

Evaluation of Two Triazole Fungicides for Postinfection Control of Apple Scab

R. D. Kelley and A. L. Jones

Graduate research assistant and professor, respectively, Department of Botany and Plant Pathology and the Pesticide Research Center, Michigan State University, East Lansing 48824.

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ABSTRACT

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The triazole fungicides 1-((2-(2,4-dichlorophenyl)-4-ethyl-1,3-dioxolan-2-yl)methyl)-1*H*-1,2,4-triazole (CGA-64251) and bitertanol effectively controlled apple scab under greenhouse and orchard conditions. In greenhouse studies, postinfection scab control by CGA-64251 at 18.7 µg/ml and bitertanol at 299.6 µg/ml applied 2 and 3 days, respectively, after inoculation were similar to fenarimol at 41.9 µg/ml, fenapanil at 617.9 µg/ml, and an organic mercury fungicide at 93.8 µg/ml. Fungicides applied 4.0, 4.5, 5.0, and 5.5 days after inoculation did not prevent establishment of lesions, but did inhibit their development and sporulation from them. In orchard studies, two sprays 7 days apart of CGA-64251 at 18.7 µg/ml or bitertanol at 299.6 µg/ml, applied to lesions either late in the incubation

period or starting 2 days after they were visible, prevented spore production in chlorotic lesions and suppressed conidial development in sporulating lesions. Fruit infections also were suppressed. Fungicides applied at 7-day intervals suppressed scab development better than those applied at 14-day intervals. Leaves sprayed with CGA-64251 were smaller, thicker, puckered, darker green, and had more layers of palisade cells than those from unsprayed trees; whether the net effect was detrimental or beneficial remains to be determined. These therapeutic fungicides should be tested further in apple scab control experiments designed to identify infection periods and to suppress established epidemics of apple scab.

Additional key words: *Venturia inaequalis*, *Malus pumila*.

Management of apple scab, which is caused by *Venturia inaequalis* (Cke.) Wint., requires fungicides for postinfection control of the disease. Currently, effective postinfection control of scab in the United States is limited because strains of dodine- and benomyl-resistant *V. inaequalis* have developed (10,20), because some fungicides can only be used early in the growing season to avoid phytotoxicity problems, and because public agencies have canceled or delayed registrations of some effective fungicides. Since 1968, several fungicides that inhibit ergosterol biosynthesis and membrane function have been evaluated for apple scab control. The pyrimidine fungicides triarimol, fenarimol, and nuarimol, and the piperazine fungicide triforine were studied extensively and found to control scab after the onset of infection (8,17). Recently, a group of triazole fungicides, which inhibit ergosterol biosynthesis and exhibit postinfection control properties (19), have become available for testing (4,16). Two of these, 1-((2-(2,4-dichlorophenyl)-4-ethyl-1,3-dioxolan-2-yl)methyl)-1*H*-1,2,4-triazole (CGA-64251 10% W, CIBA-Geigy Corp., Greensboro, NC 27409) and bitertanol (Baycor 50% W, Mobay Chemical Corp., Kansas City, MO 64210), were selected for study.

The purpose of this study was to investigate the postinfection control properties of these fungicides under greenhouse and orchard conditions and to examine their effects on apple scab lesions and on conidial development and morphology.

MATERIALS AND METHODS

Greenhouse studies. Actively growing, single-shooted McIntosh apple (*Malus pumila* Mill.) trees in pots were inoculated with a suspension of conidia of *V. inaequalis* (3×10^5 /ml). The suspension was made by washing infected apple leaves with distilled water, and was atomized onto the trees 1 hr before placing them in a mist chamber at 20 C for 47 hr. The youngest leaf on each shoot was tagged for later reference.

The trees were removed from the mist chamber and placed on a

cheesecloth-enclosed bench in the greenhouse. The cheesecloth was wetted daily to maintain the relative humidity at 80–100%. At different intervals after inoculation, four plants for each fungicide treatment were removed from the enclosure, sprayed with fungicide and, after the deposit had dried, returned to the enclosure. Eight inoculated plants were left unsprayed as controls. Data were recorded 18 days after inoculation by visually estimating the leaf area covered with sporulating lesions and the total leaf area covered with lesions, (both sporulating and nonsporulating [chlorotic] lesions) by using a standard diagram for comparison (21). The four leaves below the tagged leaf were evaluated. The experiment was done twice.

Field studies. In 1979, an orchard of 3-yr-old McIntosh apple trees on M26 rootstock was sprayed to runoff with a handgun at 28 kg/cm² (400 psi). Treatments were arranged in a randomized complete block design with four blocks and three trees per replicate. Data were taken at about 2-wk intervals from mid-June to mid-September on 20 terminals per replicate.

