The Devastation of American Chestnut by Blight

E. G. Kuhlman

USDA Forest Service, Southeastern Forest Experiment Station, Research Triangle Park, NC 27709

ABSTRACT.— A brief account of the explosive history of the classic forest tree disease, chestnut blight, in North America.

The American chestnut (*Castanea dentata* [Marsh.] Borkh.) played an important role in the history of our country. Henry David Thoreau (1854) captured some of the magic of the chestnut in his book *Walden*.

When chestnuts were ripe I laid up half a bushel for winter. It was very exciting at that season to roam the then boundless chestnut woods of Lincoln,-they now sleep their long sleeps under the railroad,—with a bag on my shoulder, and a stick to open burs with in my hand, for I did not always wait for the frost, amid the rustling of leaves and the loud reproofs of the red squirrels and the jays, whose half-consumed nuts I sometimes stole, for the burs which they had selected were sure to contain sound ones. Occasionally I climbed and shook the trees. They grew also behind my house, and one large tree which almost overshadowed it was, when in flower, a bouquet which scented the whole neighborhood, but the squirrels and jays got most of its fruit, the last coming in flocks early in the morning and picking the nuts out of the burs before they fell. I relinquished these trees to them and visited the more distant woods composed wholly of chestnut. These nuts, as far as they went, were a good substitute for bread.

For others, nostalgia may come from recollections such as those of G. H. Hepting (1974) who said,

As a boy, on cold, blustery fall and winter nights, I well remember a shivering, old Italian standing on a street corner of downtown Brooklyn before his rickety sheet metal oven-like contraption, yelling, 'Hot roasta chestnuts! Hotta roast chestnuts!' I remember the popping and crackling noises as the old fellow took off the lid to give me a nickel's worth of the sweet, hot delicious nuts....

While the fruit of the chestnut was important to man and his domesticated animals, it was even more important to the wildlife of the eastern forest. Thoreau felt smug about outsmarting an occasional squirrel or jay, but it was quite certain that wild turkeys, squirrels, jays, and other animals preferentially sought and frequently devoured this succulent fruit.

The American chestnut comprised 25 percent of the eastern hardwood forest. Its natural range included over 200 million acres of land. Mature trees were 60-120 feet tall with straight boles up to seven feet in diameter (Roosevelt, 1902). On good sites, open grown trees often added one inch in diameter per year and could sometimes sustain this growth for more than 50 years. Normal growth was 500 board feet per acre per year (Holmes, 1925). Chestnut had a faster rate of growth than its associated hardwood species (Holmes, 1925; Korstian and Stickel, 1927; Roosevelt, 1902).

Chestnut wood carried man from cradle to grave, in crib and coffin. Many homes had chestnut siding, chestnut shingle roofing, and chestnut doors and paneling. Because chestnut wood was durable and rot resistant it was used for telephone poles, ship masts, railroad ties, and farm fencing. Chestnut extracts provided tannin for the leather industry.

Three rather contrasting estimates of the value of chestnuts were made by the states of Pennsylvania, North Carolina, and West Virginia from 1909 information. Detwiler (1912) estimated 7.6 million acres of forest land in Pennsylvania with 21 percent in chestnut timber. He allowed two poles, four railroad ties, and two cords of wood per acre at a value of two dollars per pole, 33 cents per tie, and one dollar per cord. Total timber value was \$55 million. Nut crop, orchard, park, and shade trees had an estimated worth of another \$15 million. Buttrick (1925) estimated 31/2 billion feet of standing chestnut in North Carolina in an area of approximately 7.6 million acres. Only 50 percent of this timber was accessible to lumbermen. For the accessible portion the estimated value was 11/2 dollars per thousand bd. ft. for saw timber and 121/2 cents per cord for cordwood. Total value of the chestnut was therefore only \$2.5 million in North Carolina. In West Virginia there was an estimated ten billion bd. ft. of standing chestnut worth 21/2 dollars per thousand bd. ft. for a total value of \$25 million (Giddings, 1912). West Virginia reported that one railroad station shipped 155,000 lbs. of chestnuts in 1911 (Giddings, 1912). The U.S. Forest Service's estimate for chestnut timber cut in 1909 was \$20 million (Detwiler, 1912).

H. W. Merkel (1905), chief forester and constructor of the New York Zoological Society, discovered the chestnut blight disease in the Bronx parks. "During 1904," he reported, "an epidemic of a fungus disease has occurred throughout the parks of this Borough, which but for the fact that it was confined to a single species of tree, might have overshadowed in deadliness and rapid spread all other enemies of tree life." Merkel (1905) obtained an emergency appropriation of \$2,000 to treat the affected trees. With that money he trimmed out the disease from 438 individuals. In spite of this effort he reported that 98 percent of all chestnuts in Bronx parks were infected in 1905. The fungus was highly virulent; in one case only 21 days elapsed between the first symptom and the girdling of a 4-in. stem. Merkel (1905) reported the physical condition of the tree had no effect on the fungus. The disease was equally as frequent on young nursery specimens, sprouts, and young trees 30-40 feet tall as it was on old patriarchs 10-12 feet in circumference. It should be noted that although the disease frequency was similar on all trees, the response of individual trees and even limbs on the same tree varied greatly. Generally, infections rapidly girdled smooth-barked branches. However, girdling of rough-barked limbs took from one to ten years with an average of three to four years (Gravatt, 1925).

