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DISEASES OF ASPEN SUCKERS IN NORTHERN ONTARIO

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RÉSUMÉ

L'importance des maladies qui affectent la régénération du peuplier faux-tremble (*Populus tremuloides* Michx.) est évaluée sur la base des données déjà colligées, des recherches en cours et d'une enquête conduite en 1977 dans tout le nord de l'Ontario par le Relevé des insectes et des maladies des arbres forestiers du Centre de recherche forestière des Grands Lacs. Le relevé de 1977 a nécessité l'échantillonnage aléatoire de 45 peuplements et 10,685 drageons de peuplier faux-tremble.

L'analyse des défauts du bas de la tige ainsi que des appareils racinaires a été incluse dans le relevé, et un fort pourcentage des arbres échantillonnés étaient affectés intérieurement de tache colorée (61%) et de carie (32%). *Armillaria mellea* (Vahl. ex Fr.) Kummer, un agent causal de carie et de la pourriture du pied, a été extrait de 24% des appareils racinaires et était présent dans 24% des peuplements échantillonnés. D'autres organismes associés avec des défauts sont rapportés.

La brûlure des pousses du peuplier causée par *Venturia macularis* (Fr.) Müll. & Arx est considérée être une maladie grave chez le jeune peuplier faux-tremble. Elle tue fréquemment les pousses terminales supérieures, causant ainsi une perte de croissance en hauteur et l'altération du port de l'arbre. La tache d'encre (*Ciborinia whetzelii* [Seaver] Seaver), une maladie des feuilles, causait d'ordinaire des dégâts insignifiants. Le champignon, *Cytospora chrysosperma* (Pers.) Fr., agent causal de chancre, a été fréquemment trouvé au cours du relevé de 1977, mais il n'est pas considéré comme un problème important chez le jeune peuplier faux-tremble. *Hypoxylon mammatum* (Wahl.) Miller a été rarement trouvé au cours du relevé de 1977 et, bien que causant un chancre grave dans les vieux peuplements, n'est pas considéré important dans les jeunes peuplements de drageons de peuplier faux-tremble.

ACKNOWLEDGMENTS

We wish to acknowledge the assistance of William Britnell and Wayne Ingram who isolated and identified organisms associated with wood defects, and of the FIDS field staff who did the field sampling.

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INTRODUCTION

In the last decade, aspen (*Populus tremuloides* Michx.) utilization has increased in northern Ontario. Currently about 1,000,000 m^3 are harvested annually. While this volume is well below the allowable annual cut, large areas of cutover are present. Aspen is regenerating on these sites principally as suckers growing from the parent root systems that usually continue to live. The potential of these regenerating stands assumes greater importance as aspen utilization increases.

Investigations of the pathological quality of aspen suckers have been conducted by Basham and Navratil (1975) and Kemperman et al. (1976). The studies were conducted in the cutting limits of the American Can Canada Inc. in the Terrace Bay District of the Ontario Ministry of Natural Resources (OMNR). Wood stain and rot were the principal defects studied. However, considerable damage by other diseases was observed. Aspen shoot blight (*Venturia macularis* [Fr.] Müll. & Arx), for example, was especially severe in the study area in 1974 and 1975. The 1977 survey by the Forest Insect and Disease Survey Unit (FIDS) was stimulated by concern expressed about the probable importance of other aspen diseases such as shoot blight. Root and lower stem defects were included to determine if the information accumulated by Basham and Navratil (1975) and Kemperman et al. (1976) was typical throughout the range of aspen in Ontario.

METHODS

Sample Selection

Five randomly selected stands of aspen regeneration were sampled by each survey field technician located in northern Ontario. Stands were less than 10 years of age, had at least 50% aspen composition, and the majority of aspen trees present originated as root suckers. The character of the stands sampled is summarized in Table 1. Plot locations and the OMNR regional boundaries referenced in this paper are shown in Figure 1.

Each selected stand was investigated by standard procedures (Gross, unpublished) used by FIDS staff for sampling the particular disease present. Ten randomly located 3.3 m wide strip plots provided a minimum sample size of 100 aspen trees per stand. Strip length varied depending on the density of aspen encountered in a particular stand.

The first aspen sucker on each of the 10 plots was exhumed for defect analysis. The parent root was severed at a distance of about 20 cm from the sucker. Then roots smaller than 0.5 cm diam and stem

portions more than 20 cm above ground level were discarded, as earlier studies have shown that these tissues in young aspen are generally defect-free. The remaining portions were sectioned progressively toward the root collar in 5 cm segments. Sectioning of the stem or any root stopped if stain or rot was observed on a cut surface. The remaining portions were shipped immediately to the Great Lakes Forest Research Centre so that they would arrive within 48 hours. After arrival samples were stored at -5°C. Later the roots were further sectioned for defect analysis. As part of this analysis, isolations were attempted from any wood showing evidence of rot or stain. Isolates were identified from cultures grown on 2% malt agar.

Table 1. Summary data for stands sampled to rate diseases of aspen.

Region[a]	Age		Height (m)		Diameter (cm)		Trees/m^2		Percent aspen	
	Avg	Range	Avg	Range	Avg	Range	Avg	Range	Avg	Range
Northeastern	4.3	2-6	2.5	2.0-3.5	1.3	0-2.5	1.2	0.7-1.7	92	83-100
Northern	7.1	5-9	2.7	1.5-3.7	2.1	1.0-2.5	0.9	0.5-1.6	98	77-100
North Central	8.0	6-10	2.2	1.1-3.0	2.2	1.2-3.0	0.5	0.4-1.0	90	65-100
Northwestern	4.8	3-9	2.2	1.8-3.3	1.2	0.6-1.8	1.1	0.6-2.1	97	82-100

[a] See Ontario Ministry of Natural Resources regional boundaries in Figure 1.

