# Effects of Pruning Method on the Incidence of Mummy Berry and Other Lowbush Blueberry Diseases

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**ABSTRACT**

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A shift from biennial burning to mowing as the pruning technique for lowbush blueberry (*Vaccinium angustifolium*) resulted in a 90-fold increase in mummy berry disease (*Monilinia vaccinii-corymbosi*) over six crop cycles (12 yr), relative to the disease levels present under burning. After light burning was resumed in a previously mowed field, relative disease incidence was reduced twofold to threefold in each of the two following crop cycles. Two minor diseases of lowbush blueberry, red leaf (*Exobasidium vaccinii*) and powdery mildew (*Microsphaera penicillata* var. *vaccinii*), were not decreased by burning. A return to burning will reduce disease inoculum but is not likely to produce immediate control of mummy berry disease.

Lowbush blueberry (*Vaccinium angustifolium* Aiton) is most productive after severe pruning. Traditionally, fields of naturally established plants are burned in alternate years, with vegetative growth in the first summer followed by fruit production in the second (3). Because of the increase in fuel costs in the 1970s and recognition of the damage that burning does to the surface organic layer of blueberry soils, pruning by flail mowing replaced straw-fired or oil-fired burning on many of Maine's 23,000 ha (7,8). A consequence of this change was an increased potential for pest problems. Burning contributes to suppression of spanworm, which caused severe damage in certain mowed fields in 1987 (4). Burning also destroys fungal inoculum and has been valuable in controlling diseases elsewhere (5). In Maine, the change from burning to mowing was followed in some cases by an apparent increase in severity of the crop's most important pathogen, *Monilinia vaccinii-corymbosi* (Reade) Honey, cause of mummy berry disease. Pseudosclerotia formed around infected (mummy) berries can remain viable for 2 yr or more (1,10). Apothecial production coincides with budbreak and ascospores infect developing leaves or flowers. Ascospores can be dispersed to distances of more than 300 m (11). Ovaries are infected by secondary conidia spread by watersplash or pollinators (2). Infected berries mummify and fall to the ground, where they may be killed during burning (10). Controlled trials in Nova Scotia (P. Hildebrand, *personal communication*) indicate that 10% or more of the mummies survive burning. The disease is endemic in most fields, causing little loss and often going undetected. With sufficient inoculum and wet weather at budbreak, however, the disease can be severe. In these cases, areas 1–10 m in diameter centered on genetically uniform clones may be completely defoliated. This study was undertaken to determine whether mowing increases the potential for disease, how significant such a change is, and how a return to burning would affect disease incidence in the short term.

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## MATERIALS AND METHODS

To quantify differences in disease associated with mowing and burning, sampling grids were established in 1986 at two lowbush blueberry fields in Washington County, Maine. At site 1 (University of Maine Blueberry Hill Farm, Jonesboro), comparisons were made on adjacent 0.4-ha plots. One treatment had been mowed for the previous five crop cycles; the other had been pruned only by burning during the previous 40 yr. This site was used to demonstrate the cumulative effects of a long-term shift to mowing. At site 2 (Tracy Field, Cherryfield), a 7-ha portion of a larger field was burned following two cycles in which the entire field had been mowed. This site was selected to demonstrate the short-term effects of a return to burning. Treatments at both sites were repeated for an additional cycle (burning and vegetative growth in 1988, infection and fruit production in 1989). At site 1, six parallel transects (130 m long, 10 m apart) were laid out to cross from one treatment to the other. The transects were each subdivided into six subplots (10 × 10 m) with a central 10-m space between the surveyed areas. At site 2, eight transects (210 m long, 15 m apart) were each subdivided into two sets of 10 subplots with a lane separating the treatments.

Mummy berry disease was evaluated at late bloom in 1987 and 1989 in two randomly selected 0.25-$m^2$ areas in each subplot. The number of stems showing any foliar blight symptoms was recorded. Substantially higher counts in 1989 required that corrections be made to account for multiple foliar infections on single stems. Using 600 stems per square meter and 10 leaf clusters per stem as averages, the Poisson distribution was used to estimate the total numbers of infected leaf clusters from the recorded numbers of stems with infections. Corrections were made only when foliar blight occurred on more than 100 stems per square meter, and a conservative upper limit of four infected leaf clusters per stem was placed on counts from the few severely affected subplots.

