CHESTNUT BLIGHT: DEFENSE REACTIONS

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ABSTRACT.-- *Anatomical studies showed that chestnut trees have the capacity to set boundaries to resist spread of infected bark and wood. Where portions of cambium remained alive after infection, xylem rays expanded into the bark and wood formed.*

Preliminary results of anatomical studies show that the pathogen in bark and wood is walled off, and wood is formed in bark by extensions of xylem rays.

Chestnut blight was viewed from CODIT, a model for compartmentalization of decay in trees. The CODIT perspective of stem and root diseases is that trees survive after injury and infection so long as they have the time, energy, and genetic capacity to recognize and compartmentalize injured and infected tissues rapidly and effectively and to generate enough new tissues to maintain the tree.

Materials and Methods

Stem sections--5 to 15 cm in diameter--with one or more cankers were received from Kentucky, 6; West Virginia, 60; North Carolina, 4; and Virginia, 12. Also, 10 sections of similar diameter, but without obvious cankers, were received from West Virginia. The stem sections ranged from 30 to 60 cm in length. The stems were first cut into disks, 1 to 3 cm in width. The disks were then sanded and studied under a dissecting microscope at 10 to 30 X, Selected samples, approximately 30, yielded one cm^3 subsamples of bark and wood for microtome sectioning 8 to 15 pm. The blocks were fixed in 5 percent formaldehyde. Sections were stained with 0.05 percent toluidine blue 0.

Results and Discussions

Where small portions of cambium remained alive after infection, xylem rays expanded outward into the bark. The bands of xylem seemed to start in wood that had the characteristics of a barrier zone. The xylem rays grew outward in the form of a pillar or cylinder. The cells formed a cambium that produced all wood components (Figures 1 and 2).

Figure 1. Extension of xylem ray forming wood in bark. The dead bark was removed to show the pillar or cylinder of xylem ray cells (arrow). A dissecting needle is beneath the pillar. The ray cells join to form a cambium that forms all wood components in the bark.

Wound periderms set boundaries for the infected bark. When the pathogen grew from the bark to the wood, the infected wood was walled off according to the CODIT model.

The results show that chestnut trees have the capacity to set boundaries to resist spread of infected tissues. The question is, why do most trees fail to effectively wall off infected tissues associated with wild strains of the pathogen, while many trees are fairly successful in effectively walling off infected tissues associated with the hypovirulent strain?

The key factors for survival are time, energy, and genetic capacity to recognize and compartmentalize infected tissues. Our results show that the trees have the genetic capacity to compartmentalize infected tissues. But compartmentalization does not start until the infected tissue is recognized.

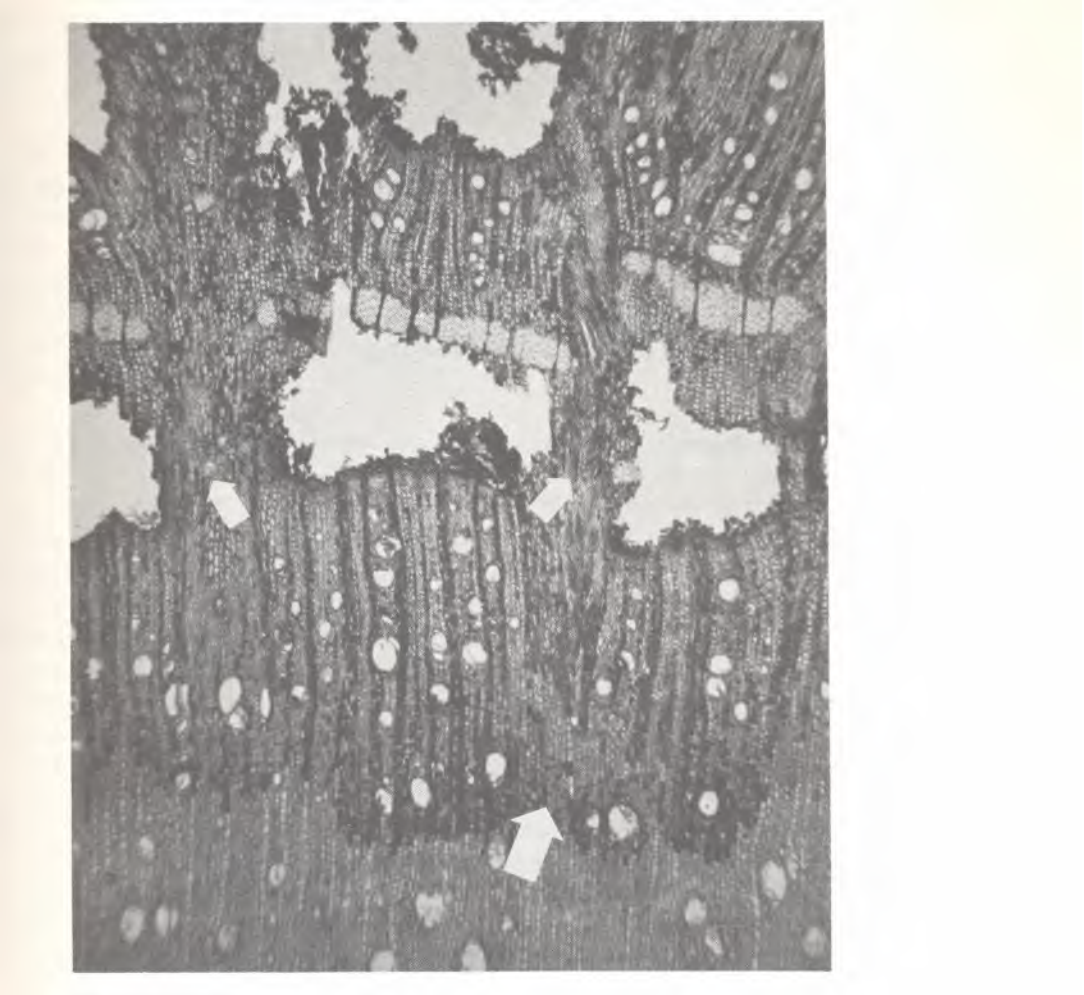

Figure 2. Portions of cambium that remain alive after infection form a barrier zone type tissue (large arrow). From this zone, ray cells divide rapidly to form pillars (small arrows) that extend into the infected bark. The xylem ray cells then form a cambium that produces all components of wood.

Time may be the key factor. One possible explanation is that the wild strains spread rapidly in outer bark first before they spread inward and are recognized by the cambium. The hypovirulent strains may spread slowly in outer bark, but rapidly inward to the cambium. If this is so, it would explain the usual ellipsoid shape of the hypovirulent-incited cankers. Such shapes of cankers or dead areas are typical of drill wounds where a portion of cambium is killed quickly.