Dogwood Anthracnose in Northeastern United States

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ABSTRACT

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Native flowering dogwood (*Cornus florida*) in parts of the northeastern United States have shown increasing dieback and mortality since the late 1970s. Symptoms consist of spots, necrotic blotches, and blight of the leaves; cankers; epicormic branching; and dieback beginning in the lower branches. From the identification of associated fungi, isolations from infected leaves and stems, and inoculation of healthy dogwoods, a fungus in the genus *Discula* was identified as the primary causal agent. Postinfection stresses may have intensified tree decline.

Native flowering dogwood (*Cornus florida* L.) in the northeastern United States have shown unusual amounts of dieback and mortality since the late 1970s. Because dogwood is such a widely used ornamental tree, this decline has aroused public concern.

Pirone (9) reported a rapid deterioration of flowering dogwood in southeastern New York and southwestern Connecticut in 1978 and 1979. He attributed the damage to infection by *Colletotrichum gloeosporioides* (Penz.) Sacc. in wet springs. In 1983, we reported (3) a lower branch dieback disease on *C. florida* in New York, Connecticut, New Jersey, and Pennsylvania with symptoms similar to those reported by Pirone. Insect pests and other diseases of dogwood (1,8) were eliminated as causal agents. A species of *Discula* Sacc. was consistently associated with the diseased trees we examined.

The disease has reappeared annually on both ornamental and woodland dogwoods. A 1984 survey (7) of lower branch dieback in Catoctin Mountain Park, Maryland, revealed that only 3% of the *C. florida* were free from symptoms, whereas nearly one-third were dead.

Byther and Davidson (2) described a similar disease of the western flowering dogwood (*C. nuttallii* Aud.) in western Washington and named it dogwood anthracnose. In 1983, Salogga and Ammirati (11) reported dogwood anthracnose in Oregon, Idaho, and British Columbia, and they associated *Discula* sp. with the disease. After a comparison of symptoms, signs, and isolates of *Discula* from *C. nuttallii* in Washington and from *C. florida* in New York, we concluded that the same disease occurs in both locations. Because the symptoms and signs on *C. florida* are consistent with anthracnose diseases, we concur

with Byther and Davidson that dogwood anthracnose is the appropriate name for this disease. Dogwood anthracnose differs from spot anthracnose caused by *Elsinoë corni* Jenkins & Bitanc., a disease of *C. florida* primarily in its more southerly range (6).

This paper presents our observations on the symptomatology and etiology of dogwood anthracnose as it affects *C. florida* in southeastern New York.

MATERIALS AND METHODS

Symptomatology. In 1982 and 1983, symptoms on *C. florida* were observed at

Planting Fields Arboretum, Oyster Bay, Long Island, and at a woodland site at the Brooklyn Botanic Garden Research Center, Ossining, New York. Foliar and shoot infections were monitored at the woodland site. Fifty leaves in the lower branches of three diseased dogwoods were selected at random and marked. Foliar symptoms were recorded on 9 June 1983 and again in August and October.

Shoot dieback was monitored on one major branch on each of five dogwoods in a semiopen site without overstory trees and in the entire branch canopy of each of 15 understory dogwoods. In December 1982, the margin between dead and live bark on shoots, twigs, and branches was marked with paint. In May and again in November 1983, dieback advancement and the occurrence of new dieback on unmarked stems were marked and measured.

A major root from each of 40 dogwoods with lower branch dieback was excavated to the fibrous roots and examined for root death and signs of root rotting fungi.

Fig. 1. Symptoms of dogwood anthracnose on *Cornus florida*: (A) Purple-rimmed necrotic spots and blotches on leaves; (B) blighted leaves that did not abscise the previous autumn; (C) shoot dieback originating from infection at node; (D) abundant epicormic branches, some with dieback; and (E) canker at base of dead epicormic branch (bark removed).

Identification and isolation. Prior to microscopic examination of fruiting structures, leaves and shoots were held in a moist chamber at 20 C for 24–48 hr. Cross sections 30–50 μm thick were cut with a freezing microtome and mounted in 1% aniline blue in lactophenol. For isolations, plant samples were surface-sterilized in 0.525% NaOCl for 10 min and incubated on potato-dextrose agar (PDA) or malt agar (MA) in petri plates held in darkness at 22 C.

