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Sapstreak Disease of Sugar Maple: Development Over Time and Space

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Abstract

Sapstreak disease is a potentially serious problem of sugarbushes and forest stands. It is caused by the fungus *Ceratocystis virescens,* which invades sapwood of roots and bases of stems through wounds created during logging, saphauling, or other activities. This report describes the results of observations and experiments to learn more about the patterns of disease development and the factors that affect them, within individual trees and within representative forests and sugarbushes.

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Introduction

During the course of our research to clarify the stress factors and organisms associated with dieback and decline diseases of sugar maple, we encountered several instances in which declining trees in managed forest stands and sugarbushes in northern New York had not been subjected to stresses usually associated with these problems.[1] The subsequent discovery that a number of these trees were affected by sapstreak disease suggested that this disease might be causing more loss than previously recognized, and that management practices were involved.

Background

Sapstreak disease of sugar maple (*Acer saccharum* Marsh) is a vascular xylem disease incited by *Ceratocystis virescens* (Davidson) C. Moreau (=*C. coerulescens* (Munch) Bakshi). See Smith (1990) for a nomenclature history of the sapstreak fungus. The disease was first noticed in North Carolina about 1935 (Hepting 1944), then in Michigan in 1959 (Kessler and Anderson 1960), Vermont in 1964 (Houston and Fisher 1964), Wisconsin in 1971 (Kessler 1972), and New York in 1978 (Beil and Kessler 1979). In each of these cases, the disease occurred in stands where activities such as logging, road building, or sap hauling had injured the affected trees.

Injuries apparently allow *C. virescens* to invade and then kill the xylem of the lower portions of stems and roots where it creates the distinctive "sapstreak" stain (Hepting 1944, Ohman and Kessler 1963, Kessler 1978, Houston 1985). The common occurrence of the fungus as a saprophyte on freshly cut bolts of sugar maple and other northern hardwood species (Shigo 1962) suggests that in managed stands inoculum is readily available.

The external syndrome begins with leaves that are small and off-color (Fig. 1), continues through progressive stages of crown dieback, and finally ends in tree death. These stages may ensue rapidly with trees dying in 1-2 years or the stages may stretch over several years. Kessler (1972) reported that no symptomatic trees recover.

Inside the tree, the diseased wood of roots and lower parts of stems exhibits a distinctive stain (Fig. 2). Freshly exposed, the stained wood is greenish yellow to yellow-tan with red flecks and the wood appears watersoaked. Often, in cross section, the stain columns appear to radiate outward and are bordered by a thin, dark-green margin. Soon after exposure, the stained wood darkens dramatically, and later still, fades to a light brown.

[1]These trees were brought to our attention by New York State Department of Environmental Conservation (NYSDEC) personnel, who earlier had confirmed the presence of sapstreak disease in a sugarbush tree (Beil and Kessler 1979).

Figure 1.--*A thin crown with leaves much smaller than normal,* the first indication that a sugar maple tree may have sapstreak disease.

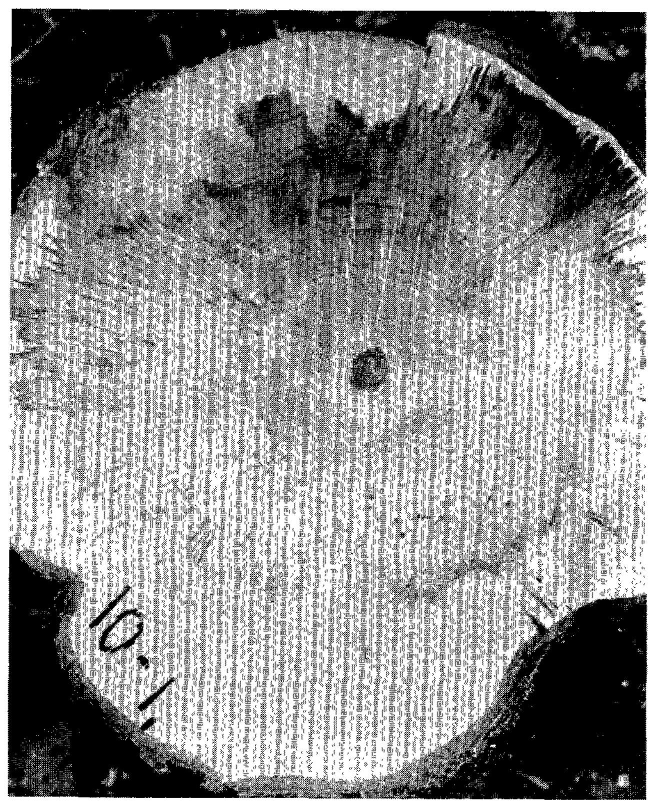

Figure 2.--The unique stain of the sapwood, which occurs most often in roots and lower stems.

The studies reported here were conducted to learn more about the patterns of disease development and the factors that affect them, within individual trees and within representative forests and sugarbushes.

Disease Development in Sugarbushes and Forests

Observations were made annually for up to 11 years in three stands in northern New York State.[2] These included a sugarbush in Diana, a forest stand near Barnes Corners logged in early summer 1981, and a second forest stand, also near Barnes Corners, thinned in 1980. While the majority of observations were made in these areas, numerous sapstreak diseased trees in other sugarbushes and forest stands in New York and Vermont were also examined, sometimes several times, and records were made of symptoms, locations, and disturbance histories.

Patterns in the sugarbush. The Diana sugarbush, a 65-acre portion of a mature, pure sugar maple stand, has been in operation for about 75 years. Sap is collected by buckets (2,000 taps) and hauled by tractor-drawn wagons. Summer activity includes felling of diseased or weakened trees and hauling the resulting wood to be stacked near the sugar house. The disease was observed first in the Diana stand in 1978 and affected a single tree located close to the sugar house.[1] A survey the next year located four additional symptomatic trees. Isolations from stained buttress-root tissues yielded *C. virescens.*

In 1980, six study plots, each containing at least one symptomatic sapstreak-diseased tree, were established to observe the progress of disease within the symptomatic trees, to observe for patterns of "spread" from diseased to healthy trees, and to monitor the appearance of symptoms in infected but initially asymptomatic trees. A seventh plot was established in 1982. Plots varied in size and shape and contained 15 to 32 trees. The locations of all plot trees > 4.0 in (10 cm) diameter at 4.5 ft (1.4 m) (d.b.h.), dead trees and stumps, and woods roads were mapped. The d.b.h., crown class, general crown condition, and the presence or effects of insects and disease agents were noted. The distances from each tree to haul roads, sapstreak-diseased trees and stumps; and the location, size, and probable cause of any visible root or buttress wounds were recorded.

In 1979, it was determined that sapstreak-stained wood characteristically was very low in electrical resistance (ER), (≤ 50 K ohms and often as low as 5-10 K ohms), compared to healthy tissue (100-700 K ohms), and that tissues infected by sapstreak disease reliably could be identified by their ER measurements (Table 1) (Houston and Schneider 1982).[3] At the outset, and periodically thereafter, ER of buttress-root wood was measured for each plot tree to detect the presence of stained tissue in otherwise asymptomatic trees. Measurements were made in up to four roots, one each on opposite sides of the tree, or until diseased tissue was encountered (Table 2).

[2]In each of these stands at least one sapstreak-diseased tree had been detected previously by NYSDEC personnel.

[3]Electrical resistance was measured with a Shigometer (Model OZ-67, Osmose Wood Preserving Co. of America, Inc., Buffalo, New York) and twisted wire probe (Shigo and Shigo 1974). The method utilizes the fact that as wood becomes discolored, cations increase in concentration and electrical resistance decreases (Tattar et al. 1972). Holes 0.10 in (2.4 mm) diameter were drilled to depths of 1 to 2 in (2.5-5.0 cm) in up to four buttress roots on opposite sides of the tree. The probe was inserted and resistance readings taken at 0.25 in (0.63 cm) intervals.

Table 1.--Comparison of electrical resistance (K-ohms) of buttress-root tissues of a healthy tree and a tree with sapstreak disease

	Electrical resistance (K-Ohms) - July 1980								
		Depth (inches) into root wood							
	Root	.25	.50	.75	1.0	1.25	1.50	1.75	2.0
Healthy	1	180	190	210	290	260	230	240	200
	2	250	230	160	210	256	200	260	260
	3	500	400	330	280	280	500	300	-
	4	320	220	230	240	240	250	340	500
Diseased:	1	21	34	18	27	22	46	21	-
Symptoms in 1980	2	15	18	23	17	13	13	9	11
Alive in 1981	3	80	80	70	80	70	-	-	-
	4	100	80	70	90	50	60	-	-

Table 2.--Electrical resistance measurements taken in 2 successive years on different sides of diseased tree 68 reveals the progression of the disease

		Depth (inches) into root wood							
Time of measurement	Side of tree	.25	.50	75	1.0	1.25	1.50	1.75	2.0
July 1980	N	24	45	35	110	80	35	-	-
(Symptoms in	W	500	500	450	380	300	500	500	-
extreme top)	S	450	250	180	170	250	190	380	200
	E	40	100	130	150	110	110	190	80
July 1981	N	55	60	70	60	55	65	-	-
(Symptoms	W	100	175	45	40	40	38	40	-
same as 1980)	S	140	180	280	280	210	220	220	190
	E	20	12	12	8	7	6	5	4

Annually through 1990, the condition of each plot tree was noted. When possible, photographs were taken of each diseased tree to provide a visual record of symptom progression. Each year the sugarbush, in general, was surveyed to detect any additional trees with crown symptoms. If such trees also possessed ER measurements and stain patterns characteristic of sapstreak disease, their locations in the stand were mapped and their conditions were followed annually until they died or were cut by the sugarbush owner.

Results. Locations of the plots and diseased trees are shown in Figure 3. Over the course of this study, 19 trees in the study plots (Table 3) and 4 outside the plots developed foliar and crown symptoms or the characteristic stain of buttress-root wood and were confirmed as having sapstreak disease. Trees in all crown classes were affected and ranged in size from 7.0 to 43.6 in (18 to 111 cm) d.b.h. Most trees were located within 5 ft (1.5 m) of haul roads, although three were 10 ft (3.1 m) away.

Although diseased trees often occurred and persisted as isolated individuals (Fig. 3: plots 1, 2, 4, 5), there were several cases where healthy trees relatively close to diseased trees became symptomatic (Fig. 3: plots 3, 6, 7). For at least two of these cases (Figs. 4, 5), it is quite probable that other diseased trees had occurred earlier as descriptions provided by the sugarbush owner suggested strongly that many of the stumps present in these areas were of trees affected by sapstreak.