The total sample consisted of 45 stands, 10,685 aspen suckers, and 450 lower stems and roots for defect analysis. The large sample of trees resulted from a tendency to select larger plots than necessary to ensure a minimum of 100 aspen trees per stand.

RESULTS AND DISCUSSION

Cankers

Cytospora canker (*Cytospora chrysosperma* [Pers.] Fr.) was encountered in most of the stands. It was frequently observed fruiting on dead suckers, stem wounds, and recently dead twigs or branches (Fig. 2). However, no instances in which *C. chrysosperma* was considered the primary cause of a canker or branch death were recorded.

Hypoxylon canker (*Hypoxylon mammatum* [Wahl.] Miller) (Fig. 3) was observed in only seven (15%) of the stands. The most severe damage was in a stand in the Northern Region where 3% of the trees had cankers.

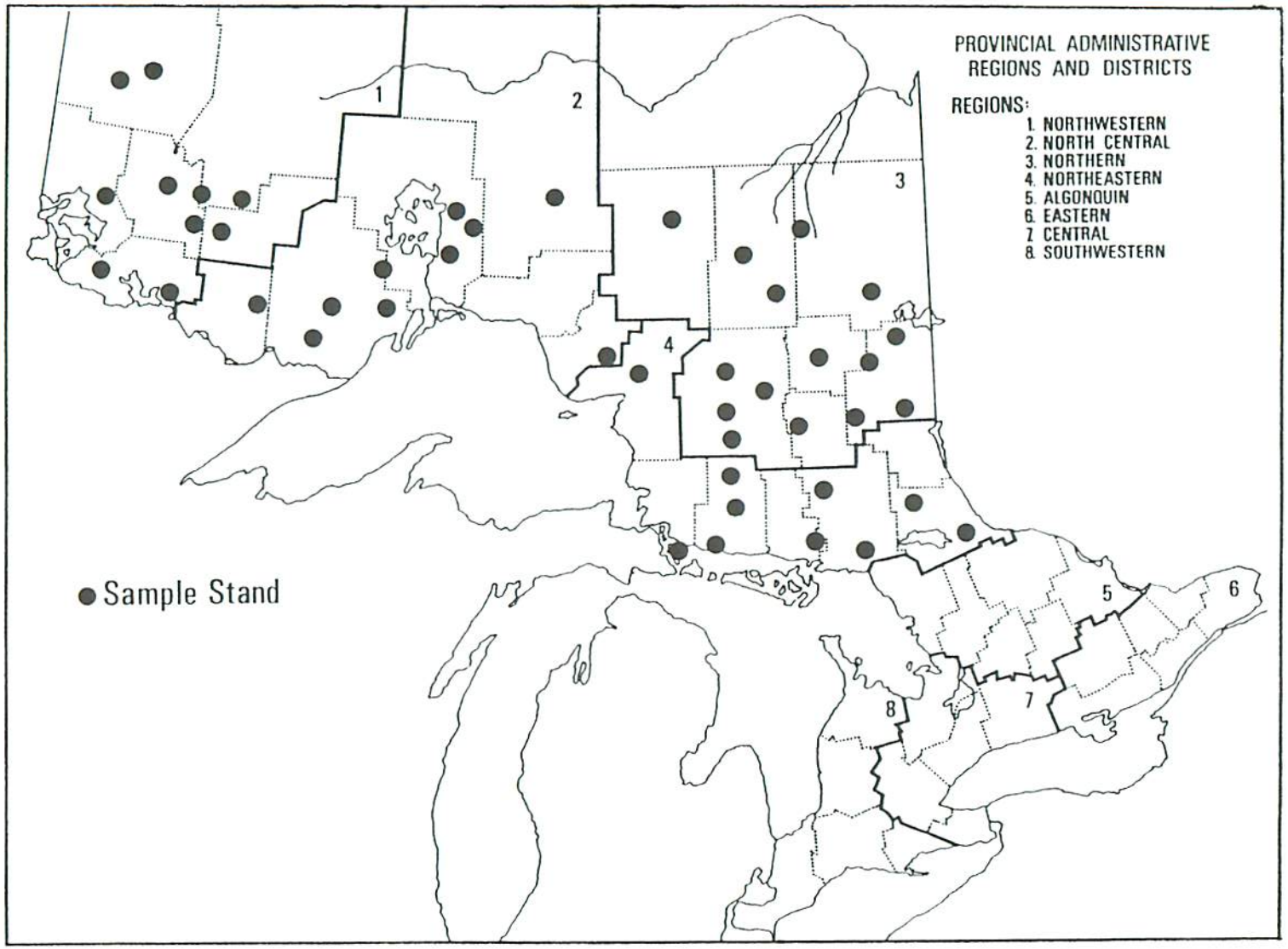

Figure 1. The location of stands sampled for the aspen disease survey, and Ontario Ministry of Natural Resources administrative regional boundaries.