A botanist, W. A. Murrill (1906), of the New York Botanical Gardens, reported that inoculation studies indicated infection probably took place only through wounds, which unfortunately were all too common on chestnut.

The causal fungus was briefly called *Diaporthe parasitica* but was soon named *Endothia parasitica* (Murr.) P. J. & H. W. And. (Shear, *et al.*, 1917).

Undoubtedly the blight had entered this country before 1904 when Merkel found the affected trees in the Bronx, but it was a few years before the source of the fungus was determined. Initially the sudden outbreak was attributed to severe drought conditions that made chestnut susceptible to an otherwise innocuous fungus. Evidence was soon presented that the disease was introduced from a foreign country. Observations in the eastern United States had indicated Chinese and Japanese chestnuts had more natural resistance to the disease than did the American chestnut (Shear et al., 1917). If the host and pathogen evolved together there would have been selection pressure on both and some resistance would have occurred. Thus natural resistance in Asiatic chestnuts indicated an Asian origin for the pathogen was likely. In the fall of 1912 diseased chestnut material from Agassiz, British Columbia, proved to contain E. parasitica (Shear et al., 1917). Chestnut was not native to British Colubbia and the Agassiz planting contained stock of American, European, and Asian origin. Although all the trees were ordered from American nursery firms, the planting supervisor remembered the Asian species were shipped to Agassiz in the original wrappings which consisted of distinctive Asian mats and casings. In 1913, Frank N. Meyer, an agricultural explorer, found the fungus in China on native chestnuts and subsequently he also found it in Japan. Isolates from these specimens caused symptoms identical to those caused by isolates from the United States on American chestnuts (Shear et al., 1917).

Most conditions appeared to favor the pathogen. A highly susceptible host evenly distributed through its range, a favorable climate, no natural barriers to limit spread, and two abundant spore forms provided an efficient means of spread.

The disease spread rapidly from the New York City area. In 1909, the USDA indicated that most chestnuts within 30 miles of New York City were infected and scattered disease centers were present in Pennsylvania, Virginia, Maryland, Connecticut, and Rhode Island (Metcalf and Collins, 1909). Two vears later, the main disease center was 150 miles south and 60-70 miles north and west of New York City, with scattered centers up to 120 miles in advance of this main center (Metcalf and Collins, 1911). The rate of spread of the main disease center was given as ten miles per year. However, spot infection centers developed up to 150 miles from the leading edge. One such large center involving three counties in North and South Carolina was first noticed in 1923, but its size suggested it started at least as early as 1912 (Gravatt, 1925). These spot infections were thought to result from spores carried on birds; however, movement of chestnut products was not restricted and probably contributed to the rapid dissemination (Detwiler, 1914; Gravatt, 1935). By 1950 the blight occurred on more than 80 percent of the trees throughout the range of American chestnut.

Efforts to control chestnut blight were started by Merkel, the discoverer of the disease. He attempted control by cutting out the affected tissue and by spraying with Bordeaux mixture (Merkel, 1905). Neither method provided control in New York City.

Regional efforts to eradicate the disease were made from 1908-1914. Metcalf and Collins (1911) of the USDA, Bureau of Plant Industry located 14 spot infection centers within a 35-mile radius of Washington, D.C., in 1908. All infected trees in these centers were felled, and the bark and brush were burned on the stumps. In 1911 no new cases of the disease were reported in the 35-mile zone (Metcalf and Collins, 1911). F. C. Stewart (1912) of the New York Agricultural Experiment Station analyzed Metcalf and Collins' work in a paper he presented at the Pennsylvania Chestnut Tree Blight Conference in 1912. His first criticism was " ... there was no check treatment and experimenters are agreed that experiments without checks have little value." Secondly, Stewart visited two centers of infection within Metcalf and Collins' "immune zone." One tree over 3 ft. in diameter was in advanced decline and must have been infected for several years, including the time Metcalf was stating that the area was apparently free from the disease. Finally, Stewart visited two treated areas and in one found the fungus in bark that had not been removed from the stump. A nearby tree also was infected. Stewart correctly predicted that eradication would not be effective in controlling the disease. Although his speech was low key and scholarly it became the target of other speeches at the Pennsylvania Conference. The prevailing mood was patriotism and was exemplified by Pennsylvania's willingness to invest \$275,000 in an effort to stop the disease. That mood and effort were not

slowed by Stewart. Although others echoed his sentiments they veiled their criticisms, whereas Stewart's title "Can Chestnut Bark Disease be Controlled?" was immediately answered negatively in his text. When the Pennsylvania effort was abandoned two years later, it was suggested the disease had been slowed by five years through the effort (Sargent, 1914).