Access roads and haul roads dissected the sugarbush, and depressions revealed the locations of many additional old roads no longer used. Most diseased trees possessed obvious and severe butt and root injuries attributable to equipment used in hauling sap or skidding logs. However, six trees bore no visible injury. Five of these were within 6 ft (1.8 m) of a road; the sixth tree, located 10 ft (3.1 m) from a major access road, was determined later to be connected by root grafts to an asymptomatic diseased tree 0.5 ft (0.1 m) from the road (Fig. 6). The possibility of root-graft transmission

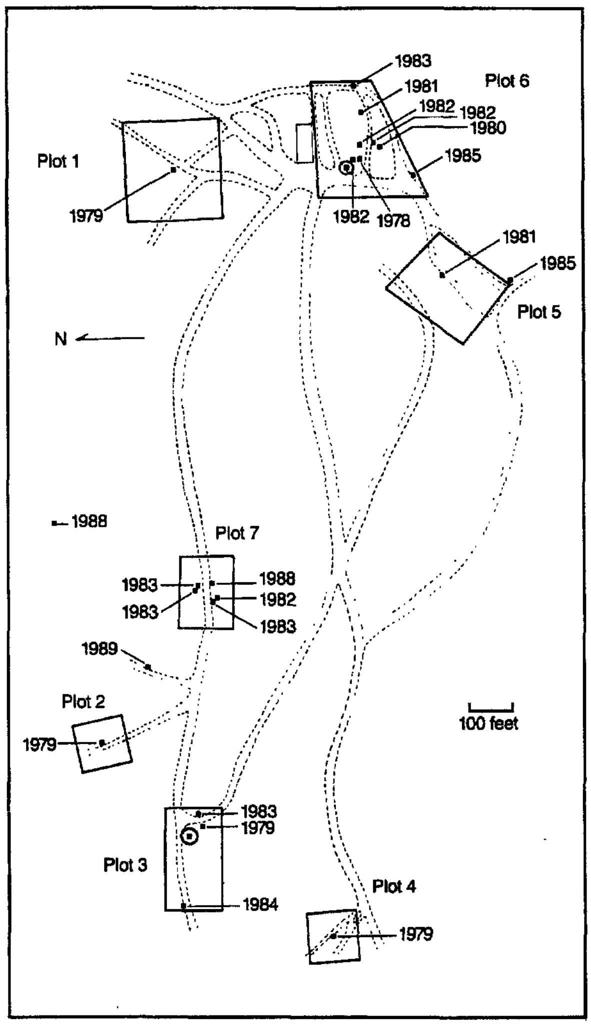

Figure 3.--Locations and years symptoms were first observed, of sapstreak-diseased trees (black dots), and stumps (circled dots), in and near study plots in a Diana, New York, sugarbush. Main access and sap hauling roads (dashed lines) lead to the sugarhouse adjacent to Plot 6.

Table 3.--Patterns of symptom expression in 19 sapstreak-diseased trees in a New York Sugarbush, 1979 to 1990

			Year			Distance (ft) to:		
Plot no.	Tree no.	D.b.h. (inches)	First symptoms	Dead, cut	Recovered (remission)	Road	Diseased tree	Associated[a] root rots
1	1	15.7	1979	1984		2.5		2,3,4
2	32	17.6	1979	1981		3.5		2,3
3	51	12.5	1984 (ER:1980)[b]			4.0		
	59	43.6	1979	1983		3.0		1
	60	15.0	1983 (ER:1982)			1.57		
4	68	20.2	1979		1980	6.0		
5	76	17.0	1981	1982		3.0		2,3
6	95	21.8	1981	1984		2.0		1
	98	19.3	1980	1983		10.0		2,3,4
	99	16.2	1982		1983	8.0	5.0	
	104	24.3	1983			2.0		
	107	16.8	1981	1982		3.0		1,3
	109	7.0	1982	1987		10.0		
	112	16.0	1982		1986	14.0		
7	120	26.3	1982	1985		10.0	7.0	1,2,3
	121	15.6	1988			3.0		
	128	14.9	1983 (ER:1982)		1986	0.5		
	133	12.9	1983 (ER:1982)			4.0	1.0	
	134	17.9	1983 (ER:1982)		1988	0.5		

[a]1: *Armillaria* sp.
2: *Xylaria* sp.
3: *Hypoxylon deustum*
4: *Ganoderma applanatum*

[b]Electrical resistance measurements in the given year were indicative of sapstreak.

was explored later in a separate study (see inoculation trial 5). Although roadways in this sugarbush were not excavated to determine if adjacent diseased trees had roots that extended beneath the road and had been wounded, we observed this to be the case in several forest stands elsewhere. Buttress-root injuries often were nearly closed over by callus by the time foliage symptoms first appeared.

In this sugarbush some of the major access roads had been used earlier for skidding logs. In wet areas, especially on slopes, skidders caused deep ruts and injured and exposed roots. Plot 3 (Figs. 3, 4), where sapstreak was severe, encompassed a road deeply rutted during the skidding. Sapstreak disease also was severe near the sugarhouse (Figs. 3, 5: plot 6). Over the course of this study, nine trees in this plot developed symptoms. Although trees in the sugarhouse area suffered the most traffic-related injuries, other factors (including a possible buildup of the pathogen on wood from diseased trees stacked near the sugarhouse) also may have contributed to infection of nearby wounded trees.

Patterns in forest stands. Disease development was followed in two forest stands on the Tug Hill Plateau near Barnes Corners, New York. The first of these (stand 1) was a red maple-sugar maple-ash stand (basal area: 120 ft^2/acre; 25 percent sugar maple), a portion of which had been harvested for sawlogs in early summer of 1981. The second (stand 2) was a sugar maple-beech stand (basal area: 131 ft^2/acre; 80 percent sugar maple) thinned during 1980.

In stand 1, skid trails radiating from the log landing site were numerous and deep. A survey in 1984 disclosed 25 trees with crown symptoms, stain, and ER readings characteristic of sapstreak. Two additional trees developed crown

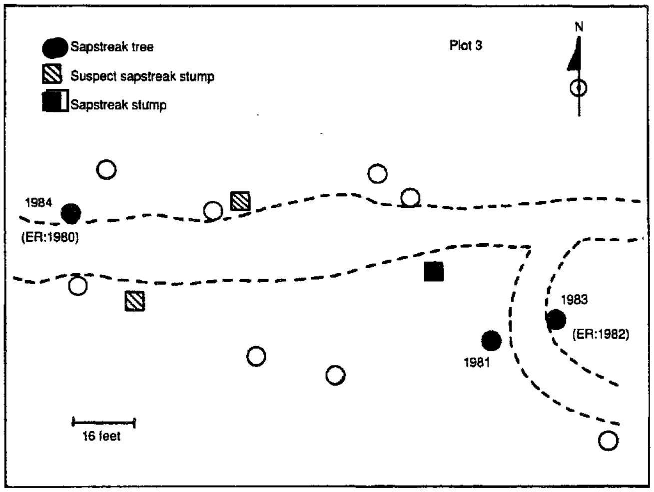

Figure 4.--Study Plot 3 in the Diana, New York, sugarbush. The enlarged section of roadway (within dashed lines) was deeply rutted during logging in 1975-1976. Dates represent years crown symptoms or electrical resistance (ER) measurements revealed sapstreak. "Suspect" stumps are of trees whose descriptions suggest disease.

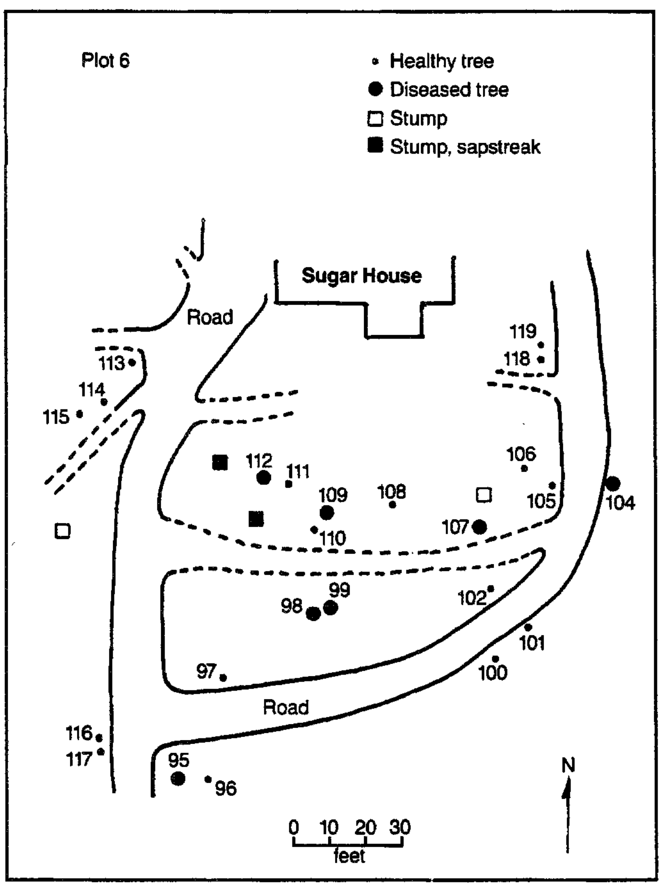

Figure 5.--Study Plot 6 in the Diana, New York, sugarbush. Numbered trees (larger solid circles) became diseased during the decade of observation (1980-1990).

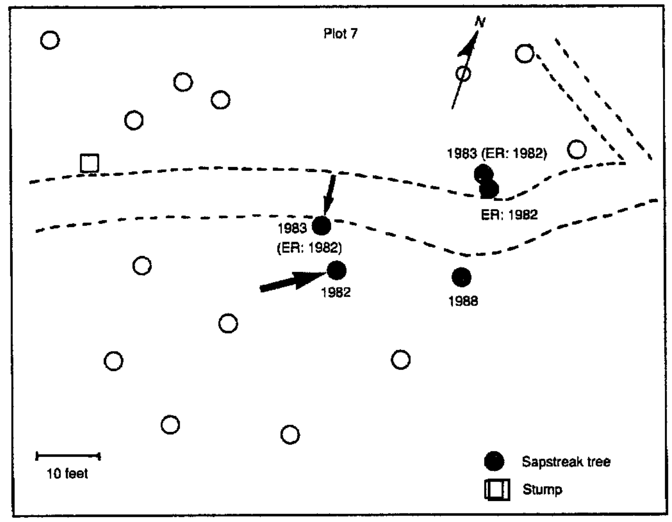

Figure 6.--Study Plot 7 in the Diana, New York, sugarbush. The diseased tree (large arrow), which first showed symptoms in 1982, had no discernible root or buttress wounds. Soil excavations revealed that its roots were grafted to the severely wounded and infected tree adjacent to the road (small arrow).

symptoms by July 1985. All of these trees were adjacent to skid trails (Fig. 7) and all bore severe buttress or root wounds, or both. No diseased trees were found in nontrafficked portions of the stand.