In 1980, an orchard of 5-yr-old McIntosh apple trees on M7 rootstock was used. Treatments were replicated four times in a completely randomized design using single-tree plots. Tags were tied onto the youngest expanded leaf after critical infection periods to identify susceptible leaves exposed to infection. Data were taken on 30 fruit spurs and 20 terminals per replicate.

Infection periods were predicted with a microprocessor-based instrument placed between two apple trees and about 1.5 m above ground level. This instrument, a modification of a unit described by Jones et al (9), monitored temperature, rainfall, leaf wetness, and relative humidity in the orchard. Incubation periods were estimated based on the average temperature during the infection periods by using Mills' table (12).

Effects of the fungicides on conidial morphology and development were determined by examining lesions with an ISI Super III scanning electron microscope (International Scientific Instrument Corp., Santa Clara, CA 95050). Leaf disks with individual lesions were cut from randomly collected leaves with a cork borer, fixed in phosphate-buffered 4% glutaraldehyde for 24 hr, washed twice in 0.1 M phosphate buffer (pH 7.3), and postfixed in 1:1 mixture of 0.2 M phosphate buffer and 2% OsO₄ for 24 hr. Samples were washed again in phosphate buffer, dehydrated in an ethanol series, and critical-point dried. Dried specimens were

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mounted on stubs and sputter-coated with 200 μm of gold. In addition, lesions were rubbed across the surface of PDA to remove the conidia and germination was evaluated after 24 and 48 hr.

To determine the viability of the fungus within chlorotic lesions, isolations were attempted by using the procedures of Hoch and Szkolnik (8). Leaves were collected on 3 June from each replicate of treatments showing chlorotic lesions and washed for 5 hr in running tap water. Twenty chlorotic lesions per treatment were removed with a cork borer, dipped briefly in 70% ethanol, and cut into quarters. Each set of quarter sections was placed in a petri dish containing 3% malt extract agar amended with 250 ppm streptomycin. Plates were examined for growth of *V. inaequalis* after 20 days at 20 C. Lesions from unsprayed trees were included for comparison.

RESULTS

Greenhouse studies. Bitertanol (149.8 and 299.6 μg a.i./ml) and CGA-64251 (18.7 μg a.i./ml) were applied to trees 2, 3, 4, 4.5, 5.0, and 5.5 days after inoculation. Three fungicides: fenarimol (EL-222 12.5% EC) at 42 μg a.i./ml from Elanco Products Co., Indianapolis, IN 46206; fenapanil (RH-2161 240 g/LEC) at 617 μg a.i./ml from Rohm and Haas Co., Philadelphia, PA 19105; and phenyl mercury triethanol ammonium lactate (Puritized Agricultural Spray [PAS] 7.5% liquid) at 93.8 μg a.i./ml from Niagara Chemical Division of FMC Corp., Middleport, NY 14105, which have exhibited eradivative properties in the past, were included for comparison.

In several of the treatments, both normally sporulating lesions and chlorotic fleck symptoms were present (Table 1). CGA-64251 and bitertanol at 299.6 μg a.i./ml were similar in effectiveness to fenarimol, fenapanil, and PAS when applied 2 and 3 days after inoculation. At 4 days after inoculation, the percent leaf area covered with chlorotic flecks was high for all fungicides, but only leaves sprayed with PAS and bitertanol at 149.8 μg a.i./ml exhibited normal lesions. Treatments continued to suppress development of normal lesions 4.5, 5.0, and 5.5 days after inoculation.

Field studies, 1979. CGA-64251 10% W (18.7 μg a.i./ml) was evaluated in five postinfection treatments (Fig. 1) as follows: a postinfection eradication schedule with treatments applied within 3 days after the beginning of wetting periods predicted to give infection; two presymptom eradication schedules (the first with treatments applied 2 days before lesions were predicted to appear and the second [which included a second fungicide] applied 1 wk later); and two postsymptom eradication schedules (the first with treatments applied 2 days after lesions were visible and the second [which included a second fungicide] applied 1 wk later). A protective treatment was included for comparison.

The presymptom and postsymptom eradication schedules with a repeat spray 1 wk after the first resulted in significantly ($P = 0.05$) less infection than the one-spray sequence schedules and were not significantly different in effectiveness from the 7-day-schedule (Table 2). Effects of the postinfection schedule was not significantly ($P = 0.05$) different in levels of infection from the 7-day-schedule on 11 July, but significantly more scab was recorded in the postinfection schedule on 1 August (Table 2).

