Ceratocystis Canker of Aspen

T. E. Hinds

Plant Pathologist, USDA, Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colorado 80521, in cooperation with Colorado State University.

Accepted for publication 6 September 1971.

ABSTRACT

Several species of *Ceratocystis* (*C. alba, C. crassivaginata, C. fimbriata, C. populina*, and *C. tremulo-aurea*) are associated with a trunk canker commonly found on quaking aspen throughout the western United States. Inoculations confirm that *C. fimbriata* is a causal agent and that *C. tremulo-aurea* is not. Virulence of four different isolates of *C. fimbriata* varied considerably. Although young stems and twigs became infected through leaves, the primary point of canker initiation is at fresh bark wounds.

Phytopathology 62:213-220.

Additional key words: canker epidemiology, *Nectria galligena*, insect transmission.

Quaking aspen (*Populus tremuloides* Michx.) stands are extensive in the central Rocky Mountains. In this region, aspen is plagued by a variety of canker diseases, and the damage they cause is probably exceeded only by heart rots. About 11% of the trees examined in five National Forests of Colorado in 1960 had one or more stem cankers (5). Black cankers and others similar to those caused by *Nectria galligena* Bres. on aspen in the Lake States region were common. One or the other was found on 4.4% of the living trees. The latter also resembled the Ceratocystis canker of aspen in Minnesota caused by *Ceratocystis fimbriata* Ell. & Halst. briefly described by Wood & French in 1963 (17). The Nectria fungus was not found on the Colorado canker, whereas *C. fimbriata* was common. Thus, the cause of both canker types remained in question.

The work reported in this study was begun in 1962 to determine the causal agents responsible for these cankers and their mode of infection.

Long (9) described a perennial canker of aspen commonly found in the mountains of Arizona and New Mexico half a century ago. Although he was unable to find fruiting bodies associated with these cankers, he realized that they were not caused by *Cytospora chrysosperma*, the organism discussed in his paper. In 1926, Baker (1) found similar aspen cankers of unknown origin plentiful in localized areas throughout the Rocky Mountain region, and Meinecke (12) published photographs of them. Boyce (2) called the canker, found in Utah and elsewhere in the Rocky Mountain region, "black canker of aspen", and pointed out that it resembled the canker on aspen in Michigan caused by *N. galligena*.

Wood & French in 1963 (17) first reported that *C. fimbriata* was associated with a similar, perennial, target-type canker on aspen in Minnesota, and that the fungus was capable of attacking aspen and causing the canker. The following year the association was reported in Pennsylvania (16), Colorado (5), and the Provinces of Quebec (13), Manitoba, and Saskatchewan in Canada (8). In 1965, Zalasky (18) described the process of infection by *C. fimbriata* in aspen through the epidermis of leaf blades, petioles, and young stems. More recently, Manion & French (10) were unable to consistently differentiate cankers caused by *C. fimbriata* and *Nectria galligena*, and concluded that the actual importance of *C. fimbriata* in causing perennial cankers was still unknown.

Aspen is host to numerous species of *Ceratocystis*, some of which are associated with cankers: *C. fimbriata, C. ambrosia* Bakshi, *C. cana* (Münch) C. Moreau, *C. serpens* (Goid.) C. Moreau (10), *C. tremulo-aurea* Davidson & Hinds (3), and *C. populina* Hinds & Davidson (7). Because *Ceratocystis* spp. are consistently associated with black cankers and the cankers resembling those caused by *N. galligena*, I will refer to such cankers for convenience in this report as "Ceratocystis" cankers.

Symptoms and distribution.—Typical oval or elliptical "target-shaped" cankers suggestive of those caused by *Nectria* spp. are found on small trees. Numerous small cankers are frequently observed on large branches and at forks, but these seldom attain the size of the trunk cankers. The cankers are perennial and enlarge slowly. They originate at some type of trunk wound which has penetrated the cambium, or at the juncture of living or dead branches with the trunk. The disease first appears as a circular, necrotic area on the trunk around the wound or branch junction. A continuous cicatricial zone, which is formed by the host during cambial activity in the spring, walls off the healthy bark from the infected area and forms a callus fold at the margin of the lesion. The mycelium gradually invades the cambium and inner bark during the tree's next dormant season and kills a new zone of tissue. This process is repeated year after year until a canker consisting of successive rings of dead bark is formed. The dead bark usually adheres to the wood for several years; it then begins to slough off, exposing successive rings of dead woody tissue. Vertical growth of young cankers is always greater than horizontal.

