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Dogwood Anthracnose
Understanding a Disease New to North America

The flowering dogwood, *Cornus florida* L., has an extensive natural range that includes most of the eastern United States. Its lovely spring bract display has made it one of the most celebrated native trees, as well as a very popular choice for ornamental landscape plantings. In the late 1970s, dogwood fanciers in the New York City area began to notice symptoms of ill health on their favorite tree. The most striking symptom was dieback of the lower branches, which limited the bract display to the upper part of the tree canopy. Dieback was conspicuous along roadways and in home landscapes, as well as within forests. The *New York Times* reported in an article by P. P. Pirone that in spring 1979, thousands of dogwoods in southeastern New York and southwestern Connecticut had developed symptoms of a "mysterious disease" (36). Leaf spotting and defoliation were noted. The disease was at the time attributed to wet springs and infection by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz., a known leaf pathogen of dogwood.

By 1983, the flowering dogwood problem was recognized as a disease new to the northeastern United States. The new disease was named "lower branch dieback," and an unidentified species of fungus in the genus *Discula* was associated with infected trees (21). The earlier report of *Colletotrichum gloeosporioides* was a misidentification of this new pathogen. Lower branch dieback was later renamed "dogwood anthracnose" (14), and *Discula* sp. was confirmed as the causal agent of dogwood anthracnose in *C. florida* as well as in *C. kousa* (Buerger ex Miq.) Hance in the northeastern United States (27,28).

Symptoms of dogwood anthracnose were actually first observed on native Pacific dogwood, *Cornus nuttallii* Audubon, in southern Washington State in 1976 and 1977 (14). The causal agent of this western disease was first reported as *Gloeosporium* sp., but later the revised designation *Discula* sp. was given for the fungus that was repeatedly isolated from symptomatic *C. nuttallii* (40). Less severe anthracnose symptoms were reported in the Pacific Northwest on ornamental *C. florida* and *C. kousa*, which are exotic to that area (41).

Redlin (38) compared leaf and twig samples from infected *C. florida* and *C. kousa* on the east coast with specimens of *C. nuttallii* from the west coast. He concluded that the dogwood anthracnose fungus was common to these hosts but distinct from other North American *Discula* spp. which cause anthracnose of ash, oak, and sycamore. In 1991, the pathogen was described and named *Discula destructiva* Redlin (38).

Fig. 1. Flowering dogwood (*Cornus florida*) along roadway in Islip, NY (May 1985). Note the severe lower canopy dieback due to dogwood anthracnose.

Symptoms

Dogwood anthracnose affects leaves, bracts, current-year shoots, localized areas of bark and cambium of trunk or branches, fruits, and seeds of *C. florida* (11,21). Symptoms are more severe in the lower canopy for *C. florida* in relatively exposed landscape plantings (Fig. 1). *C. nuttallii* is affected similarly when it is grown as an ornamental (Fig. 2) (14). Understory dogwoods, which have shaded canopies and thinner leaves, are more likely to be uniformly affected. Some of the trees infected by dogwood anthracnose are killed in 1 to 3 years; others survive and, depending on the environmental conditions, exhibit symptoms of varying severity from year to year. *C. florida* regeneration has been greatly reduced in areas where dogwood anthracnose is severe. Young saplings may be killed in a single season.

Leaf symptoms caused by *D. destructiva* on *C. florida* are usually first noted in the spring on recently expanded leaves. Necrosis at the tips of very young leaves occasionally occurs. Leaves may show three major kinds of symptoms: leaf spot, blotch, and blight. Leaf spots are scattered across the blade and are irregular in size and outline. They have a reddish to purplish rim and a light brown center, and are sometimes partly limited by veins. Spots are initially surrounded by a diffuse yellow halo (Fig. 3). Under sunny conditions, sometimes the centers of the leaf spots drop away to form "shot-holes," leaving a purple rim around a roughly circular opening.

Leaf blotch refers to lesions that are more extensive than leaf spots. Most often, blotches are seen at the leaf tip or along

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the margin (Fig. 4). The margin of blotches usually develops a reddish purple color. Leaf blight (in which the entire leaf blade rapidly becomes necrotic) can progress into the petiole, leading to the initiation of shoot infection and cankers (Fig. 5). Blighting commonly occurs throughout the canopy of understory *C. florida*, or within the interior canopy or on epicormic branches of exposed trees. Blighted leaves sometimes adhere to the shoot through the winter (Fig. 6).

