Epidemiological Studies of Blueberry Anthracnose Disease Caused by *Colletotrichum gloeosporioides*

J. S. Hartung, C. L. Burton, and D. C. Ramsdell

Graduate research assistant, research plant pathologist SEA-AR, U.S. Department of Agriculture; and professor, Department of Botany and Plant Pathology, Michigan State University, East Lansing, MI 48824.

This research was supported in part by the Michigan Blueberry Growers Association, Grand Junction, MI 49056, and reserve funds from SEA/AR, USDA.

We thank Barbara J. Dyko for assistance in fungal identification and John W. Nelson and Linda Swain for technical assistance.

Michigan State Agricultural Experiment Station Journal Series Article 9412.

Mention of a trademark, proprietory product, or vendor, does not constitute a guarantee or warranty by the USDA and does not imply approval to the exclusion of the other products or vendors that also may be suitable.

Accepted for publication 23 September 1980.

ABSTRACT

Hartung, J. S., Burton, C. L., and Ramsdell, D. C. 1981. Epidemiological studies of blueberry anthracnose disease caused by *Colletotrichum gloeosporioides*. Phytopathology 71:449-453.

Epidemiological studies of blueberry anthracnose disease caused by *Colletotrichum gloeosporioides* were conducted in a commercial highbush blueberry field (cultivar Jersey) at Grand Junction, MI, in 1978 and 1979. Conidia of *C. gloeosporioides* were collected in rainwater runoff from diseased bushes from the bud swell through the harvest stages of growth. Conidia were associated with diseased fruiting wood, blossoms, and rotting fruit. Maximum numbers of conidia appeared to coincide with probable natural fruit infection periods as predicted by in vitro conidial germination studies. Such incidents occurred from the green fruit through the harvest growth stages. Inoculation of swelling flower buds, blossoms, and immature fruit, both on mature branches of cultivar Jersey bushes in the field and on potted 3- to 4-yr-old bushes (cultivars Bluecrop and Berkeley), with 10^6 conidia per milliliter sterile distilled water, resulted in apparently healthy fruit that later developed a rapid anthracnose decay after harvest. Inoculations performed prior to and during bloom also caused a severe blossom blight. The pathogen overwintered in blighted fruiting wood which it entered via blighted blossom clusters or, perhaps, rotting fruit pedicels.

Additional key words: Vaccinium corymbosum, Gloeosporium.

In recent years anthracnose decay of blueberry fruit has become a major problem for commercial highbush blueberry (*Vaccinium corymbosm* L.) production in New Jersey (3–5), particularly on fruits harvested late in the season (3). Anthracnose decays also are of great concern to the Michigan blueberry industry. In early studies of anthracnose decay of blueberries, the causal fungus was reported to be *Gloeosporium fructigenum* (3,5,8) which is the imperfect form of *Glomerella cingulata* (Stonem.) Spaulding and Schrenk. According to von Arx (9) *G. fructigenum* is synonymous with *Colletotrichum gloeosporioides* (Penz.) Sacc. In this study we follow the nomenclature of von Arx (9) and of Mordue (6) and refer to the blueberry anthracnose pathogen as *C. gloeosporioides*.

To develop appropriate control strategies we determined the relationship between highbush blueberry phenology and infection by *C. gloeosporioides*. Spore trapping studies were done to determine when, and in what relative numbers, conidia of *C. gloeosporioides* were produced in the field.

MATERIALS AND METHODS

Characterization of *C. gloeosporioides*. A single conidial isolate of *C. gloeosporioides*, obtained from rotting blueberry fruit in 1977, was used for studies of inoculation, mycelial growth, and conidial germination. When grown on potato-dextrose agar (PDA) (Difco Products Co., Detroit, MI 48201) the fungus produced large amounts of pinkish-purple pigment in the medium and numerous acervuli containing salmon-colored spores formed on the agar surface. Colony morphology and sporulation were unaffected by light. Individual conidia measured 19.0 ± 0.6 μm long by 7.8 ± 0.3 μm wide (mean ± standard deviation of measurements of 50 conidia) were cylindrical to slightly elliptical, hyaline, and had granular inclusions.

On rotting fruit the fungus was visible only as acervuli containing salmon colored spore masses on the surface of the fruit. Hyaline setae were present in acervuli on fruit but not in acervuli on PDA.

