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Ecology of survival and recovery from blight in American chestnut trees (*Castanea dentata* (Marsh.) Borkh.) in Michigan¹

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BREWER, LAWRENCE G. (Department of Botany, Miami University, Oxford, OH 45056). Ecology of survival and recovery from blight in American chestnut trees (*Castanea dentata* (Marsh.) Borkh.) in Michigan. Bull. Torrey Bot. Club 122: 40–57. 1995.—Between 1986–1987, 259 locations with American chestnut trees were examined in Michigan's lower peninsula. One hundred forty-four locations had trees with chestnut blight disease, while at 115 locations blight was not found. All locations combined had 1492 (75%) diseased trees and 493 (25%) disease-free trees ≥ 10 cm dbh. At 52 locations with blight 9813 chestnut trees less than 10 cm dbh were located and examined, while at 22 blight-free locations 4161 chestnut trees were found.

At forty-two locations in Michigan, American chestnut trees had abnormal cankers and were recovering from the effects of chestnut blight disease. Quantitative measures were established at these locations to determine the extent of hypovirulence. At these locations there were 989 chestnut trees ≥ 10 cm dbh and 3233 smaller chestnuts. It appears that initially all locations with abnormal cankers had normal virulent blight. Only after a lag of 15–25 years did abnormal cankers appear.

The percentages of abnormal cankers and live branches above those cankers were determined for all 42 locations and compared to soil texture. Between 13% and 100% of the cankers at each location were abnormal. The percentage of live branches above abnormal cankers ranged from 41% to 100% per location. Trees on sites with sandy soils had more abnormal cankers and more surviving stems above them than those on heavier textured soils. Possible ecological factors which may explain the differential success of hypovirulence on different soil types include: (1) a better competitive advantage of the American chestnut on well drained sandy soils, (2) the origin of hypovirulence from sandy textured soils, and (3) more dispersing agents of hypovirulent strains on sandy textured soils.

Key words: chestnut blight, hypovirulence, *Castanea*, *Cryphonectria*.

The natural range of the American chestnut (*Castanea dentata* (Marsh.) Borkh.) encompassed over 200 million acres, included 24 eastern states, and contained an estimated 3.5 billion chestnut trees (Pennsylvania 1912; Beattie and Diller 1954; Little 1971; Hepting 1974). The tree reached its greatest size on well-drained soils of the oak chestnut association (Braun 1950; Whitaker 1956) where it could be found with diameters of 13 feet and heights of over 120 feet (Roosevelt 1902; Jaynes 1978; Bloomfield 1980). The American chestnut was the fastest growing hardwood tree in eastern North America. It was also the most rot-resistant, and thus its wood was used for railroad ties, shingles, fence posts, as well as fine furniture. The tannin from the bark

was used in tanning leather. Unlike the oaks, which had sporadic nut production, the American chestnut produced a dependable yearly crop which was invaluable to wildlife (Christisen 1965).

In 1904 chestnut blight, caused by the fungus *Cryphonectria parasitica* (Murr.) Barr, was found on American chestnut trees in New York City (Merkel 1905). The blight spread 25 to 35 miles per year, and by 1910 had invaded 10 states (Pennsylvania 1912). By the 1950s it had destroyed every stand in its natural ranges making it the worst disaster in forest history. Today, although the tree continues to sprout (Paillet 1984, 1988), it has been replaced in the canopy on drier sites by chestnut oak (*Quercus prinus*), red oak (*Quercus rubra*), scarlet oak (*Quercus coccinea*), white oak (*Quercus alba*), black oak (*Quercus velutina*), and pignut hickory (*Carya glabra*), while on more mesic sites it has been replaced by sugar maple (*Acer saccharum*), red maple (*Acer rubrum*), beech (*Fagus grandifolia*), hemlock (*Tsuga canadensis*), black birch (*Betula lenta*), and tuliptree (*Liriodendron tulipifera*) (Korstian and Stickel 1927; Keever 1953; Nelson 1955; Woods and Shanks 1957, 1959; Good 1968; Mackey and Sivec 1973; Stephenson 1974, 1986; McCormick and Platt 1980; Johnson and Ware

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1982; Adams and Stephenson 1983; Stephenson et al. 1991; Mikan et al. 1994).

In Michigan the natural range of the American chestnut was restricted to the southeastern portion of the state (Little 1971). The species migrated into the state less than 1000 years ago and was continuing to move northwestward (Davis 1976) when the blight first arrived in the area in 1928 (Baxter and Strong 1931). Because the climate and soil were potentially favorable, early settlers had great success in planting chestnuts on their homesteads beyond the species' natural range, especially on the western portion of Michigan's lower peninsula where the chestnut trees were protected from extreme cold by Lake Michigan. Between 1975 and 1981 I conducted a survey of 224 locations of American chestnut trees in Michigan (Brewer 1982a, 1982b). At eighty-four sites, the blight was observed, while the disease was not found at 140 sites. The blight spread from the southeast towards the northwest, and had a tendency to find the largest concentrations of trees first.

RECOVERING TREES. The natural recovery of chestnut trees infected by the blight fungus *Cryphonectria parasitica* (Murr.) Barr was first observed in 1951 when Biraghi (1951, 1953) noticed healing cankers on chestnut trees (*Castanea sativa* Miller) growing in Italy. Isolated strains of this fungus were found to be less virulent than normal strains of *C. parasitica*, and when placed in cankers incited by normal strains had the ability to convert them to less virulent forms (Greente 1965; Greente and Sauret 1969a, 1969b). Because of their reduced virulence Greente called these new strains "hypovirulent."

French hypovirulent strains sent to North America also were found to cure cankers on American chestnut trees (*Castanea dentata*) (Anagnostakis and Jaynes 1973; Van Alfen et al. 1975). Day et al. (1977) showed that these hypovirulent strains carried double-stranded ribonucleic acid (dsRNA) in the cytoplasm and that this dsRNA could be transferred to virulent strains by anastomoses. Choi and Nuss (1992) later established that this dsRNA was the causal agent for hypovirulence.

The first hypovirulent strains of *C. parasitica* in North America were isolated from abnormal cankers from a chestnut grove near Rockford, Michigan in 1976 (Day et al. 1977). These isolates also had dsRNA, as well as the ability to transfer it and convert virulent strains to hypovirulent strains.

During my 1975–1981 survey in Michigan I documented 24 locations with American chestnut trees with abnormal cankers that were recovering from blight (Brewer 1982a, 1982b). During this survey it appeared that some locations with sandy soils were recovering better than those on heavy textured soils. So far, *C. parasitica* has been isolated from abnormal cankers from at least seven of these sites and all have been found to carry dsRNA (Fulbright et al. 1983; Elliston 1985a, 1985b). In addition to the Michigan trees, abnormal cankers also have been found on isolated trees in other parts of North America (Double et al. 1985; Jaynes and Elliston 1982; Griffin et al. 1983). The unique characteristic about the Michigan trees is the large number of locations with recovering trees.

