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Long-Term Response of Precommercially Thinned Aspen Clones to Hypoxylon Canker

M.E. Ostry, N.A. Anderson, D.J. Rugg, and K.T. Ward

North Central
Research Station
USDA Forest Service

1992 Folwell Avenue
Saint Paul, Minnesota
55108

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www.ncrs.fs.fed.us

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Increasing demand for quaking aspen (*Populus tremuloides* Michx.) fiber has renewed interest in precommercial and commercial thinning to increase growth of individual trees and decrease rotation intervals. After aspen stands are harvested or killed by fire, 10,000-100,000 suckers ha^{-1} regenerate from root sprouts. Self-thinning through intraspecific competition reduces this to less than 1,000 stems ha^{-1} after 50 to 60 years. Precommercial thinning can shorten rotation length by accelerating early growth, while commercial thinning can increase total stand yield by capturing expected mortality from self-thinning.

Hypoxylon canker, caused by the fungus *Entoleuca mammata* (Wahlenberg: Fr.) J.D. Rogers & Y.-M. Ju, is a common disease killing aspen throughout the Lake States. Some research has suggested that stand density may influence the incidence of Hypoxylon canker (Anderson and Anderson 1968, Anderson and Martin 1981, Bruck and Manion 1980, Capony and Barnes 1974, Day and Strong 1959, Ostry and Anderson 1998, Schreiner 1925). However, other researchers have reached different conclusions (Anderson 1964, Pitt *et al*. 2001). The potential effect of variation among aspen clones on Hypoxylon canker prevalence was not examined in these previous studies.

The objectives of this study were to determine (i) the impact of Hypoxylon canker in a precommercially thinned stand of aspen and (ii) the influence of aspen clone differences on the effects of Hypoxylon canker. The hypotheses tested were (i) Hypoxylon canker prevalence is greater in thinned aspen relative to unthinned aspen, and (ii) Hypoxylon canker-induced tree mortality is greater in thinned aspen relative to unthinned aspen. This study was a continuation and reanalysis of the research described by Anderson and Anderson (1968).

MATERIALS AND METHODS

Study Design

This study was conducted in a quaking aspen stand regenerated after a clearcut in 1941 on the Pike Bay Experimental Forest in the Chippewa National Forest in Cass County, Minnesota. The study site was level with sandy loam soil and site index of 21-23 m. In 1951, four 4-ha areas were delineated within the study stand. Three treatments were applied: aspen thinned with all other hardwoods removed (THR); aspen not thinned with hardwoods removed (HR); and aspen not thinned and hardwoods left (control, C). For the THR treatment, two of the areas were thinned by hand to an average density of 1,831 trees ha^{-1} and all aspen with Hypoxylon stem cankers were removed. In the HR and C treatment areas, the aspen were not thinned and averaged 6,489 trees ha^{-1}. The C treatment area contained 220 other hardwood trees ha^{-1}, predominately black ash (*Fraxinus nigra* Marsh.), bur oak (*Quercus macrocarpa* Michx.), and American elm (*Ulmus americana* L.). Ten permanently marked .04-ha plots were systematically established in each of the four areas, with adjustments to avoid natural openings, roads, and trails (fig. 1). Additional details on the field design can be found in Anderson and Anderson (1968).

Data Collection

In 1953, and in seven other years through 1998, all trees on each plot were examined for the presence of Hypoxylon canker on the main stem. These assessments were performed during the dormant season with the aid of binoculars. At each examination, tree diameter at breast height (d.b.h.), number of Hypoxylon stem cankers, and cause of tree mortality were recorded. Since cankers on the lower half of stems are more likely to be fatal to affected trees than cankers higher on the stem (Day and Strong 1959), canker height was recorded.

About the Authors:

M.E. Ostry, Research Plant Pathologist; **N.A. Anderson,** Research Plant Pathologist; **D.J. Rugg,** Statistician; and **K.T. Ward,** Forester; are all with the North Central Research Station, St. Paul, MN.