Effect of temperature on mycelial growth. Disks of *C. gloeosporioides* mycelium (7 mm diameter) were cut from the margin of an actively growing culture and transferred to the center of PDA plates. Five replicate plates were maintained in continual darkness at 5, 10, 15, 20, 27, or 30 C and radial growth of the fungus was measured after 7 days. This information was used to estimate the temperature range at which fruit infection would probably be most rapid.

Effects of high relative humidity and free water at various temperatures on germination of conidia. Experiments were carried out to define the probable temperature and moisture conditions required for fruit infection in the field. Conidial germination in free water was studied by placing spore suspensions in deep well depression slides. The suspensions were prepared by flooding 6-day-old PDA cultures with sterile distilled water, rubbing the plates with a bent glass rod, and decanting the resulting conidial suspension through four layers of cheesecloth. The depression slides were placed in petri plates lined with wet paper towels and incubated at 5, 10, 15, 20, or 30 C in darkness. Percent germination was determined and germ tube elongation was measured at the meniscus of the suspensions. The first 100 conidia encountered in a ×100 microscope field in each of four replicates were recorded as germinated or ungerminated and the first 25 germ tubes encountered were measured with an ocular micrometer after 8, 16, 24, 32, and 40 hr. A spore with a germ tube the length of one dimension of the spore was considered as germinating.

Effects of temperature on conidial germination at high relative humidity (RH) without free water were determined at the same temperatures and time intervals. Conidial suspensions were sprayed onto glass cover slips by using a DeVilbiss No. 15 atomizer. The cover slips were allowed to air-dry and were placed, two per glass slide, in petri plates lined with wet paper toweling. To prevent

0031-949X/81/04044905/$03.00/0

condensation water from interfering with the experiment, a sufficient number of cover slips was prepared so that each was removed from its incubator and observed only once during the course of the experiment.

Conidial inoculation studies. Inoculation studies with the isolate obtained from rotting fruit in 1977 were carried out in the field in 1978 and 1979 to satisfy Koch's postulates and to determine which (if any) phenologic growth stage of the blueberry bush was most susceptible to infection by *C. gloeosporioides*. Six rows of 18 bushes each located on ~0.06 ha in a corner of a 16-ha (40-acre) mature commercial field of highbush blueberry (cultivar Jersey) located at Grand Junction, MI, were selected for field inoculation studies.

Conidial suspensions were prepared from 4- to 8-day-old PDA cultures as described above. Conidial concentrations in sterile distilled water were adjusted to 10^6/ml by using a hemacytometer (Arthur H. Thomas Co., Philadelphia, PA 19106). Inoculations of individual unwounded branches of selected bushes in the commercial field site were made by spraying plant surfaces with the spore suspension until runoff occurred using a DeVilbiss No. 15 atomizer. Branches selected for inoculation had no blighted wood present on the inoculation date. A wet paper towel was then placed on the inoculated branch and the branch was sealed in a 50 × 100 cm polyethylene bag for 18–24 hr to maintain free water on the plant surface during the infection process.

Identical experiments were carried out at East Lansing, MI, on 4-yr-old bushes (cultivar Berkeley). Because the in vitro studies showed that the *C. gloeosporioides* isolates being used grew poorly and their conidia germinated poorly at temperatures above 27 C, the potted bushes at East Lansing were held in a growth chamber at 22 C (Sherer-Gillette, Co., Marshall, MI 49068) for 24 hr after inoculation.

In each season and at each location a single branch on each of four bushes was inoculated with *C. gloeosporioides* or with sterile distilled water on each inoculation date in a completely randomized design. Inoculations were made at intervals starting at the bud swell phenologic stage of plant development and ending at the blue fruit stage (Tables 2–4).

At harvesttime separate disposable polyethylene gloves were used to handpick the fruit from each inoculated and control bush replicate and to place the fruit in separate 0.5-L polyethylene bags. The few fruits that had visibly sporulating *C. gloeosporioides* infections were discarded to prevent contamination.

Fruit was then incubated over water on a wire mesh screen contained in a plastic bucket. The tops of the buckets were sealed with polyethylene to insure high RH. Fruit was scored as rotted by *C. gloeosporioides* after 4–6 days if the characteristic salmon colored spore masses were present on the fruit surface.

Isolation of *C. gloeosporioides* from blighted fruiting wood. In March 1978, blighted fruiting wood from the commercial field location was brought to the laboratory for isolation of *C. gloeosporioides*. The blighted twigs were surface disinfested 10 min in 1% sodium hypochlorite containing a few drops of Tween-80 surfactant (= ~0.1%, v/v). Twigs were rinsed with sterile distilled water, four chips of blighted wood from each twig were transferred aseptically to PDA plates, and after 6–8 days the resulting fungal cultures were identified. In the spring of 1979, twigs from 10 bushes that had been inoculated in 1978 with *C. gloeosporioides* were surface disinfested and isolations were made in the same manner.