Field studies, 1980. Bitertanol 50% W (299.6 μg a.i./ml) and CGA-64251 10% W (18.7 μg a.i./ml) were evaluated in three postinfection spray schedules (Fig. 1) as follows: a postinfection eradication schedule with treatments applied within 3 days from the beginning of wetting periods predicted to give infection; a presymptom eradication schedule with a spray applied 2 days before lesions were predicted to appear and again 1 wk later; and a postsymptom eradication schedule with a spray applied 2 days after lesions were visible and again 1 wk later. Two protective treatment schedules with sprays applied at 7- and 14-day intervals were

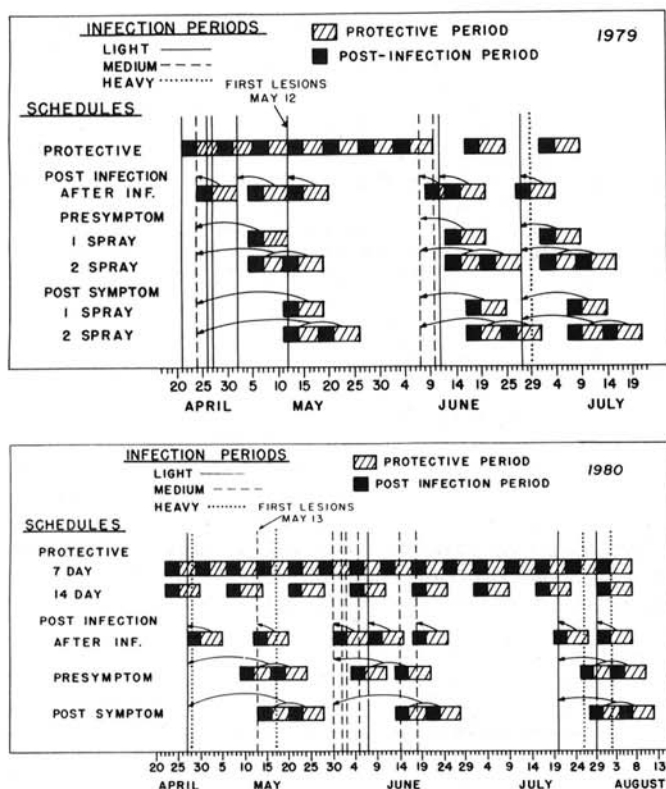


Fig. 1. Timing of apple scab spray schedules in 1979 and 1980 in relation to predicted infection periods. Right margin of the black square corresponds to the date of application for each spray. The 3-day postinfection period is based on greenhouse studies (Table 1), but the period of protective action is an estimate. Arrows indicate initial and intervening infection period(s) that each spray or spray sequence was expected to control.

TABLE 1. Postinfection control of scab on potted apple trees in the greenhouse by fungicides applied at various times after inoculation

Treatment ^y	Dosage (μg a.i./ml)	Leaf area infected 18 days after inoculation											
		2 days ^v		3 days		4 days		4.5 days		5 days		5.5 days	
		Total ^w (%)	Normal ^x (%)	Total (%)	Normal (%)	Total (%)	Normal (%)	Total (%)	Normal (%)	Total (%)	Normal (%)	Total (%)	Normal (%)
Fenarimol 12.5% EC	41.9	0	0	4	0	23	0	5	0	8	0	24	5
PAS (mercury) ^z	93.8	1	0	8	2	26	8	36	7	34	9	15	2
CGA-64251 10% W	18.7	1	0	0	0	10	0	8	1	18	4	22	7
Bitertanol 50% W	149.8	14	2	27	3	26	12	35	10	27	0	31	3
Bitertanol 50% W	299.6	11	0	6	0	42	0	48	5	42	1	33	5
Fenapanil 2 EC	617.9	0	0	2	0	8	0	11	0	15	3	35	11

^v Days refer to the interval between inoculation and application fungicide.

^w Percent leaf area covered with normal sporulating lesions and with chlorotic flecks were determined by rating four leaves per replication. Data are the means for the four replications.

^x Percent leaf area covered with normal sporulating lesions.

^y Unsprayed controls (average of eight plants) showed 49 and 37% of the leaf area covered with total normal lesion types, respectively, 18 days after inoculation.

^z Puritized Agricultural Spray (PAS) containing 7.5% phenyl mercury triethanol ammonium lactate.

included for comparison.

Many schedules permitted the formation of nonsporulating chlorotic flecks on leaves (Fig. 2A) and lesions on fruit that corked over as the fruit grew (Fig. 2B). In some schedules, particularly 14-day schedules, pinpoint lesions were observed on the fruit early in the season; as the fruit matured, however, the lesions became increasingly difficult to detect. In the presymptom and postsymptom schedules, a few yellow leaves (less than 1%) were observed on 6 June (Fig. 2C). The yellow leaves contained green circular areas that, upon clearing and treatment with basic fuchsin stain (14), were found to contain subcuticular growth of *V. inaequalis*. These leaves fell from the tree a few days later.