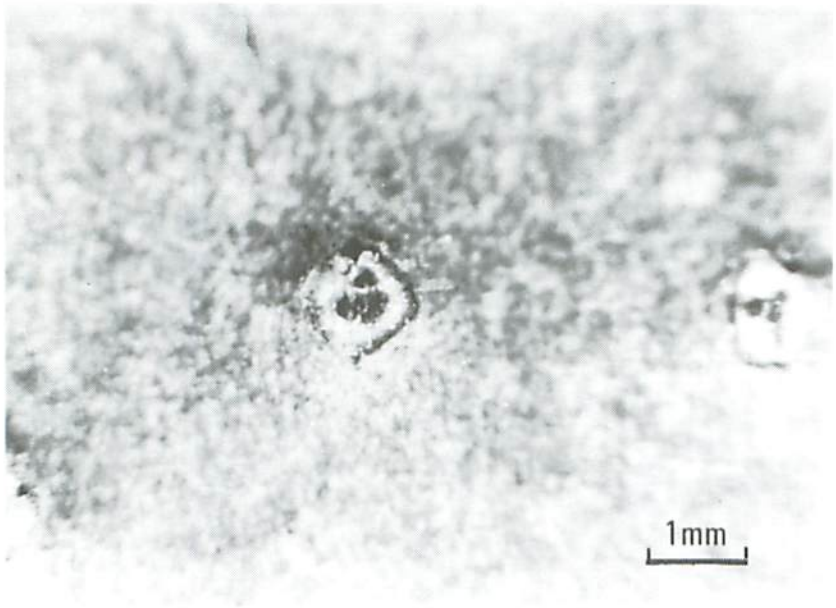

Figure 2

Spore-producing pycnidia of *Cytospora chrysosperma* on an affected aspen branch.

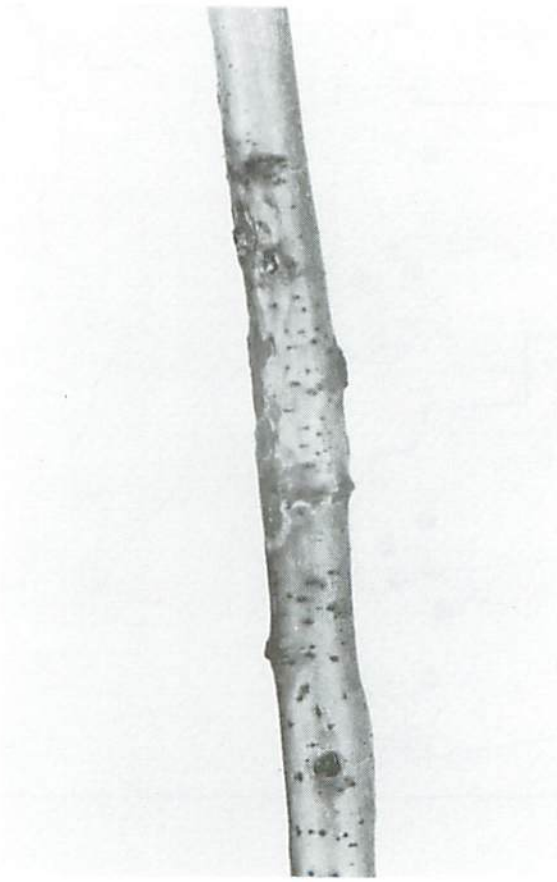

Figure 3

Hypoxylon canker (*Hypoxylon mammatum*) causing the death of a young aspen sucker.

Half of the affected trees had just died. In the other six stands, cankering occurred on 1% or fewer of the trees. Hence, Hypoxylon canker is considered to be relatively unimportant in aspen sucker regeneration. This is in contrast with the severity of the disease in older stands. The 1968 FIDS Annual Report (Anon. 1968) indicated that, among older aspen stands in Ontario, 70% have more than 5% of the trees cankered, and 29% have more than 25% of the trees cankered. There are poorly stocked stands of regeneration-size aspen growing on dry sites in which Hypoxylon canker occurs on 5 to 10% of the trees. The survey sample, however, was representative for aspen sucker stands across Ontario. Most of these stands are on average or better than average sites, possibly because aspen on poorer sites is not being utilized.

Foliar Disease

Ink spot (*Ciborinia whetzelii* [Seaver] Seaver) was not prevalent on aspen in 1977. The disease (Fig. 4) was present in 13 (29%) of the stands sampled; however, in 11 of these the percentage of foliage affected was negligible ($\leq$ 5%). One stand in the North Central Region experienced light (6-25%) foliar damage. Another stand in the Northwestern Region had heavy (76-100%) foliar damage. Disease conditions detected in the sampled stands seemed to be reflective of those in aspen stands of all sizes across Ontario. Severe damage was localized; most stands were unaffected or had negligible damage.

In most years, foliar damage caused by ink spot is similar to conditions that prevailed in 1977, the year of the survey (Anon. 1967-1981). Damage in some parts of Ontario was more pronounced in 1973, 1975 and 1978. In those years, damage ranging from 25 to 100% of the foliage affected was common in some OMNR administrative regions. Elsewhere, however, ink spot was present at typically low levels. Conditions for infection were no doubt less favorable in these areas, possibly because of the weather.

Aspen shoot blight (*Venturia macularis*) (Fig. 5) was detected in 43 (96%) of the stands sampled (Table 2). Foliar damage was light (6-25%) in four stands and trace (0-5%) in 39 stands. The disease kills succulent foliage and stem portions (Fig. 5) and progress along a shoot generally stops where woody twig tissue begins.

