# Seasonal Incidence of Fungi in Symptomless Cranberry Leaves and Fruit Treated with Fungicides During Bloom

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## ABSTRACT

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In 1987 and 1988, leaves and fruit were collected at 2-wk intervals for 20 wk from replicated plots at two commercial cranberry (cultivar Searles) marshes in central Wisconsin. Leaves were collected from budbreak, and berries were collected from 8 and 6 wk after budbreak in 1987 and 1988, respectively, until harvest. Beginning during bloom, captafol, chlorothalonil, and mancozeb were applied to plots three times at 14-day intervals to manage postharvest storage rots. Control plots received no fungicide. Fungi were isolated from surface-disinfested, symptomless leaves and berries on all sampling dates in both years. In all, 33 genera or species of fungi were identified. Of these, only seven were known pathogens (*Apostrasseria lunata*, *Botryosphaeria vaccinii*, *Glomerella cingulata*, *Godronia cassandrae*, two morphologically distinct types of *Physalospora vaccinii*, *Phytophthora* sp., and *Pyrenobotrys compacta*), and three were possible pathogens. *B. vaccinii* and *P. vaccinii* were recovered most frequently and consistently. The proportion of leaves or berries from which fungi were isolated (i.e., incidence) increased as the season progressed. Although there was no significant difference among fungicide treatments in seasonal incidences of fungi from either leaves or berries, fungicides delayed the time, by 2–6 wk, at which incidences began to increase in both leaves and berries compared with untreated controls. By the end of the season, fungicides had reduced the number of fungus colonies and the incidence of *B. vaccinii* from berries in both years and the number of fungus colonies from leaves in 1987; however, final incidences of *P. vaccinii* (either type), *Alternaria*, and miscellaneous other fungi were not affected. Incidences of most fungi began increasing in both treated and untreated leaves and berries at 10–12 wk after budbreak, after the last fungicide application had been made, and continued to increase up to harvest. Initiating fungicide applications after bloom, at approximately 10 wk after budbreak, should reduce the incidence of fungi in cranberry leaves and berries more effectively than earlier applications.

*Additional keywords*: endophytes, *Vaccinium macrocarpon*.

Fungi that cause postharvest storage rots of fresh cranberries (*Vaccinium macrocarpon* Aiton) have been isolated from symptomless, immature berries very early during the growing season (1,3,26). In two investigations, the frequency of storage rot fungi isolated from berries did not change appreciably over the remainder of the growing season (1,26). Consequently, fungicide applications for managing storage rots have been initiated around bloom (1,3,24,26).

Storage rot fungi and other minor pathogens also have been recovered from surface-disinfested, symptomless leaves (13,29). It is unknown whether the presence of fungi in leaves ultimately affects yield, but it may serve as a source of inocula. The occurrence of fungi in symptomless fruit and leaves of cranberry and the periodic incidence of various fungi in these plant organs during the growing season have been reported in Wisconsin (4,5,12). The most frequently isolated fungus species pathogenic to cranberry was *Godronia cassandrae* Peck (anamorph: *Fusicoccum putrefaciens* Shear), which causes end rot in storage and is considered the most important storage rot disease in Wisconsin (5,24). Also isolated frequently were *Botryosphaeria vaccinii* (Shear) Barr (=*Guignardia vaccinii* Shear) (anamorph: *Phyllosticta elongata* G. J. Weidemann), which can cause berry speckle and postharvest fruit rot under certain storage conditions (6,29), and *Physalospora vaccinii* (Shear) Arx & E. Müller (=*Acanthorhynchus vaccinii* Shear), which causes blotch rot in storage but is of minor importance in Wisconsin (4,12,24). The prevalence of one or more of these fungi in leaves or berries

seemed to increase as the season progressed (4,5,12). Friend (12) and Brown (4) also isolated fungi from symptomless leaves and berries collected from cranberry beds treated with fungicides; however, the effect fungicides had on fungus infection of symptomless plant parts was not determined conclusively.

Except for triforine, which is registered to manage cottonball caused by *Monilinia oxycocci*, the only fungicides registered for use on cranberry in Wisconsin are those applied to manage storage rots (17). Applications for storage rot management are initiated during bloom and are made three times at 10- to 14-day intervals (17). The effects of these applications on the seasonal incidence of fungi in cranberry leaves and fruit have not been determined. However, if such effects were known, fungicides could be applied when fungi were most likely to infect fruit and, thus, improved storage rot management might be possible. Therefore, the objectives of this research were to investigate the occurrence of fungi in symptomless cranberry leaves and fruit; to determine the seasonal incidence of these fungi, particularly known pathogens; and to determine if fungicides applied for storage rot management influence seasonal incidence. This research complemented a project on the effects of fungicides on cranberry yield and storage rot incidence (16).

## MATERIALS AND METHODS

**Treatments and experimental design.** The experimental design and plot layout are reported in detail elsewhere (16). Briefly, the experiment was conducted in 1987 and 1988 at two locations in the principal cranberry-growing region of central Wisconsin. Fungicides had not been used at either location for at least 10 yr. Four treatments were compared, including an untreated control and the three fungicides most commonly used in Wisconsin for storage rot management at the onset of the project. Fungicides and amounts of active ingredient per hectare were: chlorothalonil, 3.51 kg; captafol, 3.59 kg; and mancozeb, 3.59 kg. Aqueous suspensions of fungicides were applied to plots in a volume equivalent to 1,429 L/ha. Treatments were applied three times at 14-day intervals and at both locations on the same day. Applications were initiated 17 June in both years, which corresponded to 70% bloom in 1987 and 14% bloom in 1988. Plots were established at both locations in cranberry beds (cultivar Searles) that were at least 40 yr old. Ten replicate plots, $2.0 \times 3.5$ m each, were used for each treatment and were arranged in a completely randomized design to study the effects on yield and storage rot incidence (16). Five randomly selected plots of each treatment were sampled at each location in this investigation, because biweekly isolation and collection of fungi (see next section) from all 10 plots would have been unmanageable.

**Sampling procedure, isolation, and data collection.** Preliminary studies were conducted during the 1986 growing season to determine the most appropriate procedures to be used in 1987 and 1988 for sampling, surface-disinfesting, and isolating from leaves and berries. Samples always were collected before applications were made on dates that fungicides were applied. Plots were sampled biweekly from budbreak until within 2 wk of commercial harvest. Budbreak was defined as the time when more than 50% of the shoots had begun to elongate, but the first leaves were not fully expanded. Samples were collected at each location by arbitrarily cutting six actively growing upright shoots from the center portion of each plot to ensure that five independent leaves or berries would be available for isolation; shoots with berries were collected when berries were present. Shoots were placed in polyethylene bags, transported in a cool ice chest to the laboratory, and refrigerated. Isolations were conducted 24–48 h after sample collection.