Smaller trees are often girdled, but tree circumference growth is usually more rapid than canker enlargement. On larger trees, single cankers seldom kill a tree, but when many are present on a stem they frequently coalese and completely girdle the trunk. The vertical-to-horizontal ratio of cankers increases with age, and cankers become several feet in length. The fungus is most active at the canker top

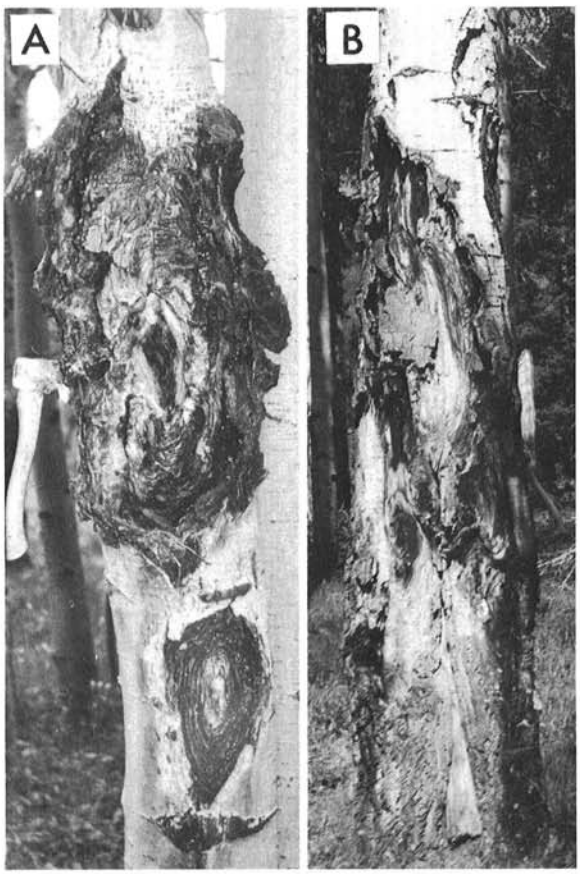


Fig. 1. Ceratocystis cankers on quaking aspen in Colorado. **A)** Nectria-type canker (bottom) ca. 24 years old and a ca. 59-year-old black canker on 83-year-old tree. **B)** Old canker which originated along the sides of a basal wound.

and bottom, but it is sometimes callused off at various places on the canker margins.

Canker morphology is an expression of host reaction and canker age (Fig. 1). Older cankers typically have a central area of dead wood surrounded by a series of bark calluses which may be regular in outline but are usually irregular in shape and ragged in appearance due to the massive callus folds and flaring dead bark. The dead bark tissue adjacent to the canker is black; hence the name "black canker".

Large basal wounds caused by fire or fallen trees frequently become infected by *Ceratocystis*. After many years of infection, the large wounds do not exhibit typical wound callusing or typical canker symptoms. Insects burrow along the canker edges; some pupate in the bark crevices, while others overwinter in cankers of all ages. Nematodes are common in dead bark tissue that is still moist (11). Canker faces become covered with amber-colored sap which exudes from the marginal regions of the canker and with perithecia give the dead wood a brown to black appearance. On older trees infected with wood decay fungi, sporophores are produced in the dead portions of the canker, and wood beneath such cankers is decayed. A brown stain and wet wood extending into the heartwood above and below the canker's limit are always present in the tree trunk regardless of decay.

Perithecia of various species of *Ceratocystis* can usually be found along the perimeter of the cankers. During moist weather, perithecia are produced on the most recently killed bark and wood tissues which have been dead at least 1 year. Although perithecia are frequently consumed by insects and some are disintegrated by sap flow and rain, remnants of perithecial bases often remain for several years.

Ceratocystis canker occurs throughout most of the range of aspen in the western United States. Cankers are common in aspen stands in Arizona, Colorado, New Mexico, Utah, and Wyoming. Specimens have also been collected from California, Idaho, Nevada, Montana, South Dakota, and the Turtle Mountains in North Dakota.

Isolation of canker fungi.—To determine canker-fungi associations, isolations were made from canker tissue and perithecia found on cankers from numerous stands of aspen throughout the Rocky Mountain region during the past 7 years. A 2% Fleischmann's diamalt, 2% Difco-Bacto agar medium was used.

Ceratocystis fimbriata was most consistently isolated, although numerous species of imperfect fungi, bacteria, and yeasts were also isolated. *Nectria* was not isolated from canker tissue, nor were its perithecia observed on cankers. No attempt was made to ascertain the frequency of recovery of the various fungi in culture because perithecia of *Ceratocystis* were almost always found on active cankers. *Ceratocystis* was isolated from necrotic bark tissue more successfully in the spring, whereas perithecia associated with cankers could usually be identified at any time of the year.

Perithecia of *C. alba* DeVay, Davidson, & Moller, *C. crassivaginata* Griffin, *C. populina*, and *C. tremulo-aurea* were often found coexisting with those of *C. fimbriata*; however, it was not unusual to find only one of these other species on a canker. Isolations from necrotic canker tissue confirmed multiple species infection.

