## APPARENT REDUCTION OF LITTLE CHERRY DISEASE SPREAD IN BRITISH COLUMBIA<sup>1</sup>

Jack M. Wilks and Maurice F. Welsh<sup>2</sup>

During the years 1933 to 1948 the virus that causes little cherry disease was distributed very rapidly through the Kootenay region of British Columbia. All sweet and sour cherry trees in most Kootenay districts became infected within this 15-year period. The virus was transmitted very efficiently within orchards and the disease made sudden appearances in districts at distances of 50 miles from the nearest known source of infection.

Extension of disease spread to the remaining Kootenay districts, to other fruit-growing regions of British Columbia, and to neighboring parts of the United States was anticipated. There was particular concern for the Okanagan Valley, where the bulk of British Columbia's cherry crop is grown. This area is connected to the Kootenays by 2 highways, each about 300 miles long, traversing 2 mountain ranges.

The disease has not appeared in the Okanagan Valley, and there has been no evidence of spread from the Kootenays to other fruit-growing regions of the Pacific Northwest. The several reported appearances of little cherry disease in other widely separated regions (7, 5) can be more logically ascribed to spread of the virus from ornamental flowering cherry to sweet or sour cherry (8).

Meanwhile, within the Kootenay region the rate of spread of little cherry virus appears to have subsided since 1948. This assessment is supported by records of spread within orchards and by data on district-to-district spread.

## Spread within experimental plantings

The rapid rate of spread pertaining during the early history of the disease was demonstrated in the isolated healthy orchard used for transmission tests in 1943 (3). In that year 20 sweet cherry trees were bud-inoculated. In 1944, 16 of these trees displayed little cherry symptoms. In 1945, the other 4 inoculated trees displayed symptoms, and 20 of the 24 uninoculated trees in the orchard were also diseased.

By contrast, spread has been slow in an experimental orchard of 52 Bing trees established in 1956 (Table 1). Sixteen of the trees have been inoculated during the years 1956 to 1963; seventeen trees have become naturally infected, and nineteen trees remained healthy in 1963. This orchard is surrounded by infected commercial orchards.

Contribution No. 158 Canada Department of Agriculture, Research Station, Summerland, British Columbia.

<sup>&</sup>lt;sup>2</sup>Plant Pathologists.

Table 1. Spread of little cherry virus disease in an experimental orchard near Creston, B.C.

Year	Trees Showing Symptoms		Trees Not Showing Sympto		Symptoms
	Inoculated	Natural Infection			
1958	8	<b>o</b>		44	
1959	4	1		39	
1960	2	4		33	
1961	0	7		26	
1962	1	5		20	
1963	1	0		19	
Total	16	17			

Spread within commercial orchards

Surveys for little cherry disease in commercial orchards have also indicated contrasting patterns of spread during the two periods. In 1946, 155 infected trees were found in a survey of 5 selected orchards in the Creston district (2). In 1947, 568 additional trees in these orchards were showing symptoms. By 1949, essentially all the cherry trees in this, and most other Kootenay districts were infected and, in newly planted orchards, young trees often produced little cherry symptoms the first year that they fruited.

During the last 10 years, however, despite recommendations discouraging new plantings of sweet cherry in the Kootenay region, a limited acreage has been replanted with young cherry trees. Most of these trees remain healthy, although neighboring sources of infection are abundant.

Spread to new districts

Surveys have shown a similar depression of the spread of the disease to new districts. By 1949 the disease had reached all Kootenay districts except several communities on the Arrow Lakes, at the western edge of the Kootenay fruit-growing region (Fig. 1). Surveys in that season indicated that all cherry trees were diseased as far west as New Denver. All orchards were healthy in Nakusp, on Upper Arrow Lake, 30 miles west of New Denver, but most trees were diseased in Makinson and Burton, on South Arrow Lake, 18 and 23 miles southwest of Nakusp. No diseased trees were found in the Fauquier-Needles and Edgewood districts,

