Effect of Thiophanate on Epidemic Development of Anthracnose and Yield of Watermelon

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ABSTRACT

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Artificial inoculation of watermelon with *Colletotrichum lagenarium* caused severe anthracnose and yield losses up to 63%. Thiophanate methyl (Topsin-M) sprays applied during early disease development reduced disease severity. Thiophanate sprays protected plants from infection by *C. lagenarium* for about 20 days, but did not eradicate the fungus from established lesions. Early infection (28 to 45 days after sowing) resulted in severe disease, premature death of plants, and substantial yield loss, while late infection (80 days after sowing) resulted in less yield loss. Protected plants yielded more marketable fruits and fewer diseased fruits. Correlation coefficients between disease indices and infected fruits were positive and significant ($P = 0.05$), whereas correlations with total fruit yield were negative and significant ($P = 0.01$). Highest correlations were found between yield loss and disease indices during flowering to fruit formation period. The apparent infection rates of anthracnose were higher on unsprayed plants than on those sprayed with thiophanate.

Additional key words: yield loss, infection rates.

Anthracnose of watermelon (*Citrullus vulgaris* Schrad. ex Eckl. & Zeyh) is a serious disease caused by *Colletotrichum lagenarium* (Pass.) Ellis & Halsted. Walker and Weber (13) reported that anthracnose was more severe and destructive than all other diseases on watermelon in the southern states of the USA. This disease also has been reported on watermelon from California and India (7,9). The same pathogen also causes anthracnose and severe losses on cucumber and other members of the Cucurbitaceae in the USA and India (5–7). Amin et al (2) showed that watermelon anthracnose is a compound interest disease and that it can be effectively controlled by benomyl and thiophanate.

The objectives of the present investigations were to determine the rate of anthracnose disease development and the subsequent yield loss in thiophanate-protected and unprotected watermelon plants.

MATERIALS AND METHODS

Field layout. Thirty seeds of watermelon cultivar Sugar Baby were sown separately on 8- to 10-cm-high ridges in field plots on 29 July 1977. The plants were 15 cm apart in rows spaced 3 m apart. The design of the field experiment was a randomized block design with four replications. Individual plot size was 5×3 m. Fertilizers totaling 50-30-30 kg/ha N-P-K, respectively, were applied.

Inoculations. The pathogen, *Colletotrichum lagenarium,* was isolated from diseased watermelon leaves by the method described earlier (1), and a pure culture was deposited at CMI, Kew, England as IMI 212952. Spore suspensions were prepared from 15-day-old cultures grown on bottlegourd leaf extract agar (BLA). *C. lagenarium* sporulates well on BLA under normal laboratory conditions. Actively growing, 1-mo-old plants in each plot were inoculated on 29 August by spraying them with a spore suspension (5.1×10^6/ml) applied with an Aspee Gator rocking-type sprayer (American Spring & Processing Works Ltd., Malad, Bombay, 400 064, India). All the leaves were covered with spore suspension.

Disease incidence and fungicide treatments. Early anthracnose leaf spots were small, brown, and necrotic; they gradually increased in size and finally developed characteristic shot hole symptoms. Initial disease incidence and further development were manipulated by spraying with thiophanate methyl wettable powder (Topsin-M 70% wp). Different spray treatment schedules were started after symptoms were noticed on the leaves. Sprays of 0.1% thiophanate (0.666 kg/ha) were applied on 27 August at 30 days after sowing (DAS), 8 September (42 DAS), 19 September (53 DAS), 29 September (63 DAS) and 10 October (74 DAS), depending upon the treatment schedule.

Evaluation and infection rate. A disease index scale of 1–9 was evolved after we observed symptoms on many plants under natural field conditions. Healthy plants were graded as 1, while severely wilted or almost dead plants were graded as 9. Disease indices along with symptoms were reported by Amin et al (2). Ten individual plants per plot were scored. Nine observations on disease incidence and severity were recorded from 31 August to 27 October 1977. Combined yield data were recorded in kg/plot. Average apparent infection rates were calculated according to Vanderplank's (11) logistic equation,

$$r = [2{\cdot}3\,(t_2 - t_1)^{-1}][\log_{10}\{x_2(1-x_2)^{-1}\} - \log_{10}\{x_1(1-x_1)^{-1}\}]$$

in which r denotes the infection rate in units/day, t_1 and t_2 denote the times (days) of first observations and subsequent observations, and x_1 and x_2 represent the proportion of diseased plant tissue on the first and subsequent observation dates. Infection rates were calculated for values of x determined as (average disease index-1)/8. Experimental results were statistically analyzed.

Yield loss. This was calculated as explained by James et al (4). Yield of plots where thiophanate was applied four times beginning at 42 DAS was considered as a base and treated as 100%.

RESULTS

Thiophanate reduced anthracnose severity on watermelon plants when applied at 30 DAS (Fig. 1). One early application spray was sufficient to keep disease levels significantly below those in unsprayed check plots for at least 32 days (62 DAS). An additional application at 42 DAS resulted in little additional reduction in disease severity, but additional early applications of thiophanate at 53 and 63 DAS kept the disease severity significantly below that in the check plots until the last disease evaluation at 91 DAS. Plots with five applications of thiophanate had significantly less disease

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at 82 and 91 DAS than did plots that received any other treatment.

The effectiveness of the first application of thiophanate at 30 DAS lasted for about 20–25 days, after which disease severity increased significantly above that in plots that received additional applications (Fig. 1). The effectiveness of the second application began to decline after 13 days.

When thiophanate application was delayed until 53 DAS, two additional applications at 10- to 12-day intervals were not sufficient to keep the disease severity significantly below that in check plots (Fig. 2). However, four applications beginning at 42 DAS did keep disease severity significantly below that in check plots until the last day of evaluation (91 DAS).

Apparent infection rates, r, were calculated on the basis of visible symptoms for each time interval of disease severity evaluation. In unsprayed check plots, apparent infection rates were high early in the season, but declined as the season progressed (Fig. 3). When thiophanate was applied early (beginning at 30 DAS), the early infection rates were usually low, but infection rates late in the growing season were as high as those in unsprayed check plots. When the first application of thiophanate was at 53 DAS or later, the infection rates were as great as those in check plots throughout the season.

Two-to-five applications of thiophanate beginning at 30 DAS significantly increased total fruit and marketable fruit production (Table 1). Fruit with a visible lesion was not considered marketable. When the first application of thiophanate was delayed, four applications were required to significantly increase the total fruit and marketable fruit weight. The greatest total fruit and marketable fruit weight occurred in plots that received four applications of thiophanate beginning at 42 DAS. Relative to this treatment, yield losses in other treatments ranged from 19–63%.

Disease indices on all but the first evaluation date (34 DAS) and the last two evaluation dates (82 and 91 DAS) were significantly positively correlated with weight of diseased fruit (Table 2). The best correlation was with disease indices on 42 DAS. Disease indices on all but the first two (34 and 42 DAS) and the last (91 DAS) evaluation dates were significantly negatively correlated with the total fruit weight. Greatest negative correlations were for disease indices at 56, 60, and 65 DAS. Because there was little variation in disease indices on the first and last evaluation dates, significant correlations would not be expected for those dates. During the period between flowering and fruit formation, 46–65 DAS, disease levels varied significantly among treatments and the disease indices were correlated with total fruit yield and diseased fruits.

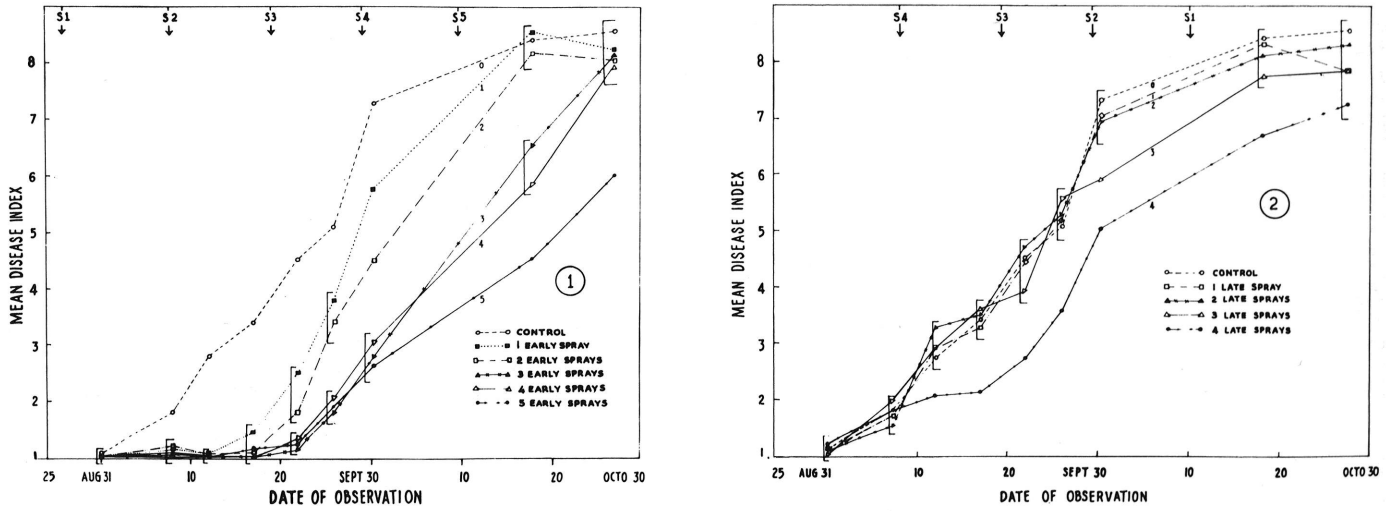
DISCUSSION

Our results indicated that the systemic fungicide thiophanate can protect watermelon plants against anthracnose for about 20 days after the application. Protection by thiophanate was temporary,

TABLE 1. Effect of thiophanate spray schedules on yield of watermelon fruits and yield loss under different anthracnose intensities

Treatments			Fruit yield kg/plot[x]			Yield loss expressed as % of four late sprays
Number of sprays	Spray applications, days after sowing[z]	Total Thiophanate kg/ha	Marketable[y]	Diseased	Total	
Unsprayed control	Control	0.000	2.48 cd	1.07 bc	3.55 c	50
5	30-74	3.330	4.76 b	0.25 a	5.01 b	30
4	30-63	2.664	5.32 ab	0.30 a	5.62 b	21
3	30-53	1.998	4.75 b	0.22 a	4.97 b	30
2	30-42	1.332	4.94 ab	0.77 abc	5.71 b	19
1	30	0.666	2.37 c	0.44 ab	2.81 c	60
4	42-74	2.664	5.75 a	1.31 c	7.06 a	00
3	53-74	1.998	3.01 c	0.60 abc	3.67 c	48
2	63-74	1.332	2.02 d	0.66 abc	2.68 c	63
1	74	0.666	1.96 d	0.92 abc	2.88 c	60
CD 5%			0.82	0.70	1.24	

[x]The treatment means followed by the same letters are not significantly different, $P = 0.05$.
[y]Marketable fruits were not infected. Fruit with a single visible lesion was considered as diseased.
[z]Sprays were applied at the interval of 10–12 days.



Figs. 1–2. Progress of anthracnose on watermelon plants. Seeds were sown on 29 July 1977. Fungicide was not applied on control plots. **1,** One, two, three, four, and five early applications of thiophanate were applied 30 days after sowing (DAS), 30–42 DAS, 30–53 DAS, 30–63 DAS, and 30–74 DAS, respectively. **2,** Four, three, two, and one late applications of thiophanate were applied between 42–74 DAS, 53–74 DAS, 63–74 DAS, and on 74 DAS, respectively. Nine disease observations were recorded between 34–91 DAS. Mean disease indices of treatments within the brackets are not significantly different, $P = 0.05$.

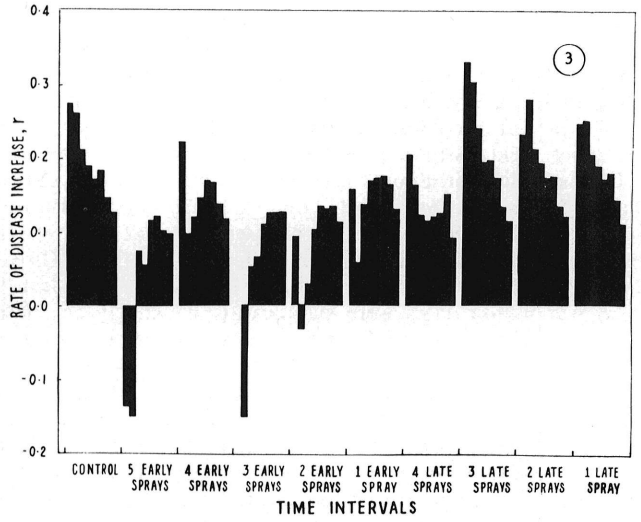


Fig. 3. Rate of watermelon anthracnose increase (apparent infection rate) during eight intervals between 34 to 91 days after sowing in unsprayed control plots, and in plots sprayed early or late with thiophanate. Each bar represents infection rate for one time interval as shown in Figs. 1 and 2.

and it did not eradicate the pathogen from established infections in plants.

Thiophanate was effective when applied immediately after the early appearance of the disease. Berger (3) postulated that fungicide sprays applied early in the epidemic were more effective than those applied later. Our results corroborated these findings.

Vanderplank (12) calls the apparent infection rate, r, the "speedometer" of the epidemic. Negative and decreased infection rates were observed in sprayed plots because thiophanate delayed further development of anthracnose while newly emerged leaves increased the proportion of healthy tissue. Strandberg and White (10) obtained negative infection rates of *Cercospora apii* in celery due to new growth and slower disease development during the cool, dry conditions in the winter months. Sasaki and Kato (8) observed that the number of susceptible and healthy host plants exposed to rice panicle blast decreased with time and became the principal factor limiting the rate of increase of disease. Our results showed that infection rates were decreased when uninfected plant parts decreased with increase in disease intensity with time. Yield loss was greater in early infected plots where thiophanate applications were delayed than late infected plots where applications of thiophanate were early. James et al (4) reported that early infection of late blight caused more yield loss in potato than late infection. The quantity of diseased watermelon fruits increased and total fruit weight decreased with the increase in disease intensity. Maximum correlation was observed with disease intensity during the flowering to the fruit formation period.

TABLE 2. Correlation coefficients between the anthracnose disease indices recorded on nine different days after sowing (DAS) with weight of diseased watermelon fruits and total weight of fruits

Disease observations DAS	Correlation coefficients[a]	
	Diseased fruits	Total fruits
34	0.0121 NS	0.0319 NS
42	0.5924 **	−0.1384 NS
46	0.3501 *	−0.4313 **
51	0.3829 *	−0.4588 **
56	0.3493 *	−0.5414 **
60	0.3275 *	−0.5279 **
65	0.3746 *	−0.5279 **
82	0.2243 NS	−0.4424 **
91	0.0192 NS	−0.3051 NS

[a]NS = not significant ($P > 0.05$). Double asterisk (**) or single asterisk (*) indicate correlation coefficients significant at $P = 0.01$ or $P = 0.05$, respectively.

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