Stem inoculations.—Because *C. fimbriata* was consistently associated with cankers, healthy aspen were inoculated with the following *Ceratocystis* isolates: *C. fimbriata* from Colorado aspen canker, Minnesota aspen canker, dying sycamore (*Platanus occidentalis* L.) in Mississippi, sweet potato (*Ipomoea batatas* [L.] Lam.), and two of *C. tremulo-aurea* from cankers on Colorado aspen. Inoculum consisted of pure cultures containing mycelium, conidia, and perithecia grown on 2% Fleischmann's diamalt agar and on sterile rye. Controls consisted of diamalt agar and sterile rye.

Trees in four different aspen clones from 39 to 83 years old near Redfeather Lakes, Colo., were inoculated in June, July, and September 1963, and May 1964. Inoculum was applied to slits (ca. 2 X 3 cm) cut into the inner bark and the cambium, and

TABLE 1. Cankers on Colorado aspen resulting from stem inoculations with *Ceratocystis* spp. in 1963-1964

Isolate	No.	Active cankers[a], 1968
	Field inoculations	
C. fimbriata from Colorado aspen	100	100
C. fimbriata from Minnesota aspen	48	32
C. fimbriata from sycamore	48	9
C. fimbriata from sweet potato	50	0
C. tremulo-aurea from Colorado aspen	102	0
Control	60	14
	Greenhouse inoculations	
C. fimbriata from Colorado aspen	16	All trees girdled
C. tremulo-aurea from Colorado aspen	19	No cankers produced
Control	9	Inoculations healed

[a] Measurable canker enlargement each year following inoculation was the criterion used to determine canker activity.

10-mm holes bored into the xylem. Inoculations were made at ca. 2 and 5 ft aboveground and in the four cardinal directions, when possible, on a tree trunk. A series of inoculations were also made on the same side of some trees. The wounds were covered with moist or dry sterile cotton, and completely covered with electrician's plastic tape. Bore holes were plugged with sterile corks which were then taped in place. Inoculum was also placed on healthy bark, dead branch stubs, and at living branch axils.

Dormant 1- and 2-year-old aspen sprouts of two different clones were transplanted from the forest into pots in early spring 1963 and placed in a greenhouse. Small slits in current and 1-year-old growth of these sprouts were inoculated with agar cultures in May 1964, 8 weeks after leaf initiation.

A total of 149 trees in the field and 30 sprouts in the greenhouse was inoculated. The numbers of inoculations and cankers resulting from the various isolates are given in Table 1. All inner bark slit, cambium slit, and bore hole inoculations with the Colorado isolate of *C. fimbriata* resulted in typical young cankers at the end of five growing seasons (Fig. 2). Cardinal direction, height, season of year, and aspen clone had no effect on infection. Ten inoculations on healthy bark, five on dead branch stubs flush with the bole, and two at live axils were negative. For the first 2 years, annual growth of the Colorado-isolate cankers averaged 20 X 73 mm; vertical growth was always greater than horizontal. Enlargement abated during the following 4 years, and averaged 19 X 46 mm. After 6 years, cankers ranged from 20 to 130 mm horizontally and from 130 to 350 mm vertically.

Average enlargement of the 32 active Minnesota-isolate cankers has been 13 X 55 mm/year; the remaining 16 inoculations are being callused over, and it is assumed that the fungus is inactive. Results of the trunk inoculations indicated that the sycamore isolate was less pathogenic. The fungus apparently failed to infect the host in 17 of 48 inoculations, and was active in only nine inoculation sites at the end of 5 years, with a yearly average development of 9 X 19 mm. The sweet potato and *C. tremulo-aurea* isolates caused a slight necrosis around the inoculation point, but these infections began callusing over the 2nd year. All were considered to be inactive, and most had been completely callused over at the end of 5 years. The relative capability of the four isolates to infect aspen is shown in Fig. 3. When inoculation points were close together, the individual cankers coalesced (Fig. 3-C). This phenomenon is common in nature.

Cankers were cut from the trees in September 1968, and reisolation of the fungus was attempted. Perithecia of *C. fimbriata* were found on 14 of 23 cankers produced by the Colorado isolate, and the fungus was recovered from the tissue of 12 cankers. Perithecia of *C. alba* and *C. crassivaginata* also were found on two of the cankers. *Ceratocystis alba, C. crassivaginata, C. populina,* and *C. tremulo-aurea* were isolated from canker tissue around five of the cankers. Twelve of the cankers produced by the Minnesota isolate were cut; perithecia of *C. fimbriata* were found on five, and the fungus was isolated from three. *Ceratocystis tremulo-aurea* was also isolated from one canker. Perithecia of *C. fimbriata* were found on two of the eight sycamore-isolate cankers cut, but only *C. crassivaginata* was isolated from tissue from three of the cankers.

Forty-six of the 60 control inoculations had completely healed over by 1968. Fourteen showed typical canker symptoms, and perithecia of *C. fimbriata* were found on six of the cankers. Canker tissue isolations were not made from the infected controls.

Small cankers were formed by the Colorado isolate within 21 days on plants in the greenhouse. The fungus girdled the current season's growth within 6 weeks; and 1-year-old growth, in 4 months. The fungus produced perithecia on most cankers, and was reisolated from tissue from all cankers at the time of stem girdling. *Ceratocystis tremulo-aurea* produced no symptoms, and the inoculation wounds, like those of the controls, were completely healed within 1 year.