Pathogenicity. Two-year-old dogwoods in dormancy were inoculated by applying mycelium of *Discula* sp. on MA to wounded (bark slit with flamed scalpel) and unwounded bark. For controls, MA only was applied to wounded and unwounded bark. The inoculation sites were wrapped with Parafilm. One-half of the inoculated trees and controls were held at 4 C in darkness for 2 mo before being moved to the greenhouse. Two-year-old dogwoods in leaf were similarly inoculated at wounded and unwounded nodes, then held at 22 C and low light (50 ft-c, 8 hr/day) for 6 wk before being

Fig. 2. Bark removed to show small cankers on dormant shoots in the autumn.

moved to the greenhouse.

For leaf inoculations, conidia were harvested from conidiomata on MA and on overwintered dogwood leaves. The spores were suspended in glass-distilled water (av. 2.3×10^6 spores per milliliter by hemacytometer counts) plus 0.1% Tween 80 and brushed onto the adaxial surface of 400 leaves on eight trees. For controls, four trees received water plus Tween 80 only. To confirm the viability of the inoculum, 48 hr after inoculation, collodion membrane prints of the epidermis were stained in 1% aniline blue in lactophenol and examined microscopically. All treated trees were held in a controlled-environment chamber for 5 days in darkness at 18 C and 95–100% relative humidity, then removed to partial shade in the greenhouse. Reisolation of *Discula* sp. from infected leaves and shoots was attempted as described above.

To test whether mycoplasmalike organisms (MLOs) might be associated with dogwoods with profuse epicormic branches (12), three epicormic branches and three root segments (2–5 mm diam) were cut from each of 20 woodland dogwoods with advanced branch dieback. Longitudinal sections (50–75 μm thick) were cut on a freezing microtome and examined for MLOs in the phloem sieve tubes by Dienes' stain (4).

RESULTS

Symptomatology. Diagnostic symptoms of dogwood anthracnose (Figs. 1 and 2) included leaf necrosis, cankers, and stem dieback, all of which began in the lower branches and progressed into the upper canopy. Dogwoods of all ages and sizes were susceptible. The rate of dieback and tree mortality varied; diseased trees in open sites usually remained alive, whereas most diseased understory trees died in 2–5 years. An unusually low population of dogwood seedlings was noted in the woodland location in 1982.

Leaves. Symptoms first appeared on new leaves, especially following periods of wet, cool weather. New lesions formed on leaves throughout the growing season. Leaf symptoms were of three types: spots, necrotic blotches, and blight (Fig. 1).

Spots ($\leqslant 7$ mm diam) consisted of bifacially necrotic brown tissue often

surrounded by a reddish brown-purple zone. Shot hole resulted when the necrotic tissue broke away. Reddish brown-purple pinpoint flecks and reddish discoloration of segments of the main vein occurred. Intermixed with spots were olive-brown necrotic blotches, irregular in outline and often bounded by a reddish brown-purple zone. Blotches formed between or along main veins and along the periphery of the blade. The foliage had a ragged appearance after the dead tissue broke away.

Blight resulted from the advancement of infection throughout the blade or after the formation of lesions on the petiole. During wet springs, the first leaf pair often became blighted, but successive leaves on the same shoot incurred only spots and blotches. Some blighted leaves remained attached, even into the following spring. By one random count, 21 of 40 blighted leaves marked in December were still attached the following May.

The increase in leaf symptoms on three dogwoods during 1983 is shown in Table 1. Spots first appeared on new foliage early in June. By late August, symptoms on the same leaves had increased markedly and spots were also evident on new leaves on these shoots. Between August and early October, new symptoms were minimal on the marked leaves, but spots continued to appear on the new foliage that developed late in the season. Trees with severe foliar symptoms showed light defoliation.

Flowers. Reddish brown-purple spots and brown necrotic blotches formed on flower bracts after rainy periods.

Stems. One apparent pathway of infection into shoots was via the petioles of blighted leaves. A brown discoloration extended into the inner bark at sites of leaf attachment. In a random sample of 200 shoots with blighted leaves, 25% showed bark discoloration around at least one node. Early symptoms of shoot dieback consisted of discoloration and shriveling of bark and darkening of terminal buds. As infection advanced basipetally, a purple-brown zone often formed between necrotic and live bark.