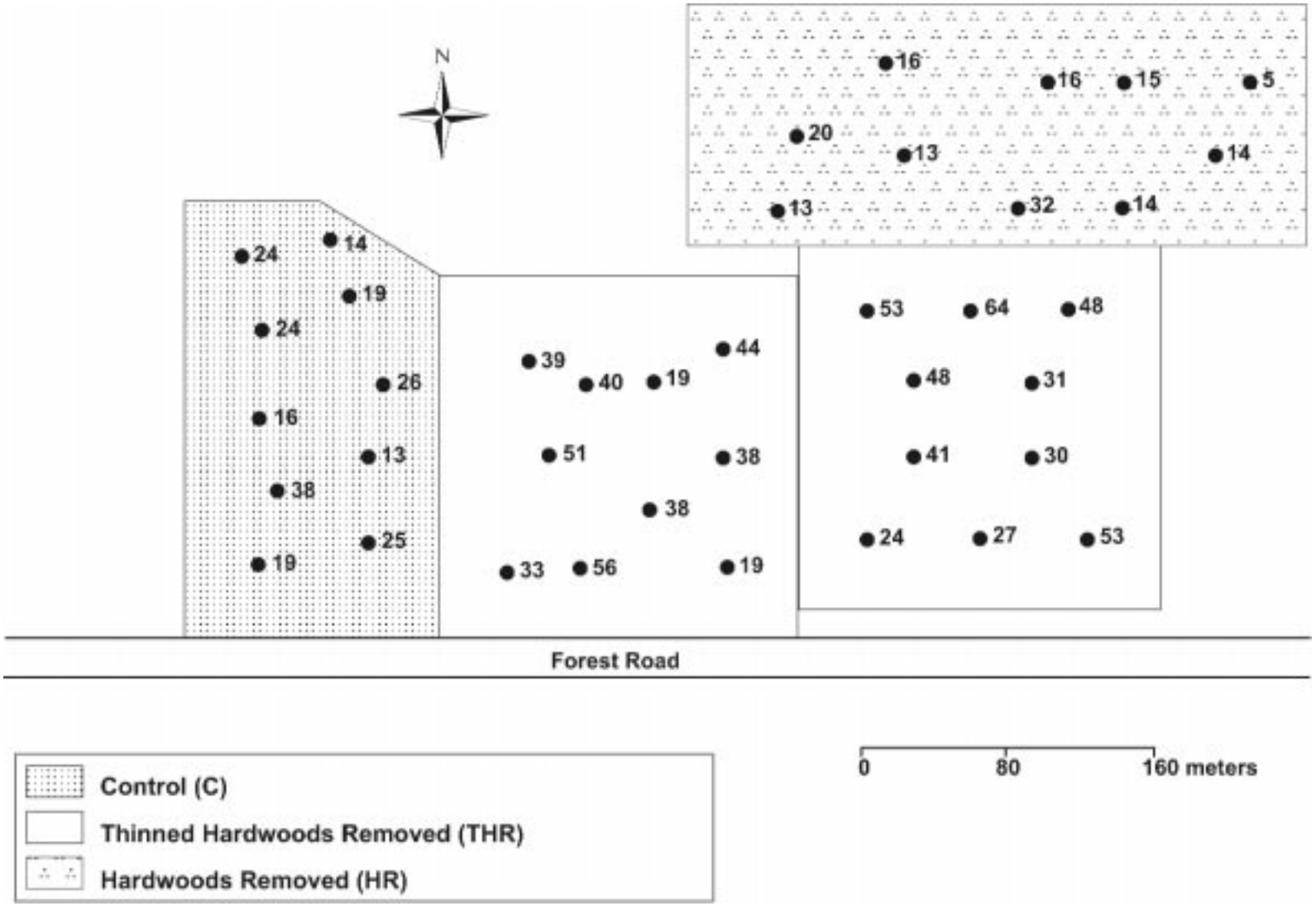

Figure 1.—*Plot (.04 ha) locations in treatment areas and percentages of trees killed by Hypoxylon canker over the 47 years on each plot. Each plot was in a single unique clone.*

Volume was calculated using Schlaegel's (1971) aspen equation developed on plots in or near the Pike Bay Experimental Forest. The equation generates cubic feet inside bark, which was converted to cubic meters.