Trapping conidia from rainwater. On 28 April 1978, 10 water traps were placed in bushes that had an abundance of dead fruiting wood similar to that from which *C. gloeosporioides* had been isolated. A water trap consisted of 15-cm (= 6-inch)-diameter funnel anchored in the bush and connected by plastic tubing to a 3.79-L (1-gallon) bottle, buried in the ground, that collected the conidia washed from the blighted tissue by rainwater. In early July 1978, three additional water traps were placed in diseased bushes that previously had been inoculated with *C. gloeosporioides*.

In April 1979 a water trap was placed in each of eight bushes that had been inoculated with *C. gloeosporioides* in 1978; the pathogen had been reisolated from six of these bushes. To enhance spore trapping consistency in 1979, the water traps were "baited" with diseased twigs, blossoms, and berries wrapped in cheesecloth and placed in the funnel orifice as the season progressed.

In each season 25 ml of a preservative solution containing ethanol, glacial acetic acid, formaldehyde, and water (50:5:10:35, v/v) was added to collection vessels weekly to prevent spore germination. Experiments showed that conidia did not germinate in this solution even when diluted 15:1.

The bottles were emptied weekly in 1978 and until 27 June 1979 after which time the bottles were emptied after individual rains. Conidial trapping was discontinued at harvesttime in both seasons. Conidial concentration in each bottle was estimated by centrifuging a 40-ml sample from each jug at 7,000 *g* for 20 min and resuspending the pellet in 10 ml of water. Three replicate counts of each sample were made by using a hemacytometer.

Monitoring of environmental conditions in the commercial field. Environmental parameters were monitored in the field in which the inoculation and spore trapping studies were carried out in order to detect probable natural infection periods. Rainfall was recorded by using a tipping bucket rain gauge accurate to 0.25 mm (0.01 inch) (Weather Measure Co., Sacramento, CA 95841). Duration of free water on leaf surfaces was recorded by using a leaf wetness meter (M. DeWitt Co., Hengelo, The Netherlands). Air temperature and RH was recorded by using a sheltered hygrothermograph 1.5 m above the ground (Belfort Instrument Co., Belfort, MD 21224). All instruments were 7-day continuously recording types and were installed within the rows of the blueberry bushes. By comparing the rain gauge and leaf wetness charts, wetting periods were attributable to rain or dew.

RESULTS

Effect of temperature on mycelial radial growth. The optimal temperature for mycelial growth of the isolate tested was 20 C, with slightly less growth at 27 C (Fig. 1). Almost no growth occurred at 30 C or at 5–10 C.

Effects of high relative humidity and free water at various temperatures on germination of conidia. The conidia of the isolate used required free water and temperatures above 10 C to germinate within 40 hr (Table 1). Germination was most rapid at 30 C; however, germ tube elongation was more rapid at 20 than at 30 C (Table 1). From these results we suggest that *C. gloeosporioides* conidia probably required a prolonged period in free water at 15–30

TABLE 1. Effect of temperature on conidial germination of *Colletotrichum gloeosporioides* in distilled water on glass slides

Temperature (C)	Percent germination and length of germ tubes (μm)									
	8 hr		16 hr		24 hr		32 hr		40 hr	
	(%)[a]	(μm)[b]	(%)	(μm)	(%)	(μm)	(%)	(μm)	(%)	(μm)
5	0.0	···	0.0	···	0.0	···	0.0	···	0.0	···
10	0.0	···	0.0	···	0.0	···	0.0	···	0.0	···
15	1.3 ± 1[c]	···	25 ± 10	13 ± 7	22 ± 7	16 ± 9	37 ± 9	22 ± 12	49 ± 3	39 ± 36
20	0.0	···	33 ± 9	49 ± 34	43 ± 14	99 ± 41	38 ± 8	100 ± 42	46 ± 13	99 ± 68
30	16 ± 4	···	50 ± 9	24 ± 14	59 ± 9	32 ± 9	56 ± 23	29 ± 13	65 ± 19	26 ± 11

[a] Percent germination of first 100 conidia encountered in microscope field.
[b] Germ tube length in micrometers (μm) (mean of 25).
[c] Mean of four replicates plus and minus (±) the standard deviations of the means.

C following rain dispersal to germinate and infect fruit.

