivars from mid-February to early March, whereas the fruit develop during March and April and reach final size and shell hardening by the end of April.

In March 1977, young almond fruits with disease symptoms not familiar to us (Fig. 1) were found in a few orchards in the two almond-growing regions in Israel. During the spring of 1977, the disease spread to adjacent orchards. In 1978, many orchards of different almond cultivars were severely affected with the disease and substantial crop losses were reported by growers. In the following years (1979–1981) the disease reached an epidemic level that threatened the almond industry and many affected orchards were uprooted.

Mycelium isolated from the inside of every diseased fruit was initially identified as *Gloeosporium* sp. Similar isolates were obtained from sporulating acervuli on the exterior of diseased fruit. The pathogen *Gloeosporium amygdalinum* Brizi has been reported as the incitant of anthracnose disease in almond-growing regions with a Mediterranean climate (1–6). The purpose of this study was to identify the pathogen and its life cycle, to follow disease development, and to find chemical means for its control.

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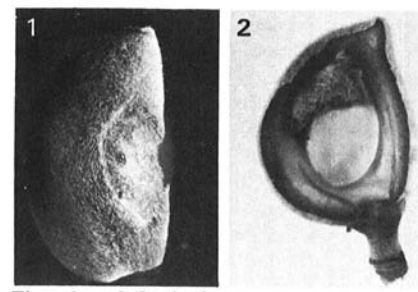
MATERIALS AND METHODS

Fruits with anthracnose symptoms were surface-sterilized by flame and cut into slices, which were placed on plates of potato-dextrose agar (PDA) and incubated at 25 C. Mycelium was also isolated directly from the internal part of the fruit onto PDA. Samples of the fungus thus isolated were identified by von Arx from the Centraal-bureau voor Schimmel Cultures, Baarn, Netherlands, as *Colletotrichum gloeosporioides* Penz., which is probably synonymous with *G. amygdalinum* Brizi (5).

Sporulating colonies were obtained by growing the fungus on Difco cornmeal agar. Spores for artificial inoculation were collected from the plates and suspended in sterile deionized water (10^5 to 10^6 spores per milliliter). Young fruits (25–40 mm long) from healthy unsprayed almond trees were laid on moistened paper towels in a shallow plastic box, and each fruit was inoculated with one drop of spore suspension, using a Pasteur pipet. The box was placed in a plastic bag and incubated at 25 C.

The effect of fungicides on fruit infection and anthracnose development was tested using artificial inoculations. Groups of 20 fruits were sprayed with water (control) or with fungicide suspensions 6 hr before or 24 hr after inoculation. Fruits were inspected daily for disease symptoms, and 10 days after inoculation, the fungicide treatments



Figs. 1 and 2. Anthracnose symptoms on almond fruits infected with *Colletotrichum gloeosporioides*. **(1)** Whole fruit; **(2)** longitudinal section showing partial colonization of kernel.

were rated as effective or ineffective according to the number of infected fruits and disease severity. The 23 fungicides tested were benomyl, bitertanol, captafol, captan, CGA 64251, chlorothalonil, copper hydroxide (Kocide), cyprex, dithianon, fenapronil, fenarimol, fluoroimide, folpet, guazatine, imazalil, iprodione, lime-sulfur, mancozeb, nuarimol, thiram, triadimefon, triforine, and vinclozolin.

Efficacy of fungicides in controlling anthracnose was tested in heavily infected almond orchards. In preliminary experiments, dormant sprays with Bordeaux mixture, copper oxychloride, or sodium pentachlorophenate in the winter (before bud burst) were ineffective. Likewise, three sprays during the spring with triforine or imazalil provided no control, whereas captan was more effective than thiram.

Based on preliminary field and laboratory experiments, five fungicides (Table 1) were evaluated further in a field test with 20-yr-old trees. Three replicates of two-tree plots of each of three cultivars (Non-plus-Ultra, Poria 10, and Um-El-Fachem) were sprayed three times at 10-day intervals during the spring, starting at the petal-fall fruit-set stage. A single spray with 4,800 mg/L captafol was also included for comparison with the regular protective treatments. Three weeks after the last spray, disease severity for each tree was assessed as follows: 0 = no infected fruits, 1 = fewer than 1% infected fruits, 2 = 1–3% infected fruits, 3 = 4–10% infected fruits, 4 = 11–20% infected fruits, and 5 = more than 20% infected fruits per tree.

RESULTS AND DISCUSSION

Laboratory and field observations indicated that the pathogen, symptoms, and development of anthracnose disease on almond trees in Israel were very similar to those reported from other countries (3,5,6). Almond fruits became infected in the spring and early summer (March through May) at various stages of their development, from fruit set until shell hardening. The fungus penetrated the husk and shell and infected the young kernel. A section along the infected fruit (Fig. 2) revealed discoloration and a dark, gummy substance under the anthracnose lesion and white mycelium on the kernel surface. A dark, gummy secretion often appeared on the surface of the green infected fruit; the husk became dry and the kernel shrank.