Leaf inoculations.—To determine the possibility of canker initiation via leaf infection, leaves and twigs were inoculated with a spore suspension of ascospores and conidia obtained by washing plate cultures with sterile water. Inoculum was applied by a hand sprayer to individual twigs with one to eight 4-week-old leaves on greenhouse plants in June 1964 and 1968. The plants were then placed in a 100% humidity cabinet with temperatures of 16-26 C for 72 hr, after which they were removed and retained in the greenhouse. Leaves and twigs sprayed with sterile water were used as controls in all tests. On 1 July 1968, when the leaves on 14-year-old aspen in the forest were about 4 weeks old, they were inoculated with a spore suspension (one isolate from canker tissue and one obtained from an insect), and sealed in plastic bags for 74 hr. Mature leaves on greenhouse plants were inoculated in October 1968 by spraying a spore suspension onto leaves which had been rubbed with Carborundum powder. Controls consisted of wounded and unwounded leaves sprayed with sterile water. All plants were sealed in plastic bags for 72 hr. Spore germination was checked after each inoculation experiment by spraying a spore suspension onto water agar in petri dishes and observing the dishes 24 hr later.

A total of 228 leaves on eight trees was inoculated with the Colorado *C. fimbriata* isolate; and 280 leaves on eight trees, with *C. tremulo-aurea* in 1964. Infections consisting of small black spots became evident after 3 days on 79 leaves and on an additional 15 leaf petioles inoculated with the *C. fimbriata* isolate. Leaf blade infections progressed to petiole infections, and premature leaf fall was common. Petiole infections reached the twigs in only four cases, and by September, after leaf fall, these necrotic twig lesions were up to 10 mm long. The necrotic spots were confined to the twigs; they did not reach the main stem, and remained the same during the winter. Necrosis did not increase with new growth during the following April and May. Unfortunately, greenhouse temperatures rose to lethal levels in June and the trees died. Leaf blade infections only occurred on 33 leaves inoculated with *C. tremulo-aurea* spores. Petioles and twigs were not subsequently infected. The control, consisting of 100 leaves on three trees, remained normal throughout the growing season.

There was no apparent difference in the infection capability of two *C. fimbriata* isolates used for leaf inoculations in June 1968. A total of 448 leaves on 72 actively growing twigs of 10 trees was inoculated. A control consisted of 432 leaves on 79 twigs of eight trees. Leaf infections appeared after 3 days. At 10 days there were 47 leaf infections, including three petiole infections which resulted in the terminal death of three twigs. After 20 days, there were an additional 10 blade infections, 27 petiole infections, and death of six terminal growths. Prior to leaf fall in October, a total of 24 twigs was killed back to the main stem, and five stem infections had resulted in death of stem tops from 5-31 cm in length. New shoot growth started below all these necrotic areas on the main stems the following spring, and the infections appeared to be arrested. The controls remained normal throughout the growing season.

A total of 650 leaves on 108 shoots of new growth on 11 branches was inoculated in the field with a spore suspension of the Colorado isolate. Sterile water was used for a control on 350 leaves on the new growth of seven branches. Hail damage to many leaves 1 week after inoculation and some leaf infection by *Marssonia* spp. precluded an accurate count of leaf infections. Petioles of 23 leaves became necrotic prior to leaf fall in September, but so did petioles of 26 control leaves. Isolations from necrotic petioles of 12 inoculated and 12 control leaves failed to yield *C. fimbriata*. Only one twig infection resulted from the inoculations, and this resulted in death of the 1968 growth (7.5 cm) back to the main branch. The main branch did not become infected, and the dead twig succumbed to abscission the following year.

