Resistance to the Fruit Infection Phase of Mummy Berry Disease in Highbush Blueberry Cultivars

A.W. Stretch¹ and M.K. Ehlenfeldt²

U.S. Department of Agriculture, Agricultural Research Service, Rutgers Blueberry and Cranberry Research and Extension Center, 125A Lake Oswego Road, Chatsworth, NJ 08019

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Abstract. Sixty-eight highbush blueberry (*Vaccinium corymbosum* L.) cultivars and selections were evaluated over 3 years for their resistance to the fruit infection phase of mummy berry disease [*Monilinia vaccinii-corymbosi* (Reade) Honey]. Average incidence of fruit infection under test conditions was 34.3% in 1995, 14.4% in 1996, and 27.9% in 1997, with significant differences occurring among clones in all 3 test years. Several cultivars exhibited consistent resistance to mummy berry fruit infection across all years of testing. ‘Northsky’, ‘Reka’, ‘Northblue’, ‘Cape Fear’, ‘Bluegold’, ‘Puru’, and ‘Bluejay’ were among the most resistant, and ‘Atlantic’, ‘Berkeley’, ‘Herbert’, and ‘E-176’ were among the most susceptible. The consistent resistant reaction of certain cultivars indicates that they may be suitable as parents for introducing resistance into a breeding program. No significant correlation was observed between blighting resistance and fruit infection resistance.

Mummy berry, caused by the fungus *Monilinia vaccinii-corymbosi*, is the most important widespread fungus disease of blueberry in North America (Eck, 1988). Pepin and Toms (1969) estimated the yearly annual crop loss to mummy berry in British Columbia to be 8% to 10%, and Wallace et al. (1976) cited a crop loss of 70% to 85% in 1974 in New Hampshire. The fungus overwinters as pseudosclerotia or mummified fruit on the soil surface, and in early spring apothecia develop from the pseudosclerotia (Batra, 1983). Primary infection occurs when ascospores, forcibly ejected from apothecia, infect noncuticularized leaf, stem, and flower-bud tissues, and cause blighting of the new growth. In the secondary infection phase, conidia produced on the surface of blighted tissue are carried by wind and pollinating insects to flower stigmata, where they germinate and grow down the style into the ovary. Fungal tissue colonizes and fills the fruit locules, with no visible exterior symptoms until near harvest when infected berries turn a salmon or cream color, begin to dry up (mummify), and drop to the ground.

Two field studies have evaluated levels of resistance to fruit infection by *M. vaccinii-corymbosi* in highbush blueberry cultivars, and several common cultivars have been rated

as susceptible. ‘Jersey’, ‘Rubel’, and ‘Weymouth’ were among those found to be susceptible by Pepin and Toms (1969) in British Columbia, and ‘Bluecrop’, ‘Bluehaven’, ‘Blueray’, and ‘Jersey’ were among those found susceptible by Nelson and Bittenbender (1971) in Michigan. However, the results of the two studies were not in complete agreement in their evaluations of several other cultivars. Our objective was to survey a wider range of cultivars under more controlled nursery conditions and to identify sources of resistance that might be used in a breeding program.

Materials and Methods

A group of 68 cultivars and selections was assembled as the screening population for this study (Table 1). Several selections not recognizable to the general public were included for their potential breeding value, including 11-104, a sibling of ‘Bluecrop’ and ‘Blueray’; 1613-A, an old selection from Whitesbog, N.J., marketed on the west coast as ‘Hardyblue’; ‘John Brown’, a drought-tolerant selection made by a New Jersey grower; E-176 (F-72 x Berkeley), a selection reported to have good overall mummy berry resistance (Nelson and Bittenbender, 1971); and F-72 (Wareham x Pioneer), a selection present in the pedigrees of several cultivars. ‘Ama’ and ‘Heerma’ are German cultivars that are reported to have resistance to *Phomopsis vaccinii* Shear (Teodorescu, et al., 1985).