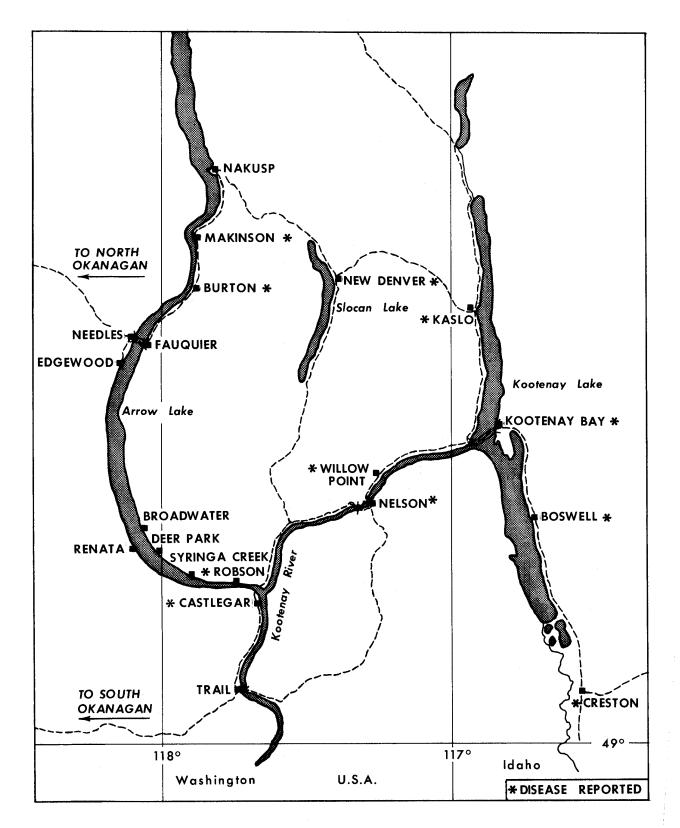


Figure 1. Distribution of little cherry virus disease in the Kootenay and Arrow Lakes districts of British Columbia.

14 and 20 miles southwest of Burton. A survey in 1960 disclosed no spread of the disease in the intervening 11 years into the Nakusp, Needles—Fauquier or Edgewood plantings. At the south end of the Arrow Lakes, by 1949, the disease had affected all trees as far west as the Robson district. There were no diseased trees at Syringa Creek, 9 miles west of Robson, or in the Renata, Deer Park and Broadwater districts, approximately 10 miles northwest of Syringa Creek. Subsequent surveys to 1962 have failed to demonstrate any spread into these 4 districts.

Thus, the disease spread during a 15-year period from its point of first discovery at Willow Point, into all trees of all orchards to points 70 miles southeast; 30 miles northeast, 40 miles west, and 65 miles northwest, yet it has not gained entry to any additional districts since 1949.

## Discussion

It seems necessary to assume that during the recent period of reduced spread, vector activity has diminished. This could be reduction in vector efficiency of the species responsible for earlier rapid spread. It could also result from the disappearance of one or more efficient vectors, as reported for mosaic of sweet potatoes in Georgia (4).

The virus causing little cherry disease has been transmitted in screenhouse and orchard tests (9) by the leafhopper, Macrosteles fascifrons (Stal). There have also been isolated experimental transmissions by Scaphytopius acutus (Say) and Psammotettix lividellus (Zett.). Orchard populations of M. fascifrons have been high in most seasons but transmission tests have indicated that its vector efficiency is so low that its ability to effect epidemic spread of the disease is hard to envisage. All of these leafhoppers were collected and tested after 1949 when the disease was no longer spreading rapidly. Thus, the collections were not necessarily representative of the orchard fauna during the period of rapid disease spread.

The populations of leafhoppers in orchards and the rate of spread of the disease have been reduced experimentally by the application of 2 sprays per year of DDT and sulphenone (10). These and other insecticides have been introduced into spray programs for apple and pear in Kootenay orchards. They should have effected some reduction of leafhopper populations on interplanted cherry trees. However, the cherry plantings in Arrow Lakes districts that have remained free from the disease are not associated with sprayed pome fruit plantings, and have themselves received no insecticide sprays.

The drop in rate of disease spread coincided with the occurrence of the most severe winter in the history of British Columbia fruit growing. On January 25, 1950 temperatures in all Kootenay districts dropped to -15°F or lower (1, 6) and thousands of fruit trees were killed. It is tempting to speculate that this uniquely rigorous winter eliminated the most effective vector or vectors from all Kootenay districts, although relatively inefficient vectors such as M. fascifrons survived to effect further slow spread of the disease.

The little cherry disease has been mentioned frequently in scientific and popular literature as an example of the epidemic proportions that can be reached by tree fruit virus diseases. Thus a report of the altered rate of spread appears to be pertinent. The cherry plantings of the Kootenays remain under surveillance, so that any return toward the former rate of spread can be detected promptly.

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CANADA AGRICULTURE RESEARCH STATION, SUMMERLAND, BRITISH COLUMBIA.