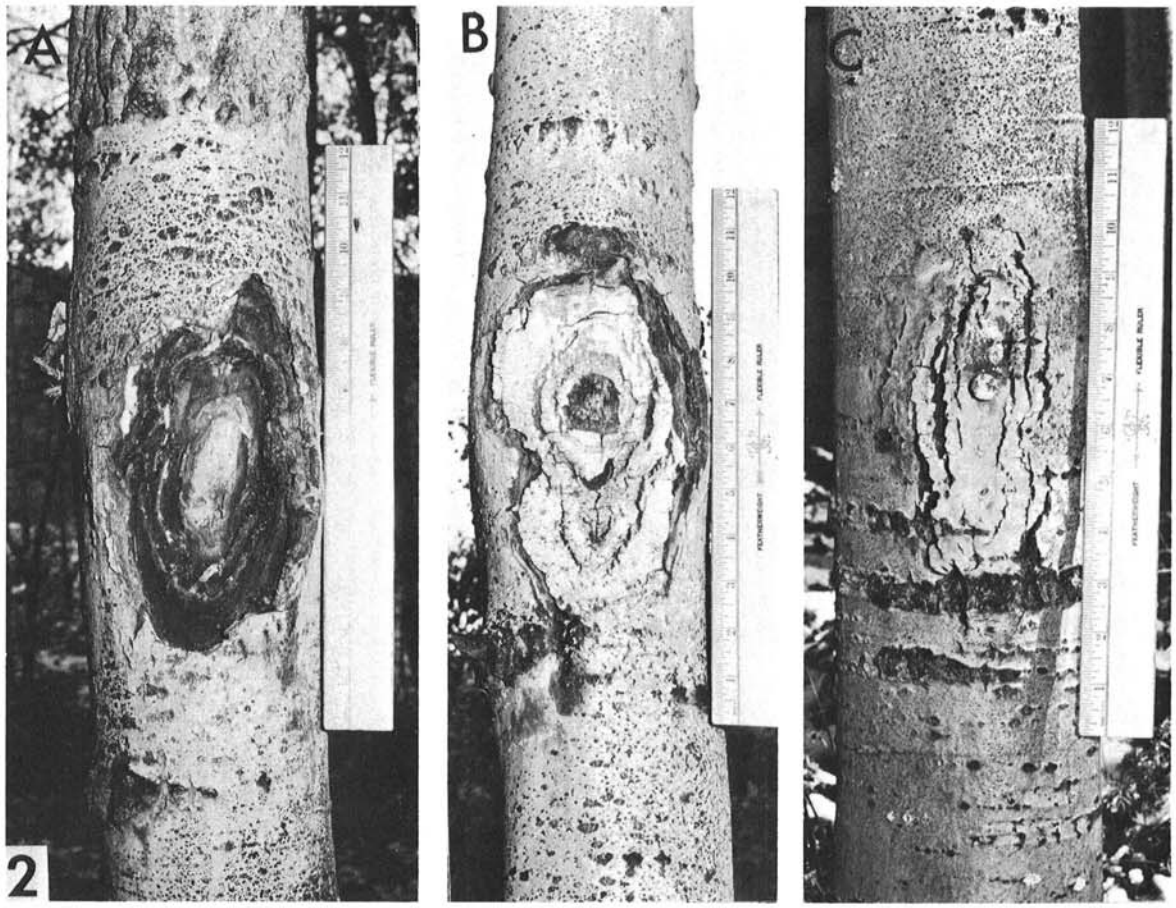
All 55 mature leaves rubbed with Carborundum powder exhibited tiny black spots ca. 1 mm in diam 4 days after inoculation. These small spots enlarged and coalesced on the 36 leaves treated with the spore suspension. One week later, the leaves began to desiccate and fall prematurely. There was no twig or stem infection. By the middle of November, the 17 leaves sprayed only with sterile water began normal leaf abscission.

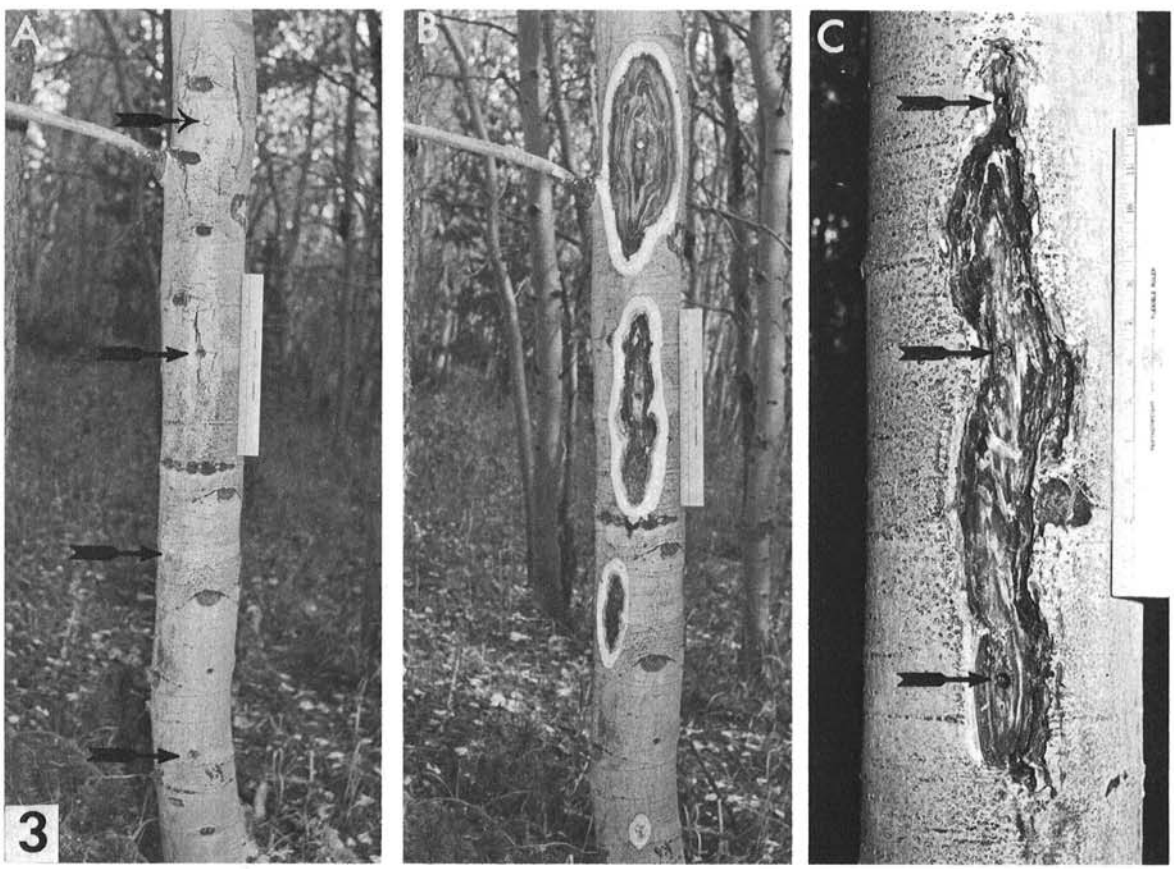
Canker initiation.—Ceratocystis cankers were examined in 12 areas in 1968 to determine the relationship of leaf and branch infections to wound infection courts (Fig. 4). All points of origin were inspected for branch stubs. Observations were confined to tree trunks from groundline to 10 ft in height (1,251 cankers); branch cankers were too high for careful inspection.