The plants were potted in 3-L pots in a 1 sand : 1 peat (v/v) planting mix. The test was conducted in a coldframe covered with shade-cloth (55% transmission) as a randomized complete-block design with four replications in 1995 and five replications in 1996 and 1997,

with one pot of each cultivar per replication. In 1995, a group of 52 cultivars and selections was arranged in a 4 x 14 grid pattern in each replication (208 plants total), with 0.36 m between plants and 0.45 m between rows. In 1996 and 1997, an additional row was added to the grid, bringing the total number to 68 (340 plants total). In all years, sporulating, blighted plants of ‘Bluehaven’ and ‘Blueray’ in 3-L plastic pots were evenly distributed between every two rows of plants to serve as inoculum sources within 0.5 m or less of target plants. In previous studies, ‘Bluehaven’ and ‘Blueray’ had averaged 63.9% and 35.6% blight, respectively, and had ranked as 2 of 47 and 11 of 47 with respect to susceptibility (Ehlenfeldt, et al., 1996). Additional plants were added as available to supplement inoculum levels. To facilitate transfer of conidia from the blighted tissue to the stigmata, two hives of honeybees were placed in the netted coldframe, one at each end of the experimental area in 1995 and 1996, and both at one end in 1997. To insure that the resultant fruit developed only under the imposed conditions of the experiment, open flowers were removed from the test plants prior to setting the inoculum plants and bees in the coldframe. The experimental area was sprinkler-irrigated (22 mm·day⁻¹) every evening during conidium production to enhance infection. Fruit were picked while still green, about 4 to 6 weeks after flowering, and stored at 2 °C until evaluated. To evaluate for infection, each fruit was sliced in half and observed for the distinctive white fungal growth pattern of mummy berry.

The incidence of mummy berry fruit infection, expressed as a percentage of the total number of fruit on a plant, was subjected to statistical analysis using the general linear model (GLM) procedure of the Statistical Analysis System (SAS Institute, 1998). An arcsine transformation of the data was evaluated, but was found to yield essentially equivalent results; therefore, results are reported as nontransformed values. Means were separated with the Waller-Duncan k-ratio *t* test.

Results

Fruit infection was successful in all 3 years, but average infection levels varied considerably (Table 1). Average infection levels in 1995, 1996, and 1997 were 34.3%, 14.4%, and 27.9%, respectively. Although significant correlations could be demonstrated between infection levels in any given pair of years ($r_{1995, 1996} = 0.67, P \leq 0.0001, r_{1995, 1997} = 0.48, P \leq 0.0001, r_{1996, 1997} = 0.48, P \leq 0.0001$), analysis of variance of combined years exhibited significant cultivar x year effects ($F = 1.89, P \leq 0.0001$). Hence, cultivar data were analyzed individually for each year. Incidence of mummy berry fruit infection differed significantly among entries in all 3 years (Table 1).

In 1995 (N = 51), mummy berry fruit infection ranged from 6.0% to 61.3% ($F = 2.81, P \leq 0.05$). The minimum significant difference for mean separation of genotypes was 23.5%. ‘Northsky’, ‘Northblue’, ‘Bluejay’,

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¹Research Plant Pathologist.

²Research Geneticist. To whom requests for reprints should be addressed. E-mail address: mehlenfe@ag.gov

Table 1. Percentage of fruit infection caused by *Monilinia vaccinii-corymbosi* on 68 highbush blueberry cultivars and selections. Cultivars arranged according to infection levels.