Although a significant amount of research has been conducted on the molecular biology and morphology of dsRNA and *Cryphonectria parasitica* (Anagnostakis 1977, 1982, 1987; Anagnostakis and Day 1979; Anagnostakis et al. 1986; Van Alfen et al. 1975; Griffin et al. 1984; Griffin 1986, 1989; Day et al. 1977; Elliston 1978, 1982, 1985a, 1985b; Dodds 1980a, 1980b; Kuhlman and Bhattacharyya 1984; Kuhlman et al. 1984; Newhouse et al. 1983; Double et al. 1985; L'Hos-tis et al. 1985; Hiremath et al. 1986; Fulbright et al. 1983; Fulbright 1984; Paul and Fulbright 1988), much less research has been undertaken on the ecology of the hypovirulence phenomena (Anagnostakis 1977, 1990; Scharf and DePalma 1981; Russin et al. 1984; Griffin 1986, 1989; Griffin et al. 1984; Griffin et al. 1991). Particularly lacking are studies on the environmental factors (climate, soils, competition, dispersing agents, etc.) which affect the interaction between the American chestnut, *C. parasitica*, and dsRNA in the field.

The goal of the present study was to return to the 224 sites initially surveyed, as well as visit new locations in order to: (1) collect information on the number of native, planted, or naturally produced chestnut trees, (2) make general assessments about the tree's ability to grow and reproduce, especially beyond the natural range, and (3) document the continued spread of chestnut blight disease in the state. For those locations with recovering trees the goal was to (1) collect data on the abundance of abnormal cankers and the branch survival above them, (2) make general assessments about the spread of hypovirulence in the state, (3) determine if there is a relationship between soil texture and the amount of recovery, and if the relationship existed (4)



Fig. 1. (A) A normal lethal canker. (B) An abnormal canker.

suggest some ecological factors for the difference in recovery on different textured soils.

Methods. The field work was conducted during the summers of 1986 and 1987. At each location, the number of trees 10 cm dbh (diameter at breast height) or greater and the number of smaller trees and saplings less than 10 cm were counted. Chestnut blight disease was confirmed at all diseased locations by looking for cankers and the distinctive fruiting bodies produced by *Cryphonectria parasitica*. After confirming that *Cryphonectria parasitica* was present at these localities, nearby residents were questioned about when the trees first began to die. In cases where there was some doubt as to when trees first started to die, no dates were recorded. Diseased trees were examined for abnormal cankers (presumed to be evidence of a hypovirulent strain) which are characterized by swollen, callused bark, in contrast to normal lethal cankers, which are flush or slightly sunken and show no signs of callusing periderm (Fig. 1).

At locations with abnormal cankers all normal and abnormal cankers were counted on all stems

>2 cm in diameter (usually more than 2 years of age). Also recorded was whether the branches were dead or alive distal to abnormal cankers. For this survey if the branches above abnormal cankers showed some life they were considered alive, while those that showed no life were considered dead. (Since normal virulent cankers are nearly always lethal, it was not recorded whether branches were dead or alive distal to those cankers.) Sixteen of the 42 sites had more than 20 trees. At eight of these sites canker data were collected from 50 meter wide transects running through the middle of the grove. At the other eight sites all of the trees were examined so that the spread of abnormal cankers within the grove could be monitored in the future.

The soil texture of the A-1 horizon was determined at all 42 sites using the method described by Mokma et al. (1983) and confirmed by comparing the results with county soil surveys. Analysis of variance (ANOVA) was used to determine if there was a relationship between sites with different textured soils and the percentage of abnormal cankers and the branch survival above them.

Table 1. Number of locations of American chestnut trees (≥ 10 cm dbh) in different size classes for diseased locations and locations where disease was not detected.

Locations	Number of trees (≤ 10 cm dbh) per location					
	1–3	4–10	11–25	26–50	51–200	Total
Number and % blighted	75 (37%)	17 (63%)	10 (77%)	8 (89%)	5 (80%)	115 (100%)
Number and % blight-free	129 (63%)	10 (37%)	3 (23%)	1 (11%)	1 (20%)	144 (100%)
Total	204	27	13	9	6	259

Bigger groves have a higher prevalence of blight

Results. A total of 259 locations with American chestnut trees were examined in 1986–1987 (Fig. 2A, B, C; Table 1). At 144 locations (56%) the trees were diseased, while at 115 locations (44%) no signs of blight were detected. Table 1 shows the number of locations of American chestnut trees (≥ 10 cm dbh) in different size classes for diseased locations and locations where disease was not detected. Forty-one locations had not been visited in the 1975–1981 survey, while six locations previously examined no longer had chestnut trees. All locations combined had 1985 trees ≥ 10 cm. Chestnut blight disease was discovered on 1492 (75%) trees, while no disease was detected on 493 (25%) trees. Locations with larger numbers of trees had a higher incidence of blight than those with few trees (Table 1). Eighty-five percent of the 28 locations with 11–200 trees had chestnut blight disease, but only 37% of the 231 locations with 1–3 trees were diseased (Table 1). Once infected with virulent isolates of *C. parasitica*, small trees in Michigan usually die within 2–4 years, while large trees may take 10 or more years (Fig. 3).

Seventy-one locations had a total of 13,973 trees, sprouts, and saplings < 10 cm dbh (Fig. 2C). Of these chestnuts, 9813 were at 52 locations where disease was present and 4161 were from 22 locations where disease was not found. Only 54 of the smaller trees were found in the southern half of Michigan's lower peninsula. This may be due to the large percentage of locations in this area with only single trees. (Since American chestnut trees require cross-pollination for nut production, these single planted trees almost always lacked naturally produced seedlings.)

Most large groves in the southern portion of Michigan's lower peninsula had chestnut blight in the late 1930s and 1940s, while *C. parasitica* reached the majority of the large groves in the northern part of the state between the late 1950s and the present (Fig. 2B). Twenty-three locations have been blighted since 1980, with most of these occurring in the northwest (Fig. 2D). This new outbreak of disease may be due in part to dis-

persal from the Chimney Corners location (Benzie County) (Thompson 1969) where blight was found in 1977. This location has over 3000 heavily infected trees and saplings. This large amount of blight, and the fact that the location lies on top of a large moraine exposed to westerly winds, may explain why many of the locations to the east now have blight.

Two locations where blight was not detected had large numbers of trees and saplings. One location in Missaukee County had 144 large American chestnut trees (≥ 10 cm), and over 4450 smaller chestnuts (< 10 cm), making it the largest known blight-free locality in North America (Figs. 2A, B, C and 4A). Originally, 24 American chestnut trees were planted at this location at the turn of the century. Since then, smaller chestnut trees produced from seeds from the original trees have invaded the nearby oak forest. At this location a 71 cm dbh chestnut was found growing in an oak forest one kilometer from the original grove. An increment boring of this tree showed it to be 35 years old with a growth rate of over 2 cm per year, which is comparable to some of the growth rates on the best sites in the natural range (Roosvelt 1902; Jaynes 1978; Bloomfield 1980). A second location in Leelanau County had 55 large trees and 1500 smaller chestnuts (Fig. 4B). This location is of interest because the chestnut trees were taking over a forest composed of American beech and red maple. In this case the American chestnut may have had a better competitive advantage over the more mesic species due to the acid, nutrient poor, sandy soils at this site.

The largest blight-free American chestnut remaining in Michigan is the National Champion in Grand Traverse County, which had a dbh of 183 cm in 1987. Forty-six trees from blight-free locations had diameters greater than 100 cm.