Data taken 22 May indicate the effectiveness of the schedules to

control scab from the two late-April infection periods. The after-infection treatments of CGA-64251 and bitertanol on 30 April were applied 63 and 36 hr from the beginning of the 27 and 28 April wetting periods, respectively. The incidence of scab in these treatments was not significantly different from the incidence in the protective schedules (Table 3). The incidence of scab in the presymptom and postsymptom schedules, regardless of chemical, was not significantly different from the incidence for untreated trees. Many scab lesions were observed on 13 May, indicating that the presymptom sprays of 12 May were applied too late in the incubation period to prevent symptoms from appearing.

Data taken 6 June indicate that the most effective treatments were the 7-day schedule with both chemicals, the after-infection

TABLE 2. Control of scab on McIntosh apple with protective and postinfection eradicator treatments of CGA-64251 10% W applied in dilute spray^x under orchard conditions in 1979

Treatments	Number of sprays	Incidence of scab on terminals ^y					
		6 June		11 July		1 August	
		Leaves infected (%)	Lesions per terminal ^z	Leaves infected (%)	Lesions per terminal ^z	Leaves infected (%)	Lesions per terminal ^z
Protective treatment							
7-day-schedule	9	0	0	0 a	0 a	3 a	2 a
Postinfection treatments							
Postinfection schedule	6	0	0	3 a	1 a	17 b	20 a
Presymptom schedules							
One-spray sequence	3	17	3	41 c	39 c	53 d	115 c
Two-spray sequence	6	1	0	5 a	2 a	8 ab	6 a
Postsymptom schedules							
One-spray sequence	3	5	1	21 b	16 b	36 c	52 b
Two-spray sequence	6	3	1	2 a	1 a	6 ab	4 a
Untreated (controls)	0	24	7	55 c	128 c	79 e	...

^x 18.7 µg a.i./ml.

^y Means followed by the same letter did not differ significantly ($P = 0.05$) according to Duncan's multiple range test on arcsine-transformed percentage data and log-transformed lesion data.

^z The mean number of leaves per terminal was 7.5, 22.0, and 23.9 on 6 June, 11 July, and 1 August, respectively. There was no significant difference between mean number of leaves on plants that received the different treatments for any of the dates.

TABLE 3. Control of scab on McIntosh apple with protective and postinfection eradicator schedules of CGA-64251 and bitertanol^y applied in dilute sprays under orchard conditions in 1980

Treatments	Number of sprays	Incidence of scab on spurs ^w				Incidence of scab on terminals ^w					Scab on fruit 5 September	
		22 May		5 June		6 June		24 June		Type of lesions ^y	Fruit infected (%)	Normal lesions (%)
		Leaves infected (%)	Lesions per spur ^x	Leaves infected (%)	Lesions per spur ^x	Leaves infected (%)	Lesions per terminal ^x	Leaves infected (%)	Lesions per terminal ^x			
Protective treatments												
7-day schedules												
bitertanol	15	0.0 ^z	0.00 ^z	1.0 a	0.05 a	1.5 ab	0.76 ab	0.8 a	0.29 a	...	1.00 a	0.0
CGA-64251	15	0.0 ^z	0.00 ^z	0.0 ^z	0.00 ^z	0.2 a	0.05 a	0.2 a	0.22 b	...	5.25 ab	0.0
14-day schedules												
bitertanol	8	1.5 a	0.16 a	5.2 a	0.48 ab	9.2 bc	11.66 abc	1.8 b	12.62 c	C/B	5.75 ab	1.0
CGA-64251	8	3.5 abc	0.37 ab	5.2 a	0.80 abc	24.5 de	29.20 d	25.8 bc	31.45 c	C/B	12.75 abc	0.0
Postinfection treatments												
Postinfection schedules												
bitertanol	7	0.5 a	0.03 a	1.2 a	0.05 a	5.5 abc	3.64 abc	9.8 b	6.89 c	C/B	5.00 ab	1.5
CGA-64251	7	5.2 ab	0.74 abc	5.5 a	0.46 ab	12.8 cd	13.20 bcd	15.2 bc	12.28 c	C/B	13.25 bc	0.8
Presymptom schedules												
bitertanol	6	16.8 d	2.27 cd	22.5 b	3.64 c	14.2 cd	21.94 cd	33.8 c	42.05 c	B/C	21.50 c	1.8
CGA-64251	6	16.0 cd	2.74 cd	20.0 b	2.47 bc	1.8 ab	0.38 ab	21.2 bc	20.52 c	B/C	8.00 ab	2.0
Postsymptom schedules												
bitertanol	6	13.5 bcd	1.79 bcd	20.2 b	2.05 bc	11.2 bc	12.71 bcd	24.2 bc	31.14 c	C/N	6.25 ab	0.0
CGA-64251	6	19.0 d	2.55 d	24.0 b	2.57 c	2.5 abc	1.02 abc	34.5 c	36.08 c	C/N	10.25 abc	0.5
Untreated (controls)	0	23.0 d	3.56 d	55.8 c	20.65 d	38.2 e	...	82.0 c	...	N	100.00 d	100.0