Cultivar	Fruit infection incidence (%) ^z				
	1995	1996	1997	1995–1997 mean	1996–1997 ^y mean
Atlantic	60.0 ^a ab ^w	36.4 a	70.3 a	55.6	53.4
Berkeley	53.5 a-d	33.8 ab	65.7 ab	51.0	49.7
Herbert	53.8 a-c	19.8 b-j	61.1 a-c	44.9	40.4
E-176	52.2 a-e	28.6 a-e	38.2 d-j	39.7	33.4
Lateblue	---	27.0 a-f	51.6 a-e	---	39.3
Bluehaven	45.0 a-j	---	33.0 e-j	---	---
Ivanhoe	45.7 a-i	16.8 d-n	50.0 a-f	37.5	33.4
Elliott	37.3 b-m	19.1 c-l	55.4 a-d	37.3	37.3
1613-A	36.9 b-m	15.4 d-o	58.8 a-d	37.0	37.1
Angola	61.3 a	19.6 b-j	29.2 g-m	36.7	24.4
Elizabeth	42.5 a-k	15.0 e-o	52.1 a-e	36.5	33.6
Croatan	52.9 a-d	36.8 a	19.2 j-s	36.3	28.0
Bluechip	47.3 a-h	15.3 d-o	45.5 b-i	36.0	30.4
Stanley	43.9 a-j	16.2 d-o	44.8 c-i	35.0	30.5
Ama	46.3 a-h	12.3 g-p	46.5 b-h	35.0	29.4
Earliblue	36.2 c-m	18.8 c-m	49.0 b-g	34.7	33.9
Murphy	47.3 a-h	29.7 a-d	26.6 h-q	34.5	28.1
Burlington	28.3 f-o	17.5 c-m	57.0 a-d	34.3	37.2
Blueray	42.2 a-k	31.8 a-c	27.7 h-p	33.9	29.7
11-104	42.2 a-k	17.7 c-m	38.4 d-j	32.7	28.0
Concord	42.7 a-k	8.8 i-p	46.0 b-h	32.5	27.4
John Brown	28.6 f-o	9.0 i-p	56.0 a-d	31.2	32.5
Dixi	31.3 c-n	17.5 c-m	44.1 c-i	31.0	30.8
Katharine	30.1 d-n	15.6 d-o	44.9 c-i	30.2	30.3
Toro	39.3 a-k	20.0 b-j	30.6 f-l	30.0	25.3
Jersey	38.6 a-l	14.9 e-o	32.8 e-l	23.8	23.9
F-72	37.4 b-m	19.0 c-l	30.1 f-l	28.8	24.5
Morrow	44.8 a-j	20.3 b-i	19.2 j-s	28.1	19.7
Rubel	51.1 a-f	9.6 h-p	21.4 j-r	27.4	15.5
Collins	36.8 b-m	12.6 f-p	30.3 f-l	26.6	21.5
Pemberton	45.3 a-j	9.6 h-p	24.8 i-q	26.6	17.2
Pioneer	30.3 c-n	20.3 b-i	22.4 j-r	24.3	21.4
Sierra	48.8 a-g	19.8 b-j	3.6 rs	24.1	11.7
Darrow	31.5 c-n	19.4 b-k	20.9 j-s	23.9	20.2
Cooper	---	20.2 b-i	26.0 h-q	---	23.1
Spartan	37.7 b-l	14.0 f-p	14.9 k-s	22.2	14.4
Blue Ridge	---	26.8 a-g	17.2 k-s	---	22.0
Bluecrop	27.4 g-o	12.5 f-p	26.0 h-q	21.9	19.2
Legacy	---	4.4 m-p	39.1 d-j	---	21.7
Rancocas	48.2 a-h	7.2 i-p	8.6 m-s	21.3	7.9
Wareham	28.0 f-o	16.4 d-o	19.2 j-s	21.2	17.8
Nelson	---	23.6 a-h	18.6 j-s	---	21.1
Bluetta	39.5 a-k	17.9 c-m	3.4 rs	20.2	10.6
Harrison	22.4 i-o	9.3 h-p	26.4 h-q	19.4	17.9
Bounty	---	10.6 h-p	27.8 h-o	---	19.2
Wolcott	36.3 c-m	8.5 i-p	12.5 k-s	19.1	10.5
Top Hat	---	7.9 i-p	28.6 g-n	---	18.2
Heerma	25.9 g-o	8.4 i-p	18.6 j-s	17.6	13.5
Duke	22.1 j-o	10.4 h-p	19.8 j-s	17.4	15.1
Sunrise	---	11.6 h-p	22.8 j-r	---	17.2
Olympia	---	6.6 i-p	26.9 h-q	---	16.7
Harding	22.3 i-o	4.7 l-p	22.5 j-r	16.5	13.6
Coville	20.0 k-o	7.4 i-p	20.7 j-s	16.0	14.0
Meador	---	12.0 h-p	19.2 j-s	---	15.6
Northland	22.6 i-o	6.0 i-p	13.7 k-s	14.1	9.8
Sharpblue	---	7.7 i-p	20.3 j-s	---	14.0
June	25.6 g-o	8.1 i-p	7.8 o-s	13.8	7.9
Cabot	28.6 e-o	2.1 op	7.1 p-s	12.6	4.6
Patriot	24.6 h-o	2.3 n-p	6.9 r-s	11.3	4.6
Reveille	---	4.9 k-p	17.2 k-s	---	11.1
Weymouth	15.2 l-o	8.9 i-p	8.5 n-s	10.9	8.7
Bluejay	13.9 m-o	8.3 i-p	6.6 r-s	9.6	7.5
Puru	---	6.1 i-p	12.8 k-s	---	9.4
Bluegold	---	4.5 l-p	12.3 l-s	---	8.4
Cape Fear	---	0.0 p	14.4 k-s	---	7.2
Northblue	12.4 no	5.4 j-p	3.6 rs	6.8	4.5
Reka	---	6.9 i-p	2.8 rs	---	4.9
Northsky	6.0 o	0.0 p	0.4 s	2.1	0.2
Average	34.3	14.4	27.9		

^zAverage percentage of incidence across four plants per cultivar in 1995, and across five plants per cultivar in 1996 and 1997.