LOCATIONS WITH RECOVERING TREES. Forty-two of the 144 locations with disease had trees that were recovering with abnormal cankers (Fig. 5A, B; Table 3). This was an increase of 18 locations since the earlier survey (Brewer 1982a,

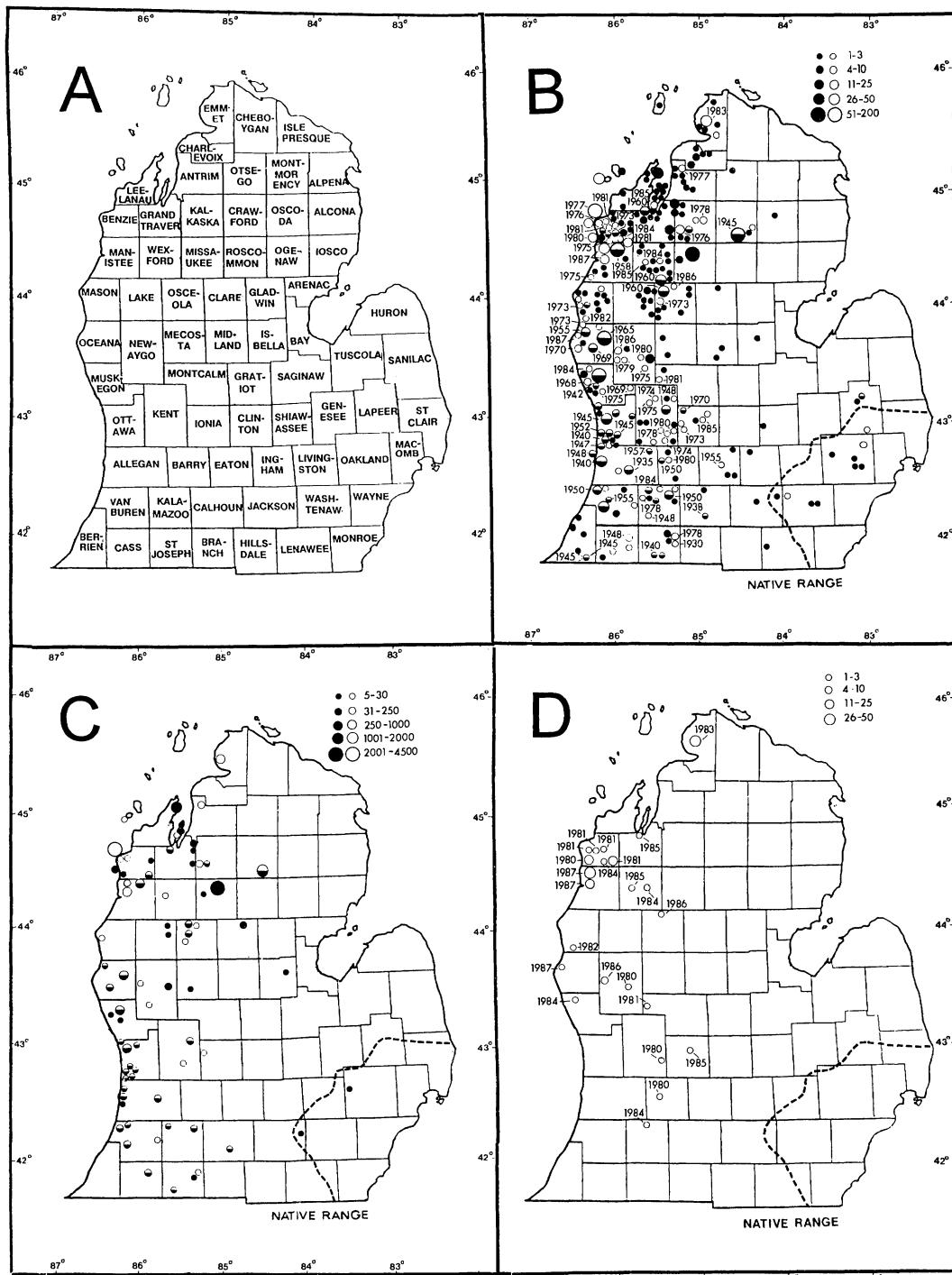


Fig. 2. (A) Michigan counties as they relate to the distribution of American chestnut trees discussed in the text. (B) The distribution and number of American chestnuts in Michigan which are ≥ 10 cm dbh. Completely dark circles indicate locations where blight was not found; open circles represent diseased locations. The circles which are half dark are diseased locations which are recovering with abnormal cankers. The size of the circles indicate relative numbers of trees at each location. The dates are the approximate times when blight entered specific locations as estimated by nearby residents. (C) The distribution and number of naturally reproduced

