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TECHNICAL NOTE

PIN CHERRY AND THE BLACK KNOT DISEASE

"Do you recollect that are tree I showed you to Parrsboro? It was all covered with black knobs like a wart rubbed with caustic. Well, the plum trees had the disease a few years ago, and they all died; and the cherry trees, I concait, will go for it too. The farms here are all covered with the same black knobs, and they do look like Old Scratch. If you see a place all gone to rack and ruin, it's mortgaged, you may depend. The black knob is on it."

Thomas Chandler Haliburton in "Sam Slick"

Black knot may be a sign of mismanaged orchards but in forestry it should be considered beneficial - as a natural control agent of unwanted pin cherry. The disease can be manipulated to some extent to enhance control and although the degree of control is scarcely comparable to that of herbicides it nevertheless has potential in situations where herbicide use is restricted.

The disease can be introduced to young (1- to 4-year-old) pin cherry thickets by spreading diseased material (cut trees or branches bearing knots) from an older stand at 2- to 3-m intervals in the young thicket. This is best done in the early spring prior to bud-break and in a year when numerous sporulating knots are present in the older stand. This will enable heavy infection of most young trees within a few metres of the introduced material (a small percentage of trees in any stand are resistant and will not become infected regardless of the inoculum load). Understanding the dynamics of pin cherry growth and the development of the black knot disease will aid in manipulation.

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Pin Cherry

Pin cherry germinates from buried seed immediately after removal of the old forest stand. For the first 3 years pin cherry grows as much as 1 m per year and can quickly occupy a site to the exclusion of most other vegetation. It is a short-lived species and within the next two decades most trees succumb to competition, defoliation by insects, stem decay, and the black knot disease. It is seldom found as a full-sized tree in mature forest stands but the forest floor will be full of seeds ready to germinate when that forest is removed.

The Disease

The black knot disease is caused by the fungus Apiosporina morbosa (Schw ex Fr) Arx [=Dibotryon morbosum (Schw) Theiss and Syd.], spores of which infect newly opening buds during the spring. In our climate, the fungus has a 2-yr life cycle. Knots first appear in the following spring after infection as olive-green swellings on the previous year's growth. These knots turn black during the ensuing summer and produce a crop of spores during the second spring.

For the fungus to have a significant impact, it should be introduced during the first or second year of pin cherry development, i.e., one or two growing seasons after fire or logging. One-year-old trees will have little receptive surface for infection, mainly a single stem with few side branches but the successful infection of the main stem will have greater impact than branch infection at later stages. The younger the tree at the time of infection the more likelihood of cankering and killing the main stem and major branches; hence the greater likelihood of control. The visible effects of early introduction of the disease are shown in Figure 1.

Figure 1. Pin cherry thickets, (A) resulting from natural infection by the black knot fungus, with most of the knots on smaller branches and twigs and (B) resulting from introduction of diseased material after 1 yr of growth and cankering of the main stem and main lateral branches.

Black knot is such a common disease that we often use it as a means of identifying pin cherry. Yet, disease intensity varies with stand origin, surrounding vegetation, and stand age. In large stands originating from wild fires, pin cherry may reach a considerable size before infection occurs and in these areas the disease has little impact on control. However, in small clear-cuts and partial cutovers infection occurs in 1- to 2-year-old trees from windborne spores from nearby older trees. I have found young trees with over 70 knots on the twigs, stems, and branches: these trees usually survived less than a decade.

Importance in Forest Development

The dynamics of pin cherry development and the growth of crop trees (conifers or tolerant hardwoods) under various control regimes are illustrated in Figure 2. These curves do not take into account the effects of other weeds, some of which may be suppressed by pin cherry. On some sites, a temporary, light cover of pin cherry should be encouraged; a strategy compatible with the use of biological controls. In other cases, complete and rapid removal of the pin cherry may be desired, requiring the use of a chemical herbicide.

Is it Practical?

Hauling diseased material from one location and spreading it around a recent clear-cut or burn is a laborious process and means of doing this on a large scale are not obvious at present. Likewise, methods of producing massive quantities of infective spores artificially have not yet been developed. Use of this disease as a control agent is best integrated into the general forest management plan. The disease can be and is manipulated silviculturally by the following practices.

1. Avoidance of the use of fire in forest renewal.
2. Small clear-cuts enabling contiguous stands of different ages to be present in the same area.
3. Building roadways several years prior to cutting to allow development of pin cherry and the disease along roadsides.