Five areas (602 cankers) in campgrounds and seven areas distant from campgrounds (649 cankers) were checked. Infection started at wounds in 97% of the cankers in campgrounds. In the areas distant from campgrounds, 87% of the cankers were associated with wounds. For all areas, 8% of the cankers were initiated at branch and trunk junctions; that is, infection may have entered the trunk through leaf and branch infection or at branch axil wounds, and 92% became established at trunk wounds.

Canker epidemiology.—Canker epidemiology was studied in a severely infected stand on the San Juan National Forest, Colo. Ten trees with 53 cankers were felled, and cuts made through points of initial

Fig. 2-3. 2) Five-year-old *Ceratocystis fimbriata* cankers produced on aspen as a result of different types of inoculation wounds: **A)** Wound to cambium. **B)** Wound to inner bark. **C)** Auger bore hole into the xylem. **3)** Five-year-old cankers on aspen formed by various *C. fimbriata* isolates. Arrows indicate point of inoculation. **A)** Source of isolates, from top to bottom: Colorado aspen, Minnesota aspen, sycamore, and sweet potato. **B)** Bark removed to show canker limits. **C)** Source of isolates, from top to bottom: sycamore, Colorado aspen, and Minnesota aspen.


A
B
C
2


A
B
C
3

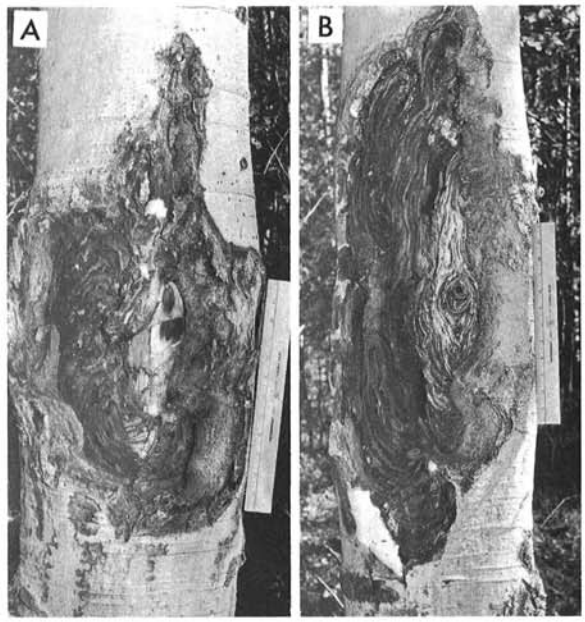


Fig. 4. A) Ceratocystis canker of aspen initiated at 25-year-old trunk wound. **B)** Thirty-eight-year-old canker that originated at a branch axil.