Conidial inoculation studies. The isolate of *C. gloeosporioides* used in this study caused postharvest fruit decay (Tables 2 and 3) when inoculations were made in the commercial field during the growing season. The fungus reisolated from the decayed fruit was indistinguishable morphologically from the isolate used to inoculate the bushes. Blueberry bushes did not differ in susceptibility to *C. gloeosporioides* throughout the growing season as shown by Duncan's multiple range test (DMRT, $P = 0.05$). Postharvest decay occurred on fruit harvested from bushes inoculated as early as the bud swell and bud break stages of growth.

The pathogen also caused a severe blossom blight when bushes were inoculated before or during bloom (Fig. 2A). Blossoms blighted by *C. gloeosporioides* produce numerous salmon colored spore masses, particularly if placed in a moist chamber (Fig. 2B). Conidia produced on such blighted flower clusters are undoubtedly important in the epidemiology of postharvest decays caused by *C. gloeosporioides*, since the blighted flower clusters persist until harvesttime (Fig. 2C).

The results of the inoculation study done at E. Lansing in 1979 were similar to the results of the field studies of 1978 and 1979 even though a different cultivar was used (Table 4). The variability of the results was reduced by performing the inoculation in a controlled environment chamber. In this experiment inoculations late in the season produced significantly more postharvest decay than did early season inoculations (DMRT, $P = 0.05$). The data from the East Lansing inoculations of 1978 were unusable due to problems with insect and bird pests.

Trapping conidia from rainwater. Conidia were trapped throughout each season whenever it rained (Figs. 3 and 4), although the differences in experimental technique used in the two seasons were such that the data should not be compared quantitatively. The proportion of traps that consistently yielded *C. gloeosporioides* conidia was much increased in 1979 by relocating and "baiting" them.

Conidia were collected each year in the preblossom period, which suggests to us that the conidia were produced on the blighted fruiting wood resembling that from which the organism had been reisolated, since no other tissue was present in the bushes at these

TABLE 2. Postharvest rot of fruit from mature cultivar Jersey highbush blueberry bushes inoculated with *Colletotrichum gloeosporioides* at Grand Junction, MI, in 1978[a,b]

Inoculation		Mean percent fruit rotted by *C. gloeosporioides*[c]	
Growth stage	Date	Inoculated[d]	Uninoculated
Bud swell	2 May	16.4 ± 8.9 z	0.0
Bud break	16 May	18.8 ± 8.9 z	0.2 ± 0.16
Pink bud	23 May	16.6 ± 6.9 z	0.0
Full bloom	30 May	5.7 ± 2.5 z	3.0 ± 2.0
Early petal fall	6 June	8.3 ± 6.6 z	0.0
Early green fruit	13 June	31.1 ± 16.5 z	0.0
Green fruit	22 June	10.3 ± 2.3 z	0.5 ± 0.57
Early blue fruit	16 July	7.5 ± 3.7 z	0.0

[a] Bushes were inoculated with a spore suspension of 10^6 conidia per milliliter in sterile distilled water or with sterile distilled water only (uninoculated).
[b] Symptomless fruit were hand harvested on 1 August 1978 and held for 4 days at room temperature in moist chambers.
[c] Percent of fruit with visibly sporulating *C. gloeosporioides* on a count basis. Numbers given are the means of four replicates plus and minus (±) the standard deviations of the means.
[d] Means within columns followed by letters in common do not differ significantly according to Duncan's multiple range test ($P = 0.05$).