These practices may be incompatible with other management objectives, but means of manipulating this and other native diseases for biological control will be improved through continued research. While these new methods are unlikely to replace chemical herbicides they will supplement them for specific weeds that are difficult to control by other means and for sites where use of chemicals is not permissible.

R.E. Wall
December 1986

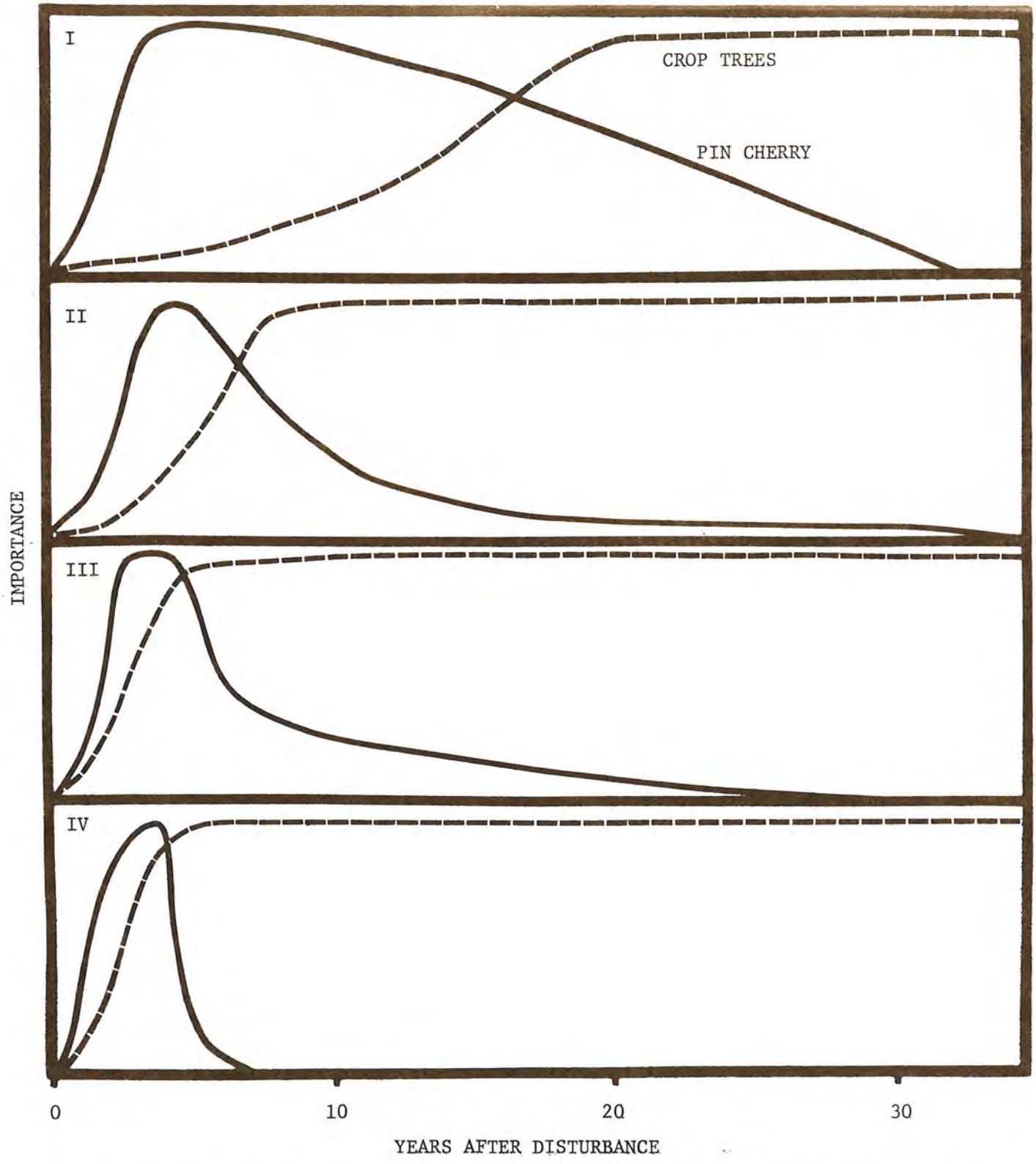

Figure 2. Pin cherry and crop tree development with different levels of control on sites where pin cherry is the dominant weed. (I) may be typical of a forest developing from wild fire, (II) from logging, (III) from artificial introduction of the black knot disease in the first or second year, and (IV) from the use of herbicides.