The irregularity and larger size of leaf spots distinguish dogwood anthracnose from the tiny, round lesions (1 to 2 mm diameter) of spot anthracnose, caused by *Elsinöe corni* Jenk. & Bitancourt (Fig. 7). Leaf infections caused by *Septoria* spp., *Ascochyta cornicola* Sacc., *Botrytis cinerea* Pers.:Fr., and *Colletotrichum gloeosporioides* are best distinguished from dogwood anthracnose by direct examination of fungal sporulation within the lesions.

Bracts infected by *D. destructiva* exhibit reddish purple spots or brown blotches (Fig. 8). These symptoms are most easily confused with those of *B. cinerea*, since infection by either fungus causes irregular brown patches on bracts as well as on leaves.

Current season's shoots may be directly infected by *D. destructiva* during the spring or fall. Cankers formed in the spring are only a few millimeters in diameter and are quickly delimited by callus as the shoot grows. Extensive cankers, however, occur as the result of fungus invasion through the petioles of blighted leaves into the twig cambium (Fig. 9). Twig dieback is greatest during the dormant season. The infected twig turns tan after it is killed (Fig. 10), and canker margins are delimited by a reddish purple zone.

Fig. 2. Pacific dogwood (*Cornus nuttallii*) with leaf blight in lower canopy caused by *Discula destructiva*.

Fig. 3. *Cornus florida* seedling with leaf spots surrounded by a diffuse, yellow halo.

Fig. 4. Leaf spot and blotch on *Cornus florida* foliage.

Fig. 5. Leaf blight on *Cornus nuttallii*.

Fig. 6. Blighted leaves of *Cornus florida* sometimes overwinter on branches.

Fig. 7. Leaf spots 1 to 2 mm in diameter are typical of spot anthracnose caused by *Elsinöe corni*.

Epicormic branches form in clusters on the trunk and main branches of flowering dogwoods following dieback due to anthracnose. Since leaf blight is likely on shaded foliage, infection of epicormic branches via petioles of blighted leaves is very common (Fig. 11). Elliptical brown cankers sometimes form where dead epicormic branches join the supporting trunk or branch (Fig. 12). These cankers are of the annual type, enlarging for only a single season. The trunk and branch cankers become delimited by callus, and the affected area becomes sunken and cracked, in contrast to the surrounding healthy bark. On a tree that survives one or more seasons of severe anthracnose, the bark covering the cankers eventually sloughs off, and if conditions for disease development are less favorable in subsequent years, the cankers may become enveloped by the expanding trunk. This results in swollen areas on tree trunks and branches.

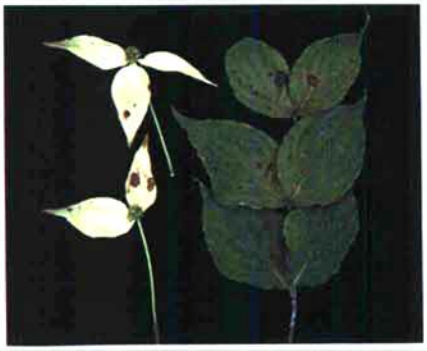

Fig. 8. Leaf and bract symptoms on the relatively resistant nonnative dogwood, *Cornus kousa* (courtesy R. L. Wick, University of Massachusetts).

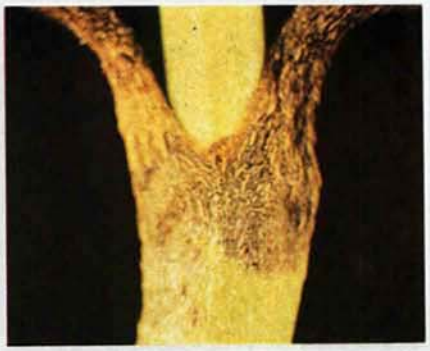

Fig. 9. Growth of the fungus from petioles into the shoot leads to canker formation.

Fig. 10. Shoot cankers are tan and covered with conidiomata of *Discula destructiva*.

The Pathogen and Disease Development

Redlin (39) described the development of asexual fruiting bodies (conidiomata). On leaves, these form most often on the undersurface, beneath a trichome (Fig. 13). Twigs with conidiomata remain on the tree over winter and provide abundant primary inoculum to begin new infection cycles in the spring. Under wet conditions, the conidia ooze from these fruiting bodies in a slimy white to beige or pinkish cirrhus (Fig. 14). It is likely that the mucilage surrounding the spores contributes to conidial survival (19). Conidia are 7 to 12 × 2.5 to 4 μm, hyaline, nonseptate, and smooth. (38) (Fig. 15). They have a distinctly truncate base and often contain polar guttules. No sexual state has been identified, but other species of *Discula* have teleomorphs in the genera *Apiognomonia* and *Gnomoniella* (*Gnomoniaceae, Diaporthales*).