Clone Delineation

In 1972 all live trees on each plot were mapped and numbered. Also beginning at this time, phenological characteristics such as date of leaf flush and leaf fall, fall coloration, bark color and texture, crown shape, and stem and branch form of aspen on each plot were recorded to delineate clone boundaries (Barnes 1969, Blake 1963). On three different dates in May 1989, color-infrared aerial photographs at a scale of 1:7920 were taken to help delineate clones within the study area using differences in the timing of leaf flush and crown shape. Each plot was located on the photographs and distinguishable clone boundaries were mapped.

Statistical Analyses

The suboptimal design of the study posed an interesting problem. Our analyses ignore the original breakdown of the thinned treatment into two replicates, treating them instead as a single large area. The technical validity of the analyses rests on the assumption that the permanent plots are spaced sufficiently far apart that the responses are independent. Responses of individual trees on a plot are clearly correlated due to both spatial proximity and genetics, so they are treated as subsamples.

We found that each plot was in a single unique clone, so we were concerned that apparent differences in canker prevalence across treatments might actually be due to the different clones in those treatments. Statistically, this would manifest itself as greater than expected variation across plots within a treatment. To address this potential problem, we analyzed the data using a categorical data method developed by Koehler and Wilson (1986), as implemented in the program TableSim (Rugg 2003).

For categorical data, there is a certain variance structure associated with a given response probability distribution. Under standard statistical tests, different aspen clones in the same treatment group are expected to have the same base response distribution and therefore the same variance structure. If the null hypothesis of no treatment differences is true, then clones in different treatment groups also have the same base response distribution and therefore the same variance structure. In such a case, the test statistic follows a standard chi-square distribution. If the null hypothesis is false, then the response distribution itself also has some variability (intertreatment variance). This extra variance inflates the test statistic. If there is enough extra variability, one can conclude that there are significant differences among the treatments.

If the treatment response varies by clone, the variance in the data for each treatment is a combination of standard response distribution-based variance (within clone) plus variance in the response distribution itself (between clones within a treatment). Just like intertreatment variance in the response distribution, this extra variability inflates the computed test statistic. In such a case, the test statistic may be large because of interclone differences within treatments, treatment differences, or some combination of the two. Clearly, inference about the existence of treatment differences becomes suspect.

While clones were our primary concern in designing the analysis protocol, putative clone effects are confounded with general plot effects. The remainder of this section, as well as the results section, will therefore refer to plot effects rather than clone effects specifically.

The Koehler and Wilson (1986) method estimates the between plot variance within each treatment and adjusts the test statistic so that it again follows a standard chi-square distribution when the null hypothesis of no treatment differences is true. Large values of the test statistic once more reflect either chance events or differences among the treatments. The size of the adjustment is related to the amount of extra,

interplot variance. One measure of the amount of interplot variation is the statistic C(j) used by the program TableSim (Rugg 2003). A C(j) value of 1.00 indicates no interplot variation; higher values indicate increasing interplot variation. In this report, C(j) is referred to as the "variance inflation factor" (VIF).

Two perspectives were taken with tree survival data. First, we examined the long-term fate of trees over the entire 47-year experiment. Second, we examined short-term changes from one sampling point to the next. For both perspectives, the data were analyzed using the same categorical data methods described above for canker prevalence data.

Tree diameter and height were analyzed using ANOVA for the 1987 and 1998 sample points. Treatments were compared using Fisher's LSD. The data consisted of the mean d.b.h. or height on each plot, which removed plot effects on the mean square error estimate. However, if all the "best" growing plots happened to be in a particular treatment, that treatment would still appear better even if it actually had no effect at all.

For each sample year from 1966 through 1998, tree density within the treatments was compared using pair-wise t-tests. The data were square root transformed prior to analysis to make the distributions more normal. Because of severe variance heterogeneity, in early years the treatments were compared using the standard t-test for unequal variances. The time trend over the entire study was assessed by plotting average number of trees per plot against year.

