

Unusual Epidemic of Tar Spot on Norway Maple in Upstate New York

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ABSTRACT

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Tar spot, caused by *Rhytisma* spp., has caused serious defoliation of Norway maples (*Acer platanoides*) in several upstate New York communities in recent years. Green- and purple-leaved varieties of all sizes and ages have been attacked; many have been completely defoliated by mid-August. Although *R. acerinum* is the only species of *Rhytisma* reported on Norway maple, the pathogen described herein differs from *R. acerinum* (common on red and silver maples in New York) in size and morphology of stromata, production of asexual spores, and growth habit in culture. The disease was controlled with benomyl, mancozeb, or triadimefon sprayed to runoff at budbreak and twice thereafter at 15-day intervals.

In August 1983, homeowners and tree care professionals in and around Utica (Oneida County), NY, expressed concern to Cooperative Extension Service specialists about a disease causing large (2-3 cm diameter) black spots on upper surfaces of Norway maple (*Acer platanoides* L.) leaves. Some premature defoliation was also noted. In 1984, disease incidence and severity in previously affected sites increased such that many Norway maples in the town were completely defoliated by mid-August, and most others suffered enough leaf necrosis to substantially reduce their value as ornamental specimens for the remainder of the growing season.

Microscopic examination of leaves infected in 1983 and collected in May and June 1984 indicated that the spots were stromata characteristic of the genus *Rhytisma* (Ascomycetidae). *Rhytisma acerinum* (Pers.) Fr. was the only member of the genus reported to occur on Norway maple (1,2,7), but there appeared to be significant differences between the pathogen causing the current epidemic and *R. acerinum* as we knew it on red (*A. rubrum* L.) and silver (*A. saccharinum* L.) maples in New York. Thus, we began an investigation to characterize the pathogen on Norway maple and to determine its relationship to other *Rhytisma* spp. on *Acer* in North America.

MATERIALS AND METHODS

Anatomy/biology. Diseased Norway maple leaves were collected from a

hedgerow in New Hartford, NY, at biweekly intervals from 1 May to 5 July 1985 and at monthly intervals thereafter until normal leaf drop in October. Diseased red maple leaves for comparison were collected from a woodlot in Ithaca, NY, where *R. acerinum* had been observed in previous years. Collections of red maple leaves were made throughout May and June within 2 days of each Norway maple collection.

At each collection date, samples from either host were taken to the laboratory, where observations of their macroscopic and microscopic features were recorded. Some stromata from each collection were moistened, kept in moist chambers for up to 48 hr, and examined microscopically to determine whether they were able to produce either ascospores or conidia. In addition, lesions on 1985 foliage were surface-sterilized by immersion for up to 2 min in 0.05% sodium hypochlorite, and parts thereof were aseptically transferred to 4% (w/v) potato-dextrose agar (PDA) (Difco Laboratories, Detroit, MI).

Geographic distribution. To learn the geographic distribution of *Rhytisma* on Norway maple in New York, questionnaires illustrating diseased leaves, briefly describing symptoms of the disease, and asking for reports of additional locations were distributed to about 1,000 tree care professionals throughout New York State. As we traveled throughout the state, we also looked for the disease wherever populations of Norway maples were found.

To determine dates and locations of previous occurrences of tar spot on Norway maples, we examined specimens deposited in the Cornell University Plant Pathology Herbarium, and Amy Rossmann checked records and specimens at the USDA National Fungus Collection in Beltsville, MD.

Chemical control. Fungicides listed in Table 1 were tested for efficacy in

preventing infection of Norway maple leaves. Branches 30-50 cm long on Norway maple saplings in a previously affected hedgerow in New Hartford were randomly selected and divided into six groups of five. Branches in each group were sprayed to runoff either with one of the fungicides plus DuPont Spreader-Sticker or with DuPont Spreader-Sticker alone at budbreak (1 May) and twice thereafter on 15 and 30 May. One group of five branches was marked at the outset to serve as a check and was left untreated throughout. Treatments were evaluated on 8 August 1985 by counting the spots on 20 leaves randomly selected on the sprayed portion of each branch. Also, all

Table 1. Efficacy of selected fungicides for control of tar spot (*Rhytisma* sp.) on Norway maple foliage

Treatment	Rate ^w (a.i./100 L H ₂ O)	Spots per leaf ^x	Leaves infected ^y (%)
Triadimefon (Bayleton 25WP)	15 g	0.0 a ^z	0 a
Benomyl (Benlate 50WP)	30 g	0.1 a	4 a
Mancozeb (Dithane M-45)	144 g	0.3 a	23 a
Copper hydroxide (Kocide 101)	370 g	1.3 b	60 b
Check	...	1.6 b	68 b
DuPont Spreader- Sticker	18 ml	1.7 b	74 b

^wDuPont Spreader-Sticker added to all fungicides at a rate of 18 ml/100 L.