Most short-distance dispersal of conidia probably occurs via splashing rain. Dissemination of conidia by convergent lady beetles (*Hippodamia convergens*) caged on *C. florida* has been demonstrated (18). The role of insects as vectors is being investigated. The fungus has also been isolated from fruit and seed of severely infected trees (11), which suggests that birds might contribute to dissemination of *D. destructiva.*

Conidial germ tubes penetrate leaves directly (26). Necrosis precedes hyphal proliferation in palisade and spongy parenchyma cells (49), indicating toxin activity. Four phytotoxic phenols have been identified in culture filtrates of the pathogen (48).

In culture, *D. destructiva* is slow growing and slow to sporulate. Colonies on potato-dextrose agar (PDA) are appressed and whitish, darkening to greenish gray, tan, or black. In one study, optimal growth of isolates from *C. nuttallii* was at 21 to 24°C; there was no growth at 27°C (40). Sporulation is enhanced by light and can be increased by growing on a medium prepared by adding dried ground oak leaves to water agar at 40 g/liter (32). This medium also reduces colony sectoring, which is common on PDA. Yao et al. (53) found that the presence and absence of double-stranded RNA (dsRNA) may explain some morphological differences observed in colonies that form sectors.

Although dsRNA has been identified in most eastern U.S. isolates of *D. destructiva* (31), banding patterns vary both within and among isolates from dogwood on East and West coasts. A collection of East Coast isolates all contained some dsRNA, while 76% of western isolates were negative (S. McElreath, *personal communication*). DNA fingerprinting analysis indicates a striking absence of genetic diversity among isolates of *D. destructiva* from both coasts (46). The results of DNA analysis, combined with the rapid spread and widespread destruction caused by the pathogen, strongly suggest a recent introduction of an exotic pathogen.

Fig. 11. Epicormic branches are produced on many diseased dogwoods and frequently become infected.

Fig. 12. Infected *Cornus florida* with the bark removed to reveal annual cankers formed at the base of killed epicormic branches.

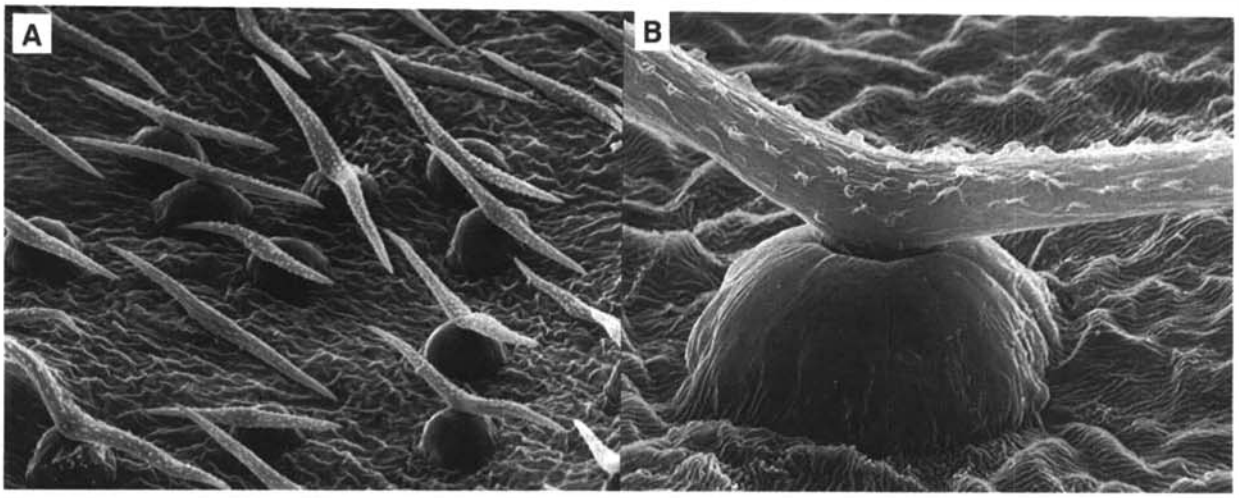

Fig. 13. Conidiomata of *Discula destructiva*. (A) Subcuticular acervular conidiomata developing under trichomes on leaf of *Cornus florida*, ×255. (B) Highly ornamented trichome above distended host cuticle and subcuticular conidioma, ×1190. Reprinted by permission from *Mycologia* 84(2):257-260, S. Redlin, Copyright 1992, The New York Botanical Garden.