In August and September of 1986 and 1987, powdery mildew (*Microsphaera penicillata* (Wallr.:Fr.) Lév. var. *vaccinii* (Schwein.) W. B. Cooke, syn. *M. alni* (Wallr.) E. S. Salmon var. *vacinii* (Schwein.) E. S. Salmon) was quantified at site 2 by rating the percentage of discolored or mildewed leaf area. Ten leaves were rated from each subplot. Red leaf disease (*Exobasidium vaccinii* (Fuckel) Woronin) was quantified at both sites in 1987 by counting all diseased stems in a strip 3 m wide through the subplots (30 $m^2$ total area per subplot). Clusters of affected stems originating from the same rhizome node were counted as a single infected plant, and where there was heavy localized spread, the count of infections per plot was limited to 50.

Both sites were laid out as split plots to accommodate a preexisting experimental design (site 1), to accommodate commercial use of the field (site 2), and to achieve a scale in which treatment-to-treatment contamination by wind or insect-borne spores would be minimized. To detect nonrandom distribution of disease in the fields, within-treatment linear regressions were run for all data sets to determine whether disease gradients existed that could confound the interpretation of treatment effects in the split-plot design. With *Monilinia*, the 1987 data were also used as a baseline for the 1989 data to compensate for any differences in disease in the two areas not

caused by treatment effects on inoculum density or microclimate. The correlations between years for treatments at site 2 were also calculated to determine whether relative disease severity within each set of 80 subplots could be used as a predictor of future disease severity in the same subplots.

## RESULTS

***Monilinia.*** At site 1, disease in the mowed treatment was 71-fold higher than that in the adjacent burned treatment after five cycles (Table 1). This is equivalent to an average treatment difference of 2.3-fold per cycle ($71^{1/5}$) compounded five times. After six cycles (1989), the difference in disease was 90-fold. Although disease incidence varied highly among the subplots, no consistent pattern of increase or decrease (disease gradient) along the transects was detected, indicating little contamination of the burned area by inoculum from the mowed area. Site 1 received 28 cm of rain in May 1989. As a result, disease increased considerably over 1987 levels; there was a 7.5-fold disease increase in the burned treatment and a 9.4-fold increase in the mowed treatment. The apparent differential between mowed and burned areas for the 1988–1989 crop cycle was 1.3-fold, considerably lower than the 2.3-fold average for the previous five cycles.

At site 2, a return to burning for a single cycle was associated with an apparent 3.6-fold decrease in disease incidence relative to the mowed treatment. However, statistically significant disease gradients were found within both treatments. These gradients decreased along the transects from the outer end of the mowed treatment, where disease was greatest, to the outer end of the burned treatment, where disease was least. In 1987, consequently, differences in disease incidence between pruning treatments could not be differentiated from gradient effects caused by variation within the field. In 1989, after two cycles of burning, the difference in disease between the two treatments was 6.6-fold. Using the 1987 data as a base, the difference resulting from burning in 1988 was 1.8-fold. In 1989, only the burned plot had a significant gradient. In this case, the 24 subplots within 25 m of mowed areas (30% of the total) accounted for 76% of the recorded disease, suggesting that the burned treatment was being contaminated by the (upwind) mowed area. If this is true, the calculated 1.8-fold difference between treatments is artificially low. Calculations using 1987 and 1989 data from only those 70% of the plots that were more than 25 m from mowed areas indicate a disease differential for 1989 of 4.1-fold rather than 1.8-fold. In 1989, several small, partially defoliated foci were present in the mowed fields at each site, but disease was not sufficent to cause significant economic loss. The correlations between square root-transformed disease counts between individual subplots in 1987 and 1989 were significant: $r = 0.437$ for the mowed plots and $r = 0.333$ for the burned plots (79 df). However, these correlations are too low to allow use of limited disease scouting as the basis for applying or scheduling of fungicide treatments in the next crop cycle.

***Exobasidium.*** Amounts of red leaf disease were not affected by pruning treatment at either site in 1987 (Table 2). Distribution of the disease within treatments was random.