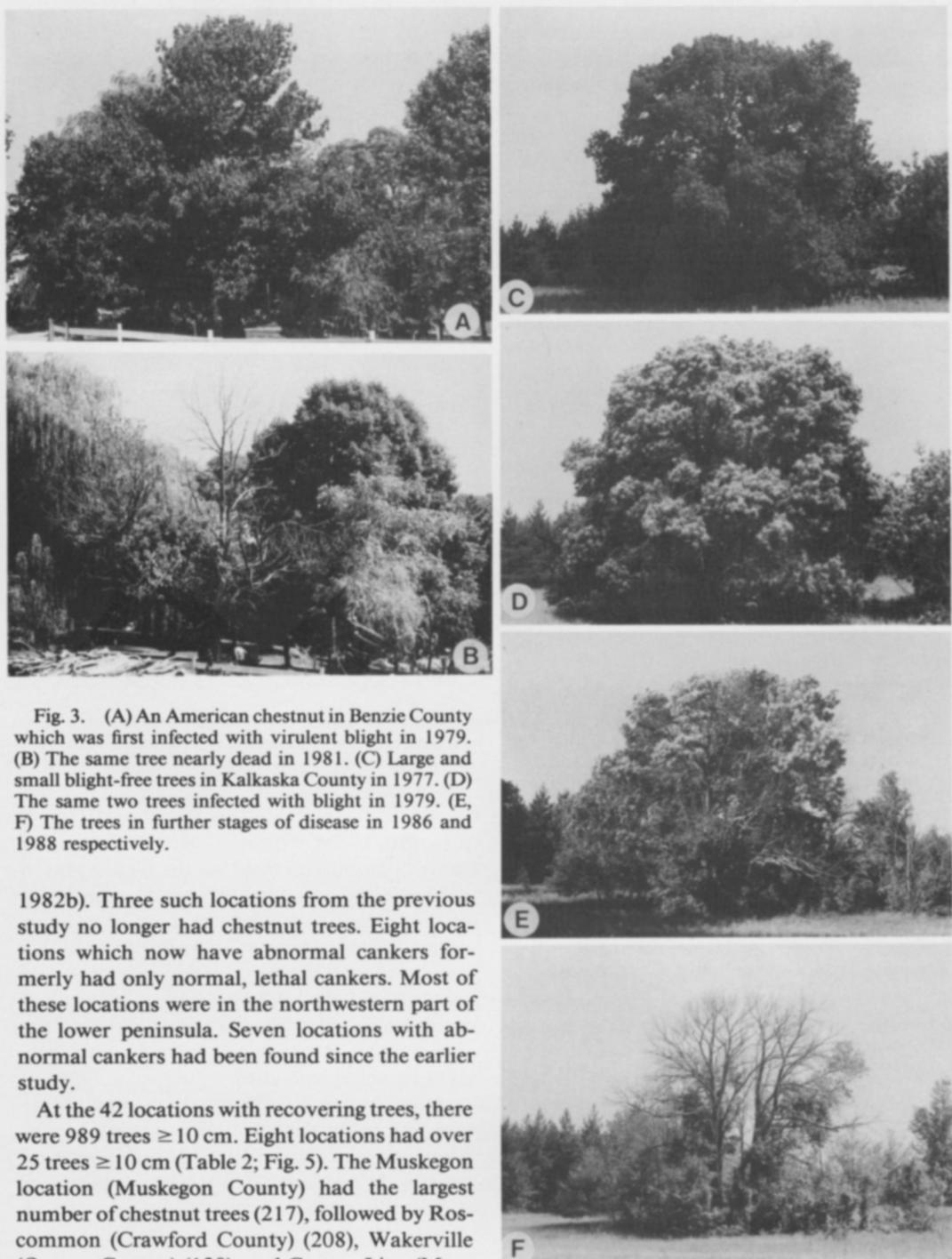


Fig. 3. (A) An American chestnut in Benzie County which was first infected with virulent blight in 1979. (B) The same tree nearly dead in 1981. (C) Large and small blight-free trees in Kalkaska County in 1977. (D) The same two trees infected with blight in 1979. (E, F) The trees in further stages of disease in 1986 and 1988 respectively.

1982b). Three such locations from the previous study no longer had chestnut trees. Eight locations which now have abnormal cankers formerly had only normal, lethal cankers. Most of these locations were in the northwestern part of the lower peninsula. Seven locations with abnormal cankers had been found since the earlier study.

At the 42 locations with recovering trees, there were 989 trees ≥ 10 cm. Eight locations had over 25 trees ≥ 10 cm (Table 2; Fig. 5). The Muskegon location (Muskegon County) had the largest number of chestnut trees (217), followed by Roscommon (Crawford County) (208), Wakerville (Oceana County) (130), and County Line (Man-

←

chestnuts <10 cm dbh. (D) The distribution and number of American chestnut trees ≥ 10 cm dbh which have been blighted since 1980. The size of the circles indicate relative numbers of trees at each location. The dates are the approximate times when blight entered specific locations as conveyed by nearby residents.



Fig. 4. Blight-free locations. (A) The Missaukee County location which has 144 large trees (≥ 10 cm dbh) and 4450 naturally produced smaller trees and saplings (< 10 cm dbh). (B) The Leelanau County location which has 55 large American chestnuts and 1500 smaller chestnuts which are taking over a beech-maple forest.

istee County) (120). These 42 locations also had 3233 smaller trees and saplings (< 10 cm) (Fig. 2B, C). The Roscommon location had the largest number (807), followed by Wakerville (550), Muskegon (533), Grand Haven (Ottawa County) (505), and County Line (197).

Based on information from nearby residents on the dates when the trees began to die and the rate of tree decline, it appears that all such locations in Michigan were initially diseased with virulent isolates of *C. parasitica*. Only after a 15 to 25 year lag period did abnormal cankers appear. In southwestern Michigan where blight was found at many locations in the 1930s and 1940s, trees at most of the locations began to recover

in the late 1950s and early 1960s. According to George Unger, who lived on the Grand Haven location (Ottawa County) for over 80 years, blight was first observed at this location in 1945, causing his 24 large trees to dieback. After 10–15 years, twelve of the trees were nearly dead and were cut. However, at the same time, on the other twelve trees, new growth began to equal the amount of dieback. In the last 25 years, only small branches have died.

In northwestern Michigan where the groves were infected in the late 1950s and early 1960s, recovery began during 1970. The Roscommon location (Crawford County) in northcentral Michigan is unusual in that it was infected by

Table 2. The number of live trees (≥ 10 cm dbh) per location at recovering locations.

Number of locations	Number of trees (≤ 10 cm dbh) per location						Total
	1–5	6–25	26–50	51–100	101–150	151–225	
42	15	11	2	2	2	2	42