Leaves were assayed on all sampling dates in both 1987 and 1988. The entire new shoot was used for the initial sample at budbreak, because leaves had not expanded. For the remaining sampling dates, only the most proximal leaf that appeared symptomless was removed from each shoot, and all six leaves for each sample were surface-disinfested together by continuous stirring in 100 ml of 0.5% NaOCl to which 1–2 drops of Tween 20 (Sigma Chemical Co., St. Louis, MO) had been added. Leaves collected up to 4 wk after budbreak were more succulent and tender than those collected later. These leaves were disinfested for 1.5–2.0 min, except that those collected 2 wk after budbreak in 1987 were disinfested for 3 min (which was phytotoxic). Leaves collected from 6 wk after budbreak to harvest were disinfested for 3 min. After disinfestation, leaves were rinsed thoroughly in sterile distilled water, dipped in 95% ethanol to displace water and enhance drying, blotted on sterile paper towels, and allowed to air-dry. Five of the six leaves in each sample were selected arbitrarily for isolation.

All isolations were conducted with half-strength potato-dextrose agar (Difco Laboratories, Detroit, MI; hsPDA; 19.0 g of potato-dextrose agar, 8.0 g of agar, and 1,000 ml of distilled water) amended with 100 mg/L streptomycin sulfate (hsPDA + S), which was added after autoclaving and then cooling to 50 C. The medium was dispensed equally into 9-cm-diameter plastic petri dishes. The five leaves from each sample were placed in one dish of hsPDA + S by pushing each leaf into the medium to ensure adequate contact between leaf and agar. Dishes were placed in clear, plastic boxes, and the boxes were placed in the laboratory near a north-facing window at room temperature (20–25 C). Dishes were observed for developing colonies, and data were recorded after 5, 8, and 12 days.

Berries were assayed when they reached 3–4 mm in diameter, beginning 8 and 6 wk after budbreak in 1987 and 1988, respectively. The largest, most mature berry that appeared symptomless was removed from each of the six shoots collected per sample, and all six berries were surface-disinfested together for 3 min as described for leaves. Five berries were selected arbitrarily for isolation after air-drying. Each was cut in half transversely, and the cut surface of the pedicel end of the berry was placed down on a dish of hsPDA + S; one dish was used for each sample of five berry halves. Isolation dishes containing berry halves were treated identically to those containing leaves.

For each leaf or berry half in a dish, the total number of discrete fungus colonies was counted, and the occurrence of each distinct morphological colony type was recorded. Data were summarized for each sample of five leaves or berry halves as the total number of colonies of all fungi isolated and the isolation frequencies of different fungi (i.e., the proportions of the sample from which different fungi were isolated). If feasible, one or more representative cultures of each unique morphological type was saved at each sampling date for later identification. Isolates to be saved were transferred to dishes of hsPDA + S and then to hsPDA slant tubes, which were stored at 2 C until fungi were identified.

**Identification of fungi.** Whenever possible, fungi were identified to genus, and those suspected of being cranberry pathogens were identified to species. All fungi were grown on potato-dextrose agar made directly from potatoes (pPDA; 250 g of potatoes, 20 g of dextrose, 10 g of agar, and 1,000 ml of distilled water), 20% V8 juice agar (V8A) (27), and cornmeal agar (CMA; Difco). Cultures were incubated under fluorescent lights with a 12-h photoperiod at 19–22 C and were examined microscopically after 2 and 3 wk.

Isolates that did not sporulate on these media were placed on water agar with sterile cranberry leaves and berry slices. Leaves were collected from greenhouse-grown plants, washed twice in sterile water plus Tween 80 (Sigma; 1 drop per 100 ml), rinsed in sterile water, air-dried, and exposed to a propylene oxide-saturated atmosphere for 15 h (27). Commercially grown cranberries were washed in a manner similar to that used for leaves, immersed in 1% NaOCl plus Tween 80 (1 drop per 100 ml) for 12 min, and rinsed twice in sterile water. Two slices, each approximately 4-mm thick, were cut from the midsection of each berry. Leaves and fruit slices were placed on pPDA to check for sterility, and, after 4 days, any that were contaminated were discarded. Leaves and berry slices then were transferred aseptically to dishes of 1.5% water agar (two leaves, one adaxial and one abaxial side up, and two berry slices per dish). Dishes were seeded individually with fungi and placed at room temperature (20–25 C)

near a north-facing window. Fungi were examined for sporulation after 4–6 wk.

**Data analysis.** In each year, four of the 11 sampling dates were selected for analyses of treatment effects on the incidences of fungi in leaves and berries. These dates and the rationale for selecting each were: the date of the first fungicide application (i.e., 6 and 4 wk after budbreak in 1987 and 1988, respectively), which was the maximum period of time elapsed after budbreak before fungicides were applied (only data from leaves were analyzed for this date); 2 wk after the third fungicide application (i.e., 12 and 10 wk after budbreak in 1987 and 1988, respectively), which was the estimated end of the period of residual fungicide efficacy; 16 wk after budbreak, which was an intermediate time between the end of fungicide efficacy and harvest; and 20 wk after budbreak and the last sample before harvest, which was the maximum length of time for infection by fungi after the last fungicide application.

For each selected sampling date, two-way analyses of variance (ANOVAs) comparing treatments and locations were computed separately for the number of fungus colonies or isolation frequencies of different fungi from leaves or berries with MINITAB (release 6.2) statistical software (Minitab, Inc., State College, PA). All proportion data were transformed to arcsine-square root values before analysis (25). For each two-way ANOVA with a significant treatment main effect, a single degree of freedom linear contrast was computed with SYSTAT (version 4.0) statistical software (SYSTAT, Inc., Evanston, IL) to compare fungicide treatments together with the untreated control. Because incidence data each year actually were the result of repeated measures on the same plots over time, the level at which computed $F$ statistics usually would be judged significant (i.e., $P \leq 0.05$) was reduced according to Bonferroni's inequalities (25). Data on four and three sampling dates were analyzed for leaves and fruit, respectively; therefore, $F$ statistics were judged significant at $P \leq 0.013$ (=0.05/4) for leaves and $P \leq 0.017$ (=0.05/3) for fruit.

## RESULTS

In both years, plots were sampled 11 times for a period of 20 wk (Table 1). Phenological development of plants progressed at a similar rate each year even though plant growth and development in 1987 were about 12 days ahead of those in 1988 based on calendar dates (Table 1).

TABLE 1. Biweekly dates that symptomless cranberry (cultivar Searles) leaves and fruit were collected for isolation of fungi in 1987 and 1988 and the associated stages of plant phenology

| 1987 | | 1988 | |
|---|---|---|---|
| Date[a] | Stage[b] | Date | Stage |
| 6 May | Budbreak | 19 May | Budbreak |
| 20 May | Hook | 1 June | Hook |
| 3 June | 15% bloom | 15 June*[c] | 9% bloom |
| 17 June* | 70% bloom | 29 June* | 80% bloom |
| 1 July* | Late bloom | 13 July* | Late bloom |
| 15 July* | Out of bloom | 27 July | Out of bloom |
| 29 July | Immature fruit | 10 August | Immature fruit |
| 12 August | Fruit development | 24 August | Fruit development |
| 26 August | Fruit development | 7 September | Fruit development |
| 9 September | Fruit development | 7 September | Fruit development |
| 23 September | Mature fruit | 4 October | Mature fruit |

[a] Fungicides were applied on dates marked with asterisks (*) after plants were sampled.

