Death of a Chestnut: The Host Pathogen Interaction

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ABSTRACT. — Endothia parasitica advances within the inner bark of its host through enzymatic maceration of sound tissues. Oxalic acid appears to play a role by acting synergistically with polygalacturonase and displaying a toxicity toward protoplasts.

With the exception of the mechanism of advance into sound host tissue the life cycle of *Endothia parasitica* (Murr.) P. J. & H. W. And. has been amply described (Anderson and Babcock, 1913; Anderson, 1914). Studies at the University of Tennessee have led to a more complete understanding of the mechanisms of pathogenesis. We hope this model will act as a basis for a better understanding of the host-pathogen interaction.

Endothia parasitica is a wound parasite requiring penetration into the inner bark to provide an infection site. The germinating spore is capable of growing saprophytically for a time on the debris of the wound. However, if the wound is on a susceptible tree, such as the American chestnut *(Castanea dentata* [Marsh.] Borkh.), the mycelium will successfully invade healthy tissue.

The mechanism for advance into sound tissue is probably enzymatic. *E. parasitica* produces a number of polysaccharide-degrading enzymes capable of depolymerizing the various components of the cell wall. The pH optima for these enzymes are equal to that of the sound inner bark (approximately 5.5) and thus these enzymes may be maximally active in this tissue. One of the first enzymes produced is polygalacturonase. It could diffuse from the mycelium into sound tissue and is known to be capable of depolymerizing the polypectate of the middle lamella which "glues" the cell walls together (Albersheim, 1975).

With degradation of the middle lamella, the other cell wall polysaccharides are exposed to attack by the remaining enzymes which are produced in addition to polygalacturonase. This activity produces a "gelatinous" zone described by Rankin (1914). *In vitro,* polygalacturonase is incapable of depolymerizing calcium salts of the polypectate. Oxalic acid produced by the fungus, however, is an effective chelator of the calcium, and may remove it from the polypectate thus exposing the substrate to enzymatic attack. Oxalic acid is also toxic to the protoplasts of chestnut and presumably plays a role in acidification of the advancing edge of the canker.

Close to the mycelium the pH drops below the optima for the polysaccharide enzymes, but comes into the range of the acid protease produced by the fungus (approximately 2.8). If the acidic conditions and oxalic acid kill the protoplast, protein constituents may then be degraded.

Degradation of tissue components in the gelatinous zone provides nutrition for the fungus. The mycelium invades the macerated tissue, more oxalate and enzymes are then produced which further advance the maceration of the tissue.

The chestnut, however, is not totally defenseless. Protoplast death initiates browning. The complex of polyphenolics produced by this activity is effective in denaturing the enzymes produced by the fungus. When tested against polygalacturonase, aqueous extracts of both Chinese *(C. mollissima* B1.) and American chestnut inner bark destroyed the enzyme activity, reaching maximal effect after approximately 30 minutes. The extracts of American chestnut, however, were only half as effective as equivalent extracts from Chinese chestnut.

Both chestnut species also contain a proteinaceous inhibitor that, when extracted by the method of Albersheim and Anderson (1971), is specific for endopolygalacturonase. Here again the inhibition from American chestnut extracts containing this component was only about 40 percent of that obtained from equivalent extracts of Chinese chestnut inner bark.

American chestnuts are ineffective in resisting the advance of the fungus. A canker will advance around the stem disrupting nutrient movement in the phloem and destroying the cambium. Also, tyloses are induced in adjacent xylem, disrupting water movement (Bramble, 1938). As the canker girdles a stem, the portion distal to the canker dies. Branches below this canker may keep the tree alive for several years, but reinfections of the same tree may result in girdling of all its shoots and eventually kill the tree.

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