SEED SOURCE AND FAMILY VARIATION IN THE INCIDENCE OF FUNGAL CANKERING IN A SUGAR MAPLE PROGENY TEST PLANTATION

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Abstract.--Sugar maples (Acer saccharum Marsh.) growing on a poor site and under stress are believed to be predisposed to infection by disease-causing organisms, including some fungi that cause cankers. In a sugar maple progeny test of 32 half-sib families from four seed sources, on a poor site with shallow soils at Underhill, Vermont, 28 percent of 594 trees have been infected by canker-causing organisms, predominately Nectria. None of the trees from 25 of the same families growing in another plantation on a good site at Williamstown, Massachusetts, have been infected. Maples that are genetically ill-adapted to stresses imposed by poor growing conditions may be more susceptible to infection. Most of the families from parent trees in northwestern Massachusetts are heavily infected, averaging 40 percent of the trees per family, as is a single family from southeast New Hampshire. Only 11 percent of the trees within families originating from the site of the test plantation are infected. Families from a second seed source in northern Vermont are intermediate. This preliminary evidence, showing that some sugar maples may be genetically predisposed to invasion by fungal pathogens, suggests that there may be a need to delineate specific seed collection areas for sugar maples that are to be planted on less than optimum sites for sugar bush replacement, replenishment, or timber production.

Sugar maple (Acer saccharum Marsh.) decline, often attributed to drought and other environmental stresses, is receiving considerable attention in Eastern North America. Cankering of stems and branches is not specifically involved, but may be an added burden for trees that already have a physiological disfunction. Hepting (1971) lists 13 fungi that cause cankering of sugar maple. Some of these fungi can cause infection by direct penetration of host tissues, but most require some form of biotic or abiotic predisposition. Sugar maples growing on poor sites are believed to be predisposed to infection by certain disease-causing organisms, including some fungi that cause cankers. The causal organism of annual canker (Fusarium solani) is more common on trees growing on shallow soils with an A horizon high in clay content (Wood and Shelly 1969). The target canker caused by Nectria galligena that usually begins at branch stubs is common on trees on poor sites, especially dry mountain-tops (Shigo and Larson 1969).

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However, high levels of genetic resistance or tolerance to infection by Nectria galligena also have been found in varietal populations of apple under field conditions and after artificial inoculations (Zagaja et al. 1971), indicating that not all individual trees may be equally susceptible to the stress/pathogen infection sequence. I report on a high incidence of stem cankering on sugar maples, predominately Nectria, growing in a half-sib progeny test plantation on a poor site with shallow soils and numerous rock outcrops in northern Vermont, and the absence of cankering on trees from the same families in a plantation on a good site in northwestern Massachusetts. I also present evidence of genetic variation in incidence of cankering, which suggests that there may be an inherent physiological or anatomical response to wounding and infection that does not occur in some sugar maples when they are growing under conditions of environmental stress, predisposing them to invasion and infection by pathogens when they are planted on less than optimum sites.

METHODS

The sugar maple progeny test plantation with a high incidence of cankering is located on the University of Vermont Proctor Maple Research Farm at Underhill. The plantation was established in 1960 and has 32 open-pollinated families from parent trees selected for sap with a high sugar content at four locations, two in northern Vermont (Jericho and Underhill), one in northwestern Massachusetts (Williamstown), and one in southeastern New Hampshire (Durham). Each family is represented by two noncontiguous two-tree plots randomly located in each of five blocks with a spacing of 10 feet between trees. A second plantation with 25 of the same families was planted in 1960 on a good site, with normally abundant soil moisture, on the Hopkins Memorial Forest, Williamstown, Massachusetts. Each tree (594) in the plantation in Vermont was examined for the presence of cankers in the fall of 1984 and placed in one of two categories, cankered and noncankered. At that time no attempt was made to differentiate between degrees of severity of cankering or size or numbers of cankers. Stems of 18 standing dead trees with cankers were dissected, and the oldest cankers were determined to have originated from infection in 1976. All trees in the Massachusetts plantation were canker free. Diameters of the trees in both plantations had been measured in most years from 1966 through 1978. Heights had been measured in most years from 1966 through 1974. Heights also were measured in the spring of 1984 in the Williamstown plantation and in the fall of 1985 in the Underhill plantation.

The observed number of cankered trees within families, the four seed sources, blocks, and plantation rows and columns were tested separately for significant deviations from expected numbers by contingency tables and chi-square tests. To determine whether relative vigor was a factor in the incidence of cankering, I made two analyses. First, I tested the significance of differences of preinfection (1974) heights and diameters among seed sources using analyses of variance. Second, I tested the significance of differences in preinfection heights and diameters of all cankered and noncankered trees and the same two groups within each seed source using a series of t-tests. The effects of cankering on subsequent growth rates were examined by comparing 1985 heights and 1974 to 1985 height-growth increment of cankered and noncankered trees.