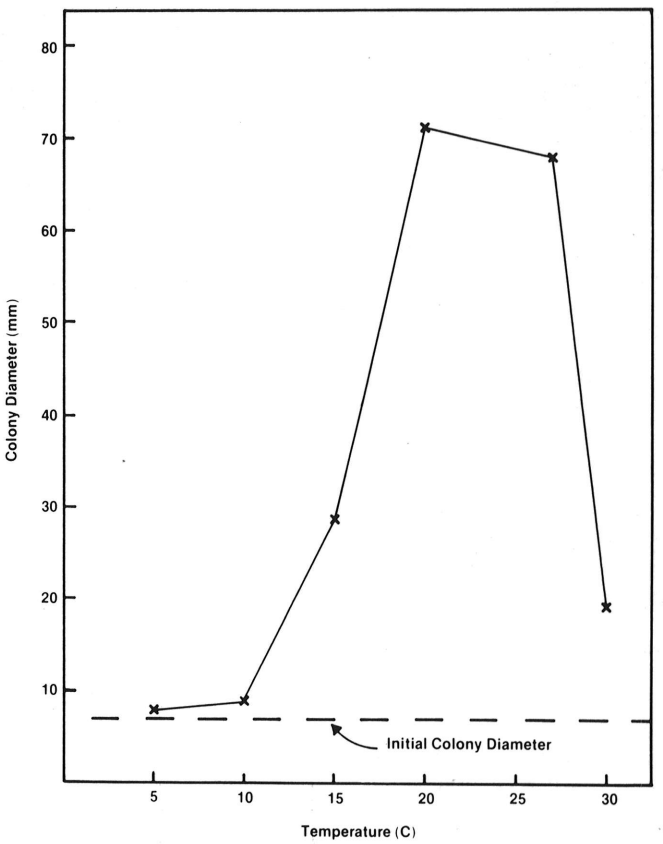

Fig. 1. Effect of temperature on mycelial growth of *Colletotrichum gloeosporioides* on potato-dextrose agar after 7 days.

TABLE 3. Postharvest rot of fruit from mature cultivar Jersey highbush blueberry bushes inoculated with *Colletotrichum gloeosporioides* at Grand Junction, MI, in 1979[a,b]

Inoculation		Mean percent fruit rotted by *C. gloeosporioides*[c]	
Growth stage	Date	Inoculated[d]	Uninoculated
Bud swell	2 May	23.3 ± 12.7 z	0.0
Pink bud	16 May	1.5 ± 0.6 z	8.5 ± 5.2
50% bloom	23 May	15.5 ± 10.7 z	14.5 ± 5.2
Early green fruit	13 June	19.3 ± 8.9 z	0.0
Late green fruit	5 July	32.8 ± 11.6 z	0.0
First blue fruit	18 July	13.8 ± 11.6 z	1.3 ± 0.6
First ripe fruit	1 August	25.1 ± 14.2 z	0.5 ± 0.2

[a] Bushes were inoculated with a spore suspension of 10^6 conidia per milliliter in sterile distilled water or with sterile distilled water only (uninoculated).
[b] Symptomless fruit were hand harvested on 15 July 1979 and held for 4 days at room temperature in moist chambers.
[c] Percent of fruit with visibly sporulating *C. gloeosporioides* on a count basis. Numbers given are the means of four replicates plus and minus (±) the standard deviations of the means.
[d] Means within columns with letters in common do not differ significantly according to Duncan's multiple range test ($P = 0.05$).

TABLE 4. Postharvest rot of fruit from potted 4-yr-old cultivar Berkeley highbush blueberry bushes inoculated with *Colletotrichum gloeosporioides* at East Lansing, MI, in 1979[a,b]

Inoculation		Mean percent fruit rotted by *C. gloeosporioides*[c]	
Growth stage	Date	Inoculated[d]	Uninoculated
Bud swell	28 Apr	5.1 ± 4.4 y	0.0
Bud break	10 May	2.8 ± 1.6 y	0.0
Early pink bud	17 May	0.0 y	0.0
Full bloom	24 May	7.1 ± 1.7 y	0.0
Green fruit	14 June	21.4 ± 5.2 z	0.0
First blue fruit	19 July	11.3 ± 7.3 yz	0.0

[a] Potted bushes were inoculated with a spore suspension of 10^6 conidia per milliliter in sterile distilled water or with sterile distilled water only (uninoculated).
[b] Symptomless fruit were hand harvested on 31 July 1979 and held for 6 days at room temperature in moist chambers.
[c] Percent of fruit with visibly sporulating *C. gloeosporioides* on a count basis. Numbers given are the means of four replicates plus and minus (±) the standard deviations of the means.
[d] Means within columns with letters in common do not differ significantly according to Duncan's multiple range test ($P = 0.05$).

times. Large numbers of conidia were collected during the bloom period in 1979, although there was no rain during this period in 1978 from which to trap conidia, and very large numbers of conidia were trapped during the green fruit stage of growth in each season. Conidial production was observed on blighted twigs, blossoms, and blue fruit in both seasons.