RESULTS

Canker Prevalence

Across the 47 years, the prevalence of Hypoxylon canker (ratio of living infected trees to total living trees) by treatment ranged from 2 to 13 percent (table 1). Of the eight sampling years, only one showed a difference in canker prevalence between THR and C plots (1956, table 2), with C having the lower rate. All other significant differences were between HR and either C or THR. Removal of hardwoods consistently produced the lowest canker prevalence over the first 15 years of growth after treatment, but the C and HR treatments were indistinguishable from each other by the 20th year (1971). No differences were found among the treatments from the 36th year (1987) onward.

Early in the study (1953-1971), most Hypoxylon cankers originated on the lower half of stems—70 percent for C and HR plots, 76 percent for THR plots. Later in the study (1972-1998), cankers on the lower half of stems declined to 38 percent for C and HR plots, but only to 66 percent for THR plots.

Tree Survival

Hypoxylon canker was clearly most damaging during the early years. Of the trees dying from Hypoxylon canker, over 60 percent died in the first 20 years and over 90 percent died within 30 years after the regeneration clearcut. Analysis of the 47-year tree survival rates and causes of mortality (table 3) showed differences among the treatments in survival rate ($P < 0.0001$). Cumulative over the 47 years, THR plots had a much greater rate of tree survival, but of the trees that did die in those plots, a larger proportion died from Hypoxylon canker than in the C or HR plots. Tree mortality caused by Hypoxylon canker among plots in the C, HR, and THR treatments ranged from 14 to 38 percent, 5 to 32 percent, and 19 to 64 percent, respectively, at the end of the study in 1998 (fig. 1). When we compare the C and HR treatments (table 3), the HR treatment was associated with less Hypoxylon canker mortality ($P = 0.052$), but equal to C in death from other causes ($P = 0.70$). Analysis of the seven intersample measures of tree survival showed that differences in mortality dissipated over time; no differences were detected 45 years after thinning (table 4).

Table 1.—*Prevalence of Hypoxylon stem cankers in thinned and unthinned quaking aspen trees per hectare from 1953 to 1998*

Year	Treatment[1]	Live stems	Prevalence of cankers[2] (%)	VIF[3]
1953	C	6,111	4	1.29
	HR	7,247	2	2.20
	THR	1,924	5	1.64
1956	C	4,658	9	3.93
	HR	5,948	5	3.56
	THR	1,897	13	1.50
1961	C	2,798	8	2.53
	HR	4,367	2	3.90
	THR	1,591	7	1.00
1966	C	1,571	7	2.22
	HR	2,586	4	1.69
	THR	1,326	8	1.54
1971	C	1,116	3	3.86
	HR	1,709	2	2.30
	THR	1,102	6	1.80
1987	C	568	8	1.04
	HR	763	9	1.00
	THR	573	10	1.14
1996	C	336	10	1.08
	HR	467	7	1.00
	THR	348	6	1.00
1998	C	314	6	1.59
	HR	430	7	1.00
	THR	331	6	1.00

[1] C = control; HR = aspen unthinned but hardwoods removed; THR = aspen thinned and hardwoods removed.

[2] The canker prevalence rates are weighted using the variance inflation factor and therefore cannot be used to compute the exact number of infected trees observed.

[3] VIF is the "variance inflation factor" and corresponds to C(j) in Rugg (2003); values greater than 1.00 indicate increasing interplot variation.

Table 2.—*P-values*[1] *of Hypoxylon stem canker prevalence analyses comparing thinned and unthinned aspen, adjusted and not adjusted for plot effects*

	Adjusted				Not adjusted			
Year	**Overall**	**THR-C**[2]	**THR-HR**	**C-HR**	**Overall**	**THR-C**[2]	**THR-HR**	**C-HR**
1953	0.002	0.26	0.0006	0.005	<<0.0001	0.17	<<0.0001	0.0002
1956	0.0001	0.032	< 0.0001	0.006	<<0.0001	0.0004	<<0.0001	<<0.0001
1961	0.0004	0.38	0.002	0.0003	<<0.0001	0.23	<<0.0001	<<0.0001
1966	0.004	0.54	0.0006	0.032	0.0001	0.37	<0.0001	0.0032
1971	0.032	0.23	0.018	0.65	0.0007	0.019	0.0003	0.43
1987	0.46				0.44			
1996	0.40				0.37			
1998	0.86				0.86			

[1] P-values are from the likelihood ratio tests (LR) provided by TableSim (Rugg 2003). When the overall test shows no differences, then no multiple comparisons are performed.