Other trees, in areas where the stand was opened severely and where water tables were high, also exhibited crown dieback, but lacked the internal sapstreak stain. While some of these trees had basal injuries, most did not, and many were not situated adjacent to skid trails. A few of these trees died during the observation period, but many recovered as they adjusted to the changed stand environment.

Twenty of the twenty-seven sapstreak diseased trees in this stand (10 in October 1984, 10 in May 1986) were felled and sawn into boards to determine the internal patterns of colonization (stain), the association of *C. virescens* with the stain, the persistence of *C. virescens* in air-dried lumber, and the appearance of diseased wood as it dried (Houston 1986). Six of the remaining seven diseased trees were dead by October 1985. All of these also were heavily colonized subcortically at their root collars by *Armillaria* sp.

In stand 2, sapstreak-affected trees were scattered over the 30-acre area (Fig. 8). Relatively few (12) symptomatic trees were discovered, and although they occurred on both upland and lower sites, five of them were concentrated in one lower and wetter area. Four diseased trees without obvious buttress or root wounds were residual members of sprout clumps. Stumps created when the sprout members were removed apparently served as infection courts; similar cases were found in two other New York stands where released, but otherwise unwounded, sprout members were affected by sapstreak disease.

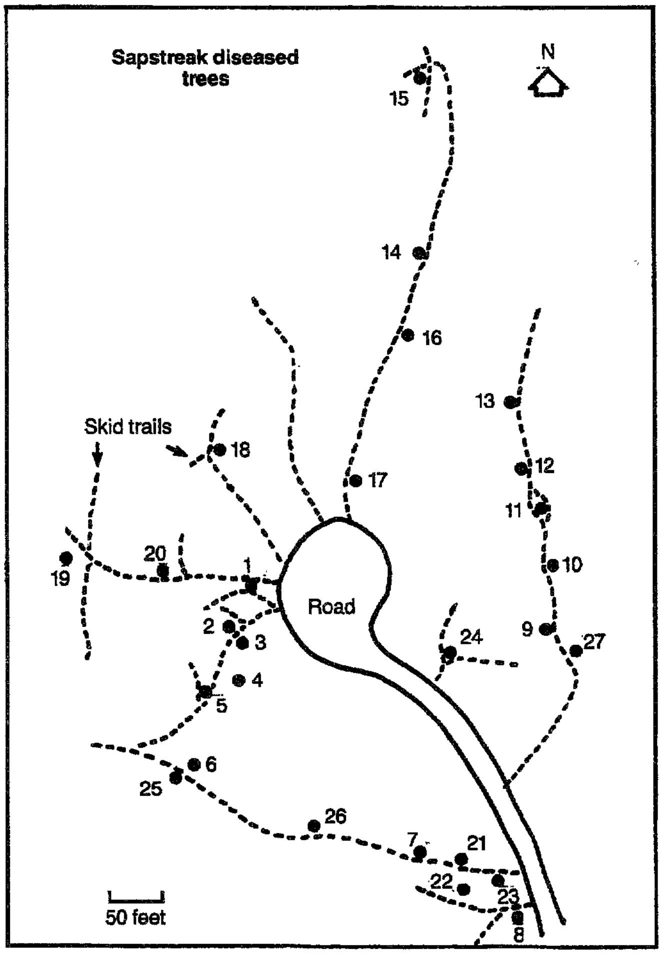

Figure 7.--Locations of sapstreak-diseased trees in forest stand 1 near Barnes Corners, New York, a portion of which was logged in 1981.

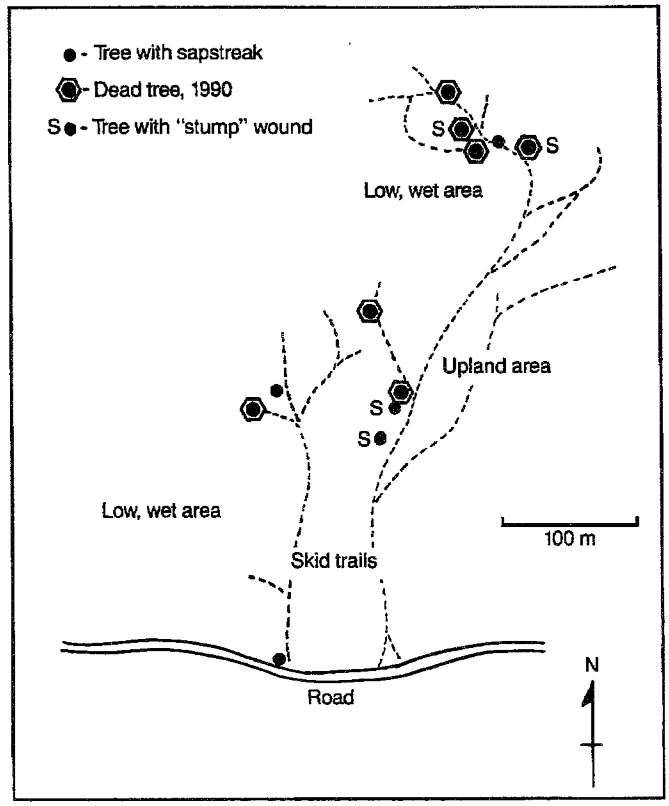

Figure 8.--Locations of sapstreak-diseased trees in a 30-acre portion of forest stand 2 near Barnes Corners, New York. The stand was thinned in 1980.

Patterns within individual sugarbush and forest trees. Within individual trees, the appearance of initial crown symptoms and the rate of their progression varied greatly (Table 3). Some trees with severe crown dieback, including some with but a few branches alive, survived for 5-6 years before they died, while others became symptomatic and succumbed rapidly, often within 2-3 years. Repeated measurements of ER on some individual trees revealed the progression of the disease. The pattern of development from July 1980 to July 1981 within the root system of tree 68 is shown in Table 2. Several very severely affected trees were harvested by the owner before they died. Disease progression, as revealed externally by crown symptoms and internally by ER measurements or root dissections, was arrested, and recovery ensued in several symptomatic trees (two with ≥ 40 percent crown dieback). At least three trees in the sugarbush, and two in forest stand 2, with root-stain patterns and ER measurements characteristic of sapstreak disease never developed severe crown foliar symptoms during the course of the study.

Usually, the first observed symptom of sapstreak was a distinctive "transparency" of the tree crown--a consequence of the presence of unusually small leaves, especially in upper branches, but sometimes over the entire crown (Fig. 1). Often, these small leaves were normal in color, shape, and number the first year, but became off-colored and sparse in subsequent years. Branch dieback often occurred where small leaves appeared the previous year, and this pattern of small leaves one year followed by death of supporting twigs and branches the next sometimes continued for several years until the tree died. Sometimes, however, symptom progression was arrested and resulted in trees with upper branch dieback or even major stagheading and lower branches fully foliated with leaves of normal size and color. While some of these trees apparently recovered with no further disease progression, others, after several years of apparent remission, again exhibited symptoms.

External symptoms were related closely to development of internal stain. Stain columns were well established, especially in root tissues, by the time crown symptoms appeared. In many trees, especially those in remission of crown symptoms, stain columns appeared to be contained by newly formed rings of healthy sapwood.

Associated organisms. In the sugarbushes and forest stands studied here, and in many others as well, trees dying of sapstreak disease also were almost always colonized at their roots or root collars by the root disease pathogens *Armillaria* sp., *Xylaria* sp., or, rarely, both (Table 3). This association was noted by Hepting (1944). Although their actual role in the disease has not been demonstrated, it is likely that these fungi contribute significantly to the death of affected trees. Their consistent presence on dying trees and their apparent absence from severely affected but recovering trees suggests that their attack may determine which sapstreak-affected trees die or recover.

Severely affected trees often were attacked, sometimes heavily, by one or two species of Ambrosia beetles. These attacks usually were concentrated, at least initially, near the buttress roots and lower bole. What role, if any, these insects or their fungal associates play in the disease is not known, but their appearance in later stages of disease development suggests that they probably are not vectors of *C. virescens*, but may hasten the demise of severely diseased trees.

Patterns within artificially inoculated trees and saplings. The preceding observations revealed much about the patterns of occurrence of diseased trees and of the factors that contributed to them. A considerable amount was learned about the progression of the disease within symptomatic trees. Even so, many questions pertaining to stand management remained unanswered. Some of these questions were addressed in a series of inoculation trials that were conducted with saplings and trees to provide information needed for developing management guidelines. Specifically, these studies attempted to determine:

• The nature and rate of symptom expression and disease development in saplings and trees beginning with the first infection,

• If trees are more susceptible or vulnerable to the disease at different seasons of the year,

• If the common stress factor, defoliation, directly influences susceptibility or vulnerability to sapstreak,

• If the rate or extent of sapwood colonization is affected by the nature (orientation) of the wound,

• If sapstreak disease can spread through root grafts, and

• If tapholes serve as infection courts for *C. virescens.*

The nature of symptoms, the rate of disease development, and the relationship of season of infection and of defoliation stress to susceptibility and vulnerability were addressed in Trials, I, II, and III; the influence of wound orientation on disease development within individual trees in Trial IV; the spread of the pathogen through root grafts in Trial V; and the role of tapholes vs. buttress-root wounds as infection courts in Trial VI.