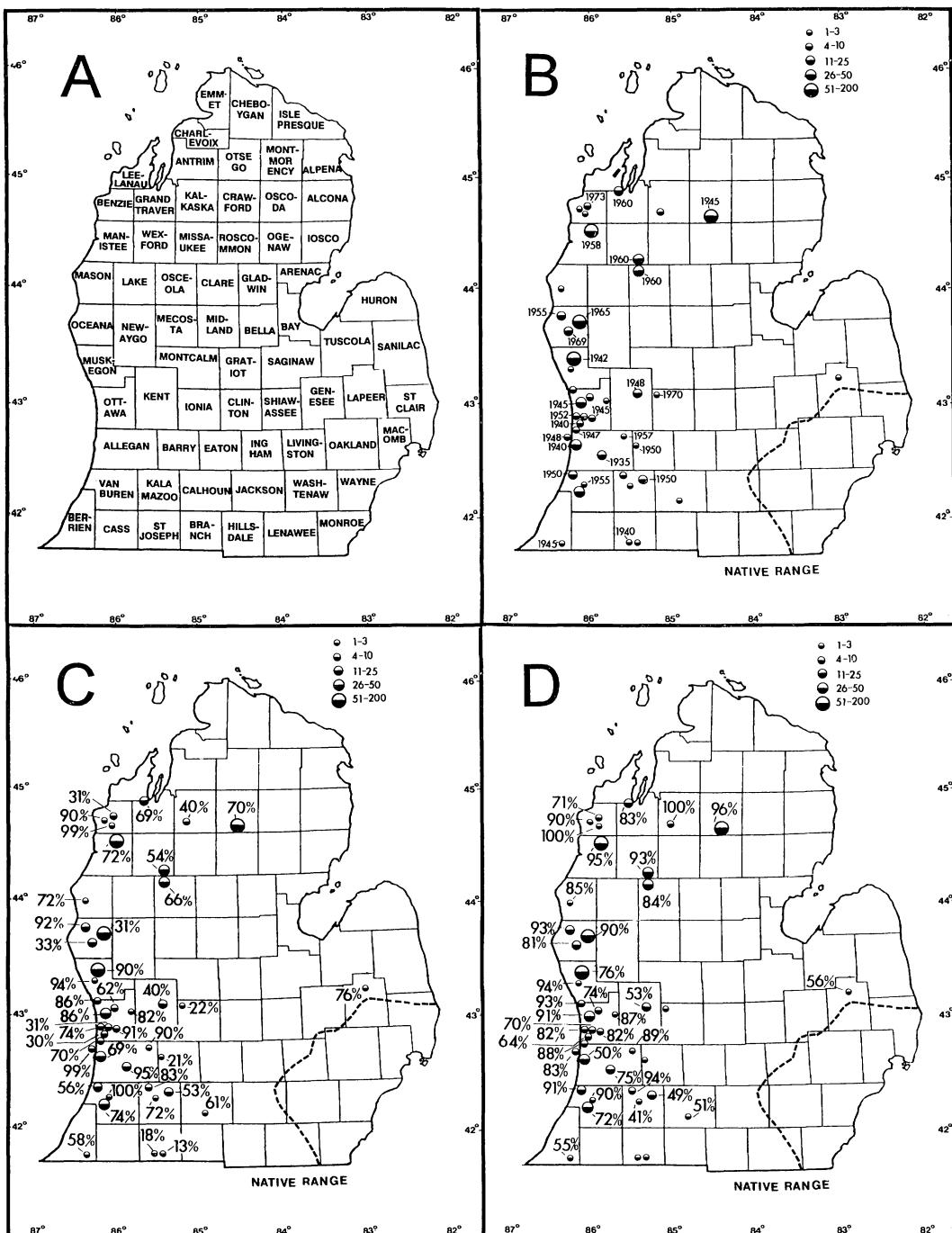


Fig. 5 (A) Michigan counties as they relate to the distribution of American chestnut trees discussed in the text. (B) The distribution and number of American chestnuts ≥ 10 cm dbh (diameter at breast height) which have abnormal cankers. The size of the circles indicate relative numbers of trees at each site. The dates are the approximate estimates of when local residents say that blight entered specific locations. (C) The percentage of cankers per site which are abnormal. (D) The total percentage of live branches above abnormal cankers. The size of each circle indicates the relative number of trees per site.

Table 3. A list of 42 locations of American chestnut trees in Michigan that have abnormal cankers. Included are the total number of trees and saplings at each site and the total number of those that were sampled, the number of cankers that were examined at each site and the percentage of those cankers that were abnormal, and the percentage of branches above abnormal cankers that were alive at each site. In addition, the dates when the trees first began to die and the soil texture at each site are listed.

Site #	Site name	County	# of trees saplings sampled (total # of trees and saplings)	Date trees began to die	Soil texture of A horizon	# of cankers examined	% of cankers abnormal	Success of abnormal cankers
1	Banger	Van Buren	(1)	955	sandy loam	401	100.0%	90.5%
2	Joyfield	Benzie	(1)	—	loamy sand	84	98.8%	100.0%
3	Lake Town	Allegan	25 (25)	1940	sand	326	98.8%	83.4%
4	Mill Grove	Allegan	23 (23)	1935	sand	285	94.7%	75.1%
5	Bear Lake	Muskegon	1 (1)	—	sand	53	94.3%	96.0%
6	Lee Curtis	Oceana	24 (24)	1949	loamy sand	425	92.2%	93.1%
7	Rose Park	Ottawa	6 (6)	1945	sand	236	90.7%	81.8%
8	Muskegon	Muskegon	26 (750)	1942	sand	777	90.3%	75.6%
9	Corning	Allegan	1 (1)	1957	sandy loam	20	90.0%	88.9%
10	Paul Rose	Benzie	1 (1)	—	loamy sand	49	89.8%	97.7%
11	St. Lazare	Ottawa	8 (8)	—	sand	268	86.2%	93.1%
12	Grand Haven	Ottawa	187 (575)	1945	sand	1833	85.8%	91.3%
13	Alamo	Kalamazoo	9 (9)	—	loamy sand	127	82.7%	94.2%
14	Tallmage	Ottawa	3 (3)	1945	loamy sand	1983	82.0%	87.3%
15	Lapeer	Lapeer	1 (1)	—	sandy loam	21	76.2%	56.3%
16	Webster Hills	Van Buren	71 (71)	1950	sandy loam	731	73.7%	72.4%
17	Riley	Ottawa	8 (8)	1952	sand	114	73.7%	82.1%
18	Eldridge	Kalamazoo	1 (1)	—	loam	25	72.0%	41.0%
19	Sounty Line	Manistee	317 (317)	1958	sand	5721	71.7%	94.7%
20	Peterson	Mason	4 (4)	—	sandy loam	440	70.7%	85.2%
21	Roscommon	Crawford	405 (1015)	1945	sand	1532	70.4%	96.4%
22	Black Bass	Ottawa	7 (7)	—	loamy sand	158	70.3%	88.3%
23	Traverse City	Grand Traverse	63 (63)	1960	loamy sand	345	69.3%	82.9%
24	Augustat	Osceola	28 (40)	1967	sand	377	65.8%	83.9%
25	Buchanan	Ottawa	8	—	sand	224	61.8%	73.6%
26	Fish Lake	Calhoun	5 (5)	1938	loam	84	61.3%	51.2%
27	Saugatuck	Allegan	47 (170)	1940	sandy loam	370	60.5%	50.3%
28	Bertrand	Berrien	2 (2)	1945	sandy loam	38	57.8%	54.6%
29	Kibbie Corners	Van Buren	19 (19)	1948	loamy sand	39	56.4%	90.9%
30	Allen	Osceola	77 (77)	1960	loamy sand	937	53.8%	93.3%
31	Kellogg Forest	Kalamazoo	45 (70)	1950	loam	324	53.0%	49.4%
32	South Bordman	Kalkaska	6 (18)	—	sand	57	40.4%	100.0%
33	Rockford	Kent	71 (150)	1948	sandy loam	538	39.6%	53.1%
34	Ferry	Oceana	45 (45)	1965	loamy sand	610	33.0%	80.6%
35	Hill and Dill	Benzie	5 (5)	1973	loamy sand	44	31.2%	71.4%

Table 3. Continued.

Site #	Site name	County	# of trees sampled (total # of trees and saplings)	Date trees began to die	Soil texture of A horizon	# of cankers examined	% of cankers abnormal	Success of abnormal cankers
36	Wakerville	Oceana	192 (700)	1965	sand	1334	31.1%	90.4%
37	Bazon	Ottawa	10 (10)	—	loamy sand	193	31.0%	70.0%
38	Pine Creek	Ottawa	14 (14)	1949	loamy sand	44	29.5%	64.3%
39	Ellis	Ionia	2 (2)	1970	sandy loam	27	22.2%	—
40	Gun Lake	Barry	1 (1)	—	loamy sand	19	21.0%	—
41	White Pigeon	St. Joseph	2 (2)	1940	sandy loam	51	17.0%	—
42	Sturgis	St. Joseph	1 (1)	1940	sandy loam	24	12.5%	—
Totals				1761 (4222)		21,288		

blight in 1945 but has only begun to recover significantly since 1980.

TREE CONDITION AND SPREAD OF HYPOVIRULENCE IN THE STATE. At the 42 sites I examined 21,288 cankers on 1761 trees. Between 13% and 100% of the cankers were abnormal (Fig. 5C; Table 3). Twenty-two locations had over 70% of the cankers abnormal, and nine sites had over 90% abnormal. The percentage of live branches above abnormal cankers ranged from 41% to 100% per site (Fig. 5D; Table 3). At 21 locations over 80% of the branches were alive above abnormal cankers, and at 14 sites over 90% were alive.

Based on the size of stump sprouts, as well as past observations of abnormal cankers at the sites, American chestnut trees in southwestern Michigan began to recover in the 1950s and 1960s. Most of these locations were infected with blight in the 1930s, 1940s and early 1950s (Fig. 5B). As a result, there appears to be a 15 year time lag between when sites first receive blight and the time they began to recover.