Most infections occur on long shoots. These continue to grow and have succulent tips as long as growth conditions are favorable. In northern Ontario, long shoot growth usually continues through most of August. This is especially true in the moist years that seem to be conducive to disease intensification and spread. In contrast, short shoots extend a short distance and produce a cluster of leaves early in the growing season. Hence, these remain succulent and susceptible to infection for a limited time. Foliar damage by shoot

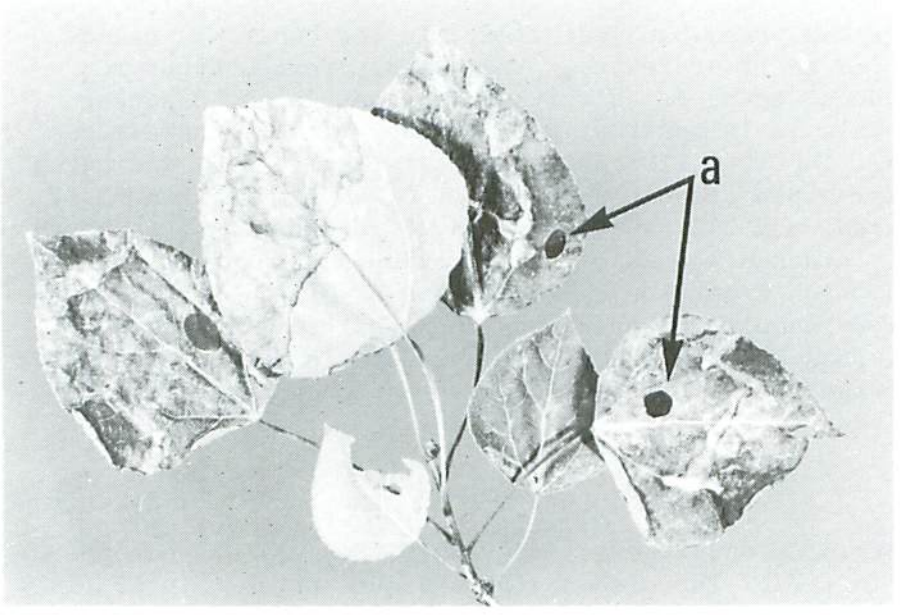

Figure 4. Ink spot (*Ciborinia whetzelii*) affecting aspen foliage. The black stromatic tissue (a) is the overwintering stage of the fungus. These often fall out, leaving holes in the dead leaf.

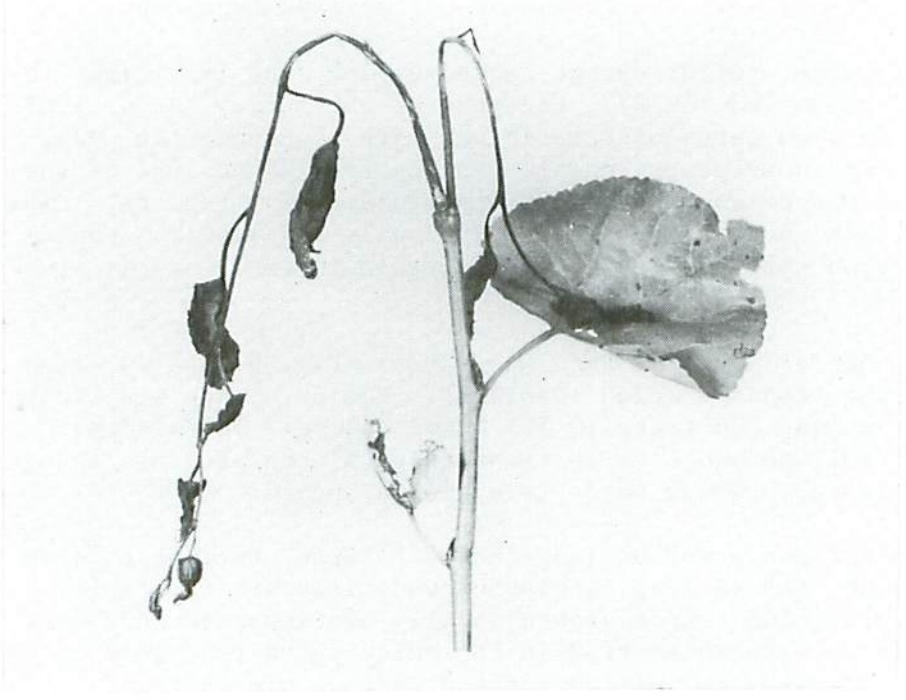

Figure 5. Aspen terminal shoot showing typical infections by aspen shoot blight (*Venturia macularis*). Affected shoots normally die to the point where woody stem tissue is encountered. Dead shoots characteristically droop, forming "shepherds' crooks". The original terminal as well as the terminal that flushed later from a lateral bud were both killed by shoot blight.

blight rarely exceeds 10% of the potential leaf surface area as the bulk of aspen foliage is on the short shoots. First year suckers, however, are an exception. Each of these develops as a single, highly susceptible, long shoot, and as such can suffer severe damage.

Table 2. The 1977 status of aspen shoot blight (*Venturia macularis*) in northern Ontario aspen sucker regeneration.

Region[a]	Trees affected		Terminals affected		Defoliation	
	(%)	Range	(%)	Range	(%)	Range
Northeastern	24	1-73	14	0-42	3	0-7
Northern	14	0-53	10	0-31	4	0-7
North Central	29	2-84	28	0-73	3	0-14
Northwestern	71	24-100	54	7-90	6	0-10

[a] See Ontario Ministry of Natural Resources regional boundaries in Figure 1.