TRIAL I (Symptom development, rate of disease development, season of inoculation vs. susceptibility and vulnerability)

• Materials and Methods. Two groups of saplings 2-4 in (5-10.2 cm) in diameter were used. The first group was growing in the understory near the edge of a stand of pole-size maple trees in southern Connecticut. This group of saplings was composed of single stems of seedling origin. The second group, growing about 50 ft (15 m) from the first, consisted of double stems of sprout origin that developed after a portion of the seedling grove was cut and thinned approximately 10 years earlier. The pole-size trees used in trial I were scattered within the stand and ranged in size from 7.5 to 10.0 in (19.0-15.4 cm) in diameter and were of intermediate or codominate crown class. The stand containing the trees and saplings used in Trial I was defoliated severely by gypsy moth (*Lymantria dispar*) in the summer of 1981. Although not observed, it must be presumed that the maples used in these studies had been defoliated to some degree.

Inoculum for these and subsequent trials was prepared by growing isolates of *C. virescens* obtained from sapstreak diseased trees on a sterile wheat grain medium for 10-14 days.[4] Groups of 10-20 saplings or sprouts were inoculated in November 1981 (10), May (20) and November (10) 1982 and June 1983 (20) by forcing inoculum into a 0.375-in (0.95-cm)-diameter hole drilled horizontally nearly through the stem at 6 in (15 cm) above ground. Bark and drill bit were sterilized before drilling by scrubbing or dipping with 95 percent ETOH. Drill holes, filled with inoculum or with sterile wheat grains for controls, were covered with masking tape. One member of each sprout clump was treated similarly. Fifteen pole-size trees were inoculated in May 1982 and 10 more in June 1983 by forcing inoculum or sterile grain into two 0.375-in (0.95-cm)-diameter holes drilled 2 in (5.1 cm) deep into buttress roots on opposite sides of the tree.

TRIAL II (season of inoculation vs. susceptibility and vulnerability)

• Materials and Methods. Beginning 1 July 1983, groups of 10 saplings each, growing in the understory of a second stand, were inoculated as above at intervals of approximately 2 weeks (1 July through 18 October 1983; 5 March through 3 July 1984) or of 4 weeks (10 November 1983 through 5 March 1984).

For both trials, all inoculated plants were examined for symptom expression and disease development at least once each growing season through 1990. ER measurements were made in buttress roots or lower stems of all trees with symptoms. Stems and buttress roots of all trees, and of representative saplings and sprouts that died, were dissected to determine the internal patterns of disease. Isolations were attempted from the tissues of all the pole-size trees that died. For saplings and sprouts, however, re-isolation attempts were made only for those that died shortly after inoculation. In saplings and sprouts that died more than a year after being

[4] 100 gm wheat grain in a 250-ml Erhlenmeyer flask moistened with enough water to equal the volume of wheat; flasks were plugged with cotton and autoclaved at 15 psi for 25 minutes.

inoculated, sapstreak-killed tissues usually were too decayed to support the pathogen.

•Results. In Trial I the rates of disease development, expressed as onset of foliar and crown symptoms and the occurrence of mortality, varied markedly with tree size (saplings and sprouts vs. pole-size trees), season, and year of inoculation. Moreover, the *nature* of symptoms between trees of the same inoculation series also was remarkably varied.

Nine of twenty saplings and 7 of 20 sprouts inoculated in May 1982, died within 1 year, and of these, six of each group exhibited complete wilt and death within 2 months. Proportionately fewer saplings or sprouts inoculated at other times during this trial developed symptoms and died (Fig. 9). In contrast, none of the 15 pole-size trees inoculated in May 1982, developed symptoms before leaf drop in 1982. However, by 18 months, six of these trees had developed crown symptoms and died. One of these trees did not leaf out in 1983, one flushed and then died suddenly in June, and another, which flushed normally, became increasingly chlorotic, then brown, and died in August. Two more of these trees had died by 1990.

Only 2 of 60 non-inoculated members of sprout clumps died even though they shared a common, and sometimes infected, root system. And, in contrast to the single-stem saplings, which continued to die through 1990, mortality among sprouts occurred only during the first 4 years after inoculation (Fig. 9).

Dissection of saplings and trees that died or possessed severe symptoms revealed extensive invasion of roots and often of stems by *C. virescens.* In most trees, vascular staining was present in, and the fungus was recovered from, xylem tissues in upper portions of stems often to 30-45 ft (10-15 m) above ground. As in naturally infected trees, dead or dying inoculated saplings and trees were attacked severely by *Armillaria* or *Xylaria,* or both. Also, as in naturally infected trees, inoculated trees and saplings dying of sapstreak frequently were infested, sometimes heavily, by one or more species of *Ambrosia* beetles. Attacks were concentrated primarily at the bases of the trees near, and especially below, the points of inoculation. Occasionally, however, these attacks occurred higher up the stems into streaks of sapstreak-killed xylem tissue that were close to the cambium.

In Trial II mortality patterns of saplings inoculated at different times during the season (July 1983 to July 1984) suggest that trees may be more susceptible or vulnerable to infection and disease development at particular times of the year (Figs. 10, 11).

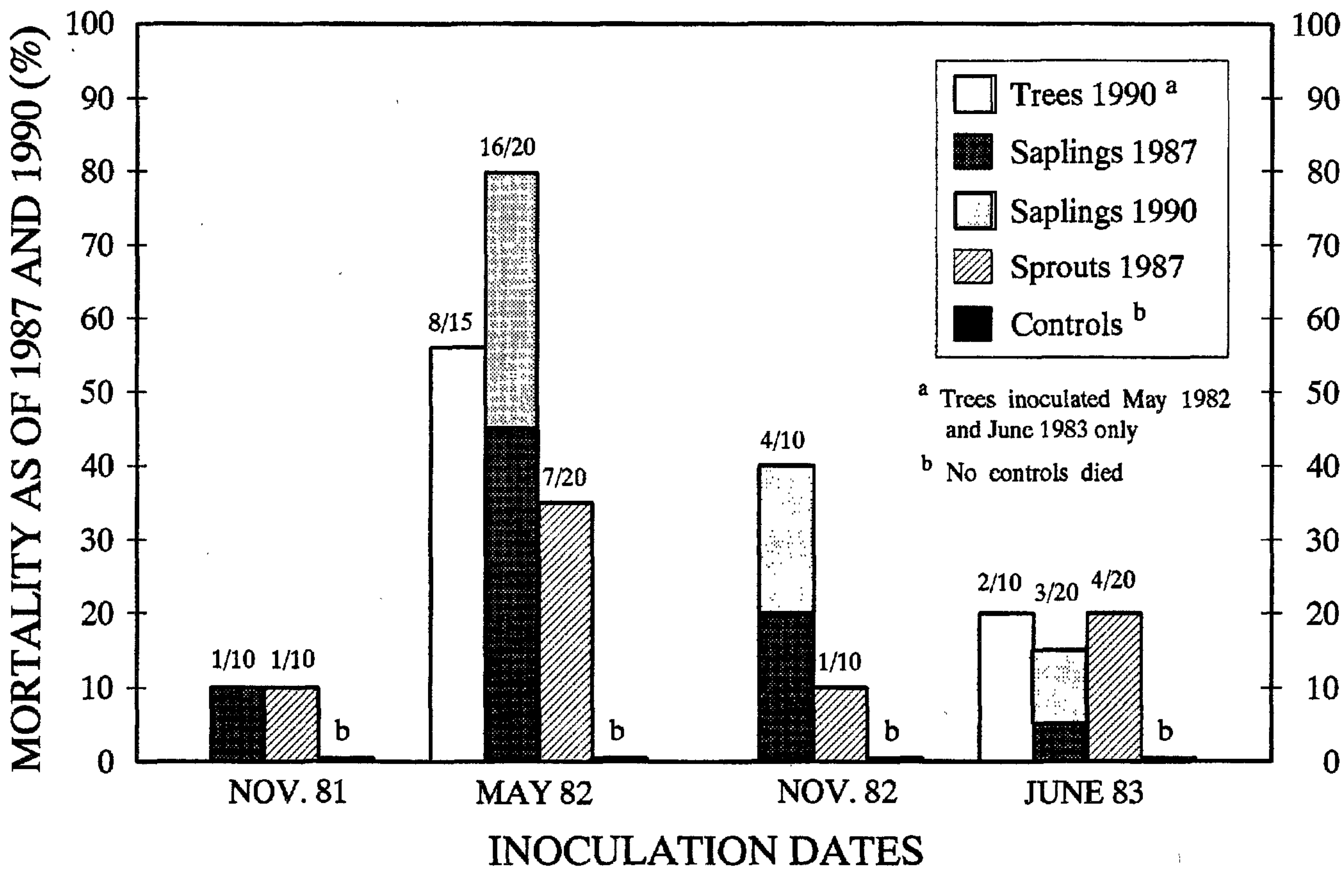

Figure 9.--Percent of saplings, sapling sprouts, or pole-size trees that died in 1987 and 1990 following inoculation on four different dates. Ratios indicate number died of number inoculated.

Mortality was highest (from 5 to 8 of 10) among each of the four sapling groups inoculated from early July through mid-August 1983, was lowest (from 0 to 3 of 10) among the four groups inoculated from late August 1983 through early April 1984, and then increased (with two exceptions) to from 4 to 5 of 10 in groups of trees inoculated from mid-April to early July 1984 (Fig. 10). The somewhat lower death rate among trees inoculated during 1984 compared to 1983 may reflect, at least in part, the 1-year shorter observation period given this group. When the 200 saplings were sorted into five groups of seasonal inoculation times, mortality patterns ranged from a low of 2.5 percent (1 of 40) in winter months to a high of 52.5 percent (21 of 40) in the summer (Fig. 11).

TRIAL III (Defoliation stress vs. susceptibility and vulnerability). The high mortality rate among trial I saplings and trees inoculated in May 1981 (Fig. 9), the first and second growing season after they had been defoliated severely by gypsy moths, prompted a trial to determine if defoliation stress before or after inoculation with *C. virescens* increased sapling and tree susceptibility or vulnerability.

• Materials and Methods. Eighty saplings, 2-3 in (5-7.6 cm) in diameter, growing in a more open portion of the same stand utilized in Trial II, were inoculated with wheat grain inoculum as described in Trial I; 40 on 15 May 1985, and 40 on 28 April 1986. Half of both groups were defoliated completely by hand on 14 June 1985 (a time chosen to reflect the period of peak defoliation activity by the gypsy moth).