In northwestern Michigan the trees only began to recover since the 1970s. All six of the locations in this part of the state were infected by *C. parasitica* in the late 1950s and the 1960s (Fig. 5B) and all had few or no abnormal cankers at the time of my earlier study in the mid to late 1970s. However, in the present study these locations had recovered greatly with significant numbers of abnormal cankers and increased stem sizes. For example, in 1975 trees at the County Line locations (Manistee County) had many normal cankers, and very few abnormal cankers (Fig. 6A). However, in 1987 (Fig. 6B, C) 72% of the cankers in the grove were abnormal, with 90% of the cankers on the older trees in the northern portion of the grove being abnormal. The recovery of the trees in this grove is shown by the increase in stem sizes. In 1975 there were only five live stems over 10 cm dbh and only one stem over 15 cm, while in 1987 there were 254 live stems (in 120 sprout clumps) over 10 cm and 101 stems (in 65 sprout clumps) over 15 cm. This site also is of interest because the cankers were less swollen than the typical abnormal cankers, and 95% of the branches above them were alive.

The Joyfield Road tree (Benzie County, northwestern Michigan) also has made a dramatic recovery in the last 13 years. Figure 7A shows the tree in 1976 when it had a large number of normal cankers and many dying branches, including



Fig. 6. (A) The County Line site in Manistee County in the fall of 1975 when the site had many normal lethal cankers. (B and C) A similar area in the grove in the fall and summer of 1987 after the trees had made a dramatic recovery.

a trunk which had bark only half way around its circumference. In 1981 (Fig. 7B) this tree had lost the bark from the branches which were dying in 1976, but few recent branches were dying and new growth had begun to appear. By 1986 (Fig. 7C) the tree had continued to recover, 90% of the cankers were abnormal with 100% of the stems distal to them being alive. In 1989 (Fig. 7D) the tree was continuing to do well despite having been stressed by the 1988 drought.

The most rapid increase in number of abnormal cankers in northwestern Michigan has been at the Augustat site (Osceola County). In 1984, trees at this site were infected and had hundreds of normal cankers. Only a few abnormal cankers (<1%) were found at the base of two clumps of sprouts at the southeast portion of the grove. However, in 1987 there were 377 (66%) abnormal cankers and only 183 (34%) normal ones. Branches were alive distal to 84% of the abnormal cankers at this location.

The Roscommon site (Crawford County) was

the only site in the northeastern portion of Michigan's lower peninsula with recovering chestnut trees. Trees at this location had few (<10%) abnormal cankers in 1980 but in 1987, 70% of the cankers were abnormal. More importantly, 96% of the branches above the abnormal cankers were alive. This is the highest percentage of non-lethal cankers of any of the major groves, even though these trees are competing in a forested location.

SOIL TEXTURE AND CHESTNUT TREES RECOVERY. Figure 8A shows the average percentage of abnormal cankers for trees on soils of different textures. (Since sites were in different stages of recovery, data were used only from 22 sites that had the blight for over 15 years and had begun to recover before 1980.) Trees in sand had the largest percentage, with 87%, while all others were in the 50 or 60 percent range. Figure 8B shows the survival of branches above abnormal cankers on different textured soils. Sand and

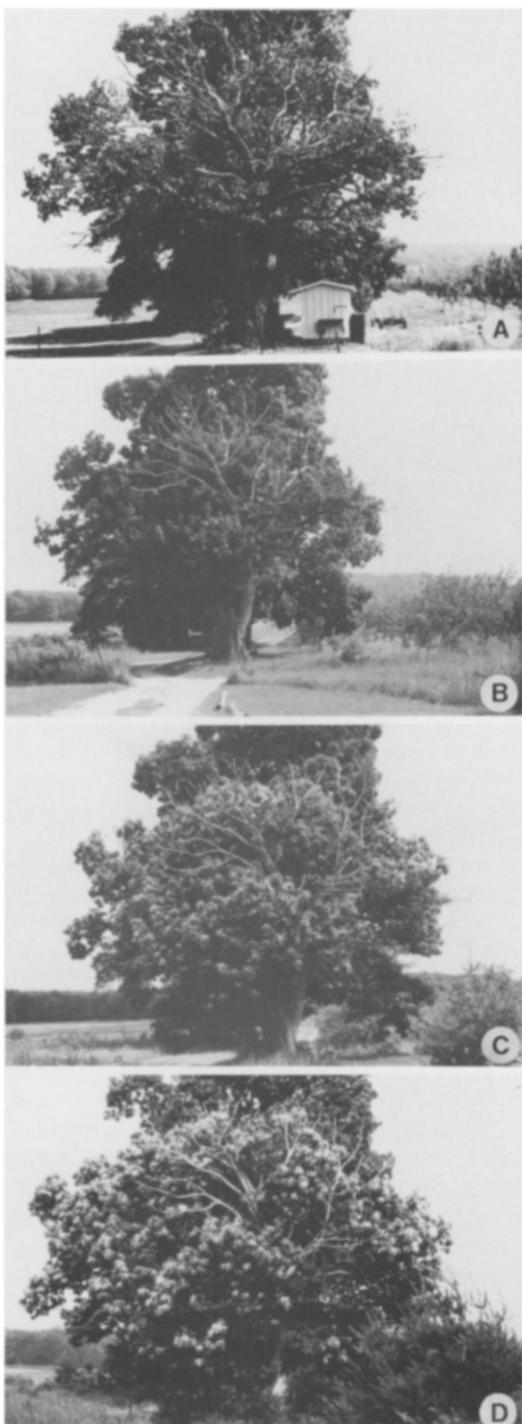


Fig. 7. (A) The Joyfield Road tree (Benzie County) in 1976 when it had many lethal cankers and had many dying branches. In 1981 (B) this tree had lost the bark from the branches which were dying in 1976, but only a few new branches with diameters less than 3 cm were dying at this time, and new growth had begun to appear.

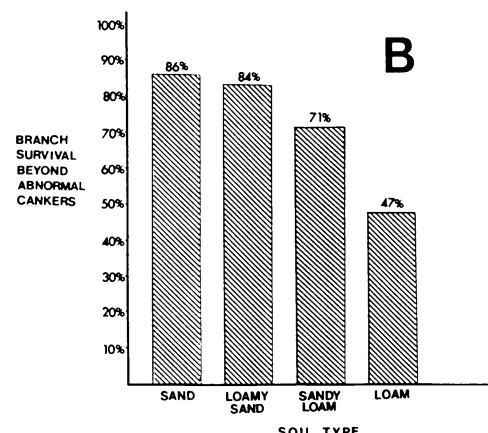
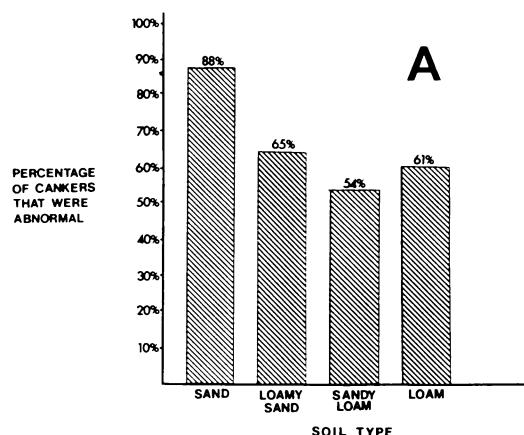


Fig. 8. (A) The average percentage of abnormal cankers for sites on different textured soils. (Since sites were in different stages of recovery, data was only used from sites that had the blight for at least 15 years and had begun to recover since 1980. This includes 22 of the 42 sites with abnormal cankers.) (B) Shows the average branch survival above abnormal cankers for sites on different textured soils.

loamy sand had the most with 86% and 84%, respectively, while loam had only 47%. Analysis of variance showed a significant difference in the number of abnormal cankers between different soil types at the 0.05 level, and a significant difference in branch survival between different soil types at the 0.01 level. These differences in re-

←

(C) The tree in 1986 after it had continued to recover and had 99% of the cankers abnormal and 100% of the stems alive above those cankers. (D) The tree in 1989 after the great drought of 1988.

covery are even more pronounced when one includes those diseased sites where abnormal cankers have not yet been found.