Recent investigations (Gross, unpublished) indicated that host impact is mostly a function of damage to the uppermost terminal shoots. Terminal infection usually reduces height growth by about one-third in the year of infection. Tree form is also affected as the dead stem stub causes a crook in the stem where the next terminal begins (Fig. 6). During years when the disease is especially severe, such as 1975 and 1978, tree terminals frequently are killed back several times.

The disease is spread by spores produced by the fungus on the dead terminals. The spores are disseminated by rain splash; hence, new shoots flushing from buds immediately below dead terminals are exposed to especially high densities of spores. Infection of these new succulent shoots is common during wet weather, and when disease incidence is high, few terminals escape infection (Fig. 6, 7 and 8). After several seasons of severe shoot blight, trees develop abnormal bush-shaped forms (Fig. 6). In the survey, stands were found in which practically all of the aspen had this distorted, bushy appearance.

Damage to terminals was directly related ($\alpha = 0.001$, F test) to the amount of foliar damage (Fig. 7). However, the relationship was not satisfactory for predictive purposes ($R^2 = 0.692$). The percentage of foliage damaged seemed to be a function of age and tree size. Shoots more than 3 to 4 m above ground had some resistance to infection, and those above 7 m were rarely affected. Also, the ratio

of long shoots to short shoots decreases as trees grow older. The proportion of foliage susceptible to infection is thus similarly reduced.

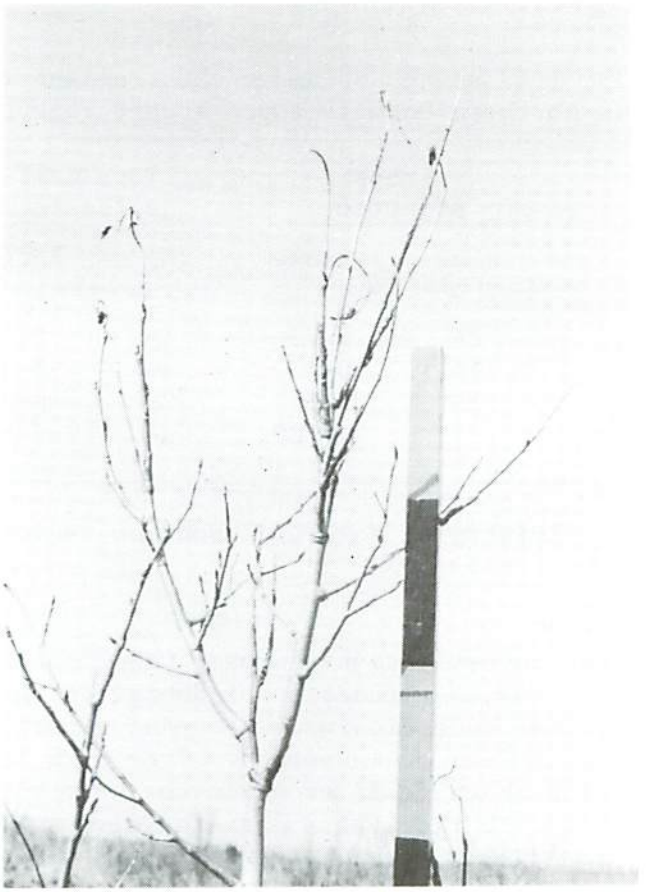

Figure 6. Distorted crown of an aspen sucker caused by several years of infection by aspen shoot blight (*Venturia macularis*).

Terminal damage was an increasing function of the percentage of trees affected in a stand (Fig. 8, $\alpha = 0.001$, F test). In this case the relationship was satisfactory for predictive purposes ($R^2 = 0.980$). Also, data (Gross, unpublished) collected in the Terrace Bay District of the North Central Region showed that a similar relationship existed for the years 1976 through 1978.

To get a clear picture of disease severity over time, the situation in 1977 was compared with that of 1967-1976. Terminal damage was estimated from the reported percentage of trees affected (Anon. 1967-1981) as indicated by the above relationship (Fig. 8). In Ontario,

