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CAUSE AND CONTROL OF DOGWOOD ANTHRACNOSE IN NORTHEASTERN UNITED STATES

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Abstract. Native flowering dogwood (*Cornus florida*) in parts of northeastern United States have shown increasing dieback and mortality since the late 1970's. Symptoms consist of necrotic spots, scorch, and blight of the leaves, non-dehiscence of some blighted leaves, cankers, epicormic branches, and dieback beginning in the lower branches. The anthracnose fungus *Discula* sp. was identified as the primary cause. Post-infection stresses may have intensified tree decline. The fungicides chlorothalonil (Daconil 2787) or mancozeb (Manzate 200) applied at 10-day intervals during leaf expansion reduced foliar symptoms. Additional recommendations are pruning, fertilization, and watering to promote tree vigor.

Résumé. Dans le nord-est des Etats-Unis, le *Cornus florida* subit une augmentation de dépérissement et de mortalité depuis la fin des années 1970. Les symptômes consistent en des nécroses, des flétrissures et des roussissures des feuilles, l'absence de déhiscence de certaines feuilles flétries, des chancres, des branches épicormiques, et un dépérissement commençant dans les branches du bas. Le champignon *Discula* sp. associé à l'anthracnose fut identifié comme la cause première. Les stress subséquents ont probablement intensifié le dépérissement des arbres. Le fongicide chlorothalonil (Daconil 2787) ou le mancozèbe (Manzate 200), appliqué aux 10 jours pendant la croissance des feuilles, a réduit les symptômes foliaires. D'autres recommandations sont l'élagage, la fertilisation et l'arrosage afin de promouvoir une plus grande vigueur des arbres.

Pirone (6) first reported a rapid deterioration of *Cornus florida* in New York and Connecticut in 1978 and 1979. He attributed the decline to infection by *Colletotrichum gloeosporioides* in wet springs. In 1983, we reported (2) a lower branch dieback disease with the same symptoms on *C. florida* in New York, Connecticut, New Jersey, and Pennsylvania. A species of the fungus *Discula* was consistently associated with diseased trees.

Byther and Davidson (1) reported a similar disease of western flowering dogwood *(C. nuttallii)* and named it dogwood anthracnose. In 1983, Salogga and Ammirati (7) reported dogwood anthracnose on *C. nuttallii* in Washington State, Oregon, Idaho, and British Columbia and they associated the fungus *Discula* sp. with the disease. We have since concluded that the same anthracnose disease occurs in both geographic locations. Dogwood anthracnose is the appropriate name for the disease of *C. florida* caused by *Discula* sp. This new disease is different from spot anthracnose caused by *Elsinoe corni*, a disease of *C. florida* primarily in its more southerly range (3).

In 1982, we began a study of declining dogwoods at Planting Fields Arboretum, Oyster Bay, Long Island and at a woodland site in Ossining, New York. This paper summarizes our observations on the symptomatology, etiology, and control of dogwood anthracnose as it affects *C. florida* in southeastern New York State.

Symptoms

Dogwoods of all ages and sizes were susceptible. Even the woodland population of dogwood seedlings was drastically reduced. Diseased trees in open sites remained alive, whereas most infected understory dogwoods in the woodlands died in 2-5 years.

The most characteristic symptom of dogwood anthracnose was the yearly twig and branch death beginning in the lower part of the canopy (Fig. 1). The disease was first called "lower branch dieback" (2).

Additional symptoms differentiated dogwood anthracnose from other diseases of dogwood. Leaves showed small purple-rimmed spots and larger brown blotches (Fig. 2). The dead tissue sometimes weathered away so that infected leaves showed "shot holes" and appeared ragged. The blotches would sometimes expand until the entire leaf became blighted. Some blighted leaves remained on the shoots through the winter. Reddish brown-purple spots and brown necrotic blotches formed on flower bracts after rainy periods. Leaf lesions were often similar to those caused by *Septoria cornicola*, but were generally larger than the tiny ($<$ 1/25 inch diam.) spots typical of the spot anthracnose disease caused by *Elsinoe corni*.