^xMean number of spots per leaf on five branches, 20 leaves per branch.

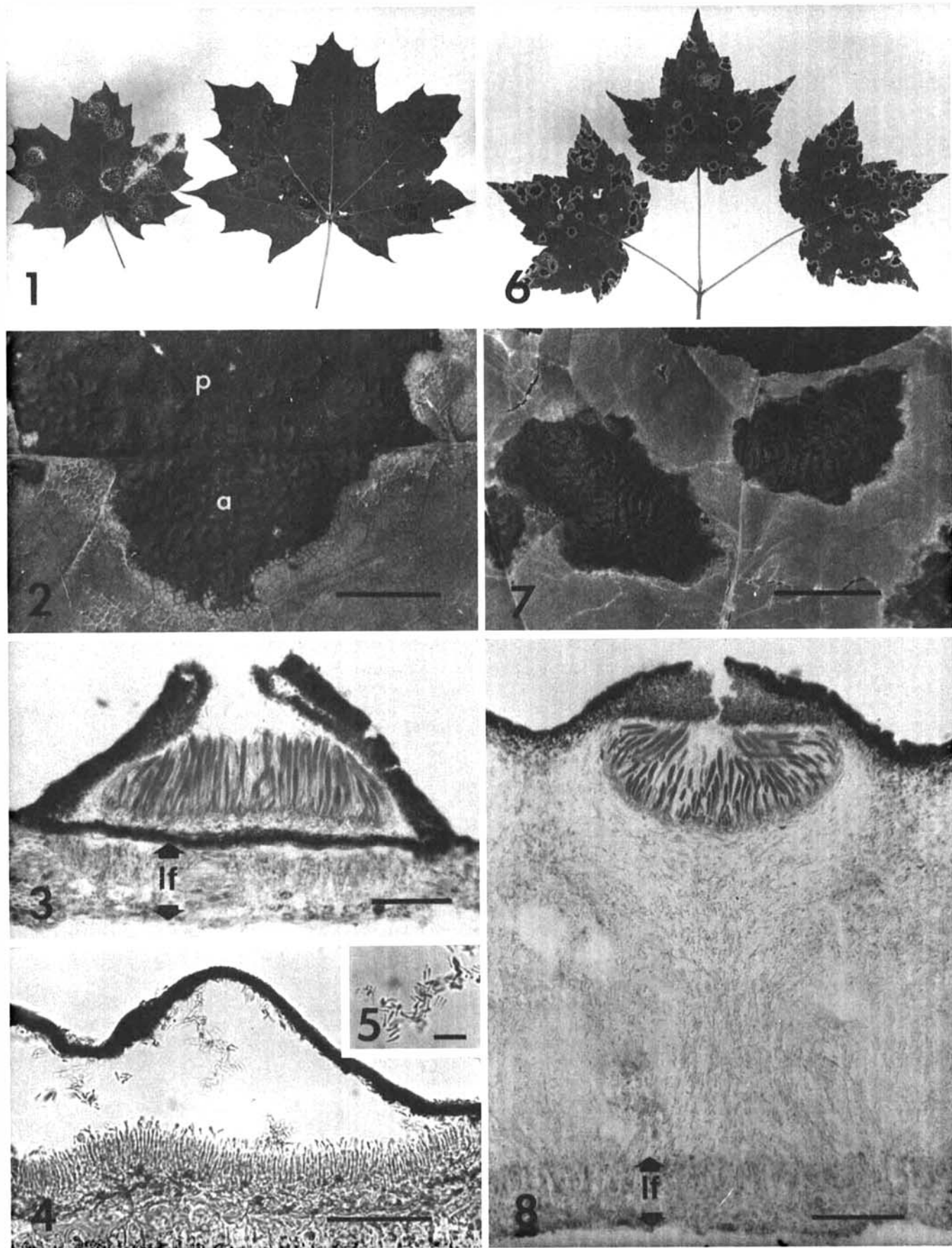
^yMean percentage of infected leaves from five branches per treatment.

^zValues in each column followed by the same letter do not differ significantly ($P = 0.05$). Means of raw data are shown here, but statistical tests for significance (LSD) were performed on ranked, transformed data after results of Bartlett's test for homogeneity of variance indicated that variances in respective columns were heterogeneous (6). Spots per leaf data were increased by 1 to eliminate computational difficulties where no spots occurred, and the data were transformed via \log_{10} . Percent leaves infected data were transformed via arc sine $\sqrt{\text{percent leaves infected}}$. In both cases, transformations resulted in homogeneous variances via Bartlett's test.