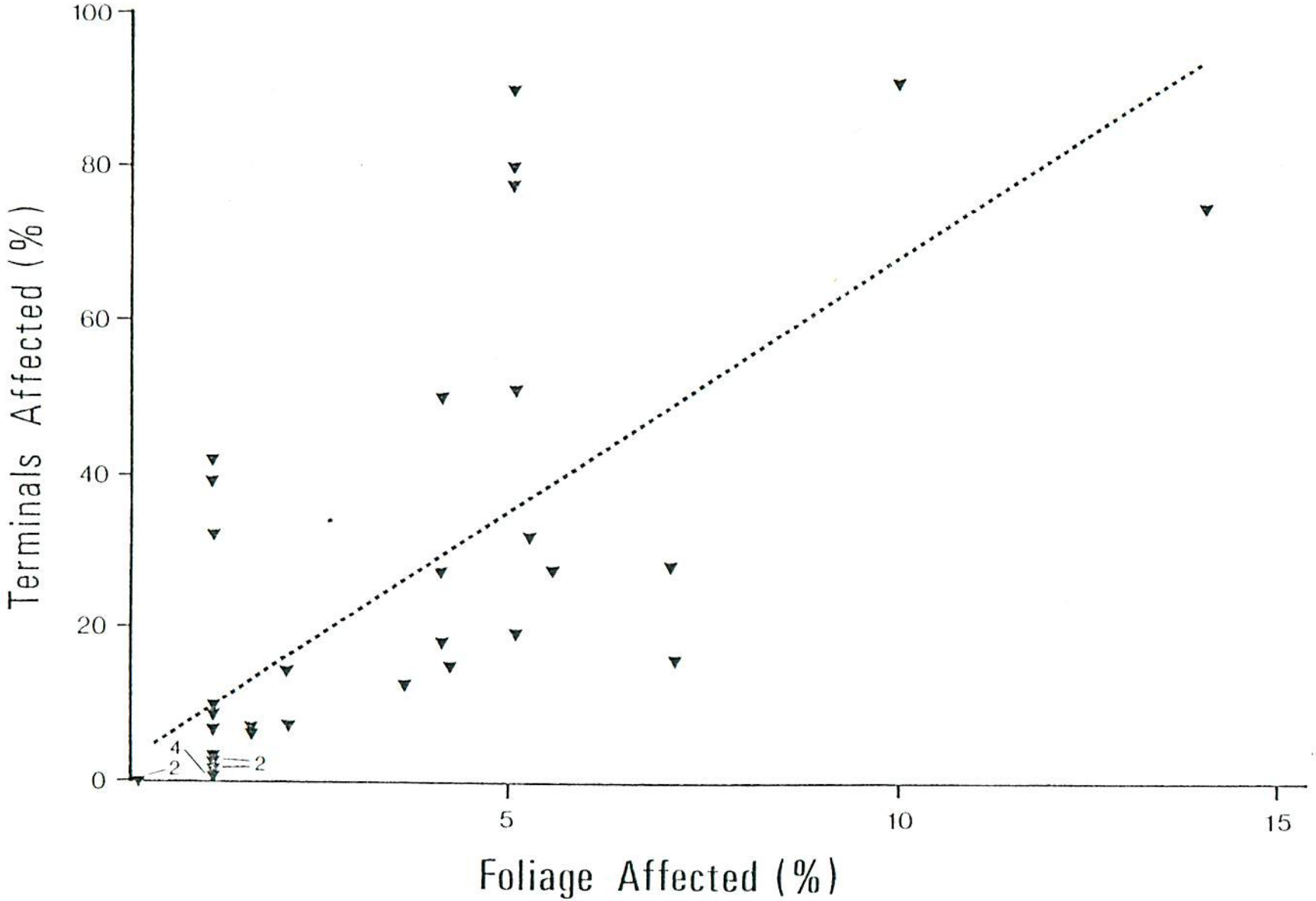

Figure 7. The relationship between the percentage of foliage affected by aspen shoot blight and the percentage of terminals affected by the disease. $Y = 6.351\ X + 0.037$ ($R^2 = 0.692$, s.e. = 23.1%).