[2] C = control; HR = aspen unthinned but hardwoods removed; THR = aspen thinned and hardwoods removed.

Table 3.—*Fate of thinned and unthinned quaking aspen at the end of the study in 1998*

Treatment[1]	**Live**[2]	**Dead/canker**[3]	**Dead/other**[3]	**n**[4]	**VIF**[5]
	Percent	*Percent*	*Percent*		
C	5	20	75	2,392	3.98
HR	6	14	80	2,886	3.94
THR	17	32	51	1,580	3.36

[1] C = control; HR = aspen unthinned but hardwoods removed; THR = aspen thinned and hardwoods removed.

[2] Live trees infected by *E. mammata* are included.

[3] The two categories of dead trees are cumulative over the 47 years of observation. "Other" refers primarily to trees dying from suppression.

[4] n is the sample size.

[5] VIF is the "variance inflation factor" and corresponds to C(j) in Rugg (2003); values greater than 1.00 indicate increasing interplot variation. The percentages are weighted using the variance inflation factor and therefore cannot be used to compute the exact number of trees observed in each category.

Table 4.—*Between sample estimates of tree survival and mortality rates by cause for thinned and unthinned quaking aspen from 1951 to 1998*

Years	Trt[1]	Tree survival	Cause of mortality: Hypoxylon canker	Cause of mortality: Other[2]	Ratio[3]	VIF[4]	P(diff)[5]
		Percent	*Percent*	*Percent*			
1951-56	C	74	5	21	19	1.07	0.0001
	HR	80	4	16	20	3.33	
	THR	91	4	5	44	2.33	
1956-61	C	63	10	27	27	2.84	0.0001
	HR	76	6	18	25	8.31	
	THR	86	13	1	93	3.39	
1961-66	C	59	9	32	22	2.55	0.0001
	HR	59	4	36	10	3.64	
	THR	83	6	10	38	2.27	
1966-71	C	75	7	19	27	3.64	0.0001
	HR	68	4	28	13	3.64	
	THR	85	8	7	53	2.76	
1971-87	C	50	1	49	2	1.00	0.0001
	HR	44	2	54	4	1.12	
	THR	53	4	43	9	1.29	
1987-96	C	60	1	39	–	1.68	0.29
	HR	63	4	33	8[6]	2.06	
	THR	65	4	31	–	2.42	
1996-98	C	94	4	1	–	1.32	0.56
	HR	92	3	5	58[6]	1.34	
	THR	94	4	2	–	1.39	

[1] C = Control; HR = aspen unthinned but hardwoods removed; THR = aspen thinned and hardwoods removed.

[2] Other mortality refers primarily to trees dying from suppression.

[3] Ratio = (Hypoxylon mortality rate/(Hypoxylon mortality rate + Other mortality rate))*100.

[4] VIF is the "variance inflation" factor and corresponds to C(j) in Rugg (2003); values greater than 1.00 indicate increasing interplot variation. The rates are weighted using the variance inflation factor and therefore cannot be used to compute the exact number of trees observed in each category.

[5] P(diff) is the overall probability of no differences among the treatments for mortality caused by Hypoxylon canker, using the likelihood ratio test generated by TableSim (Rugg 2003).

[6] In 1996 and 1998 there were no differences in mortality rate by treatment; therefore, the single value reported is pooled across treatments.