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Figs. 1-8. *Rhytisma* sp. on *Acer platanoides* leaves. (1) Typical lesions and stromata. Leaf at left shows immature stromata before they have enlarged and coalesced. (2) Close-up showing eruptions caused by maturing ascocarps (a). Spent pycnidia (p) are in center. Scale bar = 5 mm. (3) Cross section of mature ascocarp on leaf (lf). Scale bar = 75 μ m. (4) Cross section of part of a pycnidium on leaf (lf). Scale bar = 25 μ m. (5) Close-up of conidia. Scale bar = 10 μ m. (6-8) *R. acerinum* on *A. rubrum* leaves. (6) Typical stromata on leaf surfaces. (7) Close-up of stromata showing characteristic serpentine ridges. Scale bar = 5 mm. (8) Cross section of stroma on leaf (lf) to show relative thickness of stroma and location of ascocarp. Scale bar = 100 μ m.

leaves on the sprayed portion of each branch were counted, and the percentage of diseased leaves was determined.

RESULTS

Anatomy/biology. Stromata on 1984 Norway maple leaves collected from duff in May and June 1985 averaged 23 mm in diameter (range 10–32 mm) and were about 10 μ m thick. They contained numerous discrete ascocarps that swelled to 150 μ m thick when wet, thus imparting a roughened, pimplelike texture to the outer third of the stromal surface where they most often occurred (Figs. 1–3). Within 24 hr of wetting, the ascocarps ruptured via irregular slits, and hyaline, filiform ascospores (55–80 \times 1.5–2.2 μ m) were forcibly ejected from them. Ascocarps in stromata collected after 25 June failed to produce ascospores and often were overgrown by saprophytic fungi and bacteria.

First symptoms of infection of 1985 Norway maple leaves were observed on 20 June as circular, chlorotic lesions. Both green- and purple-leaved varieties of trees were affected. Within 2 wk, tiny (0.5–1.0 mm diameter), black stromata began to appear in the lesions. Eventually, as each individual lesion enlarged to a maximum size of 2–3 cm in diameter, up to 100 such stromata were observed in a single lesion. The stromata within each lesion grew and ultimately coalesced to cover each lesion with a thin, black crust of fungal tissue. Pycnidia were found in the central portions of the stromata in early August. They erupted from beneath the stromal surface and gave it a roughened texture similar to but less pronounced than that caused by mature ascocarps. Hyaline, allantoid, aseptate conidia (4.5–5.5 \times 1.0 μ m) in a milky gelatinous matrix oozed from distinct ostioles in the pycnidia whenever the stromata were moistened (Figs. 4 and 5). Pycnidia could be induced to sporulate (via wetting and incubation in a moist chamber) on stromata collected through mid-October, the last collection before this report.

In contrast, stromata on 1984 red maple foliage collected in May and June 1985 averaged 2.6 mm in diameter (range 1.0–7.0 mm) and were 500–700 μ m thick (Figs. 6–8). Ascocarps therein were in continuous, serpentine ridges rather than discrete pustules. When wet, they ruptured via slits extending the entire length of a stroma, and hyaline filiform ascospores similar to those produced on Norway maple were ejected from them. Ascospore production on red maples ceased after 25 June.

First symptoms of infection on red maples appeared on 20 June as chlorotic, circular lesions. Within each lesion, a single black stroma (1–2 mm in diameter) appeared 2–4 wk later, and in most cases, that stroma enlarged as the season progressed to a maximum diameter of 7

mm. In some instances, however, host tissue in a lesion died abruptly before the stroma could mature. This reaction often occurred in all lesions on a particular leaf but not on all leaves on a branch or plant. At maturity, most stromata were bordered by a band (1–2 mm wide) of chlorotic host tissue, but some lesions were completely covered by stromata. No pycnidia or conidia were observed. By late August, the surface of each stroma had begun to take on the serpentine pattern of alternating ridges and depressions it would have until mature the following spring.

Cultures of fungi believed to be *Rhytisma* spp. were readily and repeatedly obtained by placing leaf tissue from margins of developing lesions on cornmeal agar (Difco). However, attempted isolations from older lesions predominantly covered with stromata and surrounded with little chlorotic host tissue failed to yield any microbes.