• Results. The mortality of the trees in each of the four groups of 20 saplings through 1991 is shown in Figure 12. By August 1990, more of the saplings inoculated in May 1985 had died (16=40 percent) than those inoculated a year later (6=15 percent). No additional trees succumbed in 1991. Defoliation had no effect on mortality rates for the trees inoculated in 1985--equal numbers of trees died in each category. For those trees inoculated in 1986, however, only one of the

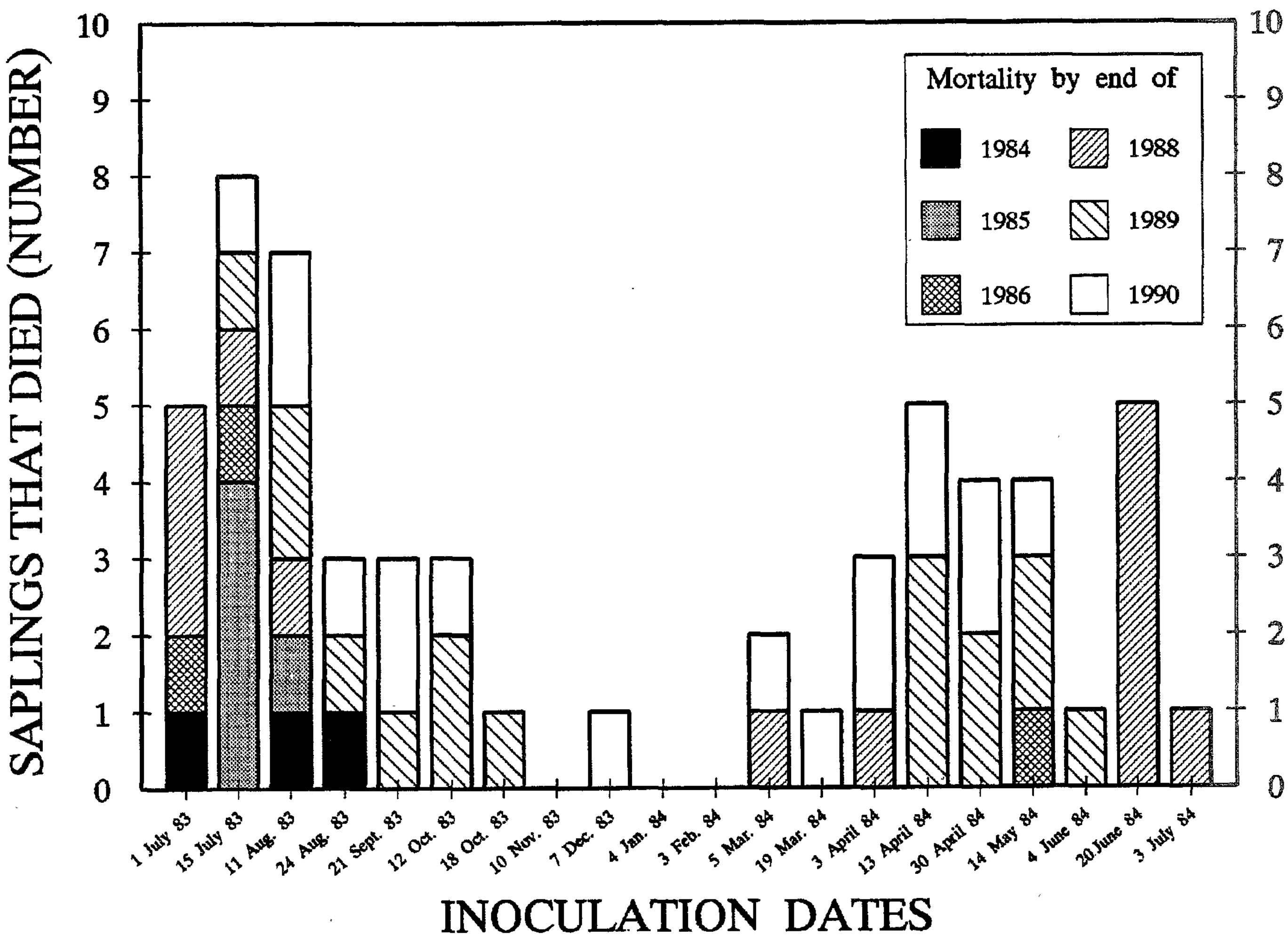

Figure 10.--The pattern of annual mortality within 20 groups of 10 saplings, each inoculated at biweekly or monthly intervals from July 1983 to July 1984.

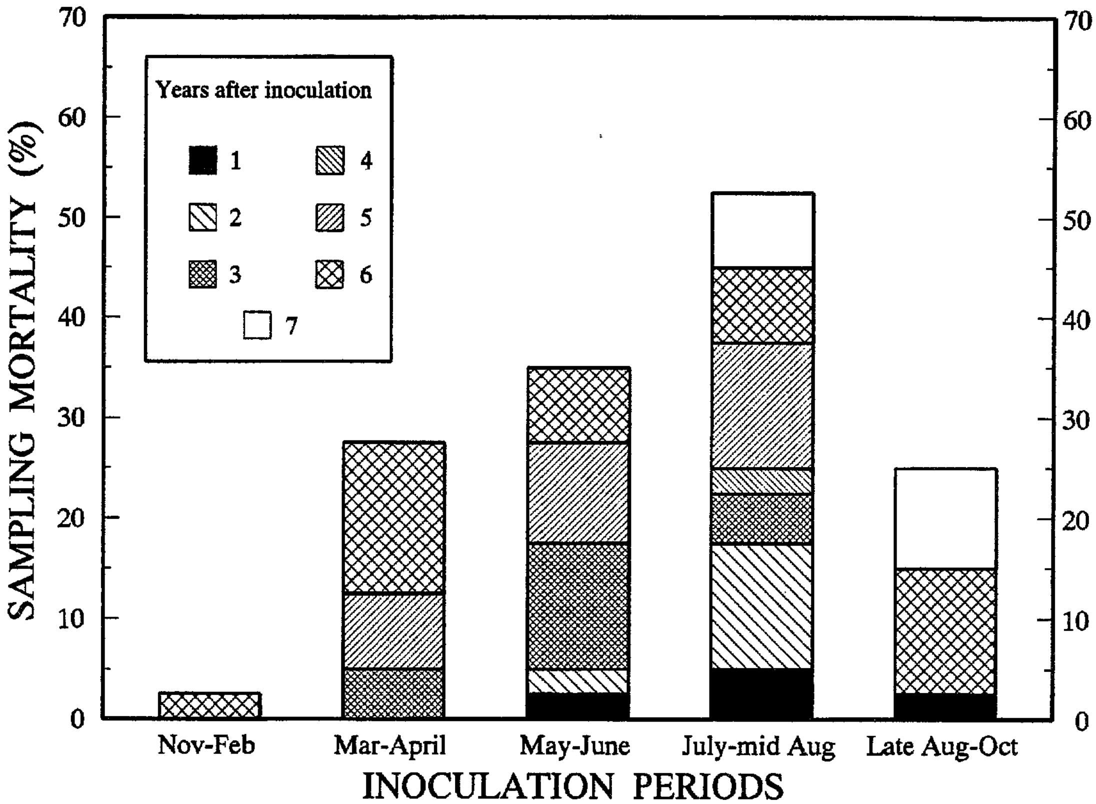

Figure 11.--The pattern of cumulative mortality, by years after inoculation, among five groups of 40 saplings inoculated at five different seasonal periods.

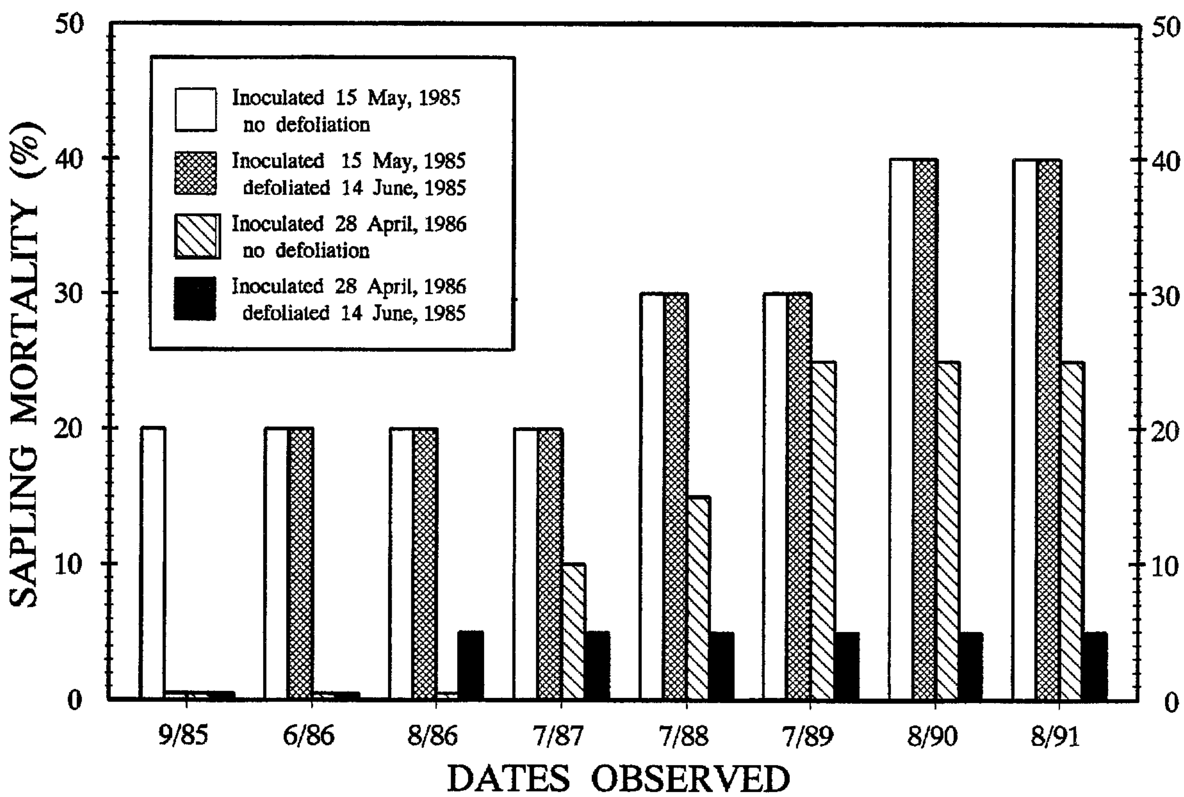

Figure 12.--Percent mortality over time, within two groups of 20 defoliated, and two groups of 20 nondefoliated saplings inoculated with *C. virescens*.

defoliated trees died compared to five of the nondefoliated series. Apparently, defoliation has no direct effect on resistance to *C. virescens.*

TRIAL IV (Wound orientation vs. pattern of disease development within individual trees). In a number of earlier studies, dissection of sapstreak-diseased trees revealed that the patterns and extent of sapstreak discoloration within individual trees varied considerably (Houston 1991, 1986, 1985). The extent of discoloration and decay within living trees following wounding is known to be related, in part, to the ability of trees to compartmentalize wounded tissues. One factor that influences this ability is the orientation of the wound relative to natural barriers within the tree, especially those created by annual rings and vascular rays (Shigo 1977, 1979). A small study was conducted to determine whether orientation of infection courts affects the pattern and extent of sapstreak development within individual trees.