About 1 wk after the appearance of anthracnose symptoms on the fruit, leaf clusters associated with the fruit (borne on the same or distal nodes) started to wilt (Fig. 3). Shortly afterward, the leaves dried out and fell off, leaving a bare twig. Similar symptoms have been reported (3,6), but unlike Dippenaar (3), we have never detected the pathogen in wilting leaves. Furthermore, attempts to trace the fungus in serial sections of the fruit peduncle showed that it was restricted to the distal (fruit) end of the peduncle and did not invade the fruit-bearing node. Therefore, wilting could not be attributed to the presence of *C. gloeosporioides* in the shoot. In severely infected trees, many shoots became bare (Fig. 4) and progressive dieback of branches in the following 2–3 yr resulted in an increased crop loss.

Young infected fruits occasionally fell off the trees, or later during April through June, anthracnose fruit became mummifed and overwintered on the tree. The following spring, when mummies from infected trees had been incubated in a moist chamber in the laboratory, spores of *C. gloeosporioides* formed on their surfaces, supporting Dippenaar's (3) suggestion about the role of the mummies in overwintering of the pathogen. Spore acervuli that formed on the surface of the mummies in the orchard constituted the source of the primary spring inoculum and were evident on rainy days during the spring. Spores produced in acervuli that develop on the anthracnose lesions of young fruits serve as secondary inoculum in the same season.

In fruits with distal infections, the pathogen seldom reached the peduncle and such fruits remained on the tree as mummies, whereas in proximal infections, the pathogen often reached the peduncle and such fruits eventually dropped off. Sporulation occurred on dry, infected peduncles when incubated in a moist chamber as well as in the orchard during rainy days and could serve as additional primary inoculum. By pruning affected twigs and branches bearing overwintered mummies and infected peduncles, the primary inoculum could be reduced.

Infection of artificially inoculated young almond fruit by *C. gloeosporioides* started as a depression at the inoculation site, followed by colonization, tissue collapse, and sporulation by the pathogen. These symptoms agree with previous descriptions (3,5). None of the tested fungicides inhibited fruit infection when applied 24 hr after inoculation, and only four of the fungicides were effective when sprayed 6 hr before inoculation. Thus, on fruits treated with captafol (1 g/L), captan (1.25 g/L), folpet (1.25 g/L), or thiram (1.2 g/L), infection was not found 10 days after inoculation.

Fungicide evaluation in a field trial is summarized in Table 1. On unsprayed trees, almost all the fruits became infected. In early spring, three applications (starting at the petal-fall/fruit-set stage)

Fig. 3. (Right) Almond twig bearing one healthy fruit and (left) two young fruits with anthracnose; note leaf wilting of the distal cluster.

Table 1. Fungicide evaluation for the control of anthracnose of almond caused by *Colletotrichum gloeosporioides*

Fungicide[a]	Rate (mg a.i./L)	Infection rate[b] (cultivar)[c] A	B	C
Captafol (Merpafol 48SC)	960	0.8	1.4	1.3
Captan (Merpan 50W)	1,250	0.9	1.6	1.6
Folpet (Folpan 50W)	1,250	1.0	1.5	1.8
Captafol (Merpafol 48SC)	4,800[d]	2.0	2.9	2.0
Prochloraz (Prochloraz 50W)	500	2.9	3.0	1.4
Copper hydroxide (Kocide 101)	1,440	3.3	3.7	2.9
Unsprayed check	···	5.0	5.0	4.6

[a] Three successive sprays on 5 (at petal fall/fruit set), 14, and 24 March. Disease incidence was evaluated on 15 April.
[b] Infection rate: 0 (no infection) to 5 (>20% infected fruits per tree).
[c] Cultivars: A = Non-plus-Ultra, B = Poria 10, and C = Um-El-Fachem.
[d] Single application on March 5 (at petal fall/fruit set).

Fig. 4. Anthracnosed mummies from the previous year hanging on dead shoots of partially desiccated canopy of an almond tree.

of captafol, captan, and folpet protected the young fruits. A single high-rate spray of captafol or three sprays of prochloraz or copper hydroxide were less efficacious. Moreover, phytotoxicity was observed whenever rain came within a few days of application of copper compounds; necrotic spots appeared on the treated leaves and subsequently caused serious leaf drop.

Our field observations during six seasons (1977 through 1982) indicate that a close relationship exists between infection and spring rains. In the spring of 1982, which was relatively dry compared with other springs, disease incidence was low; even on unsprayed trees, very few young fruits became infected and no secondary infections were evident.

Since 1979, the routine practice for anthracnose control has been pruning and removal of affected branches followed by fungicide treatment in the spring. In infected orchards, three to five protective sprays with captan (1.25 g a.i./L) in 7- to 10-day intervals starting at the petal-fall/fruit-set stage effectively reduced disease spread and crop loss. Chemical control was particularly effective when combined with pruning to remove inoculum in infected orchards.

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