[b] Stages of plant phenology were: budbreak, when >50% of the shoots had begun to elongate; hook, when unopen flowers were present on hooked pedicels; % bloom, average percentage of flowers open between two locations; late bloom, only a few flowers still open, most had set fruit; out of bloom, no flowers present; mature fruit, within 2 wk of harvest.

[c] In 1988, the first fungicide application actually was made 2 days later on 17 June at 14% bloom.

**Identification of fungi.** On pPDA, *B. vaccinii* and most hyphomycetes sporulated well, and *P. vaccinii* formed a colony with a characteristic, irregular lobate margin. However, CMA was most useful for identifying *P. vaccinii*, because it produces abundant perithecia on this medium, as noted previously (12). Some hyphomycetes sporulated better on V8A; *Colletotrichum* produced acervuli, *Phoma* and *Septoria* produced pycnidia, and several isolates of *Alternaria*, which only produced abundant aerial hyphae on pPDA, produced conidia. Water agar with cranberry leaves and berry slices was effective for identifying several other fungi. For example, *Cytospora* and *Sporormiella* produced conidia and *Pyrenobotrys compacta* (Peck) B. Eriksson (=*Gibbera compacta* (Peck) Shear) produced perithecia on the leaves.

Identification of isolates in the genera *Nodulisporium* and *Geniculosporium* was confirmed by Drs. J. D. Rogers (Washington State University, Pullman) and L. Sigler (University of Alberta, Edmonton, Canada). Isolates of *Nodulisporium* recovered in 1987 most likely had *Daldinia eschscholzii* (Ehrenb.) Rehm as a teleomorph, and the two isolates recovered in 1988 probably had *Hypoxylon fuscum* (Pers.:Fr.) Fr. as a teleomorph. Isolates of *Geniculosporium* probably had teleomorphs in the genus *Hypoxylon*.

Fungi that could not be identified were observed or isolated from both leaves and berries on most sampling dates (unknowns; Table 2). These included colonies that were overgrown by other fungi on original isolation dishes; mycelium transfers that were unsuccessful; and isolates that did not sporulate on any medium, that sporulated but still could not be identified, or that lost viability in storage. Of the fungi isolated, 8.3% were not identified.

**Isolation of fungi from leaves and berries.** Fungi were isolated from symptomless, surface-disinfested cranberry leaves and fruit at all sampling dates in both years (Table 2), except from leaves sampled 2 wk after budbreak in 1987 that were disinfested for too long. A greater diversity of fungi was recovered in 1987 compared with 1988 (Table 2). In all, 33 different genera or species were identified. Seasonal occurrence of the different fungi varied considerably. Thirteen fungi were isolated (primarily from leaves) on only one sampling date during the 2-yr period, and four were isolated infrequently in only 1 yr. The other 16 fungi were recovered at least once in both years.

Seven known cranberry pathogens (12,15,23,24) were recovered (Table 2). *Apostrasseria lunata* (Shear) Nag Raj (=*Ceuthospora lunata* Shear), *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk, and *Phytophthora* sp. were isolated on only one or two sampling dates over the 2 yr. *P. compacta* was isolated on three dates in 1987, and *G. cassandrae* was isolated sporadically in 1987 and twice in 1988. *B. vaccinii* and *P. vaccinii* were the only pathogens isolated regularly in both years. *P. vaccinii* occurred as two distinct morphological types based on colony color, i.e., dark-colored (brown or gray) or white. Three of the other fungi recovered were considered possible pathogens (Table 2), and the remaining 23 fungi were not known or even suspected cranberry pathogens.

Eighteen isolates of *Cladosporium* were tested individually for pathogenicity to cranberry because one species, *C. oxycocci* Shear, can cause leaf spot (23,24). Conidia were washed with sterile water plus Tween 80 (1 drop per 100 ml) from 10-day-old cultures grown on pPDA, and spore suspensions were adjusted to a standard concentration of $10^6$ conidia per milliliter. Spore suspensions were sprayed to runoff onto 2- to 4-mo-old cranberry plants (cultivar Searles) growing in a greenhouse, and plants were placed in a mist chamber (10 s of mist per minute) at 20–25 C for 26 days. No lesions developed on either inoculated or uninoculated plants; therefore, all isolates of *Cladosporium* were not pathogenic under these conditions.

**Seasonal incidence of fungi.** The two most consistently and frequently isolated pathogenic fungi from both leaves and berries were *B. vaccinii* and *P. vaccinii* (dark-colored colony type). *P. vaccinii* (white colony type) also occurred consistently at biweekly sample intervals but was not isolated as frequently from individual samples of leaves or berries (Table 2). The mean number

of colonies of all fungi isolated and mean isolation frequencies (i.e., incidences) of *B. vaccinii* and *P. vaccinii* (dark) from both locations combined (i.e., 10 replicate samples of five leaves or berries per treatment) were plotted over time for 1987 and 1988 (Figs. 1–3). Data from both locations were combined because treatment × location interactions were not significant, except in two instances; most interactions (86 of 98 calculated) had $P \geq 0.10$. Location main effects usually were not significant either, except for *P. vaccinii* (white). In general, seasonal progress of the numbers of colonies and the incidences of different fungi from both leaves

TABLE 2. Numbers of surface-disinfested, symptomless cranberry (cultivar Searles) leaves (L) or fruit (F) collected at biweekly intervals during 1987 and 1988 from which different genera or species of fungi were isolated[a]