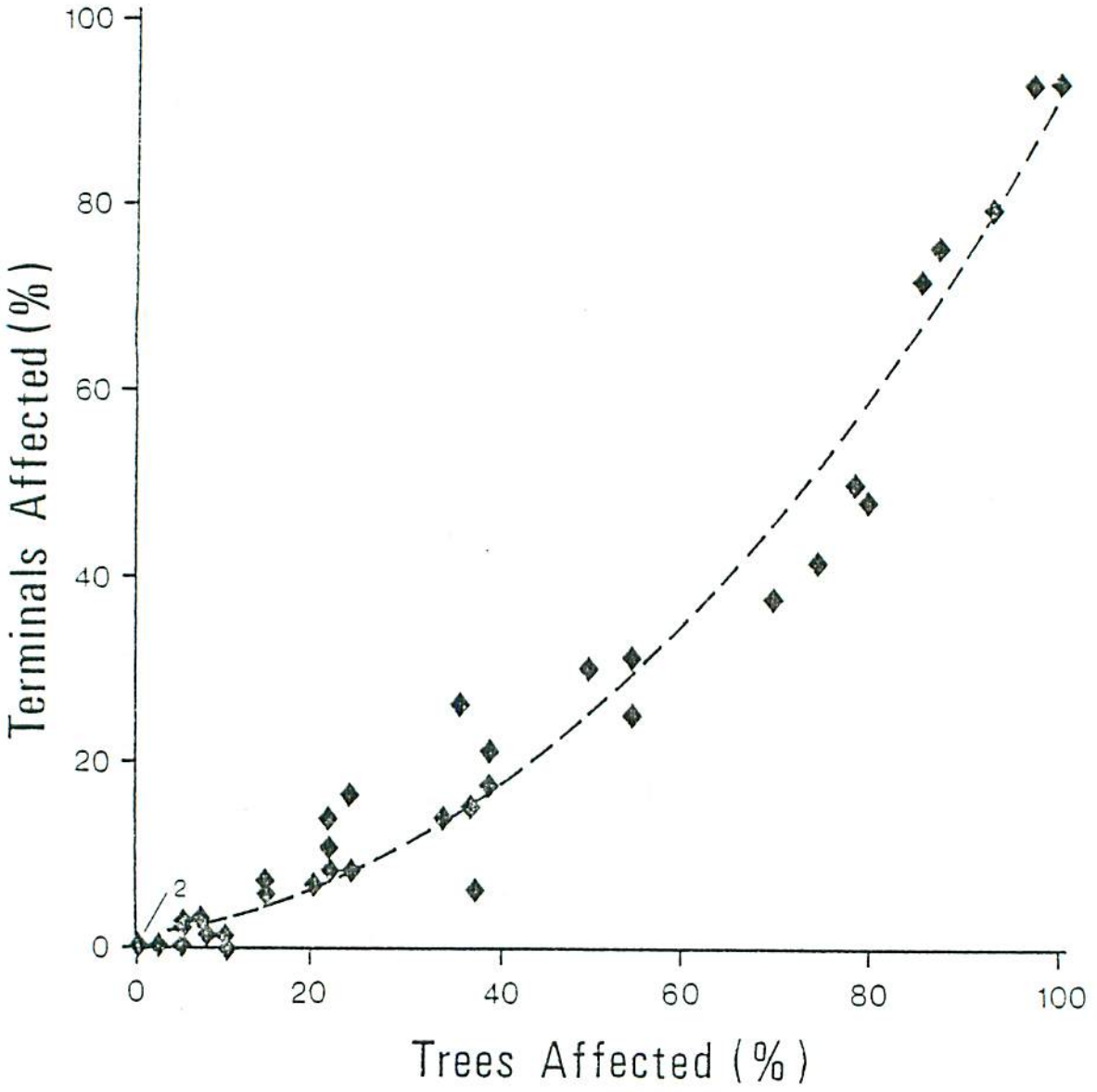

Figure 8. The relationship between the percentage of trees affected by aspen shoot blight (*Venturia macularis*) and the percentage of terminals affected by the disease. $Y = 0.878\ X^2 + 3.60$ ($R^2 = 0.980$, s.e. = 5.50%).

damage caused by the disease was especially severe in 1969, 1975 and 1978. Otherwise, light damage similar to that which prevailed in 1977 was indicated. In 1977, damage was severe only in the Northwestern Region, where over half of the terminals were killed in six of the ten stands sampled. Elsewhere damage was less severe. About half of the stands had negligible damage and the remainder had terminals killed on about 10% of the trees. In view of the dense character (Table 1) of the stands sampled, enough crop trees on these latter sites probably escaped infection that overall shoot blight impact was negligible. There was some reduction in average height growth, but this could be unimportant, particularly if sufficient crop trees continue to escape infection. Survey records for 1967-1976 indicate other relatively localized occurrences of shoot blight in years when damage was light over most of the province. The differences probably were the result of local weather-related conditions favorable to infection on these sites.

Some tree mortality directly attributable to shoot blight was observed in the North Central and Northwestern regions. Mortality, however, is unusual as woody stem portions are resistant to disease. Mortality of first-year succulent suckers flushing from parent root systems was observed in stands in which most of the suckers had emerged one or occasionally two years previously. This mortality can be important when regenerating stands are poorly stocked.

Aspen shoot blight appears to be one of the most important diseases affecting aspen sucker regeneration. In years when shoot blight is severe, sucker stands with almost 100% terminal infection are common. Large clear-cut areas are now common, and aspen regeneration throughout these vast areas can be affected. Stands tend to stagnate during periods when severe shoot blight conditions prevail. Moreover, butt log portions can be greatly distorted by several years of severe shoot blight. Also, the stubs remaining after shoots die can be infection courts for canker and rot-causing agents. This aspect of shoot blight is being investigated at the Great Lakes Forest Research Centre.

Wood Defect

A large portion of the trees sampled had defective roots (stained or decayed) (Fig. 9, Table 3). It was difficult to compare the results of this survey with those of Basham and Navratil (1975) or Kemperman et al. (1976), as sample methods differed. However, all studies detected root rot and stain defect in a large percentage of the aspen suckers. For this survey, 61% of the trees had internal stain and 32% had both rot and stain defect (Table 3). As there were no striking differences among the four regions sampled (Table 3), this disease level in aspen sucker regeneration appears to be fairly constant across Ontario. Stain defect was detected in all stands and rot defect was detected in all but two stands. In forty-two (93%) of the stands root defect was found in 30% or more of the trees.

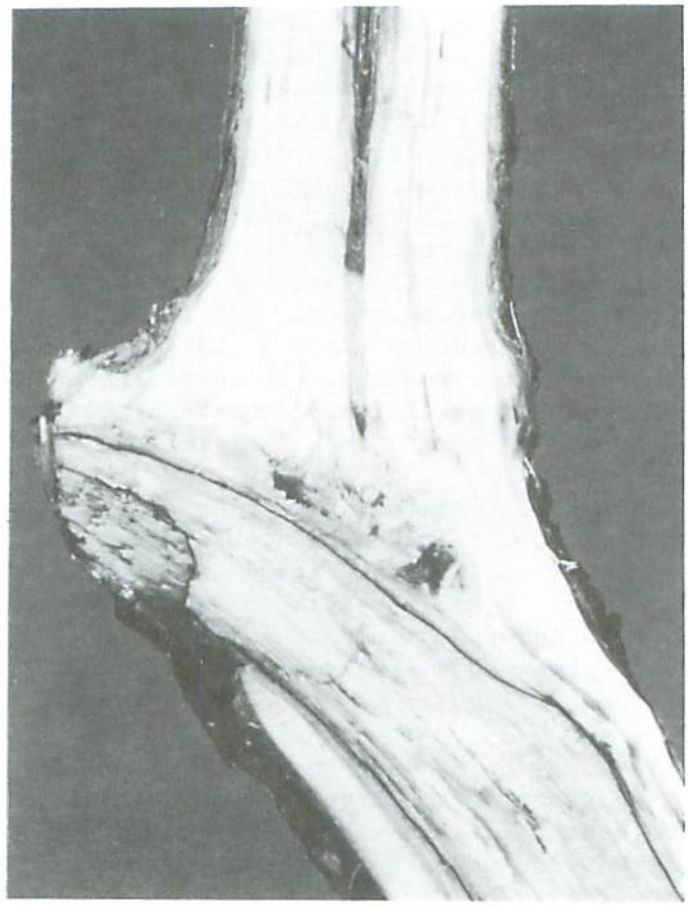

Figure 9. Portion of an aspen sucker showing rot and stain associated with a wound on the parent root system. *Armillaria mellea* and *Coryne sarcoides* were isolated from this defect.