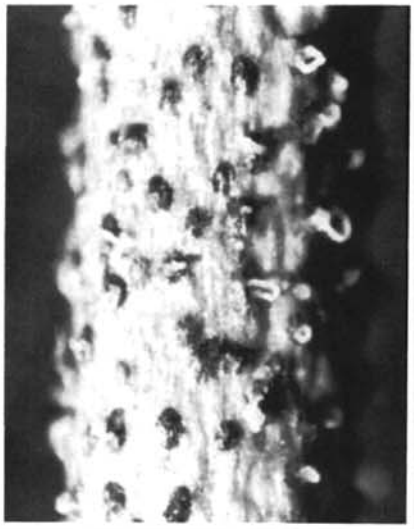

Fig. 14. Conidial ooze is produced on dogwood shoot cankers the spring following infection.

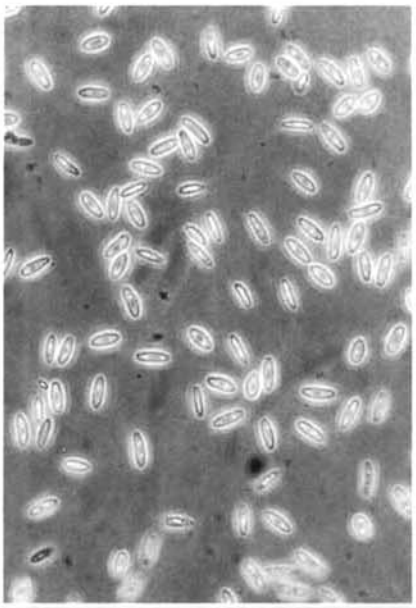

Fig. 15. Conidia of *Discula destructiva*.

Impact

The range of dogwood anthracnose is now extensive for both of its North American host trees (Fig. 16). After the reports of anthracnose on *C. florida* in New York, Connecticut, Pennsylvania, and New Jersey in 1983, the disease appeared to spread northward and southward. By 1987, with the addition of Massachusetts, Delaware, Maryland, Virginia, and West Virginia, dogwood anthracnose had been identified in more than 60 counties in nine northeastern states (2).

By 1988, the disease had spread further south, primarily along the Appalachian mountain range, and was discovered mostly in high-elevation sites and cool, wet valleys in Georgia, North Carolina, South Carolina, and Tennessee. Dogwood anthracnose was confirmed in Kentucky and Alabama in 1989 (22,30), in Ohio and the District of Columbia in 1990 (22), in New Hampshire in 1991 and Rhode Island in 1992 (A. Snyder, *personal communication*), and in Indiana (37), Vermont (A. Snyder, *personal communication*), and Michigan (K. Britton, *unpublished*) in 1993. The disease was detected in Missouri (D. Johnson, *personal communication*) and Kansas (T. Sim, *personal communication*) 1994; all of the confirmed cases in these states have been attributed to movement of nursery stock from infested areas.

After its discovery on *C. nuttallii* in Clark County (Vancouver area) in 1976, dogwood anthracnose spread further each year within Washington State, reaching Skagit County by 1981 (R. S. Byther, *personal communication*). By 1983, the disease was reported in Oregon and British Columbia, and in a coastal disjunct population of *C. nuttallii* occurring in one county in northern Idaho (along the lower Lochsa and Selway rivers, within 20 miles of their confluence) (41). In 1992, extensive tree mortality was reported in the population of *C. nuttallii* in Idaho (22). *C. nuttallii* in northern California were found to be affected in 1994 (M. T. Windham, *unpublished*). Although mortality from dogwood anthracnose has been reported for forest stands of *C. nuttallii* in British Columbia (47), western Washington State (K. Russell, *personal communication*), and northern Idaho (22), public concern and research activity in the Pacific northwestern United States has not been great (R. Byther, *personal communication*). This is partly because the Pacific dogwood is not utilized as an ornamental in the western United States as extensively as is the flowering dogwood in the east.