| | | | Number of weeks after budbreak[c] | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 0 | 2 | 4 | 6 | | 8 | | 10 | | 12 | | 14 | | 16 | | 18 | | 20 | |
| Taxa of fungi | Pathogenic nature[b] | Year | L | L | L | L | F | L | F | L | F | L | F | L | F | L | F | L | F | L | F |
| *Alternaria* | − | 1987 | 2 | | 15 | 30 | | 13 | 12 | 23 | 3 | 11 | | 14 | 3 | 20 | 3 | 34 | 14 | 16 | 5 |
| | | 1988 | | 19 | 30 | 27 | | 19 | 2 | 18 | 7 | 22 | 15 | 27 | 19 | 19 | 11 | 39 | 9 | 33 | 15 |
| *Apostrasseria lunata* | + | 1987 | 1 | | | | | | | | | | | | | | | | | | |
| | | 1988 | | | 1 | | | | | | | | | | | | | | | | |
| *Ascochyta* | − | 1987 | | | | | | | | | | | 1 | | | | | 1 | | | |
| *Aspergillus* | − | 1987 | | | | | | | | | | | 1 | | | | | | | | |
| *Aureobasidium* | − | 1987 | | | | | | | 1 | | | | | | | | | | | | |
| *Bipolaris* | − | 1987 | | | | | | | | | | | | | | | | | | 1 | |
| *Bispora* | − | 1988 | | | | 1 | | | | | | | | | | | | | | | |
| *Botryosphaeria vaccinii* | + | 1987 | 5 | | | | | | 1 | | 2 | 7 | 17 | 5 | 25 | 7 | 61 | 5 | 76 | 17 | 101 |
| | | 1988 | | | | 1 | | 1 | | 1 | 2 | 2 | 9 | 2 | 25 | 1 | 57 | 7 | 98 | 4 | 113 |
| *Chaetomium* | − | 1987 | | | | 1 | | | | | | | | | | | | 1 | | | |
| | | 1988 | | | | | 1 | | | | | | | | | | | | | | |
| *Cladosporium* | − | 1987 | 3 | | 1 | 1 | | 1 | 6 | | 2 | 2 | | | | 3 | | 2 | 2 | | |
| | | 1988 | 2 | | 2 | 2 | | | 1 | | 3 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | |
| *Colletotrichum* | ? | 1987 | | | | | | | | | | | | | | | | 1 | | 1 | |
| *Cytospora* | − | 1988 | 1 | | | | | | | | | | | | | | | | | | |
| *Epicoccum* | − | 1987 | 1 | | | 2 | | | 1 | 4 | | 1 | | | | | | | | | 2 |
| | | 1988 | | 1 | | | | | | | | | | | | | 1 | 1 | | 1 | 2 |
| *Fusarium* | − | 1987 | | | | | | | | | 1 | | | | | | 1 | | | | |
| | | 1988 | | | | | | | | | | | 1 | | | | | | | | |
| *Fusidium* | − | 1987 | 2 | | | | | | | | | | | | | | | | | | |
| *Geniculosporium* | − | 1987 | | | | | | | | | | | | | | 1 | | 5 | | 1 | |
| *Glomerella cingulata* | + | 1987 | | | | | | | | | | | | | 1 | | | | | | |
| *Godronia cassandrae* | + | 1987 | 5 | | | 1 | | | 3 | | | | | | 1 | 2 | 1 | 2 | 3 | | 4 |
| | | 1988 | | | | | | | | | | | | | 1 | | | | | | 1 |
| *Nigrospora* | − | 1987 | | | 2 | | | | | | | 1 | | | | | | | | | |
| | | 1988 | 1 | | | | | | | | | | | 1 | | | | | | | |
| *Nodulisporium* | − | 1987 | 1 | | | | | | | | | | | 1 | 1 | 3 | | 13 | 1 | 7 | |
| | | 1988 | | | | | | | | | | | | 1 | | | | | | 1 | |
| *Penicillium* | ? | 1987 | | | | | | | | | | | | | | | | | 1 | | |
| *Phoma* | − | 1987 | | | | | | | | | 2 | | | | | | | | | | |
| *Physalospora vaccinii* (d)[d] | + | 1987 | 2 | | 1 | | | 2 | | 5 | | 5 | 6 | 16 | 30 | 39 | 8 | 35 | 59 | 57 | 35 |
| | | 1988 | | | | | | 4 | | 6 | 7 | 10 | 15 | 19 | 35 | 33 | 22 | 35 | 54 | 51 | 56 |
| *Physalospora vaccinii* (w)[d] | + | 1987 | | | | | | | | | | 1 | 6 | 10 | 4 | 4 | 9 | 6 | 21 | 12 | 14 |
| | | 1988 | | | | | | 3 | | 2 | 3 | 3 | 5 | 6 | 6 | 13 | 15 | 14 | 21 | 10 | 18 |
| *Phytophthora* sp. | + | 1987 | 3 | | | | | | | | | | | | | | | | | | |
| *Pyrenobotrys compacta* | + | 1987 | | | | | | | | 1 | | | | | | | 5 | | 2 | | |
| *Pythium* | − | 1987 | | | 1 | | | | | | | | | | | | | | | | |
| | | 1988 | 1 | | | | | | | | | | | | | | | | | | |
| *Rhizoctonia* | ? | 1987 | 1 | | | | | | | | | 1 | | 1 | | | | 1 | | | |
| | | 1988 | 1 | | | | | | | | | | 1 | | | | | | | | |
| *Septoria* | − | 1987 | | | | | | | 1 | | | | | 1 | | | | 1 | | | |
| | | 1988 | | | | | | | | | | | 1 | | | | | | | | |
| *Sordaria* | − | 1987 | | | | | | | | 1 | | 1 | | | | | | | | | |
| | | 1988 | | | | | | | | | | | | | | | | | | 1 | |
| *Sporormiella* | − | 1987 | 1 | | 1 | 1 | | | | | | | | | | 1 | | 1 | | | |
| | | 1988 | | | 1 | | | | | 2 | | | | | | 1 | | 1 | | | |
| *Stemphylium* | − | 1987 | | | | | | | | | | | | 1 | | | | | | | |
| *Trichoderma* | − | 1987 | | | | | | | | | | | | | | | | 1 | | | |
| *Trichurus* | − | 1988 | | | | | | | | | | | | 1 | 1 | | | | | | |
| Unknowns[e] | | 1987 | 37 | | | | | 2 | 8 | 4 | 4 | 23 | 3 | 27 | 7 | 18 | 8 | 32 | 17 | 21 | 11 |
| | | 1988 | 1 | 1 | 1 | 1 | | | 2 | 1 | 3 | 9 | 4 | 4 | 5 | 5 | 1 | 9 | 2 | 11 | 7 |
| Totals | | 1987 | 64 | | 21 | 36 | | 18 | 33 | 38 | 14 | 53 | 34 | 76 | 71 | 99 | 96 | 141 | 196 | 133 | 172 |
| (All fungi) | | 1988 | 7 | 21 | 35 | 32 | 1 | 27 | 5 | 30 | 25 | 48 | 53 | 63 | 94 | 73 | 109 | 108 | 185 | 113 | 212 |

[a] Two-hundred entire leaves or berry halves (pedicel end) were assayed at each biweekly interval, five leaves or berries from each of five plots for four treatments at two locations. See Table 1 for collection dates and associated stages of plant phenology. Whenever possible, fungi were identified to genus; those suspected to be known cranberry pathogens were identified to species.

[b] Fungi were classified as known pathogens (+), possible pathogens (?), or not known or suspected to be pathogens (−) based on previous reports.

[c] Leaves were collected first at budbreak; fruit were collected first at 8 and 6 wk after budbreak in 1987 and 1988, respectively. Both were collected up to harvest.

[d] *P. vaccinii* occurred as two distinct morphological types that produced dark- (d) or white- (w) colored colonies.

[e] Fungi that could not be identified.

and berries were similar in 1987 and 1988 (Figs. 1–3).

Throughout both seasons, there was no consistent, discernible difference among the three fungicide treatments in their effects on seasonal incidences of fungi (Figs. 1–3), even though the spectra of activity of these fungicides are somewhat different. Consequently, statistical comparisons among fungicide treatments were not made. However, differences between fungicide-treated and untreated leaves and berries often occurred, and these differences were analyzed (Figs. 1–3). Whenever treatment main effects were significant, so were single degree of freedom contrasts for comparing fungicide treatments with the untreated control. Therefore, significant treatment effects were due largely to strong significant differences between samples from fungicide-treated and untreated plots.