Shoots became infected through the petioles of blighted leaves or from tiny sunken lesions that formed in the bark of shoots. The tips of infected shoots turned grey-tan and a purple zone formed between the dead and healthy bark.

Epicormic branches (watersprouts) formed on the trunk or main branches of diseased dogwoods. When the epicormic branches became infected and died back, annual cankers developed on the trunk or branch at the base of the dead epicormic branches. The cankers were detectable by sunken, swollen, or cracked areas in the bark. Cutting into the wood revealed brown, elliptical discolored areas beneath the bark (Fig. 3).

Cause

Tiny *conidiomata* (fruiting bodies) proliferated on the dead leaf tissues and dead bark of twigs and epicormic branches. From characteristics of the conidiomata, the fungus *Discula* sp. was identified. The conidiomata appeared as reddish brown to black bumps on twigs and leaves when viewed at high magnification. When moistened, the conidiomata exuded single-celled conidia (Fig. 4) in gelatinous masses or tendrils. These spores initiated infection on the new leaves in the spring and on leaves that formed throughout the growing season. By the use of spore traps, conidia of *Discula* sp. were shown to be wind-spread during rainy periods.

The *Discula* sp. was isolated repeatedly from portions of leaves, buds, and shoots of dogwoods showing disease symptoms. Successful inoculations of leaves and stems of healthy dogwoods with cultures of *Discula* sp. provided convincing evidence that this fungus is causing the anthracnose disease of *C. florida.*

Contributing Factors

Other diseases and insect injuries, most notably *Armillaria* root rot, *Septoria* leafspot, *Botrytis* blight of flower bracts and young leaves, dogwood borer (*Synanthedon scitula),* and dogwood club gall midge *(Mycodiplosis clavula),* were occasionally observed on dogwoods. We have interpreted these as "normal" stresses on dogwood, not responsible for the marked decline of *C. florida* since the late 1970's. Each of these agents, however, might have *contributed* to the death of dogwoods already stressed by dogwood anthracnose disease. Borer infestation has been associated with the recent incidence of decline in some dogwoods (8). Dogwood borer, however, is not considered a significant pest in woodland situations (4). We have observed only rare borer injury, but commonly severe anthracnose symptoms, in woodland sites. Native *C. florida* populations in the woodland understory in Catoctin Mountain Park, MD have also been severely injured by dogwood anthracnose (5).

There have been suggestions that stresses induced by moisture deficits, minimal snow cover, or severe winters have predisposed dogwoods to attack by an otherwise weak pathogen. The anthracnose fungi, including those attacking oak, maple, and sycamore, are strong pathogens and do not require a weakened host to become established. To reverse the predisposition scenario, it is more likely that infection by *Discula*

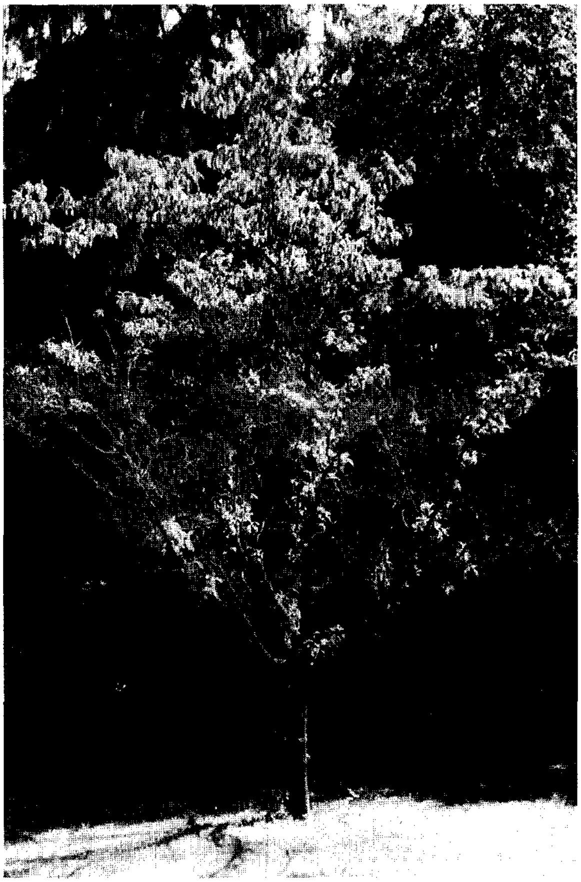

Fig. 1. Dogwood anthracnose disease causes lower branch dieback in *Cornus florida*.

