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A Severe Epidemic of *Marssonina* Leaf Blight on Quaking Aspen in Northern Utah

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ABSTRACT

The extent of Marssonina leaf blight (Marssonina populi) on quaking aspen (Populus tremuloides) was observed in northern Utah and adjacent States in 1981 and 1982. Area of the epidemic and symptoms of the disease are described. On 1,000 acres (405 ha) in northern Utah, infection levels were 6 percent slight, 12 percent light, 32 percent moderate, 16 percent moderately heavy, and 34 percent heavy.

KEYWORDS: aspen leaf blight, *Populus tremuloides*, epidemiology, symptoms

Marssonina populi (Lib.) Magn. induces a leaf and twig blight of quaking aspen (*Populus tremuloides* Michx.) that periodically becomes epidemic over extensive areas in the Intermountain Region of the Western United States (Meinecke 1929; Mielke 1957). *Marssonina* species are found on several *Populus* species in the United States and also in Europe, Japan, Canada, and New Zealand (Spiers 1978b). *M. populi*, the imperfect state of *Drepanopeziza populorum*, varies widely in morphology and host specificity. However, Thompson (1937) and Boyer (1961) did not find sufficient variability for a taxonomic separation into different species.

Early symptoms of the disease are reported as tan to brown leaf spots that later turn dark brown to black with a yellowish to golden margin. The spots range from small flecks to large discrete circular to lens-shaped lesions most prominent on the upper leaf surface. Lesions also appear on leaf petioles and young stems of current shoots (Mielke 1957; Palmer and others 1980).

With heavy infections, spots can coalesce to form large irregular-shaped necrotic blotches that may extend over most of the leaf surface. Toward the center of spots, a gelatinous mass of hyaline, one-celled spherical and two-celled ovate to pyriform conidia form in subcuticular acervuli (Hepting 1971; Palmer and others 1980). During spring, conidia and possibly ascospores from fallen leaves and shoots infected the previous season can initiate new infections. During wet periods in late May to early June spores are carried by wind and rain to developing leaves and shoots. Later in the season secondary infections may occur during favorable moisture periods intensifying an epidemic. Richard G. Krebill (unpublished) could find no ascospores of *M. populi* on overwintered leaves infected the previous season in northern Utah. He found that previous season's acervuli could produce new conidia from infected leaves and twigs that remained on the tree. In France, Pinon and Poissonnier (1975) found higher *M. brunnea* infections of cultivated *Populus* when good rains were followed by no rainfall for about a week and the average temperature exceeded 52°F (11°C).

Extent of damage from the disease is not well established. Leaves of infected trees can be smaller than normal and turn prematurely mottled-yellow to brown in July and August. With severe epidemics, premature defoliation and branch dieback can occur. Tree mortality and reduced increment growth also occur with successive annual epidemics (Mielke 1957).

The clonal nature of aspen accentuates visual evidence of variability in susceptibility to *Marssonina* leaf blight. Virtually immune to highly susceptible clones are easily identified by green to bronze color gradations according to severity of leaf blighting. With annual color photographs Mielke (1957) found year-to-year constancy in color patterns among diseased clones. Spiers (1978a) also showed in laboratory tests that poplar clones varied in resistance as related to the level of *Marssonina* inoculum applied.

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MATERIALS AND METHODS

In the summer of 1981, observations were made of development of an epidemic of aspen leaf blight in northern Utah and adjacent areas in bordering States. Survey from roads and discussions with knowledgeable people were used to determine the extent of aspen leaf blight in surrounding regions. We studied the severity of the epidemic on about 1,000 acres (405 ha) in more detail in the Tony Grove Lake area of Logan Canyon in the Wasatch-Cache National Forest of northern Utah. The boundaries of aspen clones in this area were drawn on 1:24000 U.S. Geological Survey topographical maps. The intensity of aspen leaf blight in each clone was rated on a 1 to 5 scale (table 1), visually judged by the degree of leaf bronzing. Clones placed in each disease category were checked for percentage of leaves affected. The area in each disease category was estimated using a dot grid on the topographic map.