Plot Effects

In the analysis of Hypoxylon canker prevalence, the VIF values in table 1 indicate strong plot effects. When we compare P-values adjusted for plot effects to P-values not adjusted (table 2), the reduction in significance is often on the scale of orders of magnitude. Nonetheless, the adjustment for plot effects produced only one qualitative change (1971 THR-C). The unadjusted P-values are generally either very small or very large, so the qualitative conclusions do not change much when plot effects are accounted for.

In the analysis of tree mortality caused by Hypoxylon canker, the VIF values in tables 3 and 4 show strong plot effects, and the P-values are generally very small or very large. Although we do not present the raw analysis of mortality, the parallels to the canker prevalence P-values make it clear that plot effects substantially altered the strength of the conclusions but rarely their qualitative outcome.

The likelihood ratio statistic (LR) used in the categorical data analysis of canker prevalence and tree survival can be separated into additive pieces. Therefore, we can compare the LR with and without plot effects in the model to determine how much lack of fit is due to plot effects and how much is due to treatment effects. The LR calculation shows that plot effects accounted for roughly half of the lack of fit in the first 20 years of the study for both canker prevalence and tree survival (table 5). In later years, plot effects continued to have strong impacts on survival, but only minor impacts on canker prevalence.

Table 5.—*Percentage reductions in likelihood ratio (LR) test statistics from adjusting for plot effects in the analyses of Hypoxylon stem canker prevalence and tree survival; larger reductions mean that more of the total variability in the response variable can be attributed to plot, rather than treatment, effects*

Year	LR % reduction	
	Canker prevalence	Tree survival
1953	48.4	48.3
1956	59.9	70.5
1961	69.0	61.0
1966	39.4	67.5
1971	53.6	12.4
1987	7.1	44.9
1996	5.3	25.5
1998	0.6	48.3

Tree Growth

In 1987 there were no differences among treatments in d.b.h. (P = 0.56) (table 6). Trees in the HR plots had a greater average height than trees in the THR (P = 0.05) or C (P = 0.002) plots; trees in the THR and C plots did not differ in height (P = 0.12). In 1998 trees in the HR plots had a smaller average d.b.h. than trees in the THR (P = 0.028) or C (P = 0.058) plots; trees in the THR and C plots did not differ (P > 0.90) (table 6). There were no differences in height (P > 0.90).

Tree Density

Density declined in all treatments over the 47-year growing period and approached similar levels across treatments (fig. 2). The t-tests showed that THR and C plots did not differ in density from 1966 onward (P = 0.11 in 1966, P > 0.50 thereafter). THR plots had lower density than HR plots in all years except 1998 (P < 0.002 in 1966, 1971, and 1987; P = 0.023 in 1996; P = 0.057 in 1998). Similarly, C plots had lower density than HR plots in all years except 1998 (P < 0.001 in 1966 and 1971; P = 0.035 in 1987 and 1996; P = 0.079 in 1998).

Table 6.—*Diameter at breast height (d.b.h.), height and volume of quaking aspen by treatment in 1987 and 1998; the statistics were computed using plot means as the raw data*

Year 1987 Trt[1]	D.b.h. (cm) Mean	S.E.	Height (m) Mean	S.E.	Yield (m^3/tree) Mean	S.E.	Yield (m^3 ha^{-1})[2] Mean	S.E.	Plots (n)
C	22.9	0.663	23.2	0.417	0.589	0.059	307.6	30.18	10
HR	22.2	0.572	25.1	0.243	0.580	0.034	408.1	15.80	10
THR	24.4	0.441	24.0	0.369	0.610	0.033	314.8	17.20	20
1998									
C	29.3	0.915	23.9	0.434	0.742	0.056	199.0	18.88	10
HR	27.6	0.515	24.0	0.404	0.648	0.031	261.4	16.43	10
THR	30.1	0.615	24.0	0.343	0.762	0.043	240.1	20.20	20

[1]C = Control; HR = aspen unthinned but hardwoods removed; THR = aspen thinned and hardwoods removed.
[2]Yield reductions in 1998 were the result of windthrow throughout the study site in May 1996.