Epicormic branches sometimes proliferated along the larger branches and the trunk of diseased dogwoods. Infection progressed rapidly in epicormic branches, and they often were killed back to their base.

Dieback in woodland dogwoods with anthracnose was observed December 1982 through November 1983 (Tables 2 and 3). Dieback advanced faster in the understory trees than in the semiopen trees. Seven understory trees had died back to the trunk by May, and three more by November. Stem dieback progressed year-round but accelerated during winter. Dieback typically halted at a node or at a juncture with a branch. Sudden wilting of foliage and death of
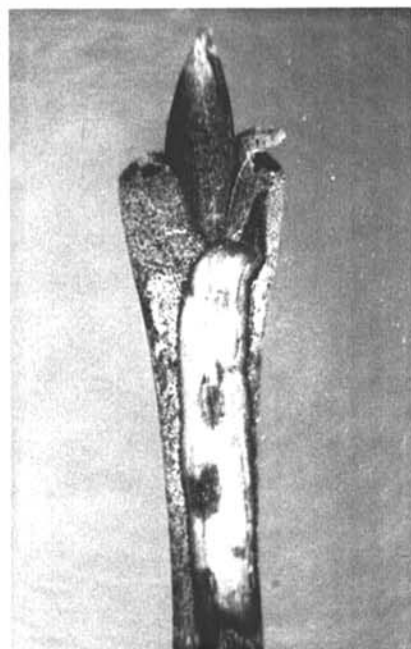
Table 1. Number of spots ($\leqslant 7$ mm diam) and area (cm^2) of necrotic blotches on 9 June and 4 October 1983 for 50 marked leaves on three woodland dogwoods with anthracnose

| | Symptoms (av./leaf) | | | | |
| | June | | October | | |
Tree	Spots	Blotches	Spots	Blotches	Spots on new leaves[a]
1	6	0	72	0.9	11
2	7	0	37	0.3	11
3	6	0	50	0	15

[a] On new shoot growth above marked leaves.

entire branches occurred occasionally during the summer, but the cause was not identified.

Cankers. Two types of canker formed on infected dogwoods: epicormic branch-associated (EBA) and shoot. EBA cankers appeared on trunks and large branches at their juncture with dead epicormic branches. Bark cracks and swelling at the canker margins were not always prominent, so EBA cankers were easily overlooked after the dead epicormic branches had broken away. When 47 woodland dogwoods with anthracnose were felled and examined, 24% of the trunks had one or more EBA cankers. Some trees with advanced dieback were free from EBA cankers, so their formation was not a prerequisite for tree decline.

Tiny cankers also formed on shoots. Small ($\leq$3 mm diam) white to brown lesions developed on new shoots early in the growing season but later often became delimited by callus. Spring shoot cankers sometimes were numerous adjacent to hanging blighted leaves, suggesting that nearby leaf conidiomata were the source of inocula. Gray to black sunken lesions ($\leq$3 mm diam) also formed on shoots and epicormic branches early in the dormant season (October–December). These lesions often were concentrated in the reddish-purple area of the bark and could be numerous—up to 100 were counted per epicormic branch. Of 520 shoots and epicormic branches collected at random from woodland dogwoods during November–December, 47% had one or more cankers.

Mortality of major roots was not detected in 33 of 40 woodland dogwoods with anthracnose. Seven dogwoods that had died back to their trunks had dead major roots bearing mycelial fans and black rhizomorphs indicative of infection by a fungus in the *Armillaria mellea* (Vahl ex Fr.) Quel. complex.

Identification. Conidiomata of the genus *Discula* were consistently associated with symptomatic leaves and shoots. Their identification was confirmed by C. Rogerson, New York Botanical Garden and A. Rossman, USDA, Beltsville. The use of the genus *Discula* is in accordance with the revision of the genus *Gloeosporium* by von Arx (14).

