

Spread and Damage of Western X-disease of Chokecherry in Eastern Nebraska Plantings

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ABSTRACT

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The western X-disease pathogen has spread rapidly from artificially inoculated chokecherry to healthy chokecherry in experimental plantings in eastern Nebraska. Symptoms appeared on more than 60% of the chokecherry trees within 3 yr and on more than 80% of the trees within 5 yr after the X-disease pathogen was introduced. Chokecherry mortality caused by this pathogen was more than 50% within 8 yr and more than 80% after 15 yr except in one planting shaded by pines, where the mortality after 15 yr was 60%. After 9 yr, none of the American plum trees planted within rows of infected chokecherry had been killed or damaged by the western X-disease pathogen.

Chokecherry (*Prunus virginiana* L.) is used in windbreaks and as a component of wildlife plantings in the central and northern Great Plains. Western X-disease, presumably caused by a mycoplasma (2), has damaged and killed chokecherry in many of these plantings. Chokecherry plantings originally made to determine if the western X-disease pathogen is seed-transmitted (3) have been used to determine the rate of spread of the pathogen and its effect on chokecherry mortality over a period of 16 yr. The pathogen is spread by leafhoppers (4).

American plum (*P. americana* Marsh.) is used in the same type of plantings on the Great Plains. American plum also is a host of the western X-disease pathogen (1). For this reason, agencies responsible for protection plantings in the Great Plains have questioned whether American plum and chokecherry should be included in the same planting. To obtain information on this question, American plums were planted adjacent to infected chokecherry and their performance was evaluated over a period of 12 yr.

METHODS AND RESULTS

Disease development in chokecherry. Nursery-grown 2-yr-old *P. virginiana* seedlings were planted at 0.7-m spacings in three rows 3.5 m apart in the horticulture garden at the University of Nebraska, Lincoln. Row one (86 trees) was planted 3.5 m from two rows of small

pines in 1960; row two (90 trees) and row three (95 trees) were planted in 1961. Weeds were controlled in early years by hoeing around trees and mowing between rows. The western X-disease pathogen was introduced into 10 trees in row one (12%) in 1960 and into four trees (4%) in row two in 1961 by grafting bark patches from infected chokecherry. No grafts were made in row three.

All trees inoculated in late summer showed typical foliar symptoms the next

growing season, consisting of reddish margins on leaves followed by yellowing and reddening of entire leaves. The rate of disease development was similar in all three rows between 1961 and 1968, and by 1968, all trees in the three rows developed symptoms of western X-disease (Fig. 1).

Mortality rate in the three rows was similar (about 15%) by 1965 (Fig. 1). By 1968, mortality was greater (55%) in rows two and three than in row one (45%). By 1977, mortality was 90% in rows two and three and 70% in row one.

Disease development in American plum. In 1969, nursery-grown 2-yr-old American plum seedlings were interplanted among the three rows of chokecherry that had been planted in 1960 and 1961. After 9 yr, there were no visible symptoms on any of the 51 American plum trees.

Symptom development in cut-back chokecherry. In 1961 and 1962, chokecherry seeds were sown in plots located 1 km from the inoculated test seedlings.