^w CGA-64251 (10% W 18.7 µg a.i./ml) and bitertanol (50% W 299.6 µg a.i./ml).

^x Means followed by the same letter did not differ significantly ($P = 0.05$) according to Duncan's multiple range test on arc sine-transformed percentage data and log-transformed lesion data.

^y The mean number of leaves per spur (or terminal) was 6.7, 5.3, 12.5, and 16.8 on 22 May, 5 June, 6 June, and 24 June, respectively. There were no significant differences between mean numbers of leaves on plants that received different treatments for any of the dates.

^z Observations of indicated lesions were: N = normal sporulating, C = chlorotic, or B = burned out. C/N indicates both chlorotic and normal sporulating lesions were present.

^z Data were not included in the statistical analysis.

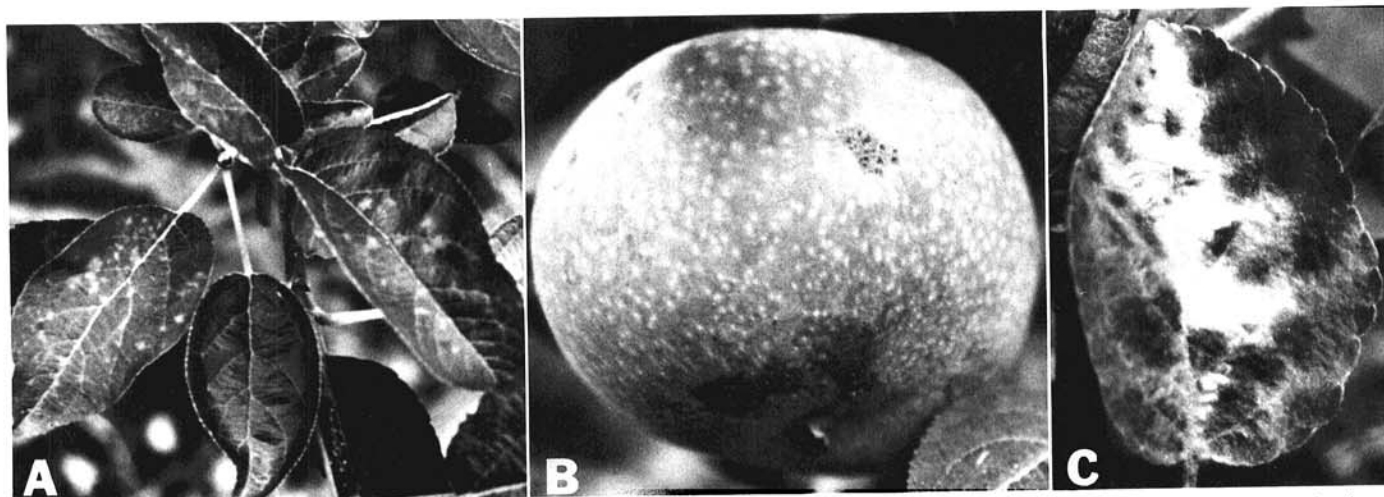


Fig. 2. Symptoms caused by *Venturia inaequalis* on apple leaves and fruit after fungicide treatment. **A**, typical chlorotic lesions on leaves; **B**, deactivation of apple scab lesions on fruit sprayed after normal symptoms were visible; and **C**, leaf from trees on presymptom spray schedule with chlorosis and green islands containing subcuticular growth of *V. inaequalis*.