In both years, the mean number of fungus colonies isolated from leaves and fruit increased as the season progressed, much more so for fruit than for leaves (Fig. 1). In either year, untreated fruit samples had approximately five times as many colonies as untreated leaf samples. In 1988, the total numbers of colonies counted on untreated leaves or fruit at season's end were reduced by approximately 33% compared with those in 1987 (Fig. 1). However, the numbers of colonies from fungicide-treated leaves and fruit were similar in both years. Fungicide-treated leaves in 1987 and berries in 1987 and 1988 tended to have fewer colonies than untreated control leaves and berries, and these differences usually were significant at the selected sampling intervals (Fig. 1A, C, D). Treatments had no significant effect on the number of fungus colonies isolated from leaves collected in 1988 (Fig. 1B).

Leaves or berries from all treatments had similar numbers of colonies at all sampling dates during the first half of the season in both 1987 and 1988 (Fig. 1). In 1987, leaves or fruit that were not treated with fungicides exhibited a marked increase in the number of colonies isolated at 12 wk after budbreak (Fig. 1A, C). Similar increases, although less pronounced, occurred for fungicide-treated leaves and berries 2 wk later, i.e., at 14 wk after budbreak or 4 wk after the last application. In 1988, numbers of colonies from untreated leaves increased almost continuously from 6 wk after budbreak throughout the season (Fig. 1B), whereas those for treated leaves began increasing continuously 12 wk after budbreak, again, 4 wk after the last application. Seasonal increases in colony numbers of all fungi isolated from fruit in 1988 were similar to those that occurred in 1987; noticeable increases in colony numbers occurred 2 wk later on treated berries than on untreated berries, 12 wk (i.e., 4 wk after the last fungicide application) and 10 wk after budbreak, respectively (Fig. 1D).

*B. vaccinii* was isolated from leaves only at low levels throughout this study (Fig. 2A, B). In both years, it was recovered more consistently and in greater proportions from untreated leaves than from treated leaves, although these differences in incidence usually were not significant when sufficient data were available for a meaningful analysis (Fig. 2A, B). Seasonal incidence of *B. vaccinii* from fruit was quite different. Frequency of isolation from untreated fruit began to increase 10 wk after budbreak and

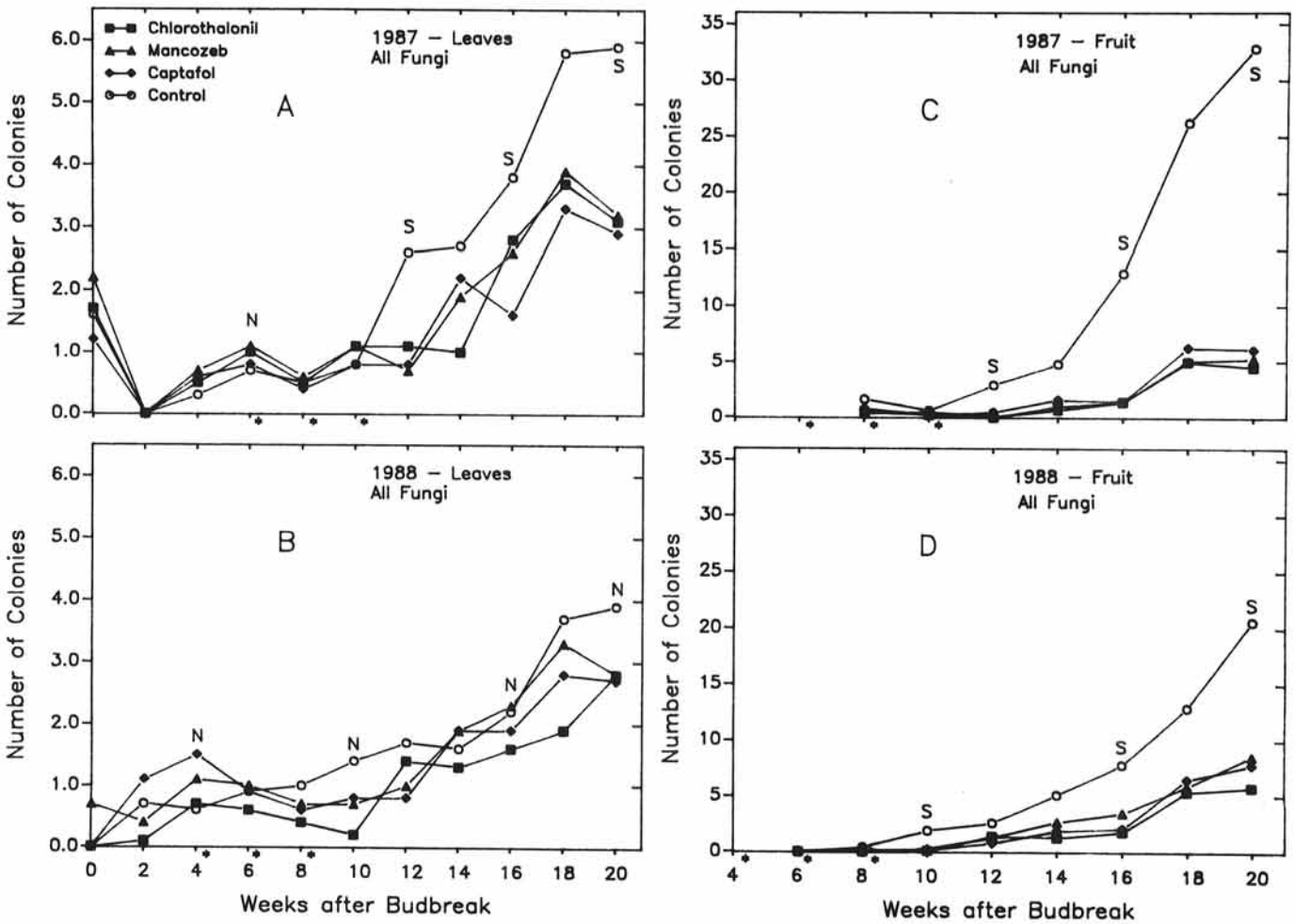


**Fig. 1.** Effects of fungicide treatments on the number of discrete colonies of all fungi growing from five surface-disinfested, symptomless cranberry (cultivar Searles) leaves or fruit halves (pedicel ends) sampled every 2 wk in 1987 and 1988. **A,** 1987 leaves; **B,** 1988 leaves; **C,** 1987 fruit; and **D,** 1988 fruit. Leaves were collected from budbreak until harvest 20 wk later; berries were collected beginning 8 and 6 wk after budbreak until harvest in 1987 and 1988, respectively. Each data point is the mean of 10 replicate samples, five from each of two locations. Fungicides were applied (*) three times each year at 14-day intervals beginning during bloom; controls received no fungicide. Based on independent two-way analyses of variance, differences among treatments at selected sampling intervals were significant (S), at $P \leq 0.013$ for leaves or $P \leq 0.017$ for fruit, or were not significant (N) (see text).

continued to nearly 100% (a proportion of 1.0) by 20 wk after budbreak in both years (Fig. 2C, D). Fungicide treatments delayed the increase in incidence of *B. vaccinii* in berries by 6 wk in 1987 and 4 wk in 1988, which was 6 wk after the last fungicide application in both years. Final incidences of *B. vaccinii* in fungicide-treated berries was approximately one-half that in untreated berries. Differences between incidences of *B. vaccinii* in treated and untreated fruit were significant at all three selected biweekly intervals in 1987 and at 16 and 20 wk after budbreak in 1988 (Fig. 2C, D).