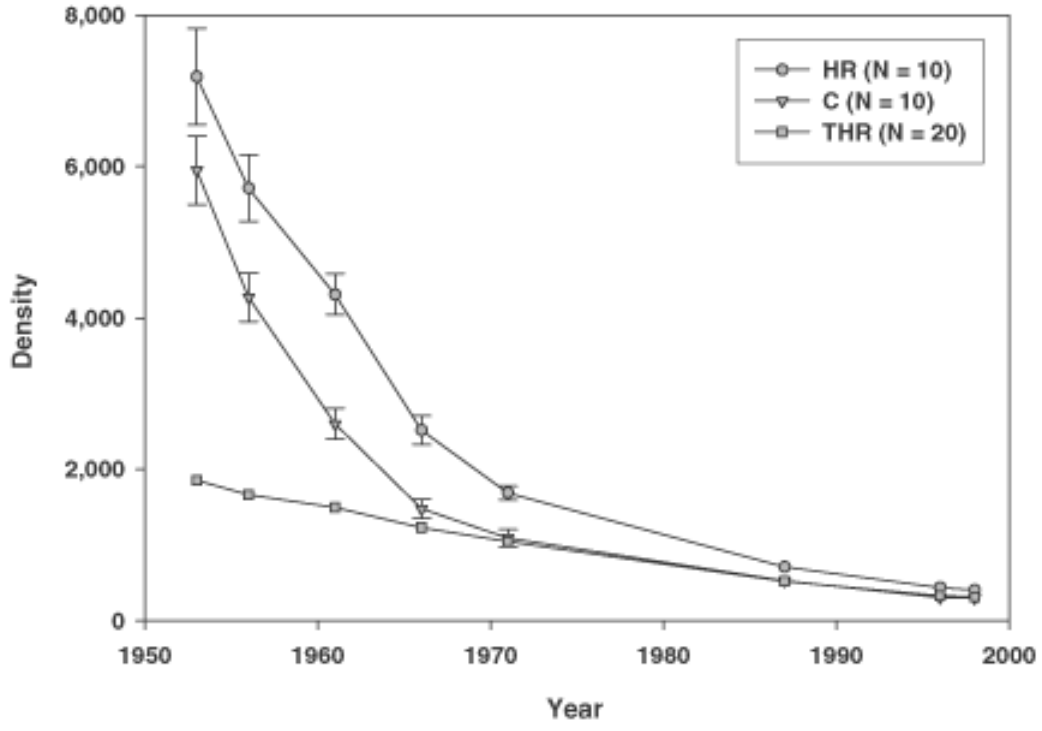

Figure 2.—*Trend in the average number of quaking aspen per hectare by treatment from 1951 to 1998. HR = aspen unthinned but hardwoods removed; C = Control; THR = aspen thinned and hardwoods removed; n = number of plots.*

DISCUSSION

Study Design

The study design is clearly not optimal by modern standards, and for some people, raises the question of whether reliable inference can be drawn from the observed data. In the context of experimental designs similar to this one, the problem is often referred to as one of pseudoreplication (Hurlbert 1984). As described by Hurlbert (1984), pseudoreplication is primarily an issue of testing hypotheses using variances that are inappropriately small. Such variances might arise from samples being dependent instead of independent or from samples being embedded in larger treatment areas that are more uniform within a treatment than across treatments. There are at least four reasons to conclude that our analyses are not particularly subject to pseudoreplication effects. First, the measurement plots were spaced far enough apart for an assumption of independence to be reasonable. (Trees within plots, on the other hand, were clearly dependent subsamples.) Second, there were no obvious differences in terrain, soil, weather regime, etc., across the large treatment areas that would affect Hypoxylon canker incidence, tree mortality caused by Hypoxylon canker, or aspen growth. Third, the removal of substantive plot-to-plot variation by the Koehler-Wilson analysis technique demonstrates the lack of inappropriate uniformity of response within treatment areas. Fourth, the undesirable effect of pseudoreplication is to increase the frequency of finding significant treatment differences when none actually exist. Even if our assumptions about processes and independence are so wrong as to affect the analyses, our conclusions are more about the lack of differences or the lessening of differences over time—the exact opposite of what would be expected from pseudoreplication.

