

CEPHALOSPORIUM WILT OF PERSIMMON

J. J. McRitchie

The genus *Diospyros* is comprised of over 200, largely tropical, tree species. Only 2, *D. virginiana* L. and *D. texana* Scheele, are native to the United States (8). The genus is found throughout the East south of a line from southern Iowa to New Jersey and extends to the Great Plains. It grows in hot dry fields, and *D. virginiana* has taken over pasture lands in the lower Mississippi Valley (5). The ripe fruit is edible, but if not fully ripe it is highly astringent. The wood of certain species is used in woodworking, and *D. virginiana* may be used as a rootstock for *D. kaki* L., a popular Japanese persimmon with large edible fruit.

According to G. H. Hepting, *Cephalosporium* wilt of persimmon ranks with chestnut blight, Dutch elm disease, elm phloem necrosis, and mimosa wilt as "among the most devastating biotic hardwood tree diseases known" (5).

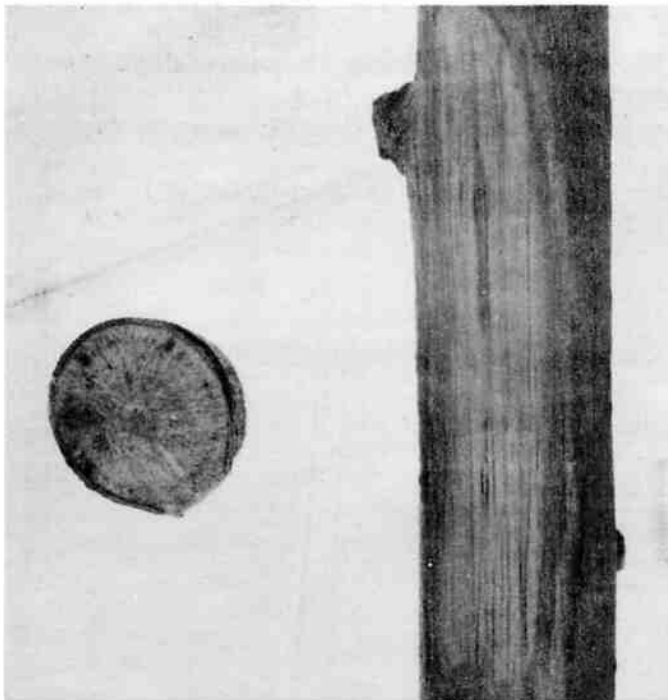


Fig. 1. Characteristic brown to black streaks in the wood of persimmon infected with

The fungus, *Cephalosporium diospyri* Crandall, virtually eliminated persimmon from one of its main habitats, the central basin of Tennessee, and has been found south to Florida and west to Oklahoma. The pathogen is so efficient that it was once proposed as a biological control agent to eliminate unwanted native persimmons (9); a questionable practice in retrospect, because the same fungus genus has since been associated with wilt diseases of oak and elm (4, 6, 7).

The disease has been found in nature on *D. virginiana*, which is highly susceptible, and on *D. kaki*, which is nearly immune but may be killed when grafted on *D. virginiana* rootstock. It was first found in Tennessee in 1936 (3), and the pathogen was

characterized as a new species in 1945 (1).

SYMPTOMS. Symptoms of persimmon wilt are typical of vascular infections: a sudden wilting which begins in the leaves in the top of the tree, and rapid defoliation and death often within 2 months. Fine brown to black streaks develop throughout the wood (fig. 1). This streaking develops before the external symptoms appear.

Pink spore masses of the fungus are produced beneath the bark of wilt-killed trees. The bark ruptures, the spores are released, and then transported by wind to healthy trees. The fungus gains entrance into healthy trees through wounds, which in many cases are produced by insects (2).

CONTROL. Control is difficult; however, removing infected trees, avoiding wounding, and controlling insects should help prevent spread.

LITERATURE CITED.

1. Crandall, B. S. 1945. A new species of *Cephalosporium* causing persimmon wilt. *Mycologia* 37:495-498.
2. _____, and W. L. Baker. 1950. The wilt disease of American persimmon, caused by *Cephalosporium diospyri*. *Phytopathology* 40:307-325.
3. _____, and R. W. Davidson. 1937. *Cephalosporium* wilt of persimmon. *Plant Dis. Repr.* 21:251-252.
4. Creager, D. B. 1937. The *Cephalosporium* disease of elms. *Contrib. Arnold Arboretum*, No. 10. 85p.
5. Hepting, G. H. 1971. Diseases of forest and shade trees of the United States. USDA Forest Serv. Agric. Handbook No. 386. 658p.
6. Van Arsdel, E. P. 1972. Some cankers on oaks in Texas. *Plant Dis. Repr.* 56:300-304.
7. _____, and R. S. Halliwell. 1970. Progress in research on live oak decline. *Plant Dis. Repr.* 54:669-672.
8. Wilson, C. L. 1963. Wilting of persimmon caused by *Cephalosporium diospyri*. *Phytopathology* 53:1402-1406.
9. _____. 1965. Consideration of the use of persimmon wilt as a silvicide for weed persimmons. *Plant Dis. Repr.* 49:789-791.