RESULTS AND DISCUSSION

Incidence of stem cankering in the sugar maple progeny test at Underhill was high. Twenty-eight percent of the trees in the plantation (168 of 594 trees) had at least one canker in 1984. Most of the cankers were the result of infection by Nectria spp., though Eutypella spp. cankers were common. Cankers caused by at least five other unidentified fungi were found in low frequency. No cankering occurred on sugar maples from 25 of the same 32 half-sib families growing in a plantation at Williamstown. Differences between the two plantations in occurrence of cankering is most likely related to site factors and perhaps severity of climate. Heavy buildup of Nectria canker most often occurs on sites with exposed slopes and shallow, infertile soils, like the plantation site at Underhill (Brandt 1964). High susceptibility to infection by facultative parasites also is associated with low host vigor (Hare 1966). Site quality is much higher in the Williamstown plantation than it is at Underhill, and growth rates are faster. In 1974, after 14 growing seasons and 2 years prior to the earliest fungal infections detected, average height of maples from 25 common families was 5.8 meters at Underhill and 7.9 meters at Williamstown. After 25 growing seasons, average height was 9.7 meters at Underhill. Average height at Williamstown was 12.8 meters after 23 growing seasons.

Most forest plantation diseases are caused by facultative parasites attacking off-site trees (Heimburger 1962). Variation in incidence of cankering among seed sources at Underhill supports this observation (Table 1). Sugar maples that are genetically ill adapted to stresses imposed by poor growing conditions may be more susceptible to infection. Most of the families from parent trees in northwestern Massachusetts (Williamstown) were heavily infected, averaging 40 percent of the trees within families. The single-family source from southeast New Hampshire (Durham) also was heavily infected. Only 11 percent of the trees within families originating from the site of the test plantation in northern Vermont (Underhill) were infected. Families from a second seed source in northern Vermont (Jericho) were intermediate.

There were no significant differences in indidence of cankering among families within seed sources (Table 1), plantation columns, or rows; but there were significant differences among plantation blocks (chi-square = 16.4 with 4df; P = <.0.01). The lowest incidence of cankering (16 percent) occurred in the block at the uppermost end of the gradually sloping site and the highest incidence (37 percent) occurred in the lowermost block. Incidence of cankering in the other three blocks was 26, 34, and 30 percent in a downslope direction.

Although differences in vigor, as measured by growth rate, that reflect site factors appear to be related to differences in infection frequency on sugar maples at the two plantation sites, vigor did not account for differences in cankering among seed sources or families at Underhill. There were no significant differences in preinfection (1974) heights or diameter among heavily and lightly cankered seed sources (Table 2).

Cankered trees in the plantation were, in fact, significantly taller than noncankered trees in 1974 and also had significantly larger diameters. Cankering did affect height-growth rate following infection. Between 1974 and 1985, average total growth of cankered trees was 3.7 meters and that of noncankered trees was 4.0 meters, a small but significant difference (t-value = 2.88; $P<0.01$). By 1985, heights of cankered and noncankered trees were near equal, 9.8 and 9.7 meters, respectively, in contrast to 1974 when cankered trees were significantly taller (Table 2). Postinfection diameters could not be measured accurately because of the swelling and distortion of the boles of cankered trees. In addition to growth reduction, cankering was the probable cause of death of several trees. Eighteen cankered trees died between 1976 and 1984, and another 10 died between 1984 and 1986. No noncankered trees died within either of these periods.

Wounds of various types are associated with infection by canker producing fungi. The fungus that causes Eutypella canker cannot penetrate the outer bark layers of sugar maple and, therefore, usually enters through a scar, broken branch stub, or any wound exposing the xylem. Cankers caused by Valsa leucostromoides are prominent on bole areas surrounding tap holes of sugar maples in Vermont (Sproston and Scott 1954). Hepting et al. (1949) found Nectria canker to be closely associated with increment borer wounds in North Carolina. Some of the wounds that I observed to be associated with cankers in the progeny test at Underhill were: branch stubs, split bark at forks, sun scald, frost cracks, insect damage (sugar maple borer), basal mouse gnawing, and 7/64-inch holes drilled for mini-tapping and sugar testing. Each tree in the plantation had been tapped once in each year between 1966 and 1976, and, therefore, had at least one potential site of infection entry in common. The noninfected trees in the plantation at Williamstown also were tapped in each of the same years, so that absence of wounding does not appear to be a major factor in differential infection at the two sites or among seed sources and families at Underhill.