In the eastern United States, *C. florida* and its many cultivars are highly valued ornamental trees, commonly used in private and public landscapes, and are thus important to the nursery and landscape gardening industries. Spring bract displays of forest dogwoods are also admired from roadways and are important for tourism. Consequently, public interest was aroused due to the sudden decline of flowering dogwood during the late 1970s to early 1980s, especially in the region that includes southeastern New York, southern Connecticut, and eastern Pennsylvania. The impact of anthracnose on dogwoods was considerable, including the dieback and disfigurement of landscape specimens as well as mortality of forest trees. Early disease management efforts were costly and often ineffective.

Many valuable specimen trees succumbed during the first decade of anthracnose in the Northeast, especially trees under additional stress from other sources. However, most well-maintained dogwoods in open to lightly shaded sites survived and recovered their aesthetic value as or-

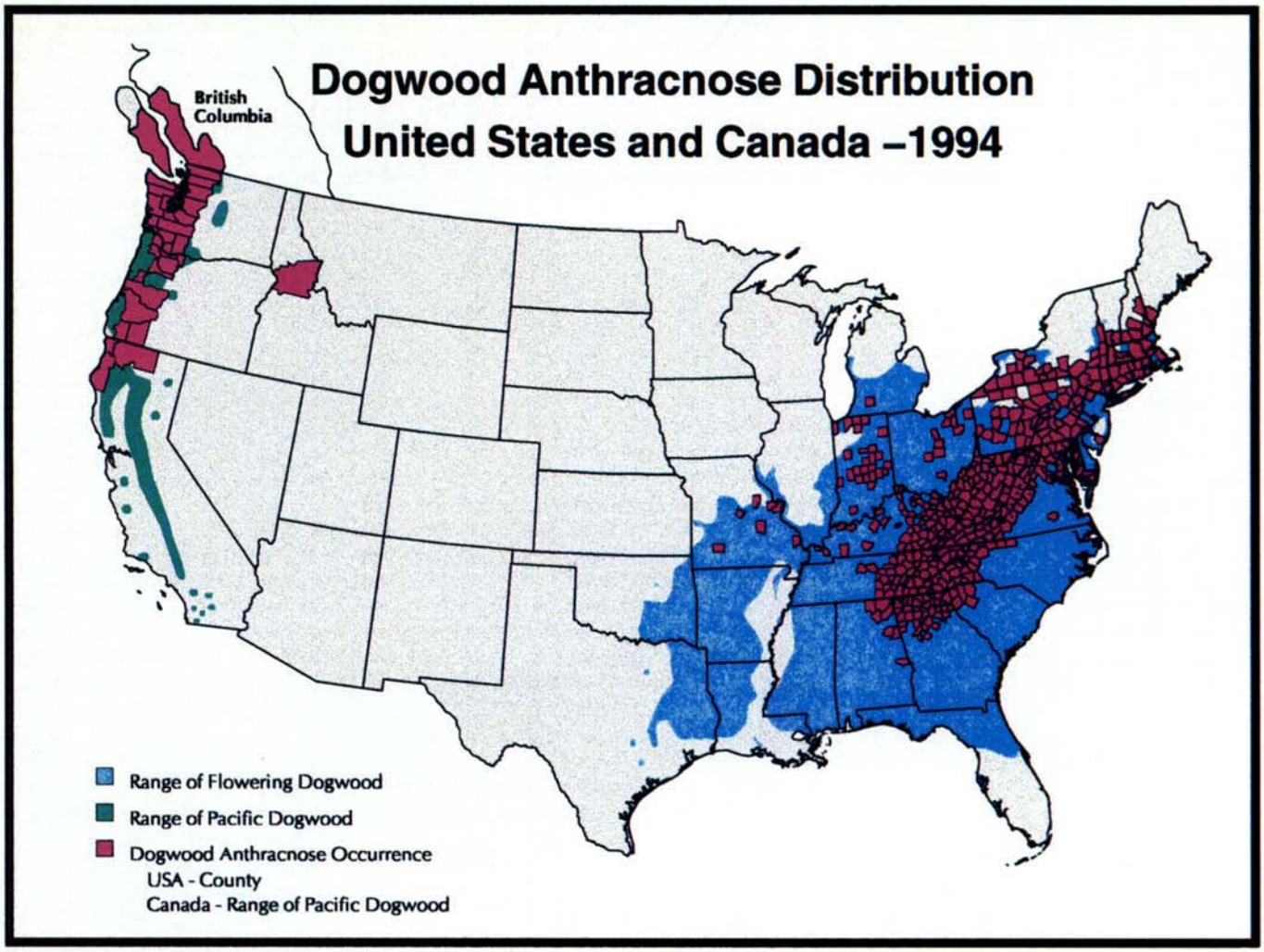

Fig. 16. Distribution of dogwood anthracnose in 1994, indicating disease on *Cornus florida* in the eastern and *C. nuttallii* in the western United States. Disease range is given by county in the United States; occurrence data include detections on out-of-state nursery stock (all cases reported in Missouri have been associated with out-of-state nursery stock). In Canada, *C. nuttallii* is affected throughout its range. Map courtesy Ed Yockey, U.S. Forest Service.