sp. has predisposed dogwoods to injury from recent climatic stresses. For example, infected *C. florida* in our woodland site showed far greater winterkill than comparable healthy dogwoods.

Why dogwood anthracnose now? Why is anthracnose suddenly affecting dogwoods? What about the coincidence of dogwood anthracnose on *C. nuttallii* in the Pacific Northwest and on *C. florida* in the Northeast? We have only educated guesses for now. The early reports of dogwood anthracnose near ports of entry on both coasts suggest the importation of the pathogen, similar to the entry of the Dutch elm disease fungus into the U.S. in the 1930's. Although this possibility cannot be discounted, the sudden and widespread distribution of anthracnose on both hosts renders this explanation less tenable. Since its initial East Coast appearance in the vicinity of New York City, the disease has been reported on *C. florida* in parts of Massachusetts, Connecticut, New Jersey, Pennsylvania, Delaware, Maryland, West Virginia, Virginia, North Carolina and Georgia.

We suspect that dogwood anthracnose was present in the Northeast prior to the recent outbreak, but at a low level of infection. Some coincidental sequence of abiotic factors, most likely including temperature and moisture, dramatically intensified the impact of this disease on dogwood. The increased leaf infection and stem dieback then allowed the rapid build-up of abundant fungus spores, which provided inoculum for repeated cycles of new infection on dogwood.

Additional abiotic influences like drought and winterkill undoubtedly have intensified the decline of dogwoods already infected. Of the biotic agents, *Armillaria mellea* is the one most often seen attacking woodland dogwoods infected with dogwood anthracnose, hastening their death.

We anticipate the continuation of dogwood anthracnose, with disease severity varying yearly depending on weather conditions, especially during the spring months. Extended rainy periods during leaf expansion will particularly favor disease development.

Fungicide Trials

We conducted fungicide trials in 1985 and 1986. In 1985, spray applications at 14 day intervals were tested. No registered fungicides performed particularly well with such a long time bet-