Isolation of *C. gloeosporioides* from blighted fruiting wood. *Colletotrichum gloeosporioides* was isolated from three of 36 (8.3%) of blighted twigs in 1978. Of 10 blighted twigs resulting from 1978 *C. gloeosporioides* inoculations, six yielded the pathogen in the spring of 1979, suggesting that such blighted twigs are a site of overwintering for the pathogens. The lower rate of pathogen recovery in 1978 may suggest that those twigs were killed by severe winter weather and not by *C. gloeosporioides.*

DISCUSSION

Colletotrichum gloeosporioides has been shown to cause latent infections on citrus, avocado, and papaya (1,2,7). The results of our study may show a similar phenomenon to be present in blueberry fruits since the inoculation of immature blueberry fruits, blossoms, and flower buds resulted in apparently healthy fruits that rapidly

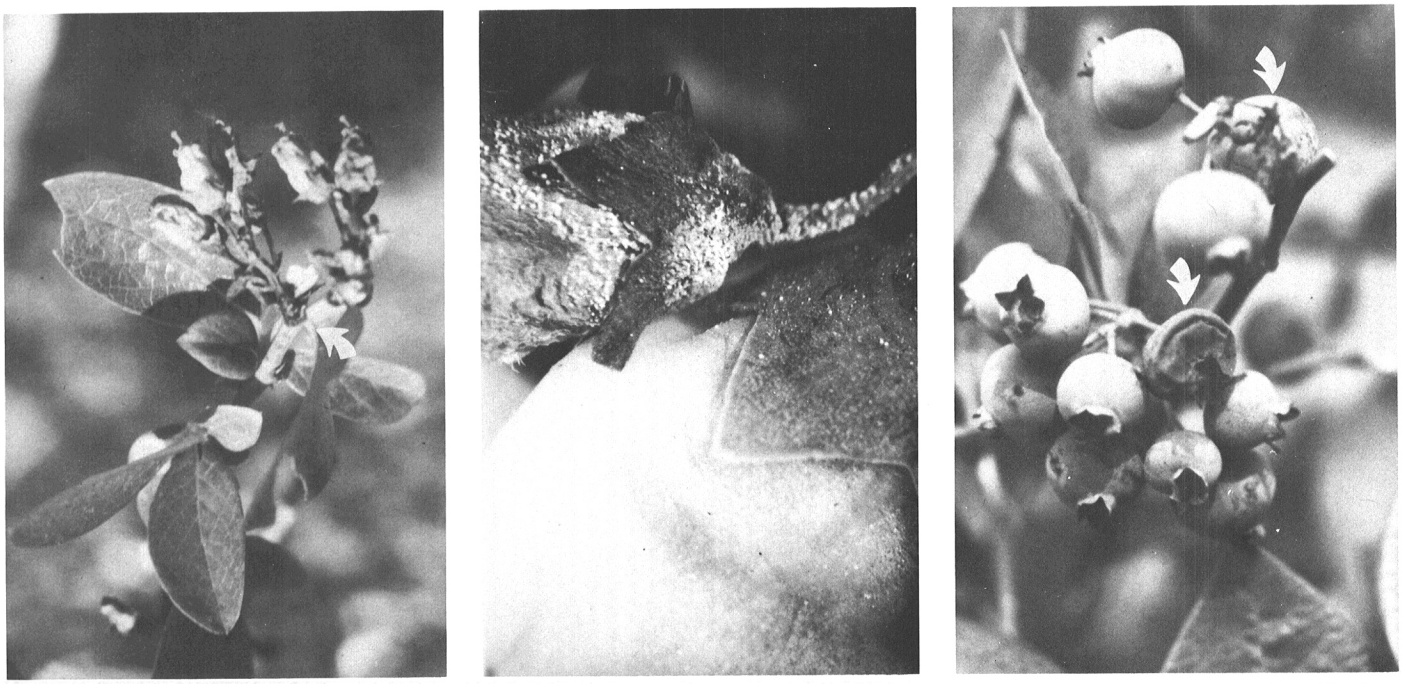

Fig. 2. Blueberry anthracnose caused by *Colletotrichum gloeosporioides.* **A,** Blossom infection entering fruiting wood. Arrow shows demarcation of healthy and diseased wood. **B,** Masses of conidia on surface of blighted blossom. **C,** Decay of ripening fruit in a cluster possibly initiated from conidia produced on a blighted blossom (arrow) persisting over a fruit cluster.