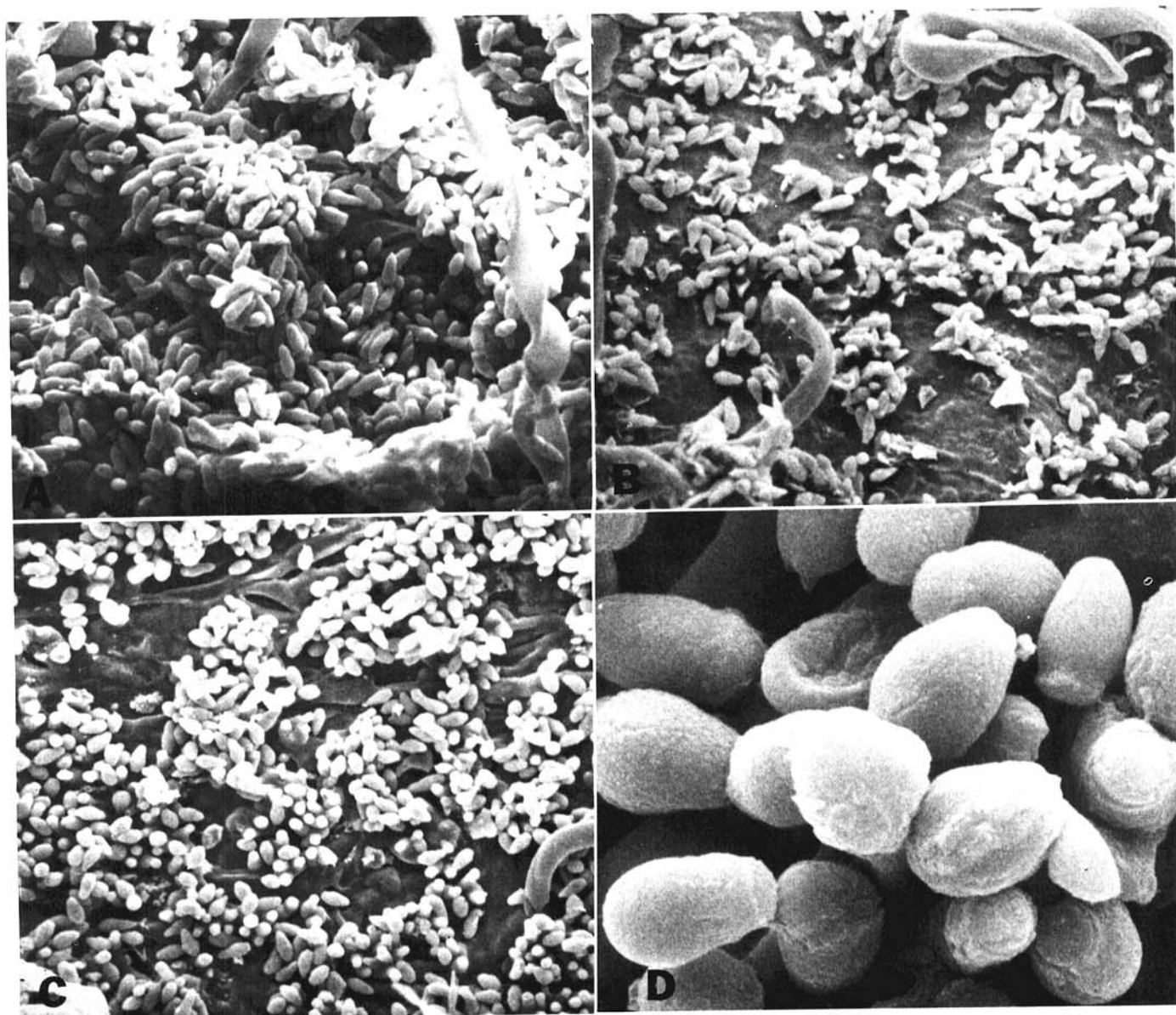


Fig. 3. Scanning electron micrographs of lesions caused by *Venturia inaequalis* on apple leaves either unsprayed or sprayed two times 7-days apart and sampled 2 days after the second spray. **A**, Unsprayed lesion with typical dense sporulation ($\times 418$); **B**, lesion sprayed with CGA-64251 at $18.7 \mu\text{g/ml}$, which reduced sporulation ($\times 424$); **C**, lesion sprayed with bitertanol at $299.6 \mu\text{g/ml}$, which reduced sporulation ($\times 444$); and **D**, ampulliform (immature) conidia in bitertanol sprayed lesion; these are more common in sprayed than in unsprayed lesions ($\times 3,043$).

schedule of bitertanol, and the presymptom and postsymptom sprays of CGA-64251 (Table 3). Large numbers of chlorotic lesions were observed in trees on the 14-day schedules, in the postinfection schedule of CGA-64251, and the presymptom and postsymptom schedules of bitertanol.

Data taken 24 June indicate the 7-day, 14-day, and postinfection schedules were the most effective treatments because the increase in number of scab lesions from 6 to 24 June were low compared to the increase in the unsprayed treatment (Table 3). The increase in the number of lesions in the presymptom schedules was high, particularly for CGA-64251, but the lesions were chlorotic and appeared to be inactive (burned out).