Table 1.—Categories for rating intensity of aspen leaf blight disease

Disease class	Disease	Percent of leaves infected
1	None or slight	< 5
2	Light	5 to 25
3	Moderate	26 to 50
4	Moderately heavy	51 to 70
5	Heavy	> 70

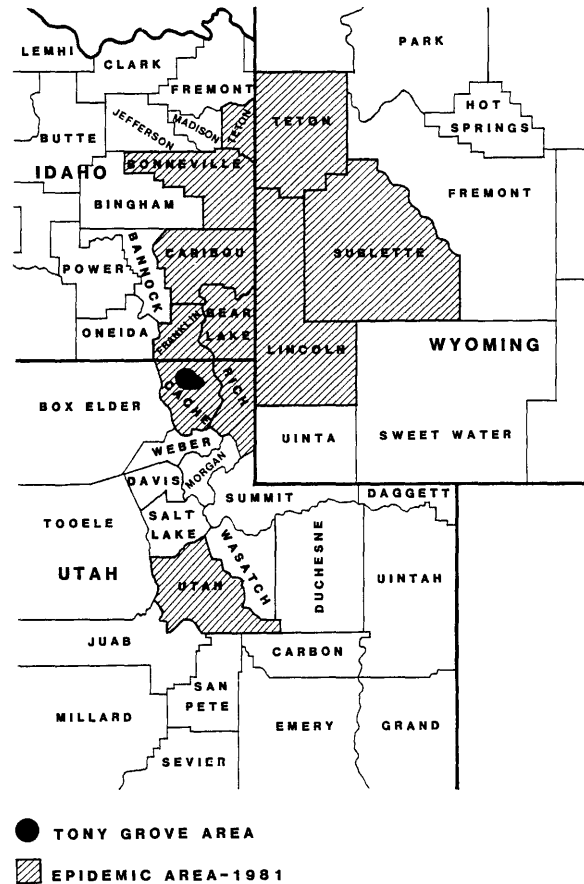


Figure 1.—The general area of severe *Marssonina* leaf blight epidemic during summer of 1981.

RESULTS AND DISCUSSION

The severe 1981 aspen leaf blight epidemic extended over large areas of northeastern Utah, southeastern Idaho, and western Wyoming (fig. 1). Leaf blight was noted in the Utah counties of Cache (fig. 2) and Rich and as far south as Utah County; in the Idaho counties of Franklin, Caribou, Bear Lake, and Bonneville; and in the Wyoming counties of Teton, Lincoln, and Sublette.

In the Tony Grove Lake area of Cache County, Utah, where we studied the 1981 epidemic in detail, it was the third year of increasing intensity of aspen leaf blight. Leaves of many infected clones were smaller than normal and entire clones had turned brown by early July. Clones that had early heavy infection were virtually defoliated by late August, and in September some of

these clones had trees with a new growth of unusually large leaves at the branch tips (fig. 3). An inspection of the more heavily infected clones suggested an increase in the number of recently dead trees.

Of the 1,000 acres (405 ha) of aspen rated for disease, only 6 percent had little or no leaf blight, 12 percent was lightly infected, 32 percent moderately, 16 percent moderately heavy, and 34 percent heavily infected. Aspen on about one-third of the area surveyed was almost completely defoliated. Although objective comparisons could not be made with previous reports of aspen leaf blight in the Western United States (Meinecke 1929; Mielke 1957), periodic outbreaks have been observed by pathologists and others in the middle Rocky Mountains since the early 1900's.



Figure 2.—View of aspen clones with various degrees of *Marssonina* leaf blight in the Tony Grove Lake area of northern Utah.



Figure 3.—Shoot regrowth on aspen tree defoliated earlier in the summer by leaf blight.