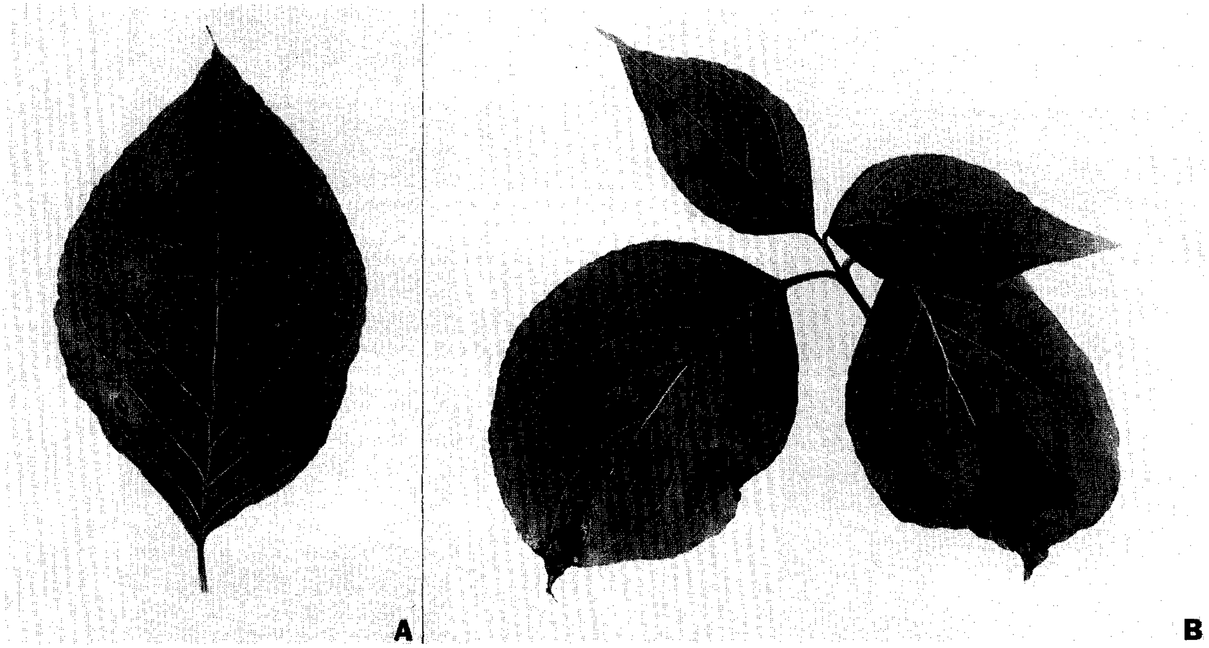

Fig. 2. Purple-rimmed leaf spots (A) and blotches (B) caused on leaves of *Cornus florida* by *Discula* sp. infection.

ween treatments. During the 1986 growing season, we attempted to identify fungicides which would be effective for leaf spot control when applied on a 10-14 day schedule. Two trials with the same fungicides but slightly different methodologies were conducted at the Long Island Horticultural Research Lab, Riverhead, New York and the Brooklyn Botanic Research Center, Ossining, New York (Fig. 5).

Second-year dogwood seedlings were in 36 2' square field plots for the Riverhead trial. Trees were inoculated by suspending epicormic branches covered with *Discula* sp. sporulation one foot above the plots. Inoculum was supplied and spray treatments were begun on May 5th. Treatments were made in 6 replications, using the following fungicides applied on a 10-day schedule: Award 10W (5 oz/100 gal), Benlate 50W (0.5 lb/100 gal), Daconil 2787 75 WP (1.5 lb/100 gal), Manzate 200 80WP (1.5 lb/100 gal) and Pratt Bordeaux (5T/gal). Leaf spot counts were made on June 16, following a natural infection period that occurred with protracted rains on June 6-12. Of the materials tested, Daconil 2787 gave the best control, providing a 64% reduction in leaf spotting compared to unsprayed control plants. Award, Manzate 200 and Benlate also showed significant disease control, not significantly different from Daconil at the 5% level (Duncan's Multiple Range Test).

In Ossining, second-year dogwood seedlings were transplanted into 38 4' square plots in a