Depending on the fungicide treatment, 78.5–99% of the fruits were free of scab lesions on 5 September (Table 3). Most of the lesions had “healed” over (Fig. 2B) and none of the spray treatments had more than 2% of the fruit with normal scab lesions.

Effect of fungicides on lesion development and isolation recovery in 1980. Lesions from the 27 and 28 April infection periods were sprayed twice in 7 days with bitertanol and CGA-64251 starting 2 days after they were visible. Two days after the second spray, lesions were collected for scanning electron microscopy. The density of conidia in lesions from unsprayed trees (Fig. 3A) was greater than that in lesions from sprayed trees (Fig. 3B-C), and many more ampulliform or immature conidia were observed in sprayed than in unsprayed lesions (Fig. 3D). When lesions from sprayed trees were rubbed across PDA, it was very difficult to dislodge the conidia, and the few conidia that were removed did not germinate. Large numbers of viable conidia were obtained from unsprayed lesions. Upon close examination, sporulation on lesions on sprayed trees in the orchard appeared to be inactive.

Chlorotic lesions on leaves exposed to the 13 and 17 May infection periods were also examined with the scanning electron microscope. Subcuticular fungal growth was observed in most chlorotic lesions. Lesions sprayed with bitertanol appeared to have more growth than lesions sprayed with CGA-64251 (Fig. 4A-B), but no mature spores were observed (Fig. 4C). Many ampulliform conidia were observed in lesions from the postinfection schedules, but only scattered abnormal surface growth was observed in lesions from the other schedules. The proportion of lesions with fungal growth to the number examined was 1 of 6 and 5 of 5 for the 14-day and postinfection schedules of CGA-64251, respectively; 3 of 6, 1 of 5, 6 of 7, and 5 of 5 for the 14-day, presymptom, postinfection, and postsymptom schedules of bitertanol, respectively.

On 3 June, isolations were made from chlorotic lesions initiated during infection periods on 13 and 17 May. The proportions of successful isolations of *V. inaequalis* to the number of lesions examined were 5 of 11 (45.5%) for the 14-day schedule of CGA-64251, 4 of 8 (50%) and 7 of 10 (70%) for the 14-day and postinfection schedules of bitertanol, and 11 of 11 (100%) for the unsprayed control.

Tree growth retardation. Retarded tree growth was observed in all CGA-64251 schedules in 1979 and 1980, and was most severe on trees sprayed with the 7-day schedule. Bitertanol influenced tree growth much less; only a slight effect was noted on trees sprayed with 7- and 14-day schedules. Elongation of lateral shoots was retarded and leaves were smaller, thicker, puckered, and darker green than those on unsprayed trees. Growth retardation was most pronounced in June and became less obvious later in the season. Leaves were taken from similar locations in sprayed and unsprayed trees and cross sections were examined with a light microscope. Leaves from sprayed trees had three to five layers of palisade cells; those from unsprayed trees had two to three layers.

DISCUSSION

The occurrence of chlorotic lesions on leaves and of nonsporulating lesions on fruit, rather than typical sporulating lesions, are evidence of a type of disease control. Infected leaves with chlorotic lesions are more functional than diseased leaves with normal lesions. The limited defoliation (<1%) noted under the high inoculum pressure in this study should not affect further crop protection. We believe fruit with corked-over lesions would be