• Materials and Methods. In July 1988, 10 trees, 4-6.7 in (10-17 cm) d.b.h., were inoculated with *C. virescens.* Wheat grain inoculum was introduced into increment borer holes. One hole made at 4.5 ft (1.4 m) was oriented radially, through the pith center and thus intersected relatively few vascular ray boundaries. The second hole, made at 3.25 ft (1.0 m) and oriented tangentially to the pith, intersected and thus disrupted nearly all of the vascular rays on one side of the tree.

Five of the ten trees were harvested after 1 year (7 June 1989), and five after 2 years (25 July 1990). Stems were sectioned at 4- or 8-in (10 or 20 cm) intervals from ground line through each of the inoculation wounds and above until stain columns disappeared. Photographs made of the ends of sections were used to characterize the cross-sectional patterns and linear extent of stain associated with each inoculation wound. The heights of the stain columns were rounded to the nearest 2 in (5 cm).

• Results. None of the inoculated trees developed foliar symptoms before they were harvested. The extent and cross-sectional patterns of the stain columns resulting from invasion by *C. virescens* varied considerably between trees (Table 4). Columns associated with the tangentially oriented wounds generally were greater, both in cross section and in linear extent than those associated with radially oriented wounds. In a few trees, these differences were striking (trees 5, 6); while in some, (trees 3, 4, 10) the columns were limited in development regardless of wound orientation.

The dramatically greater colonization in this study of wounds that disrupted more of the tree's pre-established compartment barriers suggests that the type of wounds created in logging or sap hauling can influence disease incidence and severity in a major way.

TRIAL V (Root graft transmission of *C. virescens*). As mentioned previously, a few of the sapstreak-diseased trees observed did not possess the usually obvious root or lower stem wound infection courts. Observations in permanent plots (Figs. 6, 7) suggested that some of these trees may have become infected when the fungus moved into their roots across root grafts with adjacent diseased trees. An experiment was conducted to determine if root-graft transmission does occur and if so, whether it is an important consideration in management of this disease. A preliminary report has been published (Houston 1991).

• Materials and Methods. In 1987, 10 pairs of healthy pole-size sugar maples growing in forest stand 1 near Barnes Corners, New York, were selected for the study. Members of each pair were growing in close proximity (6 to 10 ft, 1.8 to 3.1 m) and, as revealed by careful excavation, were connected by presumably functional root grafts. Electrical resistance (ER) measurements were made (Shigometer with twisted wire probe) in the roots on both sides of the graft as well as in the major buttress roots of the trees to assure that they were healthy at the time of inoculation. In July 1987, one tree of each of five pairs of trees was inoculated with *C. virescens.* Inoculated roots included the grafted root and all major buttress roots. The second group of five pairs was treated similarly in July 1988.

A conidial isolate of *C. virescens* selected for its tolerance to 2 ppm MBC (benomyl), obtained originally from diseased tissue (tree 1, plot 1, Fig. 3), was used. Use of this marked isolate made it possible to determine later whether infection and colonization had resulted from our inoculations or from natural "wild" inoculum. Inoculum consisted of 2-week-old cultures grown on and mixed thoroughly with a wheat grain substrate.

Bark surfaces were scrubbed briefly with water to remove dirt and then with 95 percent ETOH (ethyl alcohol). When dry, 0.25-in (0.16-cm)-diameter drill holes 2 in (5.1 cm) deep were made and filled with inoculum. Drill holes were covered with masking tape. Sketches were made of the excavated roots and inoculation points before roots were covered with soil and loose litter (Fig. 13).

In June 1990, the root systems of both trees in each pair were re-excavated, then dissected to determine the extent of infection and colonization. Root samples were taken for re-isolation of the pathogen where ER measurements or stain patterns, or both, suggested that the fungus had moved across the grafts. When stain columns extended above stump height, trees were felled and dissected to determine the height of the stain columns.

• Results. Results are presented in Table 5 and Figure 13. Eight of the ten inoculated trees of each pair were infected (determined by crown symptoms, ER measurements, characteristic stain, and re-isolation, or both (Table 5)). In three of these cases, *C. virescens* moved across the root grafts to the uninoculated member of the pair (Table 5, Fig. 13) (determined by crown symptoms, ER measurements, stain patterns, and re-isolation and growth of the marked isolate on malt agar amended with 2 ppm benomyl). Grafts between three of the remaining infected and uninoculated pairs were found to be nonfunctional.

Table 4.--Comparison of the pattern and extent of colonization by *C. virescens* of stem tissues, wounded radially or tangentially

Tree No.	D.b.h. (cm)	Date harvested	Height of stain above: Radial wound (cm)	Height of stain above: Tangential wound (cm)	Stain[a]
1	11.2	6/89	65	85	+ + + (both somewhat limited)
2	9.9	6/89	55	55	+ + + +
3	10.2	6/89	35	35	+ + (both quite limited)
4	10.3	6/89	35	45	+ + (both quite limited)
5	12.8	6/89	35	85	+ + + + +
Average (1 year)			45	61	
6	15.5	7/90	95	105	+ + + + +
7	17.2	7/90	?	?	+ + + +
8	14.5	7/90	70	90	+ + + +
9	10.5	7/90	50	85	+ + +
10	11.6	7/90	25	25	+ (both strongly limited)
Average (2 years)			60	76	

[a]The relative amount of cross-sectional areas discolored: tangential wound site vs. radial wound site: + indicates discolored area around tangential wound; + + + indicates approximately three times greater than around radial wound.

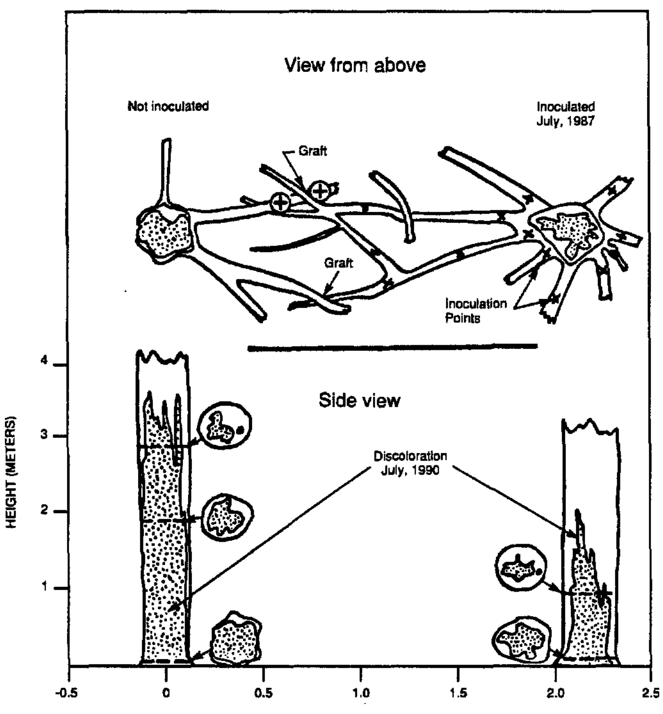

Figure 13.--A representative diagram of two sugar maple root systems showing location of potential root grafts between the systems, the points of inoculation on one of the tree's roots, and the pattern of discoloration caused by *C. virescens* following its introduction.

Dissections revealed that disease development within the inoculated trees varied greatly (Table 5). Thus, infection did not occur at all in two trees; colonization was strongly restricted and extended only a few cm in others; colonization in others was extensive with stain columns extending several m in length. In two of the three tree pairs in which graft transmission occurred, infections within the inoculated trees were less extensive than within the trees to which they spread (Fig. 13). The variable response by individual trees to inoculations made on the same day in the same way, and with the same inoculum, supports results of others (Shigo et al. 1977) that the ability of trees to compartmentalize injuries and infection by fungi is under strong genetic regulation.

The results of this experiment showed that root grafts between trees growing in close proximity can serve as conduits for the sapstreak pathogen. However, the relatively low occurrence of transmission in this study, together with the low incidence of diseased pairs and of nonwounded diseased trees in nature, suggest that infection through root grafts is much less important than direct infection through wounds above ground.

TRIAL VI (Tapholes vs. buttress wounds as infection courts for *C. virescens*). The relatively high incidence of sapstreak in some sugarbushes prompted a study to determine if tapholes serve as infection courts and if so, how important are they compared to other wounds inflicted during sap hauling or other activities. A preliminary report has been published (Houston 1992).

• Materials and Methods. In 1984, 34 groups of 10 intermediate-codominant sugar maples, 6-8 in (15-20 cm) d.b.h. growing in a 15-acre stand near Barnes Corners, New York, were wounded and inoculated as follows:

Treatment 1 (One open taphole and one taphole with a metal spout): At one of two dates (14 March, 17 April), two 7/16-in (0.43-cm)-diameter tapholes, 2.0 in (5.0 cm) deep, were made on opposite sides of the trees. One taphole (chosen at

Table 5.--Infection success, rates (extents) of colonization, and movement across root grafts in trees inoculated with *C. virescens*

Pair	Tree	Year inoc.	ER (K-Ohms) 1988	1989	1990	Infection (+,-)	Cross-over (+,-)	Height of stain (cm)	Remarks
1	Inoc.	1987	140-175	dead		+		905	Crown symptoms -
	N-Inoc.		115-700	15-50		+	+	600	1988, 1989, 1990
2	I	1987	200-400	27-190		+		27	Crown symptoms - 1988
	NI		120-130	40-100		+	+	79	No symptoms 1989, 1990
3	I	1987	160-350	60-190		-			No infection
	NI		214-440	90-140		-	-		
4	I	1987	17-80	16-30		+		208	No symptoms - 1988
	NI		290-500	40-50		+	+	390	Symptoms on I, 1989, and on both in 1990
5	I	1987	2-100	20-30		+		43	Infection did not move
	NI		420-600	70-200		-	-		into graft root
6	I	1988		10-70	33-43	+		384	Nonfunctional graft
	NI			990-170	99-236	-	-		
7	I	1988		6-18		+		25	Infection reached graft
	NI			150-250		-	-		but no cross-over
8	I	1988		6-108	10-50	+		-10	Infection well contained
	NI			130-140	200-250	-	-		
9	I	1988		83-250	10-29	+		22	Infection well contained
	NI			176-235	200-250	-	-		
10	I	1988		186-300	200-250	-			No infection
	NI			300-350	200-250	-	-		

random) was left open; a standard metal spout was inserted in the other. Both tapholes subsequently were inoculated by atomizing into them 2 ml of a conidial suspension of *C. virescens* containing 150,000 to 600,000 spores per ml Controls consisted of trees with non-inoculated tapholes. Groups of 10 trees each were inoculated once, either when tapped or at weekly intervals thereafter, until 31 May (Table 6).