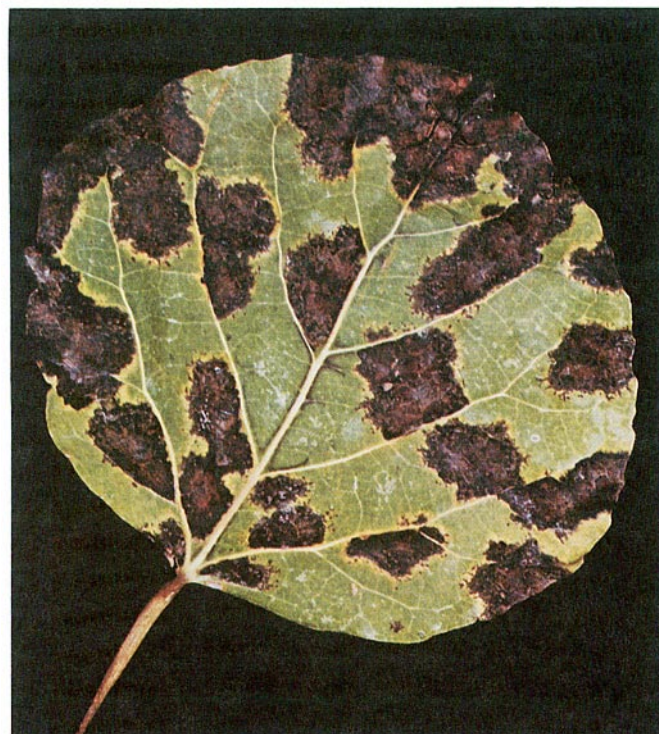
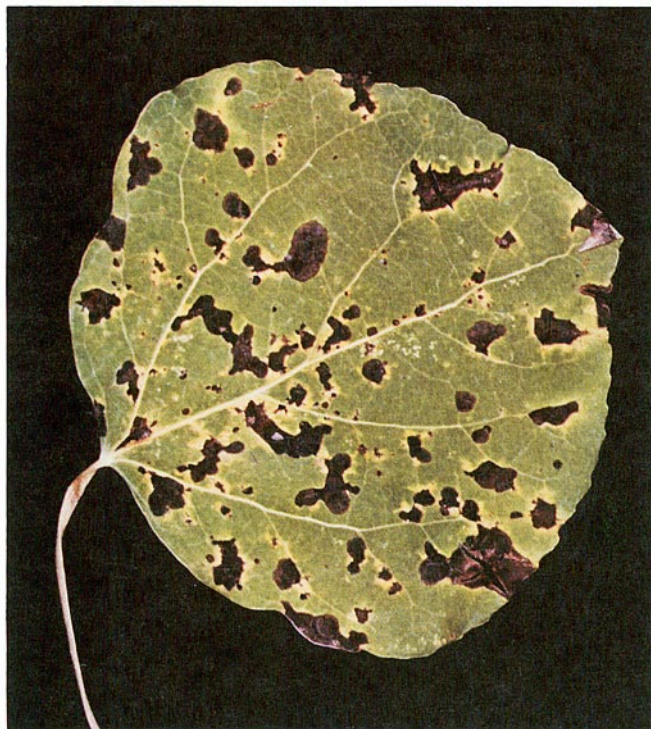
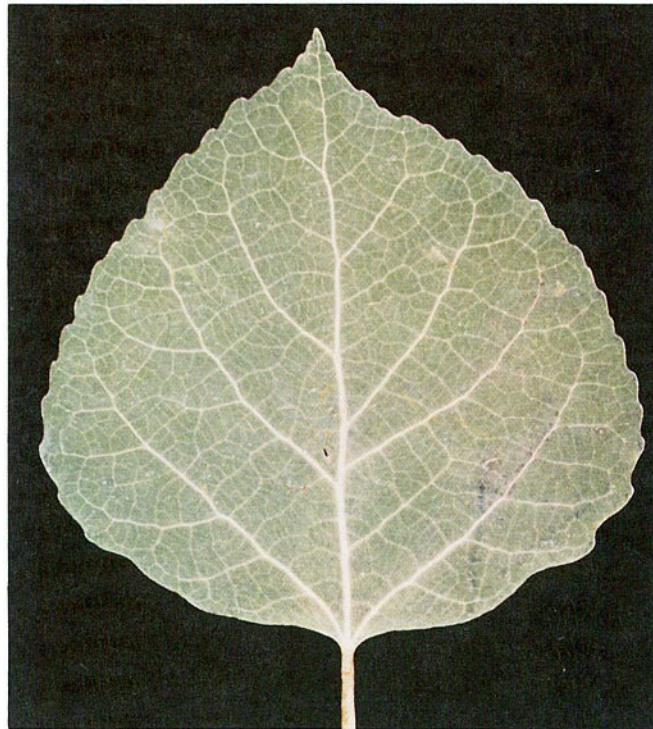
**A****B****C****D**