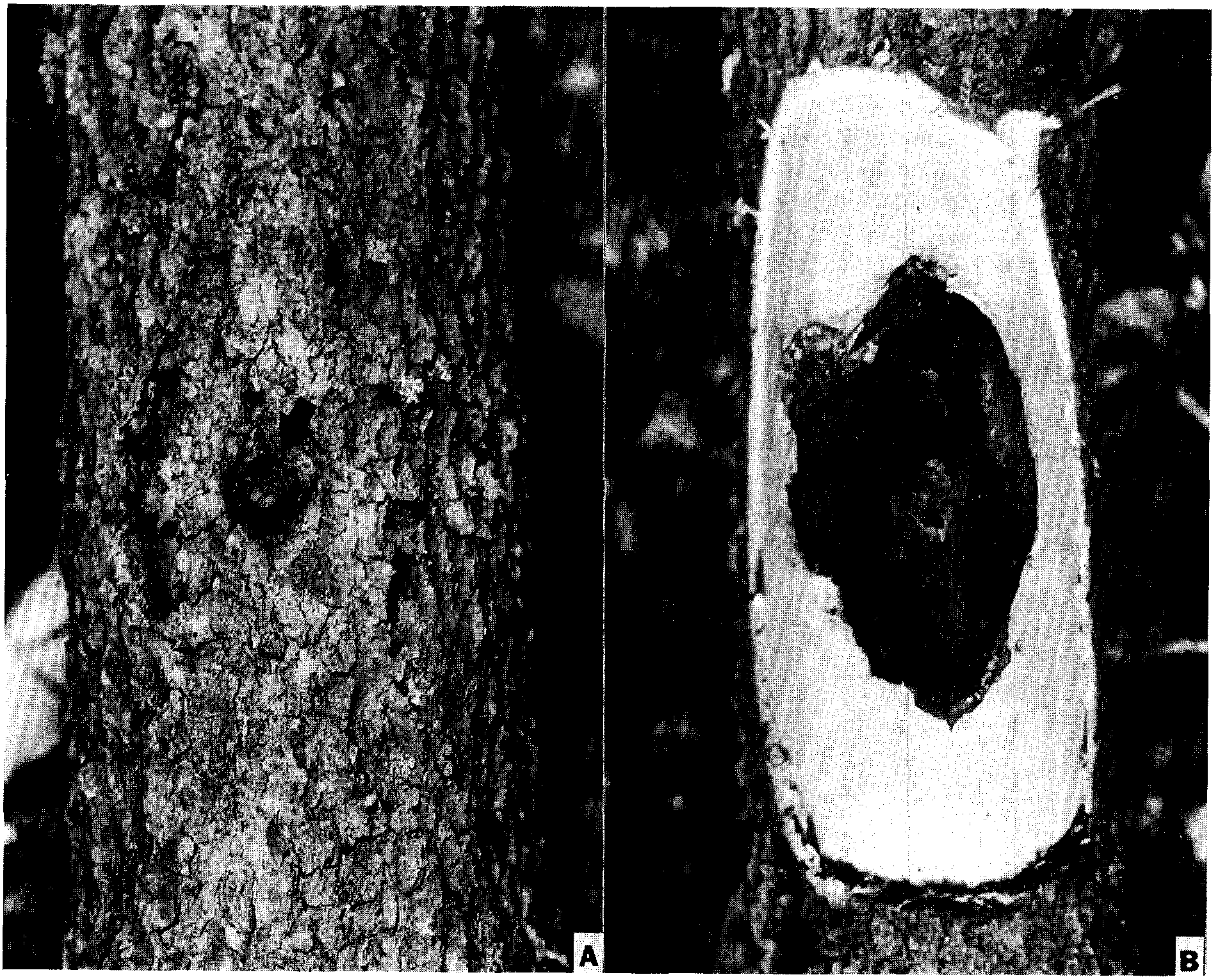

Fig. 3. Bark cracking around the base of a dead epicormic branch (A, at arrow) is a clue to the presence of a Discula canker (B).

woodland clearing where natural inoculum was provided by the diseased native dogwood population. The same fungicides were used as in Riverhead, again with 6 replications. The first spray application was made May 5 when dogwood leaves were beginning to expand, the second was made 9 days later, and the last three sprays were made at approximately 14-day intervals. Plots were rated on July 10 for percent of infected leaves per tree for 4 trees/plot. Both Daconil 2787 and Manzate 200 gave a significant reduction in percent of infected leaves per tree (Duncan's Multiple Range Test). Other fungicides also reduced leaf spotting, but the results were