^yMean separation by Waller-Duncan k-ratio *t* test, $P \leq 0.05$.

and ‘Weymouth’ were among the most resistant to fruit infection, and were included in a group of 17 cultivars that did not differ statistically. ‘Angola’, ‘Atlantic’, ‘Herbert’, ‘Berkeley’, ‘Croatan’, and ‘E-176’ were among the most susceptible cultivars in a group of 24 cultivars that did not differ statistically.

In 1996 ($N = 67$), differences also were significant ($F = 3.12, P \leq 0.05$). Infection level was lower in 1996 than in 1995, ranging from 0.0% to 36.8%. The minimum significant difference for mean separation (14.6%) encompassed a low infection group of 35 cultivars ranging from ‘Northsky’ (0%) to ‘Elizabeth’ (15%). ‘Croatan’, with a disease incidence of 36.8%, had the highest infection level in a susceptibility group that included nine cultivars.

Overall the infection level was higher in 1997 than 1996, ranging from 0.4% to 70.3%. Again, differences among cultivars for resistance to fruit infection were significant ($F = 5.23, P \leq 0.05$). The minimum significant difference for mean separation (20.6%) encompassed a low infection group of 29 cultivars ranging from Northsky (0.4%) to Darrow (20.9%). ‘Atlantic’ (70.3% disease incidence) had the highest infection level in a susceptibility group that included 10 cultivars. A highly significant difference among replications was observed in 1997, but not in 1995 or 1996. Examination of the data by replicate indicated that, generally, fruit infection levels increased from replicate 1 to replicate 5 (data not shown). Replication 5 was closest to the bee hives.

Two- and three-year means for the 68 cultivars indicated that several cultivars were consistently resistant to mummy berry fruit infection (Table 1). Among these, from most to least resistant (2.1% to 9.6%) were ‘Northsky’, ‘Reka’, ‘Northblue’, ‘Cape Fear’, ‘Bluegold’, ‘Puru’, and ‘Bluejay’. Among the most susceptible cultivars, in descending order, were ‘Atlantic’ (55.6%), ‘Berkeley’, ‘Herbert’, ‘E-176’, ‘Lateblue’, and ‘Bluehaven’ (39.0%). For many of the cultivars, there were year-to-year shifts in the percentage of fruit infection. The most dramatic susceptibility changes were exhibited by ‘Croatan’ (52.9% to 36.8% to 19.2%), ‘Sierra’ (48.8% to 19.8% to 3.6%), ‘Rancocas’ (48.2% to 7.2% to 8.6%), ‘Bluetta’ (39.5% to 17.9% to 3.4%), and ‘Legacy’ (4.4% to 39.1%).

Resistance to fruit infection did not appear to be significantly correlated with previously determined blight resistance. Comparison of 3-year average blight levels of 48 cultivars (Ehlenfeldt, et al., 1996) with their 3-year fruit infection levels, derived from this study, showed no significant correlation ($r = -0.245, P = 0.093$).

Discussion

This study, comprising 52 cultivars in 1995, 67 in 1996, and 68 in 1997, is the most comprehensive evaluation of resistance to the fruit infection phase of mummy berry in highbush blueberry to date. There appears to be substantial variability for resistance to the fruit-infect-

ing phase of mummy berry disease, although this variability appears to be influenced by environmental differences among years. However, the fact that certain cultivars consistently appeared to be resistant suggests that they would be good parents for introducing resistance into a breeding program. The levels of infection across generally unrelated cultivars formed a continuous distribution, suggesting that resistance may be quantitative and that major gene segregation is not occurring. Clones with larger percentages of *V. angustifolium* ancestry (e.g., 'Northsky', 'Northblue', and 'Bluegold') were among the most resistant types; however, an examination of ancestry across all selections did not suggest that there was any consistent correlation between the percentage of lowbush germplasm and resistance.