Figure 4.—Lesions on leaves of *Marssonina* blighted quaking aspen showing, A, heavy; B, moderate; C, light; D, none to slight infection patterns.

In our study, leaf blight symptoms varied considerably, and this variation appeared to reflect clonal differences in response to the pathogen. Enlargement of necrotic spots on the leaves of some clones was extensive and spots coalesced to cover most of the leaf blade (fig. 4A). Clones with this reaction were those most severely defoliated. In other clones, infection zones on leaves were more limited and less defoliation occurred

(fig. 4B). In some clones, leaf blight spots were markedly smaller and more irregular in size and shape (fig. 4C), possibly because of greater host resistance to the fungus. Few clones in the surveyed area were highly resistant or immune (fig. 4D); these had an occasional leaf with a possible *Marssonina* infection spot. Although it is not unusual to find variation in disease symptom expression among individual plants because of genetic

differences in susceptibility, especially in wild plant populations, these differences become more evident because of the clonal nature of aspen. Other factors such as site, time of infection, leaf maturity at infection, and variation in fungus pathogenicity could influence symptom expression. Repeated occurrence of the same symptoms in the same clone during subsequent years would more reliably establish symptom pattern as an expression of susceptibility.

A brief examination of the *Marssonina* leaf blight survey area 1 year later revealed several residual effects of the 1981 epidemic. In August 1982 leaf blight was at a typical endemic level with scattered light infection. However, clones heavily infected during the 1981 epidemic had higher rates of infection in 1982 than clones lightly infected in 1981. The more severely affected clones during 1981 tended to have fewer and more chlorotic leaves in 1982, especially those clones that appeared decadent. The most severely defoliated as well as the older less vigorous clones showed a dramatic amount of bud and branch death (fig. 5). The disease effect in this case appeared to be one of bud rather than stem mortality. The few surviving buds (fig. 6) produced vigorous shoots with unusually large leaves. Branches and twigs with no live buds remained alive during summer but were dying by fall. A likely result of the leaf blight epidemic may also be reduced stem increment growth for several years.



Figure 5.—Extensive branch and twig death in quaking aspen 1 year after the severe defoliation during the epidemic of 1981.

No fungicides are registered specifically for control of *Marssonina* on poplars in the United States (Palmer and others 1980). Spiers (1978b) in New Zealand tested 18 fungicides for their effectiveness and persistence in controlling *Marssonina* leaf blight and found 10 to give effective control ranging from 7 to 21 days. In Canada, Carlson (1972) tested seven fungicides for control of *Marssonina* and *Septoria* leaf spots on *Populus* and found two fungicides to be the most effective. The necessity for repeated spraying to achieve control throughout the season suggests that it would not be practical or economical for forest conditions but might be suitable for ornamental trees. Selection for highly resistant clones appears to be the best control alternative, especially for ornamental and revegetation purposes, because most aspen are highly susceptible to *Marssonina* leaf blight in epidemic years. Destroying leaf blight inoculum by gathering and burning leaves in the fall or tilling them into the soil and also removing clinging leaves and infected twigs should reduce infection the following spring in ornamental aspen around the home and in aspen in nurseries.