Treatment 2 (Closed tapholes--plastic spouts with tubing): Tapholes were made in six groups of 10 trees as described above and plastic spouts fitted with 12-in (30-cm) lengths of plastic tubing were installed in both tapholes. The tubing was clipped back onto the spout to form a loop. Sap-filled loops served to maintain a closed system characteristic of tubing systems. Tapholes were inoculated as above immediately after spouts were removed on 2 or 31 May (Table 7). Controls were tapped but not inoculated.

Treatment 3 (Buttress root injuries): On different groups of 10 trees each, two buttress roots, one each on opposite sides of the tree, were injured at approximately weekly intervals (Table 8) by bruising them with a heavy mallet. On one bruise the bark was removed; on the other the bark was broken and loosened but not removed. Approximately 2 ml of the previously described spore suspension were atomized onto the wounds immediately after they were made. Control trees were injured but not inoculated.

Table 6.--Schedule of wounding and inoculation, and resulting infection for trees inoculated with 2 ml conidial suspension into one taphole with metal spout and one open taphole

Treatment 1			
Date tapped (1984)	Date inoculated (1984)	Number trees harvested (1989-90)	Number tapholes infected
14 March	14 March	5	0
	19 March	5	0
	26 March	5	0
	9 April	5	0
	2 May	5	0
	31 May	5	0
	Controls	5	0
17 April	17 April	5	0
	23 April	5	1
	2 May	5	0
	31 May	5	0
	Controls	5	0

Table 7.--Schedule of wounding and inoculation and resulting infection for trees inoculated with 2 ml conidial suspension in each of two tapholes with plastic spouts and tubing

Date tapped (1984)	Date inoculated (1984)	Number trees harvested (1989-90)	Number tapholes infected
	Treatment 2		
14 March	2 May	5	0
	31 May	5	0
	Controls	5	0
17 April	2 May	5	0
	31 May	5	0
	Controls	5	0

Table 8.--Schedule of wounding and inoculation and resulting infection for trees inoculated with 2 ml conidial suspension into two buttress-root bruises

Date wounded and inoculated (1984)	Number t rees harvested (1989-90)	Number trees infected
	Treatment 3	
14 March	5	0
19 March	5	0
26 March	6	0
9 April	5	0
2 May	5	3[a]
31 May	5	4[a]
8 June	5	0
15 June	6	1[a]
Controls		
14 March	6	0
31 May	5	0

[a]Five of eight infected trees were infected through both wounds.

Treatment 4 (Root injuries): One group of 10 trees was treated on 31 May by atomizing a spore suspension onto ax cuts made on an excavated root approximately 2 ft (0.6 m) from the tree. The root was then covered with forest floor litter. Control trees similarly were injured but not inoculated.

Treatments 5 and 6 (to check on the pathogenicity of the inoculum isolate used (Table 9)): In treatment 5 groups of 10 trees each were inoculated on 14 March, 17 April, or 31 May by forcing wheat-grain inoculum into two 0.25-in-diameter holes drilled 2 in deep into buttress roots on opposite sides of the tree. Holes were covered with masking tape. In treatment 6, one group of 10 trees was inoculated on 31 May by filling the drill holes with the conidial suspension used to inoculate tapholes and buttress bruises.

Annually, from 1985 through 1989, each tree was observed for presence of foliar symptoms or other evidence of infection (poor wound closure, exudation from wounds, canker development near wounds).

In June 1989 or July 1990, 5 of the 10 trees within each treatment group were randomly selected and examined for the presence of sapstreak. Trees in treatments 1 and 2 were felled and cross sectioned through the tapholes and at 20.0-in (0.5-m) intervals above and below the tapholes to observe radial patterns of discoloration. Sections were split longitudinally to trace the linear extent of the discolored column associated with each taphole. Column lengths were measured to the nearest cm. Photographs were taken of the discoloration present in all dissected trees. Inoculated roots were excavated and dissected to determine presence and extent of infection.

Trees in treatments 3, 5, and 6 were felled, cut through the buttress-root bruises or drill holes to expose the cross-sectional infection patterns, and dissected to reveal the upward linear extent of infection.

• Results. No trees developed foliar symptoms characteristic of sapstreak during the 5-year observation period. Closure of both inoculated and non-inoculated tapholes proceeded at an apparently normal rate, although it was more rapid for tapholes without spouts. Some of the basal injuries exhibited cambial dieback, and on some trees sprouts developed near the inoculated injuries.

Incidence: Very few tapholes (3 of 146 examined) showed evidence of infection by *C. virescens* (Tables 6, 7). None of the trees in treatment 1 tapped on 14 March and inoculated then or later through open tapholes or metal spouts developed sapstreak. Controls also were not infected. Two trees in the series tapped on 17 April had stain patterns that, while limited, were suggestive of sapstreak (Figure 14). One of these had been inoculated on 23 April, the other was the open taphole of a non-inoculated control tree. Similarly, in treatment 2 none of the trees tapped on 14 March and inoculated later when the spouts and tubing were removed, nor their non-inoculated controls, developed sapstreak. However, one of the trees tapped on 17 April and inoculated on 31 May had discoloration about one taphole suggestive of sapstreak (Fig. 15). The tangential orientation of this taphole may account in part for the stain patterns (Trial IV).

Incidence of sapstreak infection in trees inoculated through buttress-root bruises (treatment 3) was considerably higher than through tapholes (Table 8). And infection was influenced strongly by treatment date. While no infection occurred in trees wounded and inoculated on 14, 19, 26 March, or 9 April, three of five, four of five, and one of five trees wounded and inoculated on 5 and 31 May and 15 June, respectively, were infected. Of these eight infected trees, five were infected through both bruise wounds. None of the wounded, non-inoculated trees was diseased. Infection ranged from

Figure 14.--"Breakout" pattern of stain around taphole wound "w", suggestive of sapstreak (cross section cut through the taphole); and the associated column of discoloration (radial section split through the discolored column).

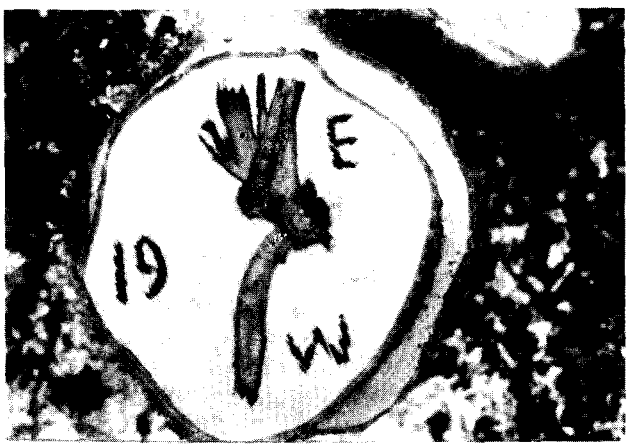

Figure 15.--A rare instance in which sapstreak disease occurred when the fungus was placed in the taphole (arrow); the fungus was contained by the tree and discolored columns were limited. The apparent ability of the fungus to spread from taphole "E" probably is attributable to the tangential orientation of the taphole, which cut across normally effective ray boundaries.

being strongly limited in some trees (Fig. 16) to extensive in others (Fig. 17). However, even in trees where extensive infection in roots had occurred, upward spread into stems was restricted to a few centimeters.

Two of the five trees inoculated through ax cuts into the roots (treatment 4) developed stain columns characteristic of sapstreak disease. Both were strongly limited and had spread only a few cm from the inoculated wound. None of the controls had stains suggestive of the disease.

Rates of infection in the trees inoculated through drill holes in their buttress roots (treatments 5 and 6) were high (Table 9), especially in trees inoculated in late May. Of the examined trees wounded and inoculated with wheat grain inoculum on 14 March, 17 April, or 31 May, one of five, two of five, and four of five, respectively, were infected. Infection occurred through both wounds on six of these seven trees. Of the five trees examined whose drill hole wounds had been inoculated with conidial suspensions on 31 May, four were infected, three through both wounds. As in trees infected through bruised bark, the extent of colonization ranged from slight to severe.

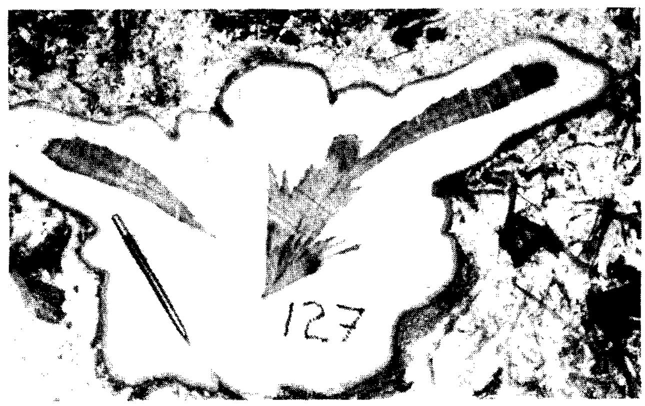

Figure 16.--In this tree, both developing columns of discoloration are limited strongly by compartmentalization.