Other *Discula* species have teleomorphs of *Apiognomonia* v. Hohn., but no such perithecia were discovered on dogwood leaves or bark in our investigation. Numerous perithecia formed, sometimes among *Discula* sp. conidiomata, on overwintered dogwood leaves, but these were identified (M. E. Barr Bigelow, *personal communication*) as *Mycosphaerella punctiformis* (Pers.:Fr.) Starb., *M. auerswaldii* (Fleisch.) Migula, and *Venturia corni* Müller.

Salogga (10) reported that the *Discula* sp. associated with *C. nuttallii* had some characteristics of *Phomopsis*. B. Sutton

(Commonwealth Mycological Institute, *personal communication*) identified the fungus causing anthracnose of *C. florida* in southeastern New York as a *Phomopsis*. We found a few ascocarps of *Diaporthe*

corni Fckl., the teleomorph of *Phomopsis corni* (Fuckel) Trav., in branch bark of *C. florida*. Neither the anamorph of *D. corni* that formed in culture nor the type specimen of *P. corni* matched the *Discula*

Table 2. Advance of dieback in marked stems during periods of tree dormancy (December 1982–May 1983) and growth (May–November 1983) in 20 woodland dogwoods with anthracnose (av./tree)

	Dormancy		Growth	
Site	Dieback advance[y] (cm)	Percent stems with advance	Dieback advance (cm)	Percent stems with advance
Semiopen	8.9 a[z]	34	3.9 b	43
Understory	13.1 c	43	4.7 b	46

[y] Includes stems on one major branch for each of five dogwoods in semiopen (no overstory trees, partial sun) and for entire canopy for each of 15 understory (shaded) trees (av. diameter 2.3 cm at 60 cm above ground).
[z] Dissimilar letters indicate significant differences ($P = 0.01$) according to standard Student's t test.

Table 3. New twig tip dieback that began during periods of tree dormancy (December 1982–May 1983) and growth (May–November 1983) in 20 woodland dogwoods with anthracnose (av./tree)

	Dormancy		Growth	
Site	Dieback distance[y] (cm)	No. twigs with dieback	Dieback distance (cm)	No. twigs with dieback
Semiopen	4.5 a[z]	18 c	6.5 a	7 d
Understory	10 b	21 c	5.8 a	8 d

[y] Includes stems on one major branch for each of five dogwoods in semiopen (no overstory trees, partial sun) and for entire canopy for each of 15 understory (shaded) trees (av. diameter 2.3 cm at 60 cm above ground).
[z] Dissimilar letters indicate significant differences ($P = 0.01$) according to standard Student's t test.
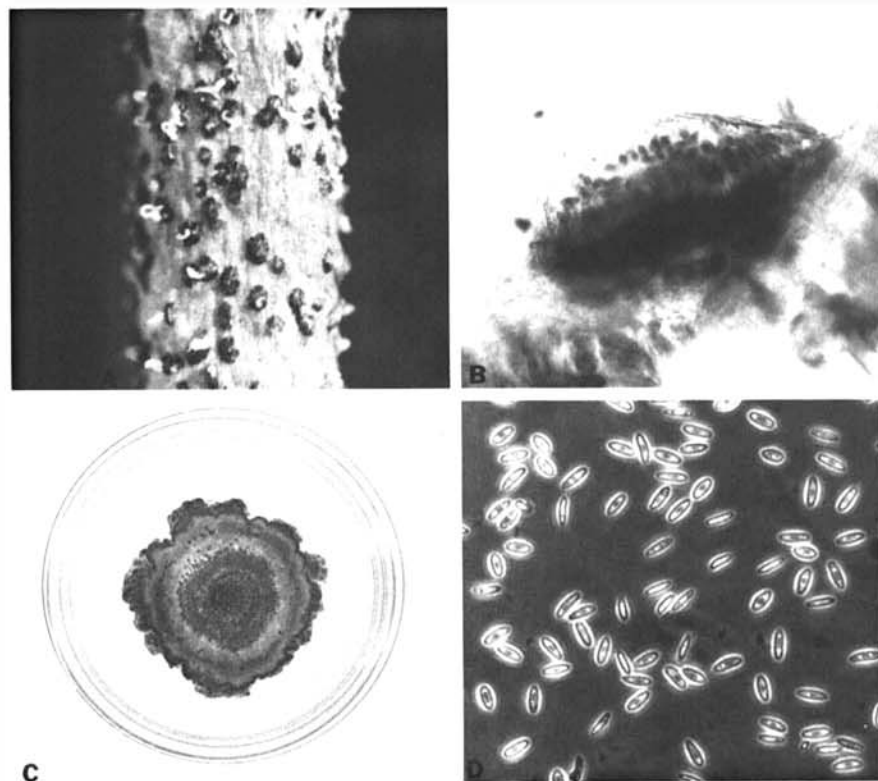
Fig. 3. Signs of *Discula* sp.: **(A)** Conidiomata on dead twig, showing oozing conidia under moist conditions; **(B)** cross section of conidioma; **(C)** 3-wk-old sporulating culture on PDA; and **(D)** single-celled, hyaline conidia (6–11 × 1–3 μm).