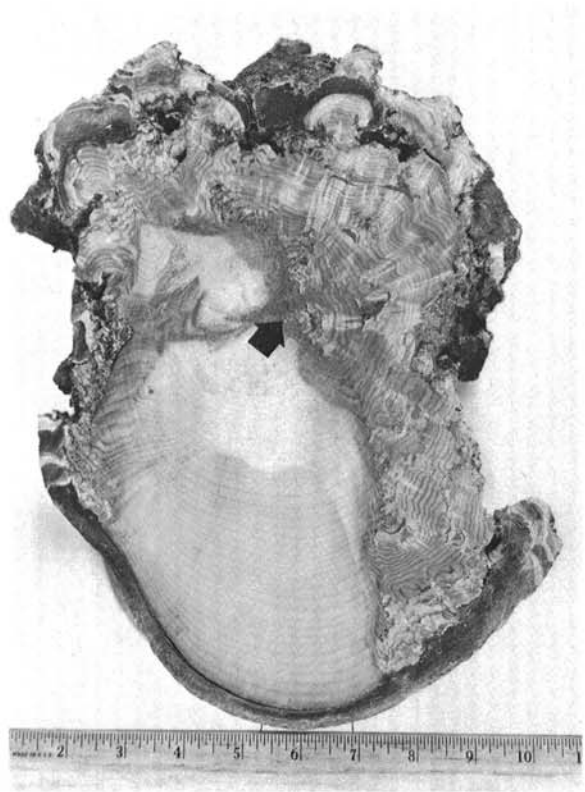


Fig. 5. Cross section through 47-year-old canker of aspen at point of origin (arrow). Canker was at 30 ft on an 85-year-old tree. The canker on the left side was 39 years old at infection point below plane of cut.

infection (Fig. 5) to determine the relationship between canker age and frequency of infection.

Tree ages varied from 73 to 103 years, and canker ages from 9 to 78 years. Figure 6 shows a histogram of the number of cankers versus age of infection (10-year classes), and a theoretical normal density curve for the population of the sample. A chi-square test indicated that canker frequency closely fitted a normal density curve. Thus, infection was a random event over the years rather than an epidemic. There was no apparent relationship between tree age and canker age.

DISCUSSION.–Inoculations in the greenhouse and subsequent reisolations in 1964 substantiate Zalasky's (18) work on the pathogenicity of *C. fimbriata* on young stems. Its ability to cause trunk cankers on older trees in the field was also demonstrated. Zalasky (18) gave an excellent histopathological account of stem infection and host-parasite interaction; consequently, these were not studied in detail. He found that three callus folds had formed around the point of inoculation 5 months after inoculation on 6- and 12-month-old stems. Development of cankers in older stems in Colorado was somewhat different. Bark tissue surrounding the inoculation became necrotic within 3 months after inoculation. The necrotic area increased during the winter months. Cambial activity during the spring and summer months callused off this infected tissue. These necrotic areas then became flat, sunken areas surrounded by the new callus tissue. Necrotic bark areas in the spring corresponded closely to the measurable canker limits early the following fall, but during the following winter the necrotic area again increased. The dead bark remained over many of the cankers, giving the appearance of a healing wound. After 6 years, tree circumference growth had caused the dead bark covering the canker to crack; on some cankers the bark sloughed off, exposing the dead wood and clearly showing annual canker growth.

Cankers formed on trees inoculated with sterile materials can probably be explained by insect transmission of the fungus. An insect vector

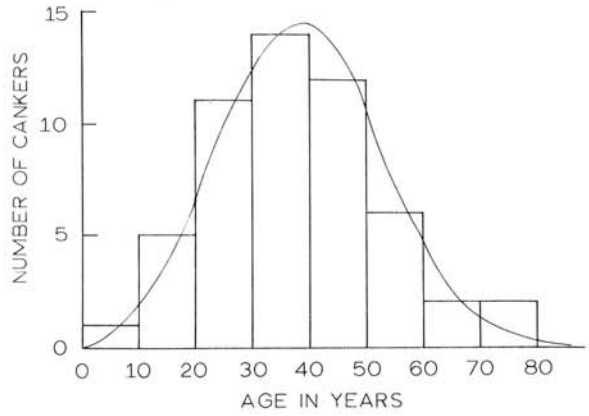


Fig. 6. Age distribution of 53 cankers on aspen by 10-year age classes. Histogram (plotted at class mean) and theoretical normal density curve.

relationship (6) was unknown at the time of the first control inoculations, and removal of the bandages in September exposed the wounds which attracted insects. These wounds became infected by various species of *Ceratocystis*. Bandages were left on subsequent inoculations for a period of 9 months, and controls did not become infected.

The fungus is reported to be difficult to isolate from canker tissue (10, 17). Isolations during the spring when bark necrosis is obvious are more successful than those made in summer when callus formation is complete. Because of this, the reisolation attempts in September did not all yield the fungus. If recovery of the fungus from trees in the field had been attempted in the spring after inoculation, however, the small cankers would have been destroyed and future observations eliminated. Canker infection by other species of *Ceratocystis* is probably also explained by insect contamination and age of canker when the reisolations were made.

Ceratocystis fimbriata is found on many hosts. Isolates from different geographic areas differ in colony type, pathogenicity, growth rate, and conidal states (15). They are highly variable in their virulence and interaction with different hosts, as has been pointed out by DeVay et al. (4) and Pontis (14), and further demonstrated in this work. Manion & French (10), using *N. galligena* and *C. fimbriata* in inoculation tests, found that cankers produced by *N. galligena* started more slowly but usually continued their development in subsequent years, whereas *C. fimbriata*, after rapid development in the 1st year, apparently ceased after 2 years. The Minnesota-aspen isolate used in this study was not as pathogenic to Colorado aspen as the Colorado isolate, and may explain why Manion & French (10) did not get comparable canker growth in their inoculation studies, and why they questioned the ability of the organism to cause perennial cankers.