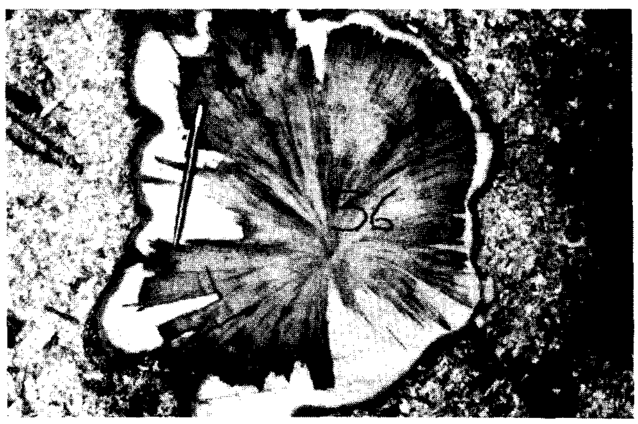

Figure 17.--The discoloration pattern in this tree indicates that *C. virescens* rapidly invaded the sapwood following infection through bark bruises.

Table 9.--Schedule of wounding and inoculation and resulting infection for trees inoculated with wheat grain inoculum (treatment 5) or conidial suspension (treatment 6) in two, 0.25-in-diameter holes drilled 2 in deep into buttress roots

Date wounded and inoculated (1984)	Number trees harvested (1989-90)	Number trees infected
Treatment 5		
14 March	5	1
17 April	5	2
31 May	5	4
Treatment 6		
31 May	5	4

The lengths of the discoloration columns (not related to sapstreak infection) associated with tapholes (treatments 1 and 2) were analyzed statistically to determine if there were differences between inoculated and non-inoculated tapholes attributable to wound date, treatment, or inoculation date. Results are presented in Table 10. Columns of discoloration associated with inoculated tapholes were no longer than those of the controls. However, significant differences did occur that were related to treatment and to the date of tapping:

- The mean length of discoloration columns about open or metal-spout tapholes (n=30) (treatment 1) was significantly longer (P=0.009) than that about closed, plastic-spout tapholes (treatment 2).

- Within treatment 1, the mean length of the discoloration columns about open tapholes (n=59) was significantly greater (P=0.013) than that about tapholes with a metal spout.

- Columns of discoloration about tapholes made on 14 March were longer than about those made on 4 April. This was significant for treatment 1 tapholes (P=0.02, n=30) but not for treatment 2 tapholes (n=61).

Thus, the mean discoloration lengths reflect gradients of taphole "openness" and time. The more open the taphole, and the longer it existed before resumption of physiological activity, the longer the columns were.

Discussion and Conclusions

Several significant relationships were revealed or suggested by the series of observations and experiments reported here. While a number of these confirm earlier reports, others

Table 10.--Comparison of lengths of discoloration in treatments 1 and 2[a] (A), the influence of tapping date (B,C,D) and of open vs. metal spout (E)

	Tapholes (no.)	Average column length (cm)	SD	P
A. All treatment 1	30	67.23	17.86	
vs.				0.009 **
All treatment 2	40	50.62	12.83	
B. All treatsment 1+2:				
14 March	60	62.30	18.24	0.098
17 April	64	55.48	13.03	
C. Treatment 1 only:				
14 March	30	72.17	18.58	0.020 *
17 April	30	57.73	13.01	
D. Treatment 2 only:				
14 March	32	50.81	12.98	0.83
17 April	32	53.37	13.11	
E. Treatment 1 only:				
Open	59	34.02	10.99	0.013 *
Metal	61	29.30	9.21	

[a]Treatment 1: Metal spouts and open tapholes;
Treatment 2: plastic spouts with tubing.
** indicates statistical significance at > 99 percent probability level.
* indicates statistical significance at > 95 percent probability level.

extend our understanding of disease expression and of factors affecting disease incidence and severity.

Importance of Wounds

Sapstreak disease rarely occurs in nonwounded trees. Only a few cases were encountered while observing disease occurrence in many sugarbushes and forests, and when such trees were excavated they were found to be connected by functional root grafts to diseased trees that had been wounded. Our studies (trial V) showed that root-graft transmission between closely adjacent trees can occur. But the low level of its occurrence in our studies, and the relatively few times adjacent trees were observed to become diseased in the field, suggest that this means of disease spread probably is not very common.

The nearly universal association of wounds and sapstreak disease is pivotal to the development of control guidelines. The understanding gained in these studies, that the suitability of wounds as infection courts depends on their type, their location on the tree, and possibly the season in which they are inflicted, provides added information for development of management guidelines.

The general levels of "natural" disease incidence, especially of trees with initial symptoms, observed in these studies was very low. Repeated observations revealed that for some sugarbushes, the disease is occurring continuously--with a few new trees becoming symptomatic every year or so. In contrast, in forest stands the majority of "new" trees often appears within a relatively short time span. This pattern of occurrence probably reflects the infrequent but severe wound-inflicting disturbances related to logging activities, and contrasts the less severe, but annual traffic-related disturbances in some sugarbushes. In forest stands, sapstreak diseased trees usually exhibit initial symptoms 3-6 years after the injury-causing event. While the period over which diseased trees die is frequently more protracted in forest stands, the trees that are going to die will have done so within 6-8 years after having become infected.

Which infected trees die and which do not is probably related to 1) the capacity of the tree to compartmentalize injuries and limit the spread of infection, 2) the condition of the tree and its position in the stand, and 3) the presence or absence of pathogens capable of attacking and killing weakened root systems. Our studies and observations suggest that each of these factors is involved. Inoculation Trials IV, V, and VI clearly showed that trees of approximately the same age, size, and vigor, inoculated with equal amounts of the same isolate at the same time and by the same method, varied greatly in their ability to resist or limit the spread of the pathogen. Indeed, some inoculated trees remained free of infections altogether. Patterns of colonization, revealed through dissection, showed that trees usually recovered if extensive spread and distribution of the fungus, particularly through the root system, had not occurred within the first year or so after inoculation.

Importance of Root Disease Pathogens

Most naturally and artificially inoculated trees and saplings with root systems well colonized by *C. virescens* died within 1 to 3 years after symptoms appeared. The nearly universal presence of *Armillaria* sp. and *Xylaria* sp. (or both) fruiting bodies, mycelial fans, rhizomorphs or decay on trees that died, and the absence of these organisms from sapstreak-infected trees that recovered suggests that their attack is a major mortality-determining factor. These root fungi are ubiquitous inhabitants of long-established northern hardwood forest stands. Successful invasion (at least for some *Armillaria* sp.) is governed by stress factors or events that, by altering tree physiology, reduce resistance (Wargo and Shaw 1985). The debilitating effect caused by extensive internal colonization of roots by *C. virescens* undoubtedly is a significant stress. Other stresses, including defoliation and drought, also weaken and predispose roots to infection. These factors, together with forest practices such as thinning, ensure a relatively continuous supply of pathogen food bases.

The fact that significant mortality occurred soon after inoculation with *C. virescens* of saplings and pole-size trees that had been severely defoliated earlier by gypsy moths probably reflects the influence of defoliation, both as a predisposing factor to these root pathogens and as a factor contributing to the buildup of their inoculum. That defoliation per se does not affect susceptibility to *C. virescens* was shown by Trial III. The saplings in this stand had not been defoliated earlier and, by consequences of its more recent origin, this stand did not harbor a significant load of root disease inoculum.

Conversely, some trees with severe sapstreak root infections also recovered. These were saplings and trees whose position in the stand was favorable for growth and that also may have escaped attack by root pathogens.

The opportunity to grow rapidly--to produce the energy required to establish barriers that restrict infection--is dependent in large part on the amount of sunlight received. Saplings in the more open stands, for example, Trial III and the sapling-sprouts of Trial I, were able to limit infection and subsequent mortality compared to those saplings in Trials I and II growing nearby in denser shade. The sprouts also probably benefited from having an energy-producing, non-inoculated sprout member. And, as discussed earlier, in addition to a better light regime, the saplings in Trial III and the sprouts in Trial I also probably benefited from a somewhat lower root-pathogen inoculum potential characteristic of young forest stands established on recently abandoned open land.

Influence of Sap Collection Methods

Very low incidences of sapstreak were observed in sugarbushes employing tubing collection systems compared to bushes where sap is collected in buckets. Whether the abundant buttress root and root injuries found in many "bucket" sugarbushes were the result of saphauling or of traffic at other times of the year is not certain. When the disease did occur in tubing system sugarbushes, it was on injured trees near sugarhouses, along logging skid trails, and in areas where cattle had trampled root systems.

Our studies examining the suitability of tapholes as infection courts (Trial VI) confirmed these observations. Tapholes, regardless of their nature, were poor infection courts compared to buttress-root bruises. Interestingly, however, the lengths of the discoloration columns (associated with all tapholes) were greater about open (metal) than about closed (plastic with tubing) spouts--and tapholes made early in the season had longer discoloration columns than those made later. The greater amount of discoloration associated with metal spouts used in bucket collection systems is perhaps another factor in favor of tubing systems.

Influence of Infection Courts Location

Injuries associated with sapstreak were almost always close to the ground. Even though stem tissues were infected following their inoculation with large amounts of a grain-based inoculum (Trial IV), and invasion of upper stems from infections originating in the roots or stem bases is common (Trial I), no cases were observed that suggested infection had occurred through broken branches or other wounds of upper crowns or stems. Tapholes inoculated with conidial suspensions very rarely became infected and when they did, the infections were strongly limited (Trial VI). In a number of cases, stump surfaces created when one member of a sprout clump was removed in early summer thinning operations appeared to be the avenue of infection for the residual stem. Such near-the-ground wounds comprise especially suitable infection courts for *C. virescens*.

Influence of Season

Late spring to midsummer appears to be a critical period for sapstreak disease. The results of several studies indicated that sugar maples are most susceptible to infection during this time (Trials I, II, and VI). Mortality was highest in trees inoculated in late spring through mid-August. Late spring and early summer is the period of greatest susceptibility of other tree species to similar vascular pathogens, including elms (Dutch elm disease) and oaks (oak wilt). Not only are trees inherently more susceptible at this time, this also is the period when heavy equipment used in the forest can create excessive rutting and cause damage to roots and massive injuries to bark and wood of lower stems. And, finally, temperatures and moisture conditions during this period usually are very favorable for rapid growth and sporulation of *C. virescens* and for growth of the associated root pathogens as well.

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Sapstreak disease is a potentially serious problem of sugarbushes and forest stands. It is caused by the fungus *Ceratocystis virescens,* which invades sapwood of roots and bases of stems through wounds created during logging, saphauling, or other activities. This publication describes the results of observations and experiments to learn more about the patterns of disease development and the factors that affect them, within individual trees and within representative forests and sugarbushes.

Keywords: *Ceratocystis virescens,* root and buttress root wounds, vascular disease, sugarbush, root grafts

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