Injuries or nearby dead tissue were observed in association with 43% of the defects (Table 4). The main associations found were: tunnels (25%) caused by root-boring larvae of a ghost moth (*Sthenopis quadriguttatus* Grote), stone bruises (22%), carry-over defect from wounds on the parent root system (21%), and dead companion stems located nearby on the same parent root (12%).

Many of the organisms isolated from aspen sucker rots and stain sites (Table 5) were also isolated by Basham and Navratil (1975). Significantly, *Armillaria mellea* [Vahl ex Fr.] Kummer was isolated from 4% of the root systems, and was present in 11 (24%) of the stands sampled. In view of the fact that only 10 root systems per stand were sampled, this seems to represent a fairly general occurrence of Armillaria root rot in aspen sucker stands in northern Ontario.

Table 3. Incidence of stain[a] and rot in aspen sucker regeneration in northern Ontario.

	Region[b]				
	Northeastern	Northern	North Central	Northwestern	Total
Lower Stem					
stain (%)	32	60	52	31	45
rot (%)	6	10	14	3	8
Root Collar					
stain (%)	50	71	64	49	60
rot (%)	4	9	8	4	5
Root					
stain (%)	42	53	52	47	49
rot (%)	22	28	24	24	25
Composite					
stain (%)	51	53	66	50	61
rot (%)	23	39	34	29	32

[a]Data for stain defect include stain associated with rot. Since stain was associated with all but two rot defects, stain-only defect can be determined by subtracting rot percentages.

[b]See Ontario Ministry of Natural Resources regional boundaries in Figure 1.

It is difficult to assess what this high incidence of internal defect means with respect to future mortality and cull as aspen stands mature. Certainly the high percentage of young aspen suckers in which rot defect is present at an early age implies that root and stem rot are potentially serious problems in this species.

Table 4. Injury and dead tissue associated with rot and stain defect in aspen sucker regeneration.

Defect associated with	Defect				Total defect	
	Rot		Stain			
	n	%	n	%	n	%
Insect tunnel	16	25	14	25	30	25
Stone bruise	6	10	20	36	26	22
Old wound on parent root	10	16	15	27	25	21
Dead companion stem	13	20	1	2	14	12
Other insect injury	7	11	1	2	8	7
Attached to dead parent root	3	5	3	5	6	5
Bark seam	5	8	0	0	5	4
Small dead root	3	5	2	3	5	4
	63	100	56	100	119	100
Injury association indistinct	132		143		275	
Total defects detected	195		199		394	

Table 5. Microorganisms isolated from rotted or stained wood in aspen suckers.

	Isolation		Area affected (n)		
	(n)	Percent with rot	Stem	Root collar	Roots
Basidiomycetes					
Armillaria mellea (Vahl ex Fr.) Kumm.	21	57	2	1	18
Coprinus micaceus Fr.	13	62	0	3	10
Fomes igniarius (L. ex Fr.) Gill.	3	33	2	1	0
Gleocystidiellum karstenii (Bourd. & Galz.) Donk	2	50	2	0	0
Peniophora polygonia (Pers. ex Fr.) Bourd. & Galz.	2	0	1	0	1
Collybia velutipes (Curt. ex Fr.) Kumm.	1	100	0	0	1
Peniophora cinerea (Pers. ex Fr.) Cke.	1	0	0	0	1
Poria subacida (Pk.) Sacc.	1	0	1	0	0
Scytinostroma galactinum (Fr.) Donk	1	100	0	0	1
Unidentified Basidiomycete	9	78	1	2	6
Ascomycetes and Imperfects					
Phialophora alba van Beyma	69	28	7	15	47
Trichoderma viride Pers. ex Fr.	45	42	1	13	31
Penicillium spp.	34	15	8	11	15
Penicillium thomii Maire	29	21	5	9	15
Cytospora sp.	20	0	13	6	1
Coryne sarcoides (Jacq. ex Fr.) Tul.	10	20	3	1	6
Pachybasium hamatum (Bon.) Sacc.	10	60	1	2	7
Fusarium sp.	4	75	1	1	2
Phlebia strigozonata (Schw.) Lloyd	4	75	4	0	0
Miscellaneous unidentified fungi	318	26	67	87	164
Bacteria	439	22	87	127	225
Yeast	27	18	15	7	5
Sterile	214	10	103	77	34
Total	1277	20	324	363	590

LITERATURE CITED

ANON. 1967-1981. Annual Report of the Forest Insect and Disease Survey. Dep. Environ., Can. For. Serv., Ottawa, Ont.

BASHAM, J.T. and NAVRATIL, S. 1975. An assessment of the pathological quality of aspen suckers established on cutovers in Ontario. Dep. Environ., Can. For. Serv., Sault Ste. Marie, Ont. Report O-X-236. 23 p.

KEMPERMAN, J.A., LYON, N.F., and NAVRATIL, S. 1976. Incidence and volume of defect in second growth aspen stands in northern Ontario. Ont. Min. Nat. Resour., Toronto, Ont. For. Res. Rep. No. 102. 24 p.