As had been noted previously for blight screening (Stretch, et al., 1995), some of our results conflict with other earlier field-based studies from various locations. Our evaluations were done in a nursery environment with abundant conidia available over a prolonged period of time, and had the advantage of more controlled infection conditions and more replications than in previous studies. If our evaluations indeed provided better inoculum, it is easy to dismiss discrepant results where we found susceptibility, but others found resistance. Several such instances exist. We found 'Atlantic' to be highly susceptible; Pepin and Toms (1969) found it resistant. We found 'E-176' highly susceptible (65 of 68 cultivars); Nelson and Bittenbender (1971) ranked it as their most resistant selection. More puzzling are the cases where we rated cultivars resistant when others found them susceptible. We found 'Bluejay' to be highly resistant (7 of 68), whereas Nelson and Bittenbender (1971) ranked it second most susceptible out of what were, eventually, 16 named cultivars. Similarly, we found 'Weymouth' resistant (8 of 68), whereas Pepin and Toms (1969) rated it as susceptible. The likely explanation for these discrepancies lies in either fungal variability or differences in environmental conditions leading to differences in plant/pathogen phenology.

The three studies cited above, represent three different geographical and climatic environments: British Columbia (Pepin and Toms, 1969), Michigan (Nelson and Bittenbender, 1971), and New Jersey. Pepin and Toms (1969) noted that the differences between long, cool springs in British Columbia and the relatively shorter springs of the eastern United States might result in escapes under one or the other set of conditions. Lehman and Oudemans (1997) have demonstrated variation between fungal populations for timing of apothecial maturation, leading to ascospore release periods that appear to coincide with their host cultivar's shoot development.

Conidium availability and stigmatic receptivity may vary in a fashion similar to that demonstrated for ascospore availability and vegetative growth stage (Ehlenfeldt, et al., 1996, 1997; Lehman and Oudemans, 1997) whereby resistance in some cases is achieved by avoidance. Such variability may account for both discrepancies between locations and for year-to-year variability observed within locations. The possibility of differentially virulent strains of the fungus would satisfactorily explain many of the discrepant results observed. To date, differential virulence has not been documented, but sufficient information is available to design an experiment to test the possibility. The differential reactions observed among cultivars across locations remains a concern for the widespread utility of our screening results; however, the results should be reliable for the northeastern United States.

Interactions of cultivar, pathogen, and environment may all affect disease levels, as may dispersion of spores. Bees are an essential factor in the spread of mummy berry disease because they act as vectors for conidial movement from blighted tissue to the flower stigma (Batra and Batra, 1985). Activity of honey bees is greatly influenced by environmental conditions. Batra and Batra (1985) demonstrated that bees were attracted by the ultraviolet radiation from blighted, conidium-bearing blueberry tissue, which was of a wavelength similar to that reflected by blueberry flower corollas. The significant replicate difference in mummy berry fruit infection in 1997 may have been a result of bee hive placement and/or the amount of sunlight reflecting off the blighted tissue. In 1997, the replicates were oriented in an east-west direction with the bee hives on the western end of the coldframe (the end that received morning sun sooner than the more shaded east end). The higher level of ultraviolet energy at the west end may have attracted more bees to the blighted tissue, and fostered more frequent transfers of conidia and pollen to the flowers in replicates 3, 4, and 5. This may have produced the observed differences among replicates in frequency of mummy berry infected fruit. Bees may also play a factor in disease frequency. Empirical observations have suggested that bees find flowers of some cultivars more attractive than others (Filmer and Marucci, 1963). It is possible (but certainly unproven) that for some cultivars, bees might be less likely to visit conidial masses on blighted tissue if the flowers were more attractive. This complicating possibility could be avoided if inoculation was done by hand; however, this would substantially limit the amount of screening that could be done. In this study, we tried to assure that conidial inoculum was abundant and present over an extended period of time so that any avoidance mechanisms would be neutralized and escapes minimized.

Resistance to fruit infection did not appear to be significantly correlated with blight resistance, although one might expect that it would be if the cultivars had undergone some empirical selection for disease resistance in the course of evaluation. However, screening for both blight and fruit infection were conducted with inoculum levels that far exceeded what might occur under average field selection conditions. Therefore, some cultivars might show better general resistance to both phases under more natural conditions than they did here. A few cultivars such as Bluejay, Duke, and Reka have shown significant resistance to both phases of mummy berry disease, even under high inoculum loads, and are promising parents for breeding future cultivars with excellent natural resistance levels.

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