sp. from *C. florida* (A. Rossman, *personal communication*).

Signs of *Discula* sp. (Fig. 3) include formation of conidiomata in necrotic tissue of leaves and stems; the conidiomata proliferate on blighted leaves, more abundantly on the abaxial surface. An average of 11 conidiomata per square millimeter were counted in a random sample of 15 blighted leaves collected in September. Leaf conidiomata are orange, reddish brown, or black, superficial or subcuticular, pulvinate-globose, and 39–96 μm ($\bar{x} = 61$) in diameter by 23–44 μm ($\bar{x} = 31$) high. The basal stroma bears lageniform to apically attenuated phialides, 5–11 μm ($\bar{x} = 8$) by 1–2 μm. Conidia are hyaline, one-celled, 6–11 μm ($\bar{x} = 8$) by 1–3 μm ($\bar{x} = 2$), and elliptical-fusiform (Fig. 3D) and often contain one or two polar oil droplets. Conidia under moist conditions are exuded in white-gray agglutinated masses or cirrhi. Conidiomata in dead bark of shoots, twigs, and epicormic branches are orange-black, erumpent (Fig. 3A), and 100–350 μm ($\bar{x} = 189$) across. When moist, they exude conidia (Fig. 3A), 5–10 μm ($\bar{x} = 8$) by 1–3 μm ($\bar{x} = 2$), in white, gold, or orange-pink agglutinated masses or cirrhi.

Conidiomata from dogwood leaves and shoots were viable (spore exudation after 2–4 days in a moist chamber at 26 C) when tested in the spring, summer, and fall. Germination (on 2.5% water agar at 26 C) of conidia was always high (80–100%), even for conidia from overwintered conidiomata.

Repeated isolations from spots, necrotic blotches, cankers, and the margin of shoot dieback consistently yielded *Discula* sp. It was also isolated from discolored main veins and petioles, brown pith beneath shoot cankers, spots on flower bracts, and nonsymptomatic vegetative and flower buds, fruits, and shoot nodes.

Cultural characteristics. *Discula* sp. on

Fig. 4. Approximate range (hatching) of dogwood anthracnose in the northeastern United States in 1986. Points indicate isolated sites where disease was identified.

PDA (Fig. 3C) or MA produces slow-growing, appressed, white colonies, granular in texture, with peripheral hyphae submerged. Colonies in natural or artificial light become gray, tan, or black with age. Conidiomata are brown-black, globose, 150–290 μm ($\bar{x} = 208$) in diameter with openings 20–100 μm ($\bar{x} = 41$) wide. Conidia are similar to those found in situ. Dogwood isolates of *Discula* sp. showed variability in colony characteristics and production of conidiomata. The formation of conidiomata on MA was enhanced by the presence of leaf, bud, or bark tissue in the media.

Pathogenicity. The pathogenicity of *Discula* sp. in *C. florida* was demonstrated. Black sunken cankers formed around nine of 53 (17%) wounds on six of 12 trees held at 4 C after inoculation and around three of 47 (6%) wounds on two of 12 trees held in the greenhouse. *Discula* sp. was recovered from discolored inner bark around the cankers. Callus formed around the cankers and there was no shoot dieback. No cankers formed at the unwounded or control inoculation sites or on trees in leaf at the time of inoculation.