namentals. Although trees at the edge of the forest have in many cases survived, extensive mortality of *C. florida* occurred in some forest sites in southeastern New York. From observations in plots in the forested Mohonk Preserve (New Paltz, NY) over a decade after the introduction of anthracnose, an estimated 11% of the original population of dogwoods remained alive in 1991, and regeneration from seed and basal sprouts was minimal (C. R. Hibben and A. J. McArdle, *unpublished*). Beyond the aesthetic loss, the absence of native dogwoods and their fruits is probably detrimental to wildlife, because the fruits are a preferred food for game species such as turkey, bobwhite quail, squirrels, rabbits, and deer, as well as for numerous nongame species of birds (20).

Three impact assessment studies documented clearly how anthracnose severely affected native populations of *C. florida* in the mid-Atlantic and southeastern United States. In a 4-year study at Catoctin Mountain National Park, Maryland, tree mortality increased from nearly 33% in 1984 to 79% in 1988. From an average of 112 live dogwoods per hectare (276 per acre) in 1984, only 13 per hectare (32 per acre) remained in 1988. Every dogwood within the study area showed some effect of the disease (33,43).

From 210 plots in Virginia, North Carolina, South Carolina, Georgia, Kentucky, Tennessee, and Alabama, the hectarage with diseased dogwoods was estimated at 0.2 million in 1988 (0.5 million acres), 0.9 million in 1989 (2.2 million acres), 2.3 million in 1990 (5.7 million acres), 3.9 million in 1991 (9.6 million acres), 5.2 million in 1992 (12.8 million acres), and 7.0 million in 1993 (17.3 million acres). Tree mortality increased from none in 1988 to 23% in 1993, and dogwoods with extensive dieback increased from 4 to 13% during the same period (2,29,30; J. Knighten and R. Anderson, *personal communication*).

From plots in the Great Smoky Mountain National Park, severe epidemics (more than 25% of foliage blighted) were detected in 88% of the plots in 1992, which represented a 638% increase in the number of plots with a severe epidemic since 1988. Tree mortality had occurred in 56% of the plots by 1992 (29).

Environmental Effects

Flowering dogwoods occur naturally in many different types of habitats, including full sun, partial shade, and understory settings. Outbreaks of dogwood anthracnose have ranged in intensity from severely blighted and cankered trees in areas with high tree mortality to trees with minimal leaf symptoms and no tree mortality. Differences in epidemic severity have often been attributed to differences in the environmental conditions in different habitats.

Dogwood anthracnose severity is affected by sunlight intensity. Parham (35) found that lesions on foliage in full sun increased in size more slowly than did lesions on foliage in full shade. In addition, inoculum necessary for secondary infections was reduced on lesions in full sun, because fewer conidiomata formed within lesions and spore exudation was reduced (50). Erbaugh (23) found that varying the intensity of sunlight in combi-

nation with drought treatments had substantial effects on disease epidemics. Under the conditions of this experiment, trees in full sun (100% ambient sunlight) developed few dogwood anthracnose lesions, regardless of whether trees received adequate moisture. Trees growing in 90% shade (10% ambient sunlight) developed the most lesions—three to six times as many as did trees in full sun. Trees in 50% shade developed little disease if they received adequate moisture; however, if weakened by drought, the number of lesions increased 50 to 500%. Chellemi and Britton (15) compared dogwood anthracnose disease severity in the interior and exterior canopies of trees in full sun and full shade. They found that disease severity was highest in foliage of shaded trees and lowest in the exterior foliage of trees in full sun. Using porcelain bulb atmometers, they found that evaporative potential (an index of the drying potential integrating air temperature, vapor pressure deficit, air movement, and solar radiation) was inversely correlated with disease severity and was lowest in the canopies of shaded trees (Fig. 17). Similarly, symptoms observed on exposed dogwoods in the Northeast and Northwest are typically more severe in the lