Mummy Berry Disease of Highbush Blueberry: Epidemiology and Control

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ABSTRACT

A Burkard recording volumetric spore trap and rainwater spore trap were operated in a highbush blueberry field in Michigan from 3 April to 11 June 1973. Ascospores of *Monilinia vaccinii-corymbosi*, incitant of mummy berry disease, were trapped from air beginning 3 April, prior to bud break, until 8 May, when bushes were at 5% pink bud prebloom stage and leaves averaged 21 mm in length. The peak ascospore count of 290 occurred on 19 April.

Conidia were first trapped from air on 7 May, 3 days prior to the appearance of leaf and shoot infections caused by ascospores. The peak conidial count of 809 from air occurred on 9 May, and conidia were caught in diminishing numbers through 11 June, when bushes were at 100% petal fall stage. Rain water collections of conidia in bushes occurred from 16 May to 11 June with peak counts of 857 conidia/ml occurring 30 May to 4 June, when bushes were at mid-petal-fall stage.

Early sprays of triforine but not benomyl applied on 17 and 25 April (new leaves 1-2 mm and 10 mm long, respectively) were effective in controlling primary infection by ascospores. Late sprays of both triforine or benomyl applied on 11, 21, and 31 May (10% pink bud prebloom, 14% bloom and 13% petal-fall stages, respectively) were effective in controlling secondary infection from conidia. There were no yield differences between treatments and control due to a relatively low level of inoculum in the test field and variation in bush size.

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An epidemiological study of the effects of various weather factors upon spore release by *Monilinia vaccinii-corymbosi* (Reade) Honey relative to highbush blueberry phenology has been reported (10). Even though eradicant methods of control are commercially used against the apothecial stage of the mummy berry pathogen (3), we have found by using trap plants that windborne ascospores are capable of traveling at least 304.8 m (1,000 ft) from a noneradicated source field (Ramsdell, Nelson, and Myers, *unpublished*). In addition, the role of honeybees has been shown (5) in the spread of conidia from ascospore infections of leaves and shoots to blossoms. Therefore, development of a well-timed protectant fungicide program directed against both primary infection from ascospores and secondary infection from conidia is necessary.

This report relates daily spore levels, host development, weather conditions pertinent to infection and protective fungicide spray timing relative to mummy berry disease control. Similar work has been done with Northern leaf blight of corn (1), Dothistroma needle blight of pine (8) and black knot of plum (11).

MATERIALS AND METHODS.—A Burkard 7-day recording volumetric spore trap was operated in a *Vaccinium corymbosum* L. 'Jersey' highbush blueberry field from 3 April to 11 June 1973 as previously described (10). Conidia were also trapped in rainwater collected within bushes using a plastic funnel, tubing and jug method (2). Relative humidity (RH), temp, leaf wetness, rainfall, bush phenology and apothecial densities were measured as previously detailed (10). Ascospore and conidial counts were done as before (10), but expressed in terms of total daily counts/24 hour day from midnight to midnight, rather than on an hourly basis.

In order to establish some precision for the timing of protectant fungicide treatments, bushes were sprayed at various stages of development, relative to spore trap and weather activity. Two early sprays were applied on 17 April and 25 April, when vegetative leaf buds were pushed to 1 to 2 and 10 mm lengths, respectively. The early sprays were aimed at controlling primary ascospore infection of leaf and shoot tissue. Three late sprays were applied on 11, 21, and 31 May at 10% pink bud prebloom, 14% bloom, and 13% petal-fall stages, respectively. The late sprays were timed to control secondary conidial infection of blossoms. Early plus late applications were also included.

The fungicides used were triforine [N, N'- 1,4-Piperazinediylbis (2,2,2-trichloroethylidene) bisformamide] 20% w/v emulsion concentrate (EC) at 1.78 and 2.34 liter/ha (24 and 32 fl oz/acre) and benomyl 50% wettable powder [methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate] at 2.24 kg/ha (2 lb/acre).

A tractor-mounted airblast sprayer (Tecnoma Co., Epernay, France) was calibrated to spray 187.1 liters/ha (20 gal water/acre) at 14.06 kg force/cm^2 (200 psi) and 5.76 km/hour (3.6 mph) ground speed. Treatments of six mature Jersey cv. bushes were replicated five times in a completely randomized design (4).

Primary infection levels were estimated on 24 May as previously described (10). Secondary infection was measured on 14 and 28 August by hand harvesting bushes and determining percent infected berries.

RESULTS.—*Spore trap counts relative to weather activity and host phenology.*—Ascospores were first caught on 3 April when bushes were at the bud-swell

stage, but prior to the appearance of noncuticularized susceptible leaf bud tissue (Fig. 1-B). Ascospores were caught daily until 8 May, except during a snowfall on 10 April, when leaves were well developed and blossom buds were at 5% pink-bud prebloom stage. The first susceptible green vegetative tissue appeared on 17 April when vegetative buds were showing 1-2 mm green tip. Peak ascospore counts occurred on 19 April when 290 were caught during the 24-hour period. The ascospore liberation period lasted for 36 days. Apothecial densities

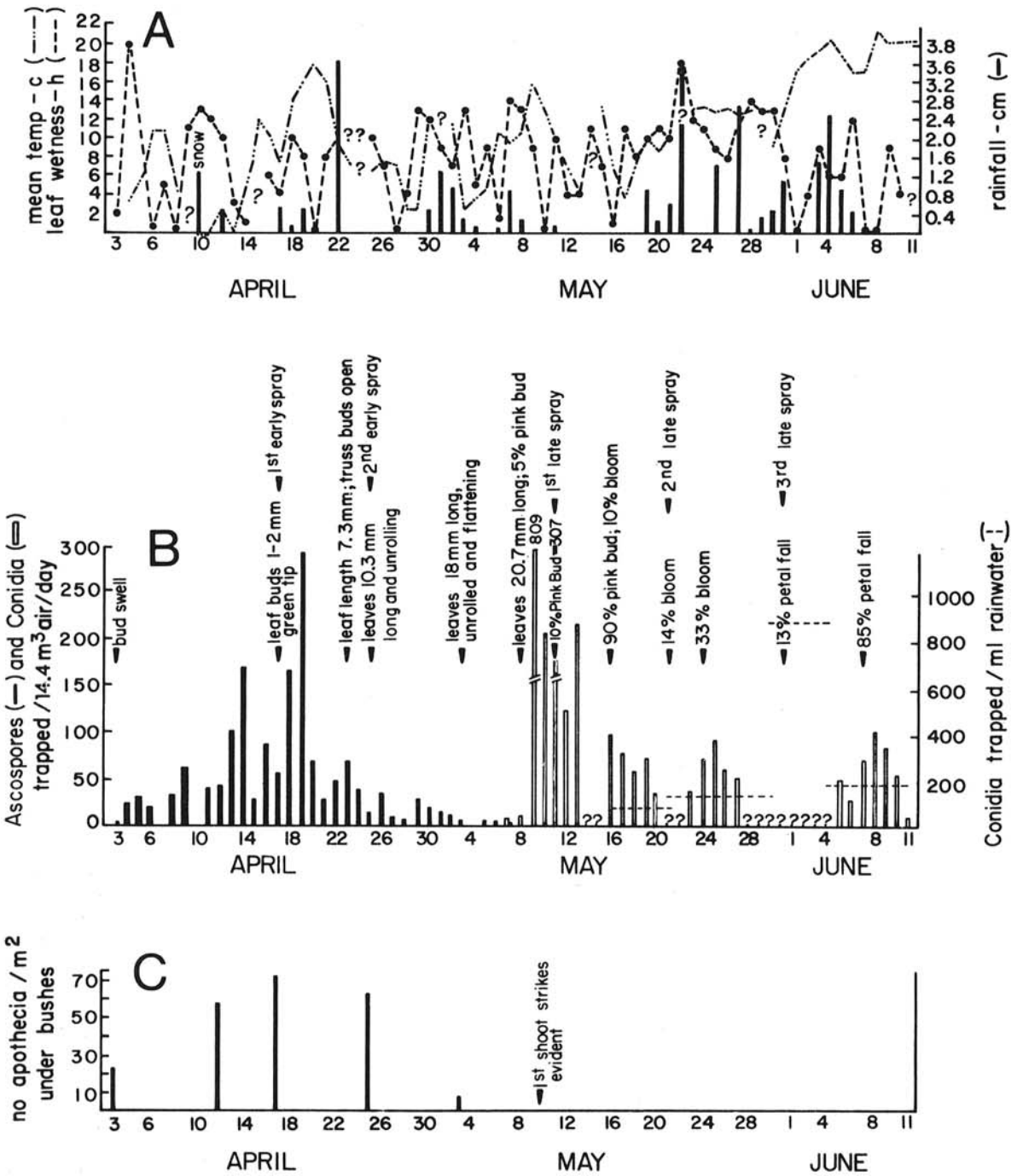

Fig. 1-(A to C). Mummy berry disease epidemiology and control data from a 'Jersey' cv. highbush blueberry field, Pullman, Michigan, 1973. Question marks (?) indicate periods of missing data due to equipment malfunction. **A)** Daily recorded mean temperature, hours of continuous leaf wetness and rainfall-cm. **B)** Daily ascospore and conidia counts, bush phenology and protectant fungicide spray dates. **C)** Apothecial development and date of appearance of primary infection symptoms ('shoot strikes').

(Fig. 1-C) closely paralleled ascospore counts. During the period of susceptibility to primary infection there were eleven 24-hour periods of continuous leaf wetness at 5 C or above for 6 hours or more (Fig. 1-A). Previous research (10) has demonstrated that ascospores germinate in 6 hours in free water at 5 to 20 C. Thus, numerous chances for primary infection occurred. A total of 4.56 cm of rain fell during this period and about half of the leaf wetness periods were caused by dew.

The first leaf and shoot infections ('shoot strikes') were evident on 10 May, 24 days after the first susceptible tissue was exposed. The first conidial catches occurred on 7 May when bushes were at 5% pink-bud prebloom (Fig. 1-B). The peak conidial catch occurred on 9 May when 809 conidia were caught by the volumetric air trap. Conidial catches occurred daily in diminishing numbers through 11 June, when bushes were at 100% petal fall. Rainwater traps placed in bushes 16 May through 11 June and collected at about weekly intervals yielded from 65-857 conidia/ml with the highest number caught during the period of 30 May to 4 June when bushes were at mid-petal fall (Fig. 1-B). The total conidial release period lasted for at least 35 days.

Preliminary conidial infection studies indicate that blossom infection can occur within 12 hours in temperatures as low as 5 C (Ramsdell, Nelson, and Myers, *unpublished*). There were eight 12-hour periods of continuous leaf wetness accompanied by temperatures above 9.5 C when the bushes were susceptible to conidial infection (Fig. 1-A). A total of 9.12 cm of rain fell during this latter period and accounted for about 40% of these leaf wetness periods.

Disease control as affected by fungicide spray timing.—Benlate 50W at 2.24 kg/ha (2 lb/acre) gave no control of primary infection caused by ascospores (Table 1). However, early sprays of triforine 20 EC at 1.78 and 2.34 liter/ha (24 and 32 fl oz/acre) resulted in 92 and 98% control of primary infection, respectively (Table 1). Late triforine sprays applied for the first time on 11 May, when leaves were 21 mm long failed to control primary infection.

Early sprays of triforine or Benlate failed to control secondary infection as measured by percent mummy berries at harvest (Table 1). Both late and early plus late sprays of triforine 20 EC at 1.78 and 2.34 liter/ha (24 and 32 fl oz/acre) gave 87 and 93% reduction of secondary infection, respectively. Both late, and early plus late, sprays of Benlate 50W at 2.24 kg/ha (2 lb/acre) gave 93 and 87% control of secondary infection, respectively, for these two treatment timings.

Yield data taken at harvest showed no significant differences between treatments. This was due to relatively low inoculum levels and size variation between bushes in the test field.

DISCUSSION.—Ascospore liberation began very early relative to bush phenology during the 1973 season. Spores were liberated for 14 days prior to the first appearance of susceptible tissue. This was due to abnormally warm spring temperatures during these two weeks. During the 1972 season, there was a period of 8 days between the last ascospore catch and the first conidial catch; whereas, during the 1973 season, there was a slight overlap between these two events. Cool, wet weather extended the period of apothecial production in 1973. Although wind is very important in the dissemination of conidia (10), the water trap counts indicate that rain and free water in bushes can effectively spread conidia within the bush from primary infections to cause secondary infection of blossoms, resulting in mummy berries.

Early timing of fungicide application is important for the prevention of primary infection. The first spray should be timed to coincide with the appearance of green tissue protruding from vegetative buds. A second spray should follow in 7 to 10 days to cover new leaf tissue. A short-term residue study was conducted in blueberries during 1972 with triforine 20 EC. Residue analyses were done by the Pesticide Residue Laboratory (N.Y. Agric.

TABLE 1. Timing of protectant fungicide application for mummy berry disease control in 'Jersey' cv. highbush blueberry, Pullman, Michigan, 1973[a]

Chemical	Rate[b]	Time of application[c]	Leaf and shoot infections/bush[d]	Control (%)	Mummy berries[e] (%)	Control (%)
Nontreated			66.5		1.5	
Triforine 20EC	24 fl oz	early	5.3 y	92	0.9	
Triforine 20EC	24 fl oz	late	84.9		0.2 z	87
Triforine 20EC	24 fl oz	early + late	5.0 y	93	0.2 z	87
Triforine 20EC	32 fl oz	early	1.3 y	98	0.3	
Triforine 20EC	32 fl oz	late	57.0		0.1 z	93
Triforine 20EC	32 fl oz	early + late	1.4 y	98	0.1 z	93
Benlate 50W	2.0 lb	early	58.0		1.1	
Benlate 50W	2.0 lb	late	69.9		0.1 z	93
Benlate 50W	2.0 lb	early + late	42.5		0.2 z	87

[a]Six mature bushes per replication; five replications per treatment in a completely randomized design.
[b]Sprays applied in 187.1 liters/ha (20 gal water/acre) with a small air-blast sprayer.
[c]Early spray = 17 Apr 1973 (1-2 mm green tip) and 25 Apr 1973 (10 mm green tip); late sprays = 11 May 1973 (10% pink bud), 21 May 1973 (14% bloom) and 31 May 1973 (13% petal fall).
[d]Numbers followed by letter y are significantly different from nontreated [LSD($P = 0.01$) = 44.6]. Infections determined 24 May 1973.
[e]Numbers followed by letter z are significantly different from nontreated [LSD($P = 0.05$) = 1.3]. Percent mummy berries determined at harvest, 4 Aug and 28 Aug 1973.

Exp. Stn., Geneva, N.Y.). It was found that triforine has a short residual life in the field; 7 to 10 days is about the maximum effective protectant period in the field (Ramsdell, Nelson, and Myers, *unpublished*). Assuming that our last late application protected for 10 days, or until 5 May, the last protection against ascospore infection was when the leaves were about 20 mm long, unrolled and flattening out.

Both triforine and Benlate as late sprays effectively controlled secondary infection, even though conidial inoculum was present throughout the blossom period. It is interesting to note that while Benlate was very effective against conidial infection, it was totally ineffective against ascospore infection. This phenomenon was previously reported by us (9) and Pepin (6). Pepin (6) has recently reported that triforine only controls ascospore infections, and is ineffective against conidia. This is in contrast with our data presented here. It is important to time the first spray for prevention of secondary infection of blossoms prior to the first blossom opening and then to apply subsequent sprays to ensure active fungicidal residues through petal fall.

Although yields were not significantly decreased by disease in this study, Pepin and Toms (7) have shown 8.14% total crop loss in blueberries with substantial loss of yield due to blossom cluster kill resulting from primary infection. Therefore, to avoid possible yield losses, it appears essential to control both primary and secondary infection.

This research has shown that good control of both stages of the mummy berry disease is achievable with well timed protectant sprays applied at low gallonages per acre with a commercially available air blast sprayer. No apothecial eradicant chemicals were applied prior to the protectant sprays in this study. Such a combination of treatments would be the most effective method of control when high initial inoculum levels exist.

LITERATURE CITED

1. BERGER, R. D. 1973. Helminthosporium turcicum lesion numbers related to numbers of trapped spores and fungicide sprays. Phytopathology 63:930-933.
2. DUBIN, H. J. 1972. Epidemiology and factors affecting fungicidal control of European apple canker. Ph.D. Thesis. Univ. of Calif., Davis. 82 p.
3. FULTON, R. H. 1958. Controlling mummy berry disease of blueberries by soil treatment. Mich. Agric. Exp. Stn. Quart. Bull. 40:491-497.
4. LITTLE, T. M., and F. J. HILLS. 1972. The completely randomized design. Pages 37-40 *in* Statistical methods in agricultural research. Agric. Ext. Publ. AXT-377, University of California, Berkeley, 242 p.
5. NELSON, J. W., and H. C. BITTENBENDER. 1971. Mummy berry disease occurrence in a blueberry selection test planting. Plant Dis. Rep. 55:651-653.
6. PEPIN, H.S. 1974. Mummy berry of highbush blueberry: A control dilemma. Can. Agric. 19:34-35.
7. PEPIN, H. S., and H. N. W. TOMS. 1969. Economic losses from mummy berry of highbush blueberry in coastal British Columbia. Can. Plant Dis. Surv. 49:105-107.
8. PETERSON, G. W. 1967. Dothistroma needle blight of Austrian and Ponderosa pines: Epidemiology and control. Phytopathology 57:437-441.
9. RAMSDELL, D. C., J. W. NELSON, and R. MYERS. 1973. Small fruit disease reports. Page 42. *in* E. I. Zehr, et al., eds. Fungicide and nematicide test results of 1973. Vol. 29. Amer. Phytopathol. Soc., St. Paul, Minnesota. 208 p.
10. RAMSDELL, D. C., J. W. NELSON, and R. MYERS. 1974. An epidemiological study of mummy berry disease of highbush blueberry. Phytopathology 64:222-228.
11. SMITH, D. H., F. H. LEWIS, and S. H. WAINWRIGHT. 1970. Epidemiology of the black knot disease of plums. Phytopathology 60:1441-1444.