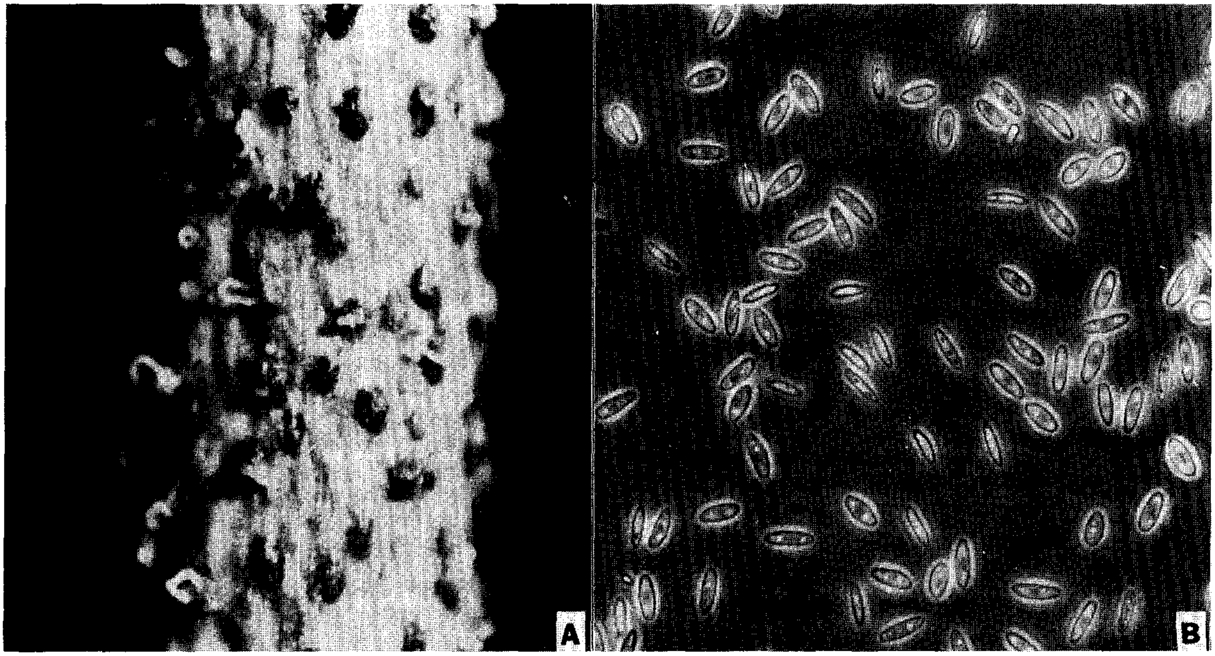

Fig. 4. A closup of a dead dogwood twig covered with Discula conidiomata oozing spores under moist conditions (A) and a microscopic view of *Discula* sp. conidia (x = 8.0 × 20um).

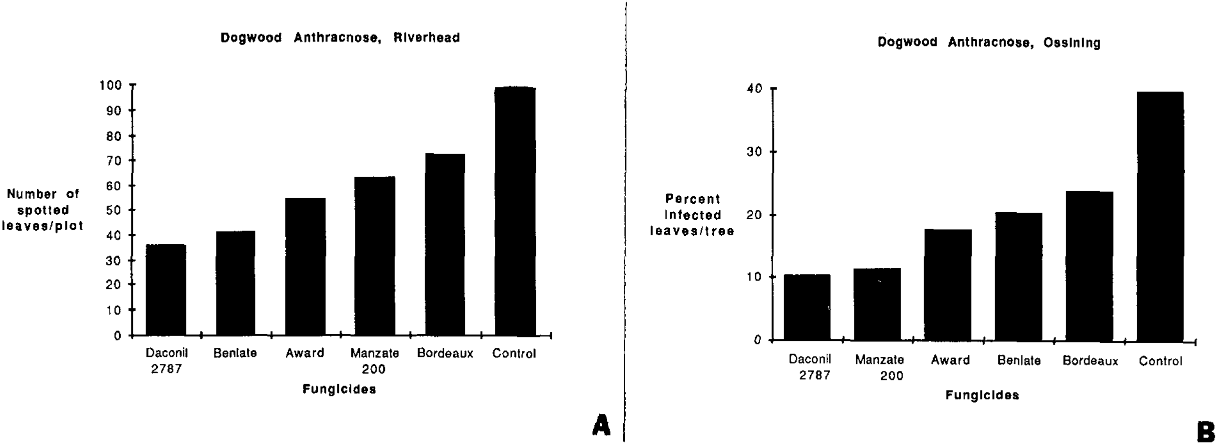

Fig. 5. Results of fungicide spray trials in (A) Riverhead and (B) Ossining, NY during 1986. Sprays were applied at a 10-14 day interval with the materials indicated.