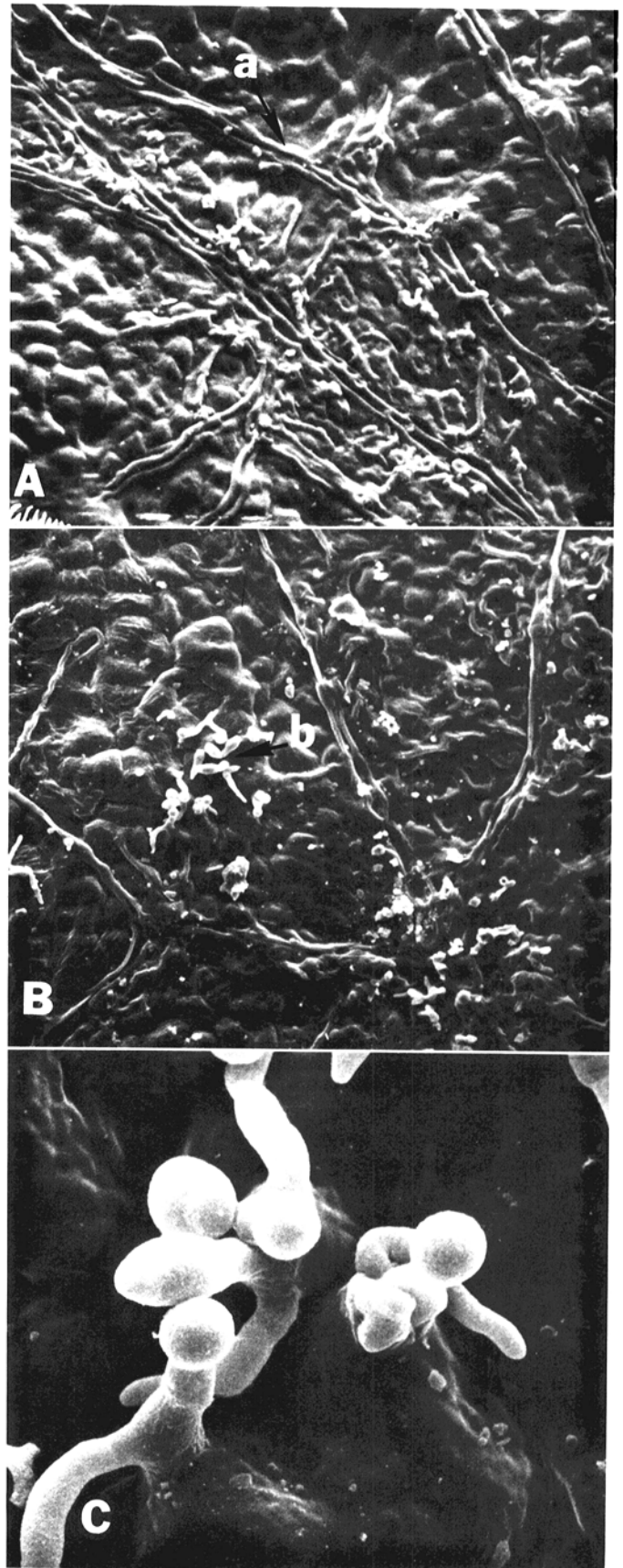


Fig. 4. Scanning electron micrographs of chlorotic lesions of *Venturia inaequalis* on apple leaves 4 days after treatment in a 14-day spray schedule. **A**, subcuticular (a) and infrequent deformed surface growth (b) in a lesion sprayed with CGA-64251 at 18.7 $\mu\text{g/ml}$ ($\times 280$); **B**, subcuticular (a) and surface growth (b) in a lesion sprayed with bitertanol at 229.6 $\mu\text{g/ml}$ ($\times 400$); and **C**, immature surface growth in a lesion sprayed with bitertanol.

commercially acceptable for processing, but not for the fresh market, except where the lesions were suppressed when they were quite small. In addition, the suppression of lesions on fruit and leaves should reduce secondary spread.

The development of chlorotic lesions suggests that the rates were too low to completely prevent scab symptoms, the treatments were applied too late to prevent establishment of lesions but soon enough to control lesion development, or environmental factors (eg. moisture and temperature) modified their effectiveness. Although subsequent lesion development or sporulation were not observed, recovery of *V. inaequalis* from chlorotic lesions indicated the fungus had not been killed and possible overwintering of the fungus in leaves with chlorotic lesions needs to be determined.

The suppression of sporulation and viability of *V. inaequalis* conidia when CGA-64251 and bitertanol were applied after symptom expression is equivalent to "postsymptom" control activity as described by Szkolnik (18). Also, the high proportion of immature conidia in sprayed lesions indicated that development of the fungus in the lesions was arrested by the fungicides. Suppression of conidia in leaf lesions by postsymptom applications of benomyl and dodine, particularly where more than one spray is applied, is well documented (1-3,6,7,11). Inactivation of lesions on fruit was reported for dodine (13), but it has received little attention even though it might effectively increase storability and marketability of the fruit.

Growth retardation effects similar to those exhibited by CGA-64251 and bitertanol in this study were with triarimol in bean plants (15) and with triadimefon in tomato and cotton plants (5). The effect has been attributed to inhibition of gibberellin biosynthesis and can be overcome by applications of gibberellic acid (5,15). It is not clear if these growth-retarding effects are detrimental to yields; they may even stimulate yields by increasing the number of fruit spurs.

CGA-64251 and bitertanol should be useful tools in apple scab control programs based on the identification of infection periods (9). Previously, when weather conditions or equipment failure prevented timely applications of a fungicide with postinfection activity, normal disease development was likely. Due to the therapeutic action of sprays applied late in the incubation period or following symptom expression, the economic risks of postinfection spray programs are greatly reduced. CGA-64251 and bitertanol will be most useful where dodine- and benomyl-resistant strains of *V. inaequalis* are a problem and no alternative fungicide is available commercially for postinfection control.

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