*P. vaccinii* (dark) was isolated rarely from leaves for the first 8 wk after budbreak; however, isolation frequencies from both leaves and berries then slowly increased over time (Fig. 3). Treatments had no significant effect on the incidence of *P. vaccinii* (dark) from either leaves or fruit, with two exceptions (Fig. 3); incidence from treated fruit was lower than that from untreated fruit at 2 wk after the last fungicide application in both years. However, fungicide treatments delayed the increase in isolation frequency of *P. vaccinii* (dark) from fruit by 2 wk in both 1987 and 1988. Even on untreated fruit, incidence did not begin increasing until after the last fungicide application. Incidences of this species in leaves and fruit at season's end were similar in both years, approximately 20–30%.

Seasonal incidence of *P. vaccinii* (white), *Alternaria*, and miscellaneous other fungi are not presented graphically. Seasonal incidence for *P. vaccinii* (white) was similar to that of *P. vaccinii* (dark), but the proportions of leaves or berries from which the white colony type was isolated typically were less (Table 2), ranging from 0.0 to 0.1. The proportion of leaves or berries from which *Alternaria* was isolated varied from 0.0 to 0.2 for most sample intervals, with no apparent trend as the season progressed. The proportion of leaves in 1987 from which other miscellaneous fungi were isolated usually ranged from 0.0 to 0.25 and tended to increase as the season progressed. However, the proportion of leaves in 1988 and fruit in both years from which other miscellaneous fungi were isolated usually ranged from 0.0 to 0.1 and remained fairly constant throughout the season. Treatments had no significant effect on the seasonal incidence of *P. vaccinii* (white), *Alternaria*, or miscellaneous other fungi from either leaves or berries with three exceptions: the incidence of *Alternaria* from leaves at 16 wk and from fruit at 10 wk after budbreak in 1988, and the incidence of other fungi from fruit at 16 wk after budbreak in 1987. However, these effects on incidence could not be attributed to significant differences between fungicide-treated and untreated samples.

## DISCUSSION

Fungi were isolated consistently from surface-disinfested, symptomless cranberry leaves and fruit throughout two

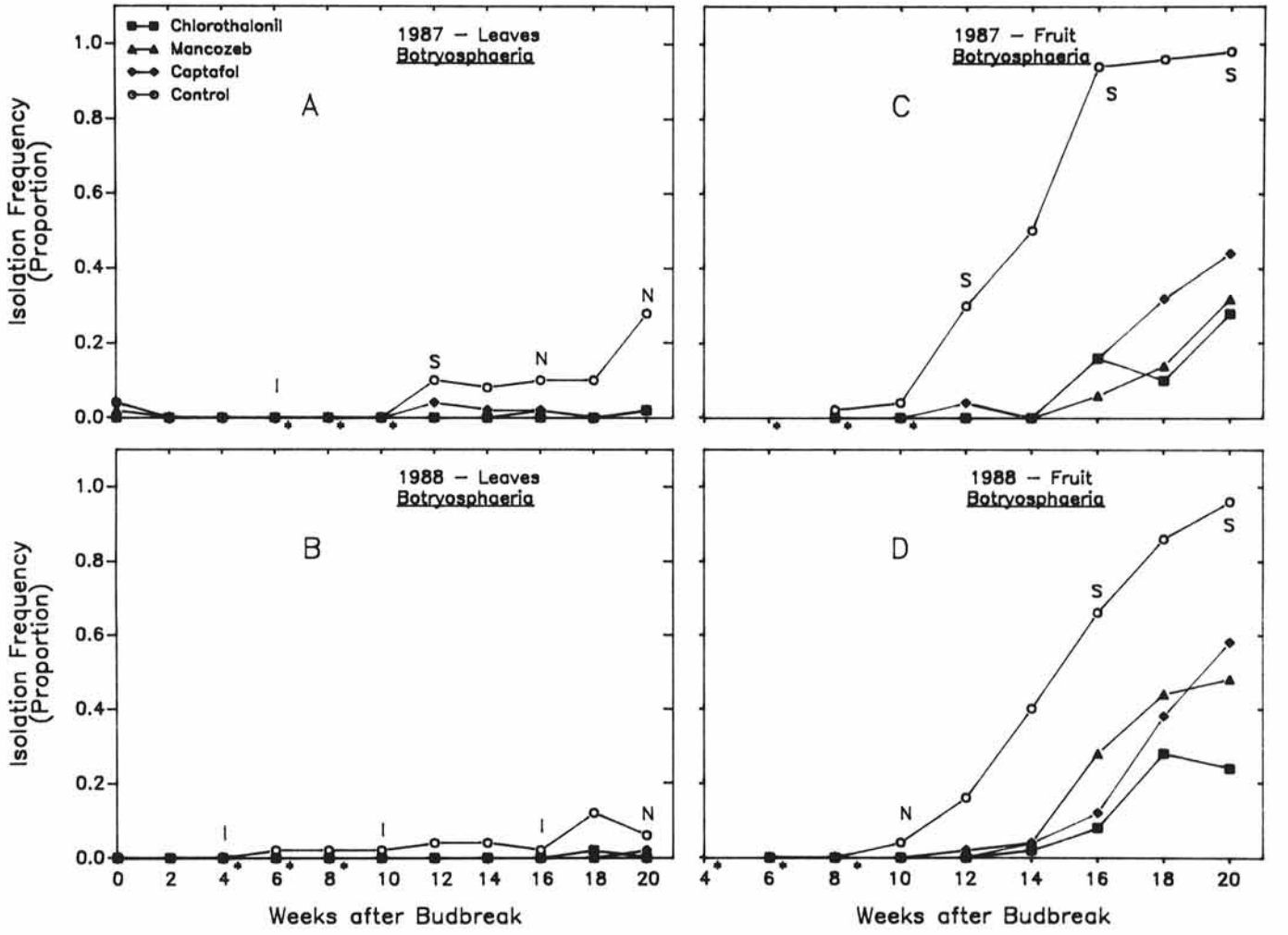


**Fig. 2.** Effects of fungicide treatments on the frequency of isolation (incidence) of *Botryosphaeria vaccinii* from five surface-disinfested, symptomless cranberry (cultivar Searles) leaves or fruit halves (pedicel ends) sampled every 2 wk in 1987 and 1988. **A,** 1987 leaves; **B,** 1988 leaves; **C,** 1987 fruit; and **D,** 1988 fruit. Leaves were collected from budbreak until harvest 20 wk later; berries were collected from 8 and 6 wk after budbreak until harvest in 1987 and 1988, respectively. Each data point is the mean of 10 replicate samples, five from each of two locations. Fungicides were applied (*) three times each year at 14-day intervals beginning during bloom; controls received no fungicide. Based on independent two-way analyses of variance, differences among treatments at selected sampling intervals were significant (S), at $P \leq 0.013$ for leaves or $P \leq 0.017$ for fruit, or were not significant (N) (see text). Occasionally, insufficient data were available for analysis (I), because fungi were isolated too infrequently.

consecutive growing seasons. Similar results were obtained from the preliminary investigation conducted in 1986. Fungi generally were recovered more frequently and in greater numbers as the season progressed, which is contrary to some earlier reports from other geographical regions (1,26) but consistent with reports from Wisconsin (4–6,12). The relative abundance of different fungi in leaves at budbreak (0 wk; Table 2) compared with other early sampling dates probably is because these samples consisted of the entire new shoot (5–10 mm in length) instead of individual leaves. In addition, fungi consistently were isolated more often and in greater numbers from fruit than from leaves. These results are the most thorough account of the seasonal incidence of fungi in cranberry leaves and fruit. Previously, only Friend (12) had attempted to survey all fungi associated with leaves, fruit, and other aerial organs of cranberry periodically during the growing season, but his samples were not collected as often or as systematically as those in this study. Several others also have isolated selected fungi from fruit and occasionally from leaves of cranberry periodically during the season (1,4–6,26). Because there were fungi that could not be identified at most sampling intervals, the diversity and incidence of fungi reported here are likely to be conservative.

Results of this research must be qualified by the potential limitations of the isolation procedures. Procedures employed throughout this study were selected based on preliminary research conducted in 1986 and, generally, were similar to those employed to isolate fungi from cranberry leaves and fruit by others (1,3–5,7,12–14,26,29). In fact, the standard procedure for isolating fungi that occur internally in symptomless plant parts is by rigorous surface-disinfestation and culturing on a nonselective agar medium (8,20). However, surface-disinfestation is not always complete, and epiphytic contaminants can survive (8,20,22). Propagules that are not killed by the disinfestant are more likely to occur on plant surfaces that are pubescent or have cracks and crevices (8,22), neither of which is typical for cranberry leaves and fruit. Another possible limitation is that isolation frequencies determined from cultured plant parts may underestimate the actual frequency of internal fungi (8), which, again, suggests the results reported here probably are conservative. In addition, use of a different medium or incubation temperature for isolations may have produced different results. However, preliminary research indicated that hsPDA was at least as effective as several other commonly used isolation media and that the addition of streptomycin sulfate did not adversely affect recovery of fungi.

Only 24% (8 of 34, including both types of *P. vaccinii*) of the fungus taxa recovered were known pathogens, and five of these, including the only storage rot fungi isolated, were recovered irregularly or infrequently over the 2-yr period. The infrequent occurrence of *G. cassandrae*, reported to be the predominant cause of storage rots of cranberry in Wisconsin (5,24), was

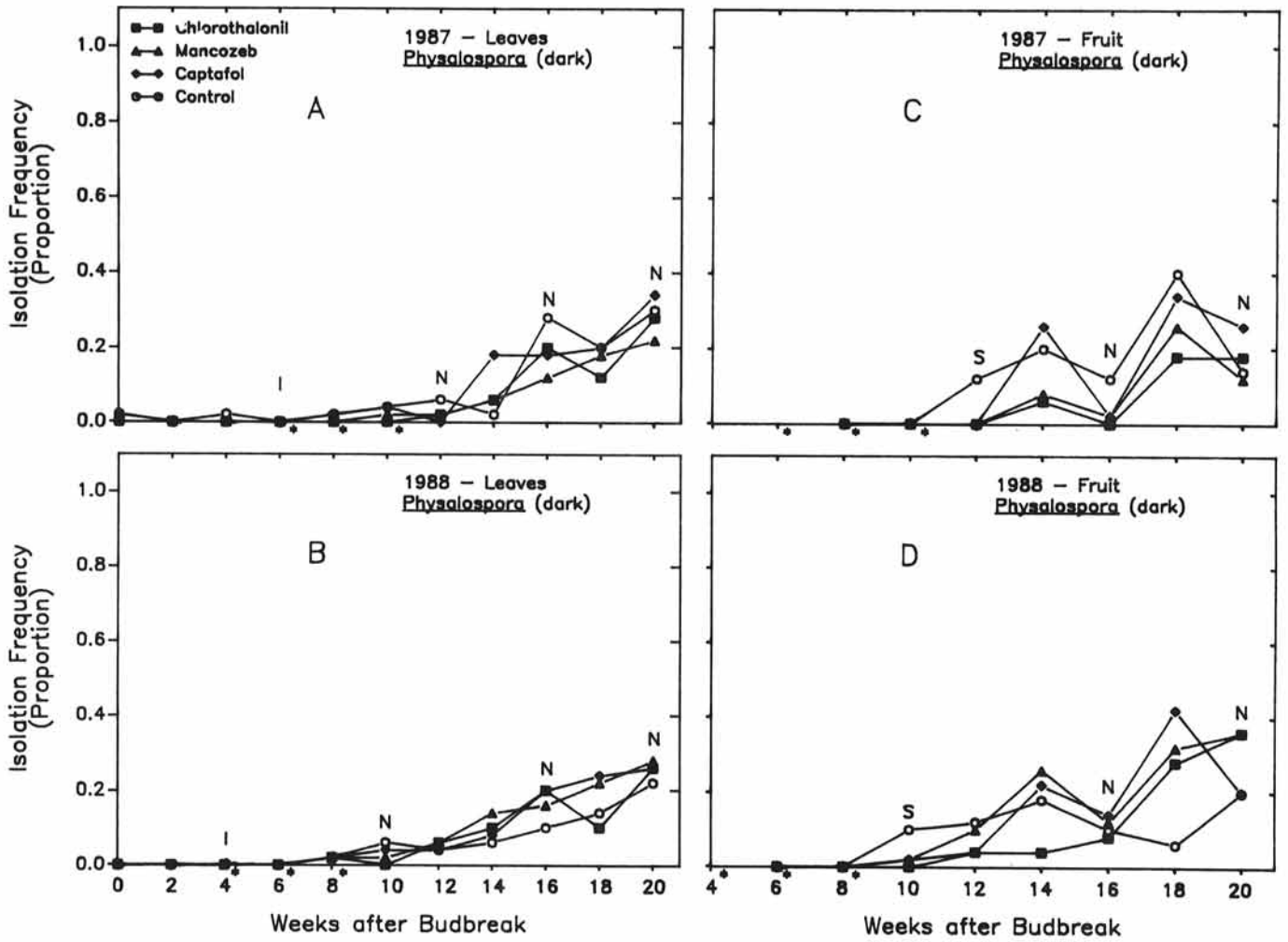


**Fig. 3.** Effects of fungicide treatments on the frequency of isolation (incidence) of *Physalospora vaccinii* (dark-colored morphological type) from five surface-disinfested, symptomless cranberry (cultivar Searles) leaves or fruit halves (pedicel ends) sampled every 2 wk in 1987 and 1988. **A,** 1987 leaves; **B,** 1988 leaves; **C,** 1987 fruit; and **D,** 1988 fruit. Leaves were collected from budbreak until harvest 20 wk later; berries were collected from 8 and 6 wk after budbreak until harvest in 1987 and 1988, respectively. Each data point is the mean of 10 replicate samples, five from each of two locations. Fungicides were applied (*) three times each year at 14-day intervals beginning during bloom; controls received no fungicide. Based on independent two-way analyses of variance, differences among treatments at selected sampling intervals were significant (S), at $P \leq 0.013$ for leaves or $P \leq 0.017$ for fruit, or were not significant (N) (see text). Occasionally, insufficient data were available for analysis (I), because fungi were isolated too infrequently.

unexpected and in contrast to Carlson's previous findings (5). However, Carlson reported that the incidence of *G. cassandrae* varied considerably from year to year and among locations. It is likely that the isolation method employed in this research did not favor recovery of *G. cassandrae*. This fungus and certain others typically occur more frequently in the blossom end of the berry rather than in the pedicel end (1,5,26). In preliminary research in 1986 when berries were cut in half lengthwise from pedicel to calyx instead of transversely, *Alternaria* spp. originating from the calyx region frequently overgrew isolation dishes and obscured other fungi. Consequently, it was decided to avoid the calyx portion of the berry to maximize the diversity of fungi isolated in this study.