The 1964 leaf inoculations were inconclusive, but canker initiation via leaf infection was later shown by Zalasky (18). When repeated in 1968, the experiment produced results similar to Zalasky's. He readily obtained leaf infection on 1-week-old leaves, whereas 4-week-old and mature leaves used in this experiment were not as readily infected. This difference in leaf age could significantly influence the rapidity with which the mycelium advances through petiole, twig, and stem.

Leaf susceptibility to infection may decrease with leaf age. Results obtained in the greenhouse on older leaves on 1- and 3-year-old stems showed that leaf infection progressed into twigs in only 24 of 72 instances, and only five of these progressed to the main stem (only one twig dieback occurred in the field). These leaf and petiole infections resulted in stem girdling and dieback, not perennial cankers. Zalasky's conclusion that the fungus progresses from leaves and petioles to twigs and to the stem is certainly valid for young cuttings and stems in the greenhouse. However, the assumption that infection progresses in a similar manner into branches and then into the main tree trunk, and subsequently causes trunk cankers in the field was not corroborated. But again, age and stage of development of all tissues may be significant. Petiole and leaf blade infections appear to be important as points of entry, but only in twig and small stem infections which kill the terminal growth. This type of damage has not been attributed to *C. fimbriata* in the field.

Although the presence of a dead branch stub in the center of a canker may indicate that the fungus invaded the unwounded stem through a petiole infection as postulated by Zalasky (18), the results of the canker initiation study suggest that this type of infection is limited. Branch axil damage occurs in many ways, and is common in nature. The subsequent infection of such a wound and the girdling of the affected branch at the trunk junction could erroneously indicate that the trunk infection did enter through the branch, particularly for old branch stubs with surrounding infections. Results of the stem inoculations in trunk wounds, leaf inoculations, and the number of cankers found to originate at wounds leave little doubt that trunk wounds are the primary courts of infection.

LITERATURE CITED

1. BAKER, F. S. 1925. Aspen in the central Rocky Mountain region. U.S. Dep. Agr. Bull. No. 1291. 46 p.
2. BOYCE, J. S. 1948. Forest pathology. McGraw-Hill Book Co., New York. 550 p.
3. DAVIDSON, R. W., T. E. HINDS, & E. R. TOOLE. 1964. Two new species of Ceratocystis from hardwoods. Mycologia 56:793-798.
4. DEVAY, J. E., F. L. LUKEZIC, H. ENGLISH, E. E. TRUJILLO, & W. J. MOLLER. 1968. Ceratocystis canker of deciduous fruit trees. Phytopathology 58:949-954.
5. HINDS, T. E. 1964. Distribution of aspen cankers in Colorado. Plant Dis. Rep. 48:610-614.
6. HINDS, T. E. 1972. Insect transmission of Ceratocystis species associated with aspen cankers. Phytopathology 62: 221-225.
7. HINDS, T. E., & R. W. DAVIDSON. 1967. A new species of Ceratocystis on aspen. Mycologia 59:1102-1106.
8. LAUT, J. G., & V. HILDAHL. 1965. Annual report of the forest insect and disease survey 1964. Can. Dep. Forest. p. 90-95.
9. LONG, W. H. 1918. An undescribed canker of poplars and willows caused by Cytospora chrysosperma. J. Agr. Res. 13:331-343.
10. MANION, P. D., & D. W. FRENCH. 1967. Nectria galligena and Ceratocystis fimbriata cankers of aspen in Minnesota. Forest Sci. 13:23-28.
11. MASSEY, C. L., & T. E. HINDS. 1970. Nematodes from aspen cankers in Colorado and New Mexico. Can. J. Zool. 48:97-108.
12. MEINECKE, E. P. 1929. Quaking aspen. A study in applied forest pathology. U.S. Dep. Agr. Tech. Bull. 155. 33 p.
13. OUELLETTE, G. B. 1965. Annual report of the forest insect and disease survey 1964. Can. Dep. Forest. p. 47-49.
14. PONTIS, R. E. 1951. A canker disease of the coffee tree in Colombia and Venezuela. Phytopathology 41:178-184.

15. WEBSTER, R. K. 1967. A morphological and biological concept of the species Ceratocystis fimbriata. Can. J. Bot. 45:1457-1468.
16. WOOD, F. A. 1964. Ceratocystis canker of aspen in Pennsylvania. Plant Dis. Rep. 48:441.
17. WOOD, F. A., & D. W. FRENCH. 1963. Ceratocystis fimbriata, the cause of a stem canker of quaking aspen. Forest Sci. 9:232-235.
18. ZALASKY, H. 1965. Process of Ceratocystis fimbriata infection in aspen. Can. J. Bot. 43:1157-1162.