Cultures from Norway maple lesions resembled those described by Schweizer (5) from Norway maple. Brownish black hyphae grew appressed to the agar surface and away from points of origin at variable rates to produce colonies with irregular, feathery margins (Fig. 9A). Groups of thick-walled chlamydospores were common within the hyphae. Cultures incubated in continuous fluorescent light (about 1,000 lux) at 18 C for 3–4 wk produced pycnidia that were spherical at first (0.5–1.0 mm diameter) but flattened with age and ruptured by way of an irregular slit. Ellipsoid conidia (1.5–2.4 \times 6.0–8.0 μ m), hyaline individually but pink in mass, oozed from the pycnidia in a gelatinous matrix. These conidia were distinctly different from those observed on leaves.

Cultures of the fungus from red maple lesions also produced brownish black mycelium that grew appressed to the surface of PDA. However, rather than having the feathery growth, hyphal growth in these cultures was much denser (Fig. 9B). When the cultures were held up to a bright light, little or no light passed through them. Chlamydospores were usually clustered in groups of 20–50 or more and were produced in such abundance that 4-wk-old cultures had uniformly roughened surfaces. No other sporulation was observed.

Geographic distribution. Tar spot was found on Norway maples in the towns of Utica, New Hartford, and Rome (Oneida County); Frankfort and Illion (Herkimer County); Gloversville (Fulton County); and Aurora and Orchard Park (Erie County). Reports of nonoccurrence of the disease were received from the municipalities of Buffalo, Clarence Center, Rochester, Syracuse, Binghamton, Vestal, and Chenango Forks; from Westchester and Nassau counties; and from northern New Jersey.

Herbaria at Cornell and at Beltsville

both had numerous specimens labeled *R. acerinum* on Norway maple, and those specimens resembled the disease on Norway maple in New York. However, all such specimens were from western Europe; there were no collections of *Rhytisma* spp. on *A. platanoides* from North America.

Chemical control. All of the fungicides tested, except copper hydroxide, gave satisfactory control of the disease on Norway maple. Triadimefon was superior in that there were no spots on treated leaves, but control afforded by mancozeb and benomyl was also satisfactory (Table 1).

DISCUSSION

We are not the first to note striking differences in fungi identified as *R. acerinum* and occurring on different species of maples. J. Müller (3) recognized that a fungus causing tar spots on Norway maples differed from that on field (*A. campestre* L.) and sycamore (*A. pseudoplatanus* L.) maples and through cross-inoculations determined that there were at least two races of the pathogen. K. Müller (4) and von Tubeuf (8) also conducted cross-inoculations as part of their studies and concluded that a second species, *R. pseudoplatani* Muller, caused the spots on field and sycamore maples, but *R. pseudoplatani* has not been

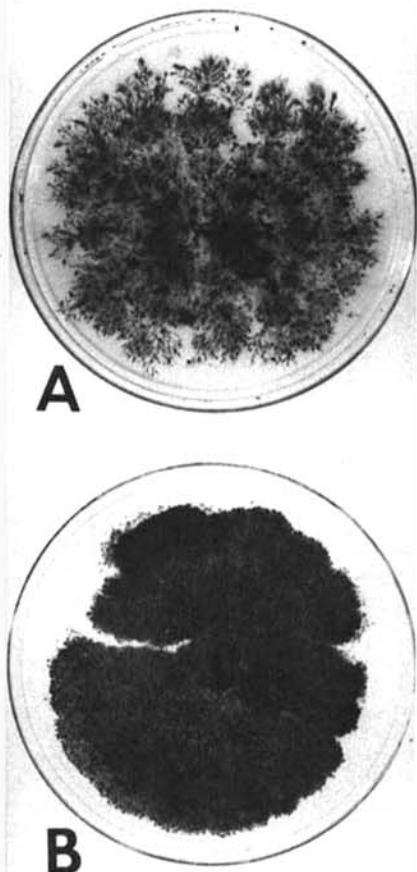


Fig. 9. Typical 28-day-old cultures of suspected pathogens from tar spots on (A) Norway and (B) red maples.

accepted as a valid species by subsequent workers. Clearly, the taxonomy of these organisms is in disarray and we plan further work, including cross-inoculations, to further elucidate and clarify differences between the pathogens.

As far as we can determine, this is only the second report of tar spots on Norway maples in North America. The first was in 1941 by Waterman (9), who examined specimens from Lake County, Ohio, and noted that the pathogen thereon did not correspond with previously known species of *Rhytisma* on *Acer*. We were unable to locate the specimens upon which Waterman's report was based, but her brief description suggests that she was working with the same pathogen we have found in New York.

Tar spot on Norway maple can be controlled easily with timely applications

of one of several conventional fungicides. However, the pathogen apparently has only one generation of inoculum per year, and homeowners theoretically may obviate the need for chemical control simply by raking and destroying diseased leaves before inoculum is produced from them each spring.

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