Discussion. This study has shown that when planted beyond its natural range in Michigan the American chestnut has grown and reproduced well. In fact, in a number of locations the tree is spreading into forests and is outcompeting oaks, and even beech and maples. Similar replacement of oak by introduced American chestnut has also been observed in southwestern Wisconsin by Paillet and Rutter (1989). The slow post-glacial migration of the American chestnut into Michigan was due in part to the tree's inability to reproduce and compete successfully in places like Ohio where the glacial soils are fine textured with a high lime content (Russell 1987). In its natural range the American chestnut was most abundant on well drained acidic soils (Braun 1950; McCormick and Platt 1980; Russell 1987; Paillet 1988; Stephenson et al. 1991). The fact that much of northern Michigan is composed of well drained acidic soils (Dorr and Eschman 1970; Veatch 1953) may in part explain the tree's success in the state. The ability of the American chestnut to grow and reproduce 200–300 miles beyond its natural range supports the theory that the tree would have eventually migrated into these areas if blight had not entered the state (Davis 1976; Paillet 1982).

RECOVERING TREES. The results of this study show that abnormal cankers have developed at 42 locations, and that they have developed approximately 15–25 years after blight entered these sites. The results also indicate that the development of hypovirulence at sites in Michigan appears to be following a different pattern than the blight. Blight spread from southeastern Michigan to northwestern Michigan and had a tendency to strike the locations with the largest concentration of trees first. In contrast, hypovirulence appears to have developed first in the southwestern Michigan and gradually appeared further north as time went on, with no real preference for sites with greater numbers of trees. The recovery for most sites in southwestern Michigan began in the 1950s and 1960s, while in northwestern Michigan it began in the 1970s. In addition, comparisons from thirty-nine sites which were examined in my 1975–1981 survey have indicated that the numbers of abnormal cankers and the number and size of large sucker

sprouts had increased at most locations since the earlier survey.

We may never know the exact time and place of the origin of hypovirulence in Michigan. In fact, the origin may very well be from a location not yet found, or from a location where the trees have now been removed. However, since in this survey the trees which first received blight, and subsequently abnormal cankers, were from southwestern Michigan, there is a good possibility that hypovirulence first arose from this part of the state. Since the Mill Grove location in Allegan County was the earliest of the 42 known sites with abnormal cankers in this survey to be infected with blight (1935), this location may have been the first location of those discovered to develop abnormal cankers. Because the largest sprouts at this location are approximately 35 years old, one can estimate that recovery might have begun around 1950.

The present study shows a relationship between the soil texture of sites with the American chestnut trees and the success of hypovirulence in Michigan. This relationship indicates there are environmental factors associated with soils which are involved in the success of hypovirulence. However, these results do not indicate the actual cause(s) for the differences in success. At present very little work has been conducted on the ecology of the hypovirulent phenomena (Anagnos-takis 1977; Scharf and DePalma 1981; Russin et al. 1984; Griffin et al. 1984, 1991; Griffin 1986, 1989). As a result, much is lacking in our knowledge of the environmental factors (climate, soils, competition, dispersing agents, etc.) which affect the interaction between the American chestnut, *C. parasitica*, and dsRNA in the field. In order to provide possible explanations for the success of hypovirulence on different textured soils, as well as to stimulate further research into these causes for the hypovirulence phenomena, three hypotheses that could explain this difference in recovery are described below.

(I) The first hypothesis states that the American chestnut grows best in Michigan on well drained sandy soils where it does not have to compete with more mesic and wet species. Consequently trees on these soils are better able to withstand stress from abnormal cankers, which results in a greater success of hypovirulence. During the presettlement in Michigan the upland sites on heavy textured soils were dominated by mesic forest species such as American beech and sugar maple (Veatch 1953; Brewer et al. 1994), while those on well drained sandy soils were oc-

cupied by oak and pine communities. In its natural range in southeastern Michigan the American chestnut was most abundant in oak communities (Brewer 1982a; Voss 1985), as was the case in the natural range in the Appalachian Mountains (Braun 1950; Whittaker 1956). Although the American chestnut could attain great size in mesic forests in its natural range, the tree was less able to compete with the more mesic species in this environment.

In general, planted or naturally produced trees with abnormal cankers growing in open areas were more successful than those that were growing in forest conditions, and trees in oak and pine forests were more successful than those growing in mesic forests. These observations are similar to the results from experimental plots reported by Griffin et al. (1991) in Virginia and West Virginia, and Anagnostakis (1990) in Connecticut. As was pointed out in this study, as well as in the study by Griffin et al. (1991), there is a latency period before hypovirulence develops on American chestnut trees. As a result, American chestnut trees that are under less stress and are able to stay alive longer have a better chance of developing hypovirulence. Although the competitive stress hypothesis may be important in determining the cause for the difference in success of hypovirulence on different textured soils in Michigan, it is unlikely that it is the only cause since a number of sites with American chestnut are located in areas where there is little or no competition from other plants but there is still a difference in success of hypovirulence.

(II) The second hypothesis for the greater recovery on sandy soils involves the origin of the dsRNA particle which causes most of the hypovirulence in *C. parasitica* (Day et al. 1977; Choi and Nuss 1992). In contrast to the belief that the hypovirulent strains of *C. parasitica* are being dispersed from grove to grove, this hypothesis states that the dsRNA may have existed in some soil fungi (or other organism) before *C. parasitica* arrived, and that the transfer of this particle to *C. parasitica* comes only after virulent strains of *C. parasitica* enter a site. If such soil fungi do exist, and are site specific for certain sandy soils, this could result in differential rates of recovery between sites. The fact that abnormal cankers are now known to be present at 42 sites throughout the state, and yet none have been found in a site without virulent strains first establishing an epidemic, supports this idea. While hypovirulent strains of *C. parasitica* may travel slower than virulent strains due to a lack of wind

dispersed ascospores (Elliston 1978), one would expect to find at least one infected site that started with abnormal cankers. In addition, there appears to be no clear preference for hypovirulence developing first at locations where there is a greater concentration of American chestnut trees, such as was the case for the spread of chestnut blight disease itself.