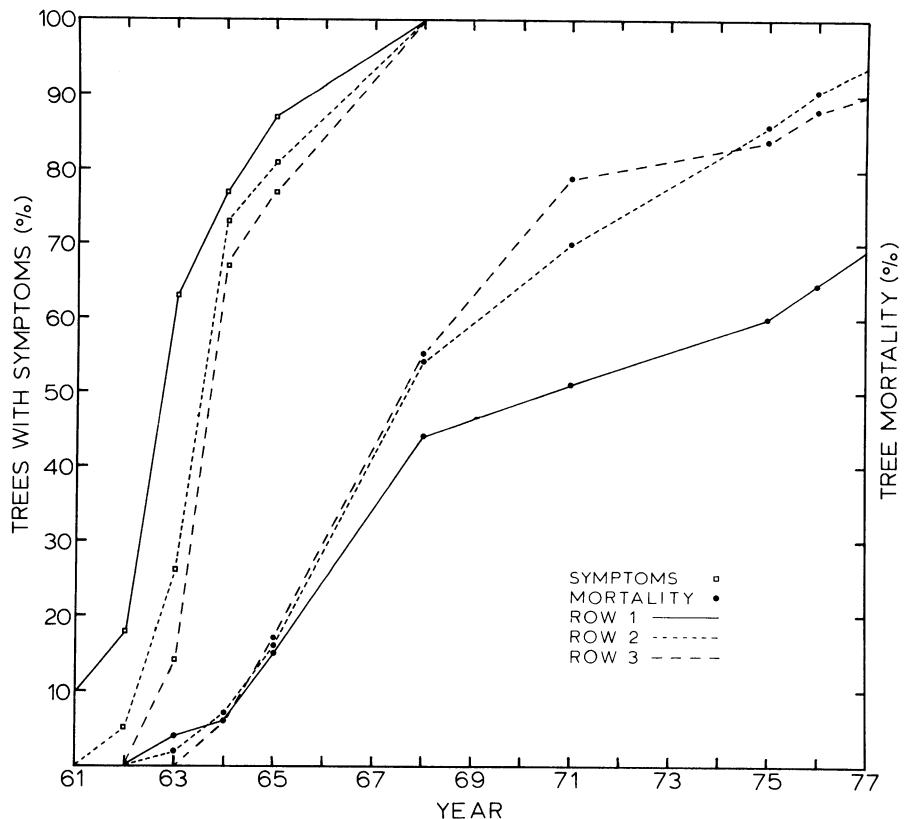


Fig. 1. Infection and mortality of chokecherry trees in eastern Nebraska experimental plantings. The western X-disease pathogen was introduced into 10 of 86 trees in row one in 1960 and into four of 90 trees in row two in 1961; none of 95 trees in row three were inoculated.

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The 174 seedlings that developed were not artificially inoculated. No infected trees were found in this plot until 1969, when two trees showed symptoms. By 1971, symptoms had appeared in 20 trees. In March 1972, all trees, except the two initially infected, were cut back to within 2 ft of the soil surface. There were no symptoms on any of the cut-back trees in 1972, but there were symptoms on three trees in 1973 and on 10 in 1974. The trees were again cut back in March 1975. There were no symptoms on the cut-back trees in 1975, but there were symptoms on two trees in 1976 and on 40 in 1977.

DISCUSSION

The results reveal that once introduced into a chokecherry planting in eastern Nebraska, the western X-disease pathogen spread rapidly. There are a number of vectors of this pathogen (4), but those involved in the rapid dispersal of the pathogen in these plots have not been identified.

The life expectancy of infected chokecherry can be estimated from the

mortality data, thereby providing information that can be used to determine when replacement plantings should be made. Mortality of heavily shaded chokecherry was lower than that of unshaded chokecherry, possibly because development of foliar symptoms was less intense on the shaded chokecherry.

The results on chronology of symptom development in previously infected chokecherry that have been cut back are consistent with the evidence that the western X-disease pathogen is present in the roots of chokecherry (1). The movement of the pathogen from roots and/or 2 ft of stem apparently was not rapid enough to produce symptoms during the growing season following the cutting back of tops. Cutting back chokecherry is not a practical method of reducing damage by the pathogen, because by the third growing season, symptoms were present on a large number of the trees that had symptoms before they were cut back.

The results with American plum trees planted adjacent to infected chokecherry

indicate that American plum could be included in plantings containing chokecherry with confidence that the western X-disease pathogen would not seriously damage the American plum. No symptoms of western X-disease were found on the American plum, and no damage was apparent. The failure to find symptoms on American plum is in accord with the findings of Gilmer et al (1). They reported that the pathogen was isolated from American plum but infected trees did not develop symptoms.

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