Vulnerability of Stem-End Scars of Blueberry Fruits to Postharvest Decays

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ABSTRACT

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The principal locus of infection in harvested blueberry fruits was the stem scar. Ninety percent of all decays that developed in blueberries held for 2, 4, and 6 days at 21 C occurred at stem scars. The incidence of stem-end decay in stemless berries was 10-fold greater than in berries with their stems attached.

Additional key words: Alternaria rot, gray mold rot, *Vaccinium*.

Decay is the most important factor that shortens the shelf life of fresh-market blueberry fruits (*Vaccinium corymbosum* L.). Although the causal agents of the important postharvest diseases of blueberry fruits have been identified (1, 2, 4), the loci of infection on the fruit have not been evaluated. Such an evaluation has become especially important with the advent of machine harvesting of blueberries. Until recently, mechanically harvested fruit was used only for processing. However, machines now are used to harvest blueberries for the fresh market, and this practice is expected to increase significantly in the future. Mechanical harvesting increases bruising of the berries and, hence, predisposes fruit to postharvest decay (3).

Most postharvest decays are caused by pathogens that infect blueberry fruits through wounds and other weakened sites. Although the stem scars of blueberries frequently appear to be infected by postharvest pathogens, the incidence and identification of the agents causing decay at the stem scar have not been documented. Our study was made to evaluate the role of the stem scar in postharvest decay of blueberry fruits.

MATERIALS AND METHODS

Three separate tests were conducted on New Jersey-grown berries during 1975. A different blueberry cultivar was used in each test. Hand-harvested berries from commercial plantings of the Collins, Coville, and Jersey cultivars were used in tests done in early, middle, and late July, respectively. Decay development was determined in each test sample of stemless berries and berries clustered on stems which were held in conventional, molded, pulpboard blueberry containers.

In each test, 12 samples, each comprising 100 fruits, were divided into three sublots. The samples from two sublots were transferred to plastic, open-mesh, 0.473-liter baskets and immersed for 30 seconds in tap water or 0.025% chlorox solution; each contained 0.01% (v/v) Tween-80 to facilitate wetting. The remaining sublot was maintained as an untreated dry control. After several minutes during which excess moisture drained from the treated samples, the berries were transferred back to the pulpboard containers. The dry and treated samples were stored at 21 C and 85% relative humidity. Four replications of each treatment were examined after 2, 4, and 6 days.

RESULTS AND DISCUSSION

Regardless of treatment and incubation time, decay was six to 10 times more prevalent on stemless blueberries than on berries attached to stems (Table 1). Although a total of 59 decay spots on stemless berries originated at wound sites and skin breaks, the most frequent loci for initial infections were stem scars. For example, stem-end infections totaled 913 (8.5%) in stemless berries and only 82 (0.8%) in berries with stems. Berry decay initiated by fungus growth from infected stems was negligible. The small amount of stem-end infections in attached berries generally occurred in cracks at the stem end or at exposed stem scar tissue at the interface of the stem and berry.

Decay caused by *Alternaria* sp. was the most prevalent and gray mold rot (*Botrytis cinerea* Pers. ex Fr.) was the next most prevalent. Most of the rots in the "other" category were not identified, but Alternaria and gray mold rots predominated in a few small samples from this category that were held until recognizable signs of the causal agent had developed. Also identified in these samples were species of *Pestalotia, Rhizopus, Aspergillus,* and *Phoma.*

These data indicate the need for overcoming the vulnerability of stem scars to postharvest decay organisms. Wetting the berries increased the incidence of decay which was not controlled effectively by dipping in chlorine solution. Others (5) have reported that, under certain conditions; e.g. hydro-separation of mechanically harvested berries, a chlorine dip decreased decay. However, all available data indicate that keeping the

TABLE 1. Incidence of decay of treated and nontreated blueberry fruits with or without stems after 2, 4, and 6 days of storage at 21 C[a]

Berry treatment	Days at 21 C											
	2				4				6			
	AR[b] (%)	GMR[c] (%)	Other[d] (%)	Total (%)	AR[b] (%)	GMR[c] (%)	Other[d] (%)	Total (%)	AR[b] (%)	GMR[c] (%)	Other[d] (%)	Total (%)
With stems												
Dry	0.1	0.0	0.0	0.1[e] a	0.2	0.2	0.2	0.6[e] a	0.2	0.1	0.6	0.9[e] a
Chlorine	0.0	0.0	0.2	0.2 a	0.3	0.1	0.5	0.9 ab	0.8	0.8	0.8	2.4 ab
Water	0.1	0.0	0.1	0.2 a	1.2	0.2	1.0	2.4 ab	1.8	0.5	1.3	3.6 ab
Without stems												
Dry	0.1	0.0	0.6	0.7 a	1.9	0.4	1.5	3.8 b	2.3	0.6	3.8	6.7 b
Chlorine	0.3	0.0	1.1	1.4 b	3.9	0.2	2.4	6.5 c	5.8	1.5	8.3	15.6 c
Water	0.5	0.3	1.3	2.1 c	7.9	1.2	7.7	16.8 d	10.2	3.0	14.1	27.3 d

[a]Rot incidence based on average of three tests of twelve 100-berry samples per storage period for all treatments.
[b]Alternaria rot caused by *Alternaria* spp.
[c]Gray mold rot caused by *Botrytis cinerea*.
[d]Mostly unidentified rots, but Alternaria and gray mold rots predominated in a few small samples from this category that were held until recognizable signs of the causal agent had developed.
[e]Means in column followed by the same letter are not significantly different ($P = 0.05$).

blueberries dry and protecting the stem scar from fungal invasion are requisites for prevention of postharvest decay.

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