Of the other known storage rot pathogens, *A. lunata* and *G. cingulata* were isolated very infrequently, as expected (5,12), and *Diaporthe vaccinii* (anamorph: *Phomopsis vaccinii*) was not isolated at all, which was not expected based on previous research in Wisconsin (5). The isolates of *Phytophthora* sp. recovered were morphologically similar to those of the unidentified species previously isolated from cranberry plants with decline symptoms in Wisconsin (15), which now are believed to be pathogenic (S. N. Jeffers and M. J. Drilias, *unpublished*). *P. compacta* is parasitic on leaves and fruit of cranberry but does not cause economic damage (24). The seasonal occurrence of this fungus (Table 2) undoubtedly was underestimated because it grew very slowly and deep into the agar. Consequently, colonies that probably were *P. compacta* frequently were overgrown on isolation dishes before they could be transferred and positively identified.

*B. vaccinii* and *P. vaccinii* were, by far, the most frequently encountered pathogens during this study. Previously, they have been isolated commonly from symptomless leaves and berries by others (4,6,12,29). *B. vaccinii* causes subcuticular latent infections of cranberry leaves (30) and typically is associated only with the epidermis of symptomless and speckled cranberry fruit (6,14). Conversely, *P. vaccinii* does not cause latent infections but apparently persists as dark-pigmented appressoria on the surfaces of leaves and berries without causing infection (4,23). The two distinct color types of *P. vaccinii* that were observed have been reported previously (4,12) and probably should be separated into two species (4). Fortunately, neither *B. vaccinii* nor *P. vaccinii* presents a serious economic threat to cranberries grown in Wisconsin (4,12,29).

Three fungi isolated from leaves and berries were listed as possible pathogens (Table 2). *Colletotrichum* could be the anamorph of *G. cingulata*, but this connection could not be confirmed because perithecia were not produced on any medium. *Penicillium* spp. have not been confirmed as primary pathogens, although they are isolated commonly from rotted fruit in storage (1,14,16,24). Recently, binucleate *Rhizoctonia*-like fungi have been implicated as root pathogens, but pathogenicity has not been demonstrated (9).

The presence of numerous nonpathogenic fungi in surface-disinfested, symptomless leaves and berries could be explained to some degree by insufficient or improper disinfestation as discussed above; by latent infections (28), such as those by *B. vaccinii* (30); or even by latent propagules, such as the appressoria of *P. vaccinii* (4,23). However, because of the diversity of fungi and the consistency of their isolation, a more likely possibility is that at least some of these fungi were endophytes. As defined by Carroll (8), endophytes are fungi that cause asymptomatic infections entirely within plant tissues and may serve as mutualistic symbionts. Previously, only Rayner (21) and Carris (7) had identified endophytes in cranberry; although Ericaceous plants, including several species of *Vaccinium*, are known to harbor large numbers of these fungi (18–20,31). In fact, some of the genera isolated in this study (e.g., *Apostrasseria*, *Geniculosporium*, *Nodulisporium*, *Sordaria*, *Sporormiella*, and *Septoria*) also were commonly associated with and were considered to be endophytes of these other Ericaceous hosts. However, it must be demonstrated microscopically that these fungi are actually within (i.e., have infected) cranberry plant tissues to confirm that they are truly endophytes (8). If it can be confirmed that endophytic fungi commonly occur within cranberry tissues, their role in plant health should be investigated. A beneficial role for endophytic fungi has been suggested (8).

*Alternaria* and *Cladosporium* were the most commonly occurring nonpathogens isolated from leaves and berries. Both have been isolated frequently from surface-disinfested, symptomless cranberry leaves or fruit by others (4,5,12,13,26). Although these fungi commonly occur as epiphytes, their potential role as endophytes can not be excluded (20,31). Eight genera of fungi that were not believed to be pathogens have not been reported previously from cranberry (1,10–12,23,24). These were *Ascochyta*, *Bipolaris*, *Bispora*, *Fusidium*, *Geniculosporium*, *Nodulisporium*, *Sporormiella*, and *Trichurus*. However, it is likely that *Geniculosporium* has been isolated before but was identified as *Sporotrichum* (12). *Fusidium* was identified according to Barron's description (2), although this genus is considered by some to be synonymous with *Cylindrocarpon* (11). Only *Nodulisporium* and *Sporormiella* were isolated in both years and with any regularity; *Geniculosporium* was isolated on the last three sampling dates in 1987.

Throughout this study, there was no discernible difference among the three fungicide treatments on any measure of fungus incidence. However, there were significant differences among these three treatments in the amount of rot that occurred when berries harvested from the same plots used here were put in storage (16). This discrepancy may be due to the use of pedicel ends as opposed to blossom ends of berries in isolations, as explained previously.

However, fungicide treatments as a group did affect seasonal incidence of at least some fungi. Samples of leaves and berries collected from fungicide-treated plots compared with those from untreated plots tended to yield fewer fungus colonies, a reduced incidence of *B. vaccinii*, but similar incidences of *P. vaccinii* (dark or white), *Alternaria*, or other miscellaneous fungi. The significant effect fungicide treatments had on the number of fungus colonies isolated from berries (Fig. 1C, D) may be due, in part, to the effect they had on *B. vaccinii* (Fig. 2C, D), which always was the fungus recovered most abundantly from berries during the latter half of the season (Table 2). Fungicide treatments also delayed the onset of increase in seasonal incidences of fungi by 2–6 wk, if such an increase occurred at all. This increase occurred in samples from untreated plots at 2 wk or in samples from fungicide-treated plots at 4–6 wk after the last fungicide application.

It is apparent from this research that fungicides were applied too early to most effectively reduce the seasonal incidence of fungi in cranberry leaves and fruit. Fungi were present at low levels from budbreak up to 10–12 wk later, regardless of the treatment applied. After that time, the incidence of fungi in leaves and berries began to increase. Consequently, if the three biweekly fungicide applications were started after plants had bloomed (e.g., at 10 wk after budbreak), as suggested previously (16), instead of during bloom, plants would have been protected when the incidence of fungi in leaves and berries was increasing markedly. Beginning fungicide applications after bloom also should avoid the deleterious effects that fungicides have on yield and yield components and may improve storage rot management (16).

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