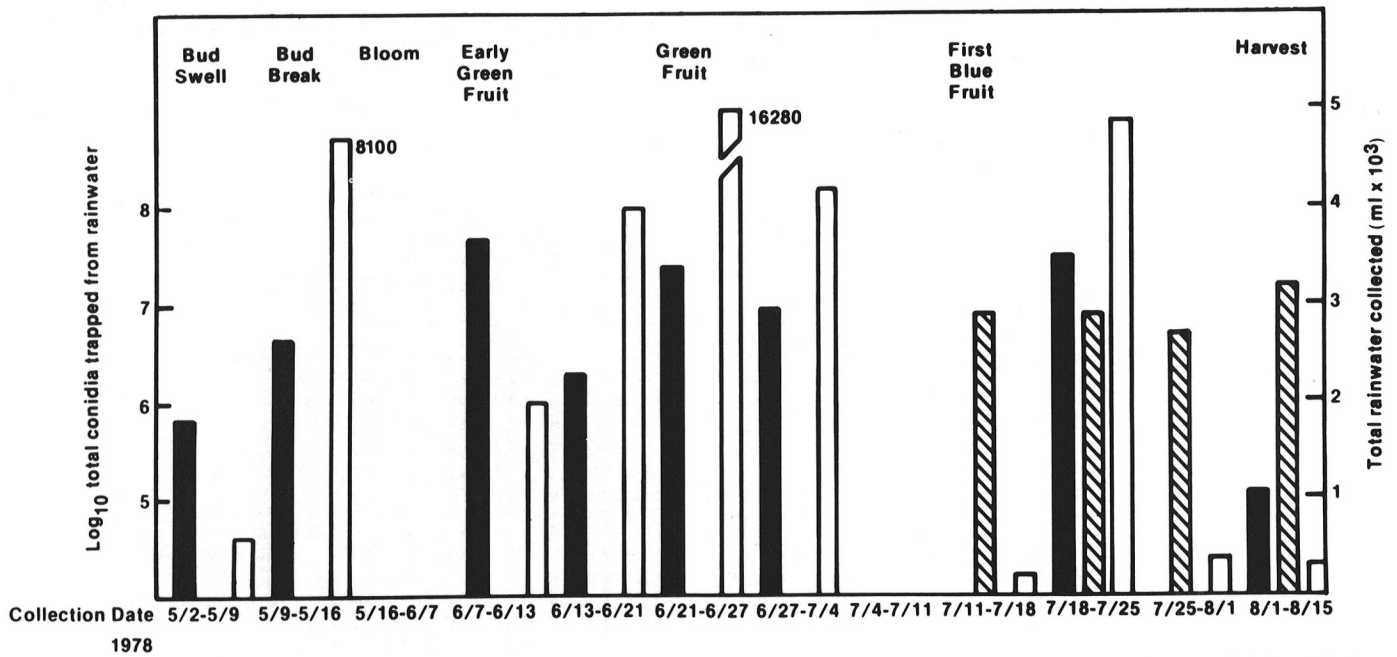

Fig. 3. Collection of *Colletotrichum gloeosporioides* conidia from commercial blueberry bushes (cultivar Jersey) at Grand Junction, MI, in 1978. Solid bars show the total number of conidia collected in 10 water traps placed in naturally infected bushes. Crosshatched bars show the total number of conidia collected in three traps placed in symptom-bearing bushes that had been inoculated earlier in the season. Open bars show the total rainwater collected in the traps. The time periods included in each collection are shown on the horizontal axis.