Phellogen is a single layer of meristematic cells in bark that produces all other bark constituents except phloem. When the phellogen is disrupted by wounding it must be restored before new suberized layers (phellem) can be produced that prevent desiccation and infection (Hudler 1984). The time period required for restoration of phellogen and the occurrence of nonspecific plant defense reactions can be critical. Biggs (1986) demonstrated that production of lignified and lignosuberized tissues of peach bark following wounding and inoculation with Cytospora leucostoma significantly decreased the rate of fungal colonization, and that production of at least three cell thicknesses of new phellem resulted in complete inhibition within a critical period of 10 to 14 days postwounding. One major consequence of water stress, and possibly other inducers of stress such as infertile soil or chronic exposure to air pollutants, in trees is to slow phellogen restoration following wounding and allow pathogens to continue to advance unimpeded into healthy tissue (Hudler 1984; Puritch and Mullick 1975). Trees suffering from water stress, as the sugar maples in the Underhill progeny test undoubtedly do, are more susceptible to canker diseases, especially those caused by facultative parasites (Bier 1961; Bloomberg 1962a, 1962b; Parker 1961).

Moderate to strong genetic control of wound closure and compartmentalization of discolored wood associated with tree wounds, including wounds on sugar maples, have been demonstrated (Gallagher and Syndor 1983; Garrett et al. 1976, 1979, 1984; Shigo et al. 1977). Seed source and family differences in susceptibility of sugar maples to infection by canker-causing organisms reported here may involve a genetically-controlled anatomical, physiological, or biochemical response that affects the rate of the phellogen restoration process following wounding. The response may be of importance only when sugar maples are growing on a poor site and under water stress, and when normal phellogen restoration is slowed. The phellogen restoration process includes several phases or simultaneous events. Fungitoxic and other chemicals accumulate near the wound (Hudler 1984; Moore 1978), flow of nutrients to invaders is cut off by production of nonsuberized impervious tissue (Puritch and Mullick 1975), and new phellogen produces protective phellem, a tissue composed of short-lived cells whose walls are impregnated with suberin as the cells mature and die (Hudler 1984). I plan to examine resistant and susceptible histological and histochemical reactions to wounding and inoculation of clonal propagules of sugar maples subjected to artificial stress to identify the specific mechanisms that may be under genetic control.

In the meantime, this preliminary evidence, showing that some sugar maples may be genetically predisposed to invasion by fungal pathogens, suggests that there may be a need to delineate specific seed-collection areas for sugar maples that are to be planted on less than optimum sites for sugar bush replacement, replenishment, or timber production.

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Table 1.--Seed source and family variation in the incidence of cankering on sugar maples in a half-sib progeny test at Underhill, Vermont

Seed source	Family	Number of trees	Number cankered	Percent cankered	Seed source	Family	Number of trees	Number cankered	Percent cankered
Jericho, VT	226	17	2	12	Williamstown, MA	545	20	1	5
	227	19	6	32		572	19	4	21
	232	20	1	5		573	20	10	50
	234	19	6	32		574	18	10	56
	238	20	3	15		575	16	6	38
	263	17	2	12		576	19	9	47
	268	18	5	28		577	20	8	40
	269	21	6	29		583	20	8	40
	303	19	2	10		585	20	8	40
						619	19	11	58
	Total	170	33	19		624	18	6	33
						632	17	5	29
Underhill, VT	397	18	2	11		640	19	8	42
	457	20	3	15		645	19	9	47
	463	20	1	5		664	17	8	47
	491	20	3	15					
	499	10	0	0		Total	281	111	40
	500	18	2	11					
	526	17	3	18	Durham, NH	005	20	10	50
	Total	123	14	11		Total	20	10	50

Chi-square for seed source differences = 46.0 with 3 df (P = <.001).
Chi-square for family differences = 34.7 with 28 df (Not significant at $P \leq .05$).

Table 2.--Preinfection diameters and heights (1974) of cankered, noncankered, and all sugar maples within seed sources in a half-sib progeny test at Underhill, Vermont

Seed source	Diameter (1974) cm				Height (1974) m			
	All trees	Noncankered	Cankered	t-value	All trees	Noncankered	Cankered	t-value
Jericho, VT	8.2	7.9	9.4	2.89**a/	5.8	5.6	6.2	2.27*a/
Underhill, VT	8.2	8.2	8.8	0.91	5.9	5.8	6.1	0.81
Williamstown, MA	8.2	7.8	9.0	3.62***	5.8	5.3	6.1	4.03***
Durham, NH	8.8	7.8	9.8	1.85	5.7	5.3	6.1	1.62
Total	8.3b/	7.9	9.1	4.88***	5.8b/	5.5	6.1	4.38***

a/ Indicates significance of t-value for differences between cankered and non-cankered trees: * = $P<0.05$; ** = $P<0.01$; *** = $P<0.001$.

b/ Differences between seed sources were not significant.