***Microsphaera.*** At site 2 in 1986 (the vegetative year), powdery mildew was 2.5-fold higher in the burned treatment than in the mowed treatment (Table 2). Leaf phosphorus concentrations, a factor associated with susceptibility to mildew (12), did not differ significantly between treatments (0.146% for the mowed treatment, 0.144% for the burned treatment). Distribution of disease within treatments was random. In the subsequent crop year, differences between treatments were not significant.

## DISCUSSION

Results at site 1 indicate that past burning has kept *Monilinia* levels low by destroying overwintering pseudosclerotia. Minimal burning, intended only to scorch blueberry stems (and perennial weeds), has a modest effect on disease in single cycles. The calculated 2.3-fold average difference between treatments in disease per cycle at site 1 and the similar values obtained at site 2 indicate that such burns reduce disease and, presumably, the number of pseudosclerotia by only 50–60% on average. As the differences in disease differentials for 1987 and 1989 indicate, the efficacy of burning varies from year to year; efficacy is lower when the soil is wet. However, the compounded effect of repeated burnings or mowings is substantial, resulting in a 90-fold difference in disease after 12 yr. Conversely, a return to light burning may be expected to reduce disease as slowly as the shift from burning to mowing increased it. Results from site 2 indicate that the differential between mowing and burning develops at about the same rate regardless of the previous pruning practice on which these treatments were superimposed. More intensive burning should hasten this process, but a decision to change pruning practice should not be made on the basis of disease control alone.

*E. vaccinii*, the cause of red leaf disease, is systemic and overwinters as mycelium in rhizomes (9). Sprouts on burned plants develop directly from nodes along the subterranean rhizomes, whereas mowed plants sprout from aboveground stubs (3). Apparently, stems left after mowing are not an important reservoir for the disease organism.

The relative importance of foliar cleistothecia vs. stem mycelium or cleistothecia as overwintering structures is not known for powdery mildew in lowbush blueberry. Both sources are destroyed by burning. However, disease

**Table 1.** Differential effects of pruning method on incidence of *Monilinia vaccinii-corymbosi* for two consecutive crops at two sites

| | | Diseased buds/m$^2$ [a] | | | | Disease gradient[d] | |
|---|---|---|---|---|---|---|---|
| Site | Year | Burned | Mowed | Difference[b] | Crop cycles[c] | Burned | Mowed |
| Site 1 | 1987 | 0.55 | 39.50 | 71.1 | 5 | None | None |
| | 1989 | 4.12 | 369.39 | 89.8 | 6 | None | None |
| Change | | 7.5 | 9.4 | 1.3 | | | |
| Site 2 | 1987 | 5.56 | 19.85 | 3.6 | 1 | B ⟶ M | B ⟶ M |
| | 1989 | 6.85 | 44.97 | 6.6 | 2 | B ⟶ M | None |
| Change | | 1.2 | 2.3 | 1.8 | | | |

[a] A unit of infection is made up of all leaves developing from a single foliar bud. Differences between treatments were highly significant ($P < 0.001$) at both sites in both years.
[b] The ratio of disease in the mowed treatment to that in the burned treatment. Change is the ratio of disease in 1989 to that in 1987.
[c] Number of 2-yr crop cycles following establishment of the mow or burn treatments.
[d] Direction along transects in which a statistically significant increase in disease incidence occurred. Regression analyses were done within treatments and evaluated at the $P < 0.05$ level. B ⟶ M indicates that disease increased along the transects in the burned-to-mowed direction.

**Table 2.** Incidence of red leaf disease (*Exobasidium vaccinii*) and powdery mildew (*Microsphaera penicillata* var. *vaccinii*) as affected by pruning treatment

| | Red leaf[a] | | Powdery mildew[b] | |
|---|---|---|---|---|
| Treatment | Site 1 | Site 2 | 1986 | 1987 |
| Mowed | 0.29 | 0.34 | 10 | 20 |
| Burned | 0.34 | 0.36 | 26 | 25 |

[a] Infected stem clusters (rhizome nodes) per square meter. Treatments surveyed in 1987.
[b] Percentage of leaf area affected. Treatments surveyed at site 2. The difference in powdery mildew between treatments was significant at $P < 0.05$ in 1985 but not 1986.

severity is not higher in mowed areas, despite the greater potential for inoculum survival. Visual estimates of powdery mildew at both sites in 1988 did not indicate that the greater incidence of mildew on newly burned land, seen in 1986, is a consistent phenomenon.

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