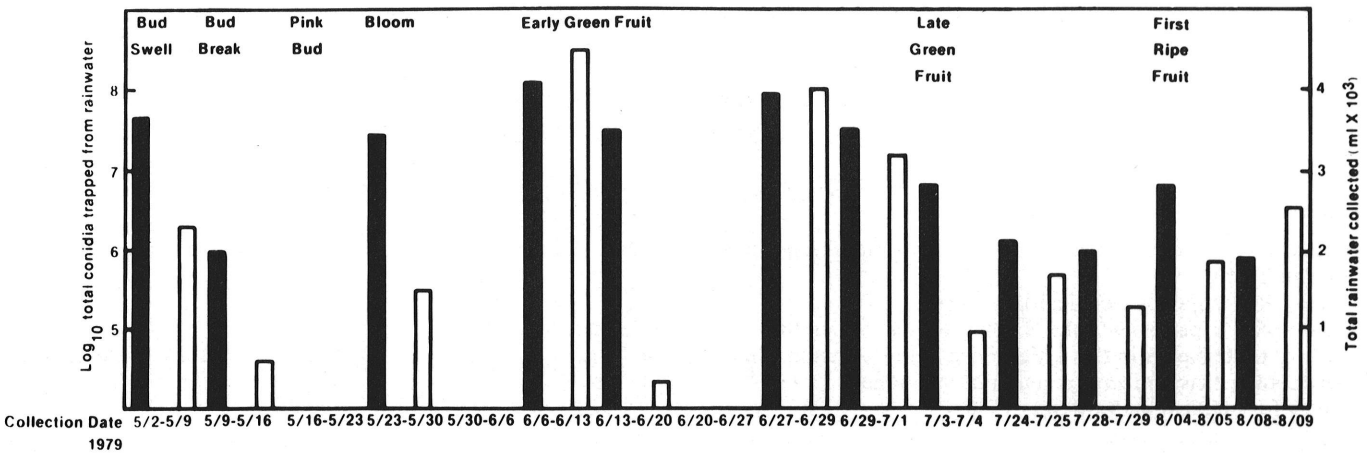

Fig. 4. Collection of *Colletotrichum gloeosporioides* conidia from commercial blueberry bushes (cultivar Jersey) at Grand Junction, MI, in 1979. Solid bars show the total number of conidia collected in eight water traps placed in bushes inoculated with *C. gloeosporioides* in 1979. The water traps were 'baited' by placing diseased twigs, flowers, and fruits in the funnel orifices as the season progressed. The open bars show the total rainwater collected in the traps. The time periods included in each collection are shown on the horizontal axis.

decayed after harvest.

Flower buds and blossoms inoculated with *C. gloeosporioides* produced apparently healthy fruit that rapidly decayed postharvest. It is not certain that all postharvest decay of fruit obtained from bushes inoculated before or during bloom was due to latent infections established on the date of inoculation. Secondary spread of the pathogen may have occurred (Fig. 2C), and the fruits were not surface disinfested at harvest to distinguish between latent infections and superficially borne conidia. However, inoculations after petal fall consistently caused postharvest fruit decay without causing any visible symptoms in the bushes. Such fruit carried either latent infections or superficially borne conidia on their surface for 6–12 wk. Further research is needed to determine unambiguously if latent infections occur with this disease.

Colletotrichum gloeosporioides is not only present in the field, but also is potentially destructive to blueberries from the prebloom stage through the harvest period. Results in the in vitro conidial germination studies show that conidia require free water and moderate temperatures for germination. We suggest that about 12 hr of continual leaf wetness due to rain at temperatures of 15–27 C are necessary for establishing a *C. gloeosporioides* conidial infection. Of 17 such periods recorded in this study, only one occurred prior to the green fruit stage of growth, which may suggest that low early season temperatures lessen the danger of *C. gloeosporioides* infection until the green fruit stage.

The inoculation study done at East Lansing using controlled environmental conditions suggests an increased susceptibility to *C. gloeosporioides* at the green fruit growth stage. The results of our inoculation study suggest to us that postharvest anthracnose decays of blueberries often can be attributed to inoculum of *C. gloeosporioides* produced earlier in the season. Confirmation of this and the determination of the precise environmental requirements for infection would be of great use in timing fungicide applications. Postharvest handling methods could be modified to eradicate latent *C. gloeosporioides* infections or kill superficially borne conidia if anthracnose decays were likely to be a problem.

LITERATURE CITED

1. Binyamini, N., and Schiffman-Nadel, M. 1972. Latent infection in avocado fruit due to *Colletotrichum gloeosporioides*. Phytopathology 62:592-594.
2. Brown, G. E. 1975. Factors affecting postharvest development of *Colletotrichum gloeosporioides* in citrus fruit. Phytopathology 65:404-409.
3. Capellini, R. A., Stretch, A. W., and Maiello, J. M. 1972. Fungi associated with blueberries held at various storage times and temperatures. Phytopathology 62:68-69.
4. Ceponis, M. J., and Capellini, R. A. 1976. Relative importance of Alternaria rot in postharvest disease development in New Jersey blueberries. Plant Dis. Rep. 60:569-571.
5. Ceponis, M. J., Kaufman, J., and Tietjen, W. H. 1973. Decays of New Jersey blueberries in marketing channels. Plant Dis. Rep. 57:487-488.
6. Mordue, J. E. M. 1971. *Glomerella cingulata*. No. 315 in: Descriptions of pathogenic fungi and bacteria. Commonw. Mycol. Inst., Kew, Surrey, England.
7. Stanghellini, M. E., and Aragaki, M. 1966. Relation of periderm formation and callose deposition to anthracnose resistance in papaya fruit. Phytopathology 56:444-450.
8. Stretch, A. W. 1967. Occurrence and control of *Glomerella cingulata* on highbush blueberry. Plant Dis. Rep. 51:401-404.
9. von Arx, J. A. 1970. A revision of the fungi classified as *Gloeosporium*. 2nd ed. J. Cramer Co., Lehre, W. Germany. S-H Service Agency, New York. 203 pp.