The failure to eliminate the disease by eradication in New York, Pennsylvania, and the District of Columbia can be understood on the basis of the large numbers of trees involved. Furthermore, evidence from several outbreaks in ornamental and orchard plantings in the western United States proved that even limited infestations were impossible to eradicate. At the Agassiz, B.C., site, all infected trees were destroyed in 1912; however, the disease appeared on other trees in 1934 (Gravatt, 1935). In Gunter, Oregon, the disease was found on two trees in 1929; these trees were cut and burned. However, in 1934 the fungus was still active on one stump a foot below ground (Gravatt, 1935). In California, the disease persisted from 1934 until at least 1945 in spite of meticulous eradication and sanitation efforts in the orchards on an annual basis (Milbrath, 1945).

The second major emphasis in control has been on efforts to breed chestnuts that are resistant to blight. Because four other papers are on the subject of resistance, it is sufficient to indicate that the earliest papers (Murrill, 1906) broached the subject of resistance and that it has remained a promising hope.

The American chestnut has been devastated by blight. This once prominent species has reverted to a very minor role in the eastern forests. The hopeful leads for the revitalization of this species will be the topic of other papers.

LITERATURE CITED

Buttrick, P. L.

1925. CHESTNUT IN NORTH CAROLINA. *In* Chestnut and the Chestnut Blight in North Carolina. N.C. Geol. and Econ. Surv. Econ. Pap. 56. p. 7-10.

Detwiler, S. B.

1912. THE PENNSYLVANIA PROGRAMME. Pa. Chestnut Blight Conf., Harrisburg. p. 129-136.

Detwiler, S. B.

1914. OBSERVATIONS OF SANITATION CUTTING IN CONTROLLING THE CHESTNUT BLIGHT IN PENNSYL-

VANIA. Final Rep. Pa. Chestnut Tree Blight Comm., 1913, Harrisburg. p. 61-94.

Giddings, N. J.

1912. (Untitled report on chestnut blight in West Virginia.) Pa. Chestnut Blight Conf., Harrisburg. p. 26, 173-174.

Gravatt, G. F.

1925. THE CHESTNUT BLIGHT IN NORTH CAROLINA. *In* Chestnut and the Chestnut Blight in North Carolina. N.C. Geol. and Econ. Surv. Econ. Pap. 56. p. 13-17.

Gravatt, G. F.

1935. CHESTNUT BLIGHT IN CALIFORNIA. I. DEVELOP-MENT OF THE DISEASE. Mon. Bull. Calif. Dept. Agr. 24: 173-177.

Hepting, G. H.

1974. DEATH OF THE AMERICAN CHESTNUT. J. For. Hist. 18:60-67.

Holmes, J. S.

1925. FOREWARD. *In* Chestnut and the Chestnut Blight in North Carolina. N.C. Geol. and Econ. Surv. Econ. Pap. 56. p. 5-6.

Korstian, C. F. and P. W. Stickel. 1927. THE NATURAL REPLACEMENT OF BLIGHT-KILLED CHESTNUT. USDA Misc. Circ. 100. 15p.

Merkel, H. W.

1905. A DEADLY FUNGUS ON THE AMERICAN CHEST-NUT. N.Y. Zool. Soc. 10th Ann. Rep. p. 97-103.

Metcalf, H. and J. F. Collins.

1909. THE PRESENT STATUS OF THE CHESTNUT BARK DISEASE. USDA Bull. 141. p. 45-54.

Metcalf, H. and J. F. Collins.

1911. THE CONTROL OF THE CHESTNUT BARK DIS-EASE. USDA Farmers' Bull. 467.24 p.

Milbrath. D. G.

1945. BUREAU OF PLANT PATHOLOGY. Calif. Dept. Agr. Rep. 34: 213-227.

Murrill, W. A.

1906. FURTHER REMARKS ON A SERIOUS CHESTNUT DISEASE. J. N.Y. Bot. Gard. 7:203-211.

Roosevelt, T. R.

1902. MÉSSAGE FROM THE PRESIDENT OF THE UNITED STATES, TRANSMITTING A REPORT OF THE SECRE-TARY OF AGRICULTURE IN RELATION TO THE FOR-ESTS, RIVERS, AND MOUNTAINS OF THE SOUTHERN APPALACHIAN REGION. Govt. Print. Off., Washington, D.C. 210 p.

Sargent, W.

1914. LETTER OF TRANSMITTAL. THE COMMISSION FOR THE INVESTIGATION AND CONTROL OF THE CHESTNUT TREE BLIGHT IN PENNSYLVANIA. Final Rep. Pa. Chestnut Tree Blight Comm., 1913, Harrisburg. p. 9-13.

Shear, C. L., N. E. Stevens, and R. J. Tiller.

1917. *ENDOTHIA PARÁSITICA* AND RELATED SPECIES. USDA Bull. 380. 82 p.

Stewart, F. C.

1912. CAN THE CHESTNUT BARK DISEASE BE CON-TROLLED? Pa. Chestnut Blight Conf., Harrisburg. p. 40-45.

Thoreau, H. D.

1882. WALDEN. 357 p. Houghton, Mifflin & Co., Boston.