Impact of Thinning (THR)

Resource competition among shade intolerant aspen suckers results in significant self-thinning among aspen (Bella 1975). Consequently, it is not surprising that the proportion of trees dying from Hypoxylon canker was higher in the THR plots than in the C or HR plots because thinning removed trees that most likely would have been lost over time to self-thinning. This implies that even if precommercial thinning had no effect on tree mortality caused by Hypoxylon canker the ratio of trees dying from Hypoxylon canker compared with trees dying from other causes would always be higher in the THR plots. Therefore, this often-reported ratio cannot, on its own, answer whether precommercial thinning causes disproportionate tree mortality caused by Hypoxylon canker.

The hypothesized damage caused by Hypoxylon canker should result in effects on tree density or dimensions. At the end of this study, the THR and C treatments had very similar tree densities (fig. 2); furthermore, the mean dimensions of individual trees were also very similar (table 6). Consequently, the study's original hypothesis that thinning would result in greater damage from Hypoxylon canker was not borne out. Rather, tree mortality from Hypoxylon canker appears to be just one of a number of interchangeable biological agents that, together with intraspecific competition (self-thinning), reduce quaking aspen stand density over time. Given the lack of stand differences in tree size in the THR and C treatments at the end of this study, this interchangeability of mortality factors extends to equivalent effects on stand health and productivity. Although these conclusions differ from some studies and from analyses reported earlier in the lifespan of this study, they are similar to the conclusions reported by Pitt *et al*. (2001). It is possible that short-term effects occur, but by the time a stand matures and is ready for harvest, any short-term effects are no longer discernible.

Impact of Hardwood Removal (HR)

The HR treatment had the greatest number of living trees throughout the study, producing a clear yield advantage at the end of the 47 years

of observation. It also had the lowest prevalence of Hypoxylon canker through the 1966 observation period. This may have been the result of earlier natural lower stem branch pruning resulting from shading and thus the presence of fewer potential infection sites (Anderson and Martin 1981, Ostry and Anderson 1979, Ostry and Anderson 1998) than on trees in the THR and C treatments.

Aspen Clone Effects

Apart from treatment effects, the impact of plot variability on canker prevalence and canker-induced tree mortality was quite striking. The apparent effect of thinning on canker prevalence was much less when plot effects were included in the analysis. Recall that each plot was in a single unique clone. We are not aware of any research showing differences in the incidence or severity of Hypoxylon canker on a set of common clones on multiple sites. Experimental evidence has, however, demonstrated varying levels of resistance to Hypoxylon canker among common sets of aspen clones when challenged with the pathogen in field and greenhouse tests (Bucciarelli *et al*. 1998, 1999; Enebak *et al*. 1997, 1999). This leads us to believe that the high interplot variability in canker prevalence and canker-induced mortality (table 5) was primarily due to clonal differences in canker resistance rather than to site differences. The biological reasons for the observed decrease in clone effects on canker prevalence after 1971 (tables 1, 5) are unknown; however, the portion of plot variability in tree survival of diseased trees continued to be large. This may be explained by the ability of resistant clones to inactivate cankers by producing callus barriers (Ostry and Anderson 1998). Given their magnitude, these clone effects should be taken into account in future studies. The definitive test of the effects of thinning aspen on the prevalence of Hypoxylon canker would be to evaluate the same clones across the various treatments to eliminate the confounding genetic effects on disease susceptibility.

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KEY WORDS: *Populus tremuloides*, *Entoleuca mammata*, *Hypoxylon mammatum*, stem disease.

MISSION STATEMENT

We believe the good life has its roots in clean air, sparkling water, rich soil, healthy economies and a diverse living landscape. Maintaining the good life for generations to come begins with everyday choices about natural resources. The North Central Research Station provides the knowledge and the tools to help people make informed choices. That's how the science we do enhances the quality of people's lives.

For further information contact:

North Central
Research Station
USDA Forest Service
1992 Folwell Ave.
St. Paul, MN 55108

Or visit our web site:

www.ncrs.fs.fed.us