Further support for the hypothesis that dsRNA already existed at sites before virulent strains of *C. parasitica* arrived comes from the fact that abnormal cankers usually develop first in those parts of a grove where virulent strains initially appeared 15–25 years earlier. If hypovirulent strains were coming from outside the grove it would be hard to explain similar entry points, since most of the living chestnut biomass by the time hypovirulent strains arrived would be on trees in the outlying portions of the grove. If the dsRNA was coming from outside the grove, then chestnuts initially infected by virulent *C. parasitica* years earlier would be the last to acquire hypovirulence, since they would be nearly dead (*C. parasitica* have not been found to grow on dead wood (Anagnostakis 1987)). In contrast, if the dsRNA already existed in other fungi at most sites before virulent strains of *C. parasitica* arrived, this could explain the 15 to 25 year time lag between observations of virulent and hypovirulent strains. If one assumes that virulent strains of *C. parasitica* enter the tops of large trees first and then produce successive infections down to the base, it might take several years for the blight fungus to make its way down to the base of the tree, and an even greater number of years before the virulent blight would come in contact with fungi located on the soil. Also, the chances of virulent strains of *Cryphonectria* reaching the base of a tree and being converted to hypovirulent strains would be greatest on those trees which first received *C. parasitica*.

There is also some support for the hypothesis that hypovirulence agents already existed at sites based on where abnormal cankers initially appear on trees in those groves which have just developed hypovirulence. While it is too late to determine where abnormal cankers first appeared on trees at most sites, as mentioned above, abnormal cankers were first observed at the Augustat site at the base of two trees located at the southeast end of the grove. If the dsRNA with hypovirulence genes was being transferred from other soil fungi already at the site to incoming virulent strains of *C. parasitica*, the most logical portion of the tree for abnormal cankers to de-

velop first would be at the base. However, if the dsRNA was coming from hypovirulent strains of *C. parasitica* outside the grove one would most likely expect to find the first abnormal cankers at the top of the trees which have more chestnut biomass and are more exposed.

While soil fungi (or other organisms) from these sites have not yet been tested for dsRNA, it is known that soils from these types of sites tend to be acidic and contain large numbers of fungi (Brady 1974).

(III) A third hypothesis for why sites on sandy textured soils are recovering better states that dispersing agents may be more common on sandy soils. I have noticed particularly large numbers of ants in the genus *Formica* traveling across the ground and up and down chestnut trees at a number of recovering sites on sandy soils. While no one has yet studied the dispersal of *C. parasitica* by ants in the genus *Formica*, carpenter ants (*Camponotus* spp.) have been shown to be capable of dispersing *C. parasitica* (Anagnostakis 1982a). If the origin of the biological control agent is in soil fungi, or in fungi growing on nearby plants, then carriers such as ants, mites (Wendt et al. 1983), or insects (Russin et al. 1984), which travel across these fungi and up and down trees could be a link needed to transfer this agent. If the biomass of other fungi containing the agent is large in the grove, and a large number of carriers are present, there could be a fairly rapid change in the *C. parasitica* population.

Whether hypovirulence is more successful on sandy soils due to (1) the American chestnuts ability to compete better on sandy soils, (2) the origin of dsRNA being from fungi of sandy soils, or (3) there being more dispersing agents on sandy soils is unknown at this time. It may be that at different sites each of these factors may have a varying influence depending on the conditions.

There are also other factors which may be affecting the rate of recovery of various locations in Michigan. For example Anagnostakis (1977, 1982b), Anagnostakis et al. (1986), Anagnostakis and Kranz (1987), and Kuhlman and Bhattacharyya (1984) have suggested that there is a variation in the expression of hypovirulence in the field that is related to the genetic variability of blight. This difference in strains of blight could result in some locations of American chestnut trees in Michigan having a greater recovery rate. In view of the disjunct nature of the chestnut populations in Michigan, one might expect the isolated "islands" of populations might even serve to amplify genetic variability in blight and thus

enhance the differential recovery rates in the state. The influence of climate on the growth of *C. parasitica* and the American chestnut (Fulton 1912; Stevens 1917; Burke et al. 1976; George et al. 1979; Anagnostakis and Aylor 1984), and the extent of dispersing agents such as birds and mammals in the area (Heald and Studhalter 1914; Scharf and DePalma 1981) may also affect the recovery of different locations in Michigan. However, while these other factors may affect the recovery they are not likely to be more important on one type of soil than another, and thus would not likely explain the difference in recovery between sites on different textured soils.

Conclusion. Despite the spread of chestnut blight disease in Michigan, the state still has a large number of blight-free American chestnut trees which are reproducing naturally beyond their natural range. However, based on the present rate of spread of chestnut blight (21 out of the 144 diseased locations were infected since 1981) it is possible that all but a few isolated American chestnut trees (≤ 10 cm) in the state will be infected by the year 2020. Also, since results showed that the spread of blight is greater in locations with larger numbers of trees, all locations with more than 5 trees (≥ 10 cm) may be diseased by the turn of the century. Over the last fifteen years several hundred thousand seedlings have been grown from seeds collected by a group of people working with the Soil Conservation Service near Cadillac, Michigan. These seedlings have been distributed across Michigan and should provide a number of additional mature, blight-free trees in twenty-five years. However, with the blight that already exists in the state, many of these trees also will become infected before, or soon after, reaching maturity.

Fortunately, American chestnut trees at many locations are developing abnormal cankers, which allow them to mature and reproduce. With the success of these trees brings new hope for the future of the American chestnut in the state of Michigan. Based on the present study, as well as observations made over the last eighteen years, it is clear that a biological control of chestnut blight has been successful in Michigan's American chestnut trees growing on sandy soils. It now appears that the production of large stands for the purpose of harvesting nuts may soon be a reality on these sites. Possible ecological factors which may explain the differential success of hypovirulence on different soil types include: (1) a better competitive advantage of the American

chestnut on well drained sandy soils, (2) the origin of hypovirulence from sandy textured soils, and (3) more dispersing agents of hypovirulent strains on sandy textured soils. At this point it is unclear just how important each of these factors is in the hypovirulence phenomenon. Hopefully, with a better understanding of how *Cryphonectria parasitica*, dsRNA, and *Castanea dentata* interact we will be better able to assess the potential of hypovirulence as a biological control.

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ANNOUNCEMENT

Symposium on Population Biology of Grasses. The Torrey Botanical Club is pleased to announce that they are co-sponsoring the symposium: "Population Biology of Grasses", organized by Greg Cheplick. This symposium will take place at the annual AIBS Meeting to be held in San Diego, August 6-10, 1995. The following speakers have agreed to participate.

Baskin, C. & J. Baskin (U. of Kentucky)—Ecophysiology of seed dormancy and germination in grasses.

Briske, D. (Texas A & M U.)—Demography of clonal grasses.

Clay, K. (Indiana U.)—Influence of fungal endophytes on the population dynamics of grasses.

Cheplick, G. (City U. of NY)—Dispersal ecology of the grasses.

Davis, J. (Cornell U.)—Genetic structure of grass populations.

Detling, J. (Colorado State U.)—Morphological and physiological responses of grasses to grazing.

Jain, J. (U. of California)—Phenotypic plasticity and life history evolution of grasses.

Quinn, J. (Rutgers U.)—Ecological aspects of sex expression in grasses.

The Torrey Botanical Club will sponsor a mixer (cash bar) immediately following the symposium; everyone is welcome.