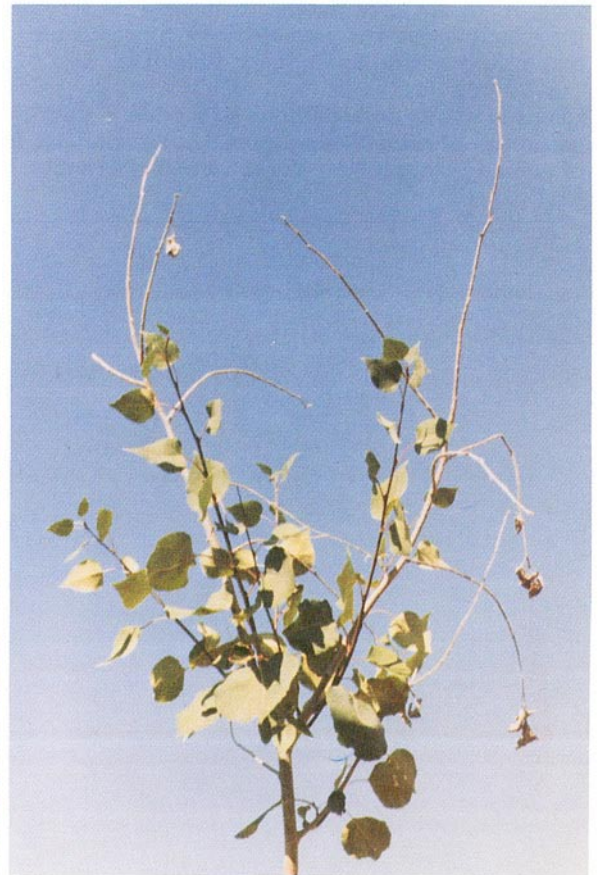


Figure 6.—Vigorous shoot growth and unusually large leaves from surviving buds 1 year after the 1981 severe defoliation.

REFERENCES

- Boyer, M. G. Variability and hyphal anastomoses in host-specific forms of *Marssonina populi* (Lib.) Magn. Can. J. Bot. 39: 1409-1427; 1961.
- Carlson, L. W. Fungicidal control of poplar leaf spots in Alberta and Saskatchewan. Plant Dis. Surv. 52(3): 99-101; 1972.
- Hepting, G. H. Diseases of forest and shade trees of the United States. Agriculture Handbook 386. Washington, DC: U.S. Department of Agriculture, Forest Service; 1971. 658 p.
- Meinecke, E. P. Quaking aspen: a study in applied forest pathology. Technical Bulletin No. 155. Washington, DC: U.S. Department of Agriculture, Forest Service; 1929. 33 p.
- Mielke, J. L. Aspen leaf blight in the Intermountain region. Research Note 42. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station; 1957. 5 p.
- Palmer, M. A.; Ostry, M. E.; Schipper, A. L., Jr. How to identify and control *Marssonina* leaf spot of poplars. How-to Leaflet. St. Paul, MN: U.S. Department of Agriculture, Forest Service, North Central Forest Experiment Station; 1980.
- Pinon, J.; Poissonnier, M. Epidemiological study of *Marssonina brunnea* (Ell. & Ev.) P. Magn. Eur. J. For. Pathol. 5(2): 97-111; 1975.
- Spiers, A. G. An agar leaf-disc technique for screening poplars for resistance to *Marssonina*. Plant Dis. Rep. 62(2): 144-147; 1978a.
- Spiers, A. G. An agar leaf-disc technique for screening the effectiveness and persistence of fungicides for control of *Marssonina* on *Populus*. Plant Dis. Rep. 62(2): 148-151; 1978b.
- Thompson, G. E. Contributions to the life history and pathology of some *Septoria* and *Marssonina* leaf fungi of poplars. Ithaca, New York: Cornell University; 1937. Ph.D. thesis.

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