Conidia applied to leaves showed 80–100% germination on the epidermis, as determined by collodion imprints. After 5 wk, the six trees inoculated with conidia from field-collected conidiomata showed anthracnose symptoms. Of 306 inoculated leaves, 207 (68%) had spots, necrotic blotch, or blight, and for five of the trees, one to four shoots per tree showed tip dieback ($\leq$6 mm). Symptoms on two trees inoculated with conidia from conidiomata on MA consisted of marginal scorch of immature leaves. None of the controls showed anthracnose symptoms. Only *Discula* sp. was repeatedly isolated from inoculated leaves with anthracnose symptoms. Conidiomata of *Discula* sp. formed on necrotic leaf tissue.

No MLOs were detected by Dienes' stain in the phloem sieve tubes of epicormic branches or roots of dogwoods with anthracnose.

Range. Figure 4 shows the approximate range of dogwood anthracnose in northeastern states as of 1986. This is based in part on cooperator reports that were confirmed by identification of conidiomata and/or isolation of *Discula* sp. from leaves or twigs.

DISCUSSION

From fructifications on necrotic tissues, isolations from infected leaves and stems, and inoculation of dogwoods, we have concluded that the anthracnose fungus *Discula* sp. is the primary causal agent of the lower branch dieback disease reported (3) on *C. florida* in the Northeast. From a comparison of symptoms, signs, and isolates of *Discula* sp., this is the same disease as the dogwood anthracnose described on *C.*

nuttallii, C. florida, and *C. kousa* Hance in the Pacific Northwest (10).

The reason for the sudden onset of anthracnose over part of the northeastern range of *C. florida* and for its coincidental outbreak in *C. nuttallii* in the Pacific Northwest is unknown. The periods of prolonged rainfall common to the Pacific Northwest and the cool, wet springs occurring irregularly in the Northeast provide conditions conducive to anthracnoses (13). A shift in spring rainfall and temperature patterns might account for the outbreaks in both geographic regions.

The outbreak of the disease near ports of entry on both coasts of the United States raises the possibility of *Discula* sp. being an imported pathogen. The sudden and widespread distribution of anthracnose in *C. florida* and *C. nuttallii* renders this a less likely explanation, however.

We propose that dogwood anthracnose was present in the Northeast before the recent outbreak but at a low level of infection. Abiotic factors conducive to a significant increase in infection could have led to a rapid buildup of inoculum, thereby perpetuating new cycles of infection in dogwood.

Anthracnose can become a persistent disease in *C. florida* because of the diverse infection sites and the production of abundant inoculum. After initial leaf infection, the disease progresses in several ways: by secondary infection of leaves, by extension from blighted leaves into shoots, by canker formation on both elongating and dormant shoots, and by way of infected epicormic branches into the trunk and main branches. The year-round contact between pathogen and host makes interruption in the disease cycle unlikely. This disease can be grouped with the anthracnoses of sycamore and white oak, in which the pathogen attacks both leaves and stems and overwinters in infected stem tissues.

The abundance of viable conidiomata on stems and on attached and fallen leaves provides ample inoculum for infection whenever moisture and temperature are favorable. The long retention of blighted leaves that bear conidiomata and the proliferation of highly susceptible epicormic branches, both uncommon in other tree anthracnoses, provide conveniently positioned inoculum and substrates for the establishment of new infection.

Hudler (5) discussed the possible predisposing effects of recent droughts and abrupt temperature fluctuations on dogwood. Dogwood having severe leaf infection would be vulnerable to cold injury because of depleted photosynthates and the loss of leaf receptors for the short-day stimulus that triggers cold hardiness (14). Diseased dogwoods in our woodland site incurred greater winterkill during the winter of 1982–1983 than did trees with no symptoms in 1982. Defoliation of overstory trees by the

gypsy moth and root mortality from consecutive winters with light snow cover are additional stresses that may have hastened the decline of diseased dogwoods. Armillaria root rot commonly attacks and kills severely stressed dogwoods in northeastern woodlands.

Dogwood anthracnose has now been established in natural and ornamental populations of flowering dogwood in parts of the Northeast for about a decade. There have been no systematic surveys to ascertain disease spread, but our observations and the unpublished reports of others indicate a gradual increase in the range since 1981. We anticipate that *Discula* sp. will continue to be a significant pathogen of *C. florida* in the Northeast, with disease severity varying considerably from year to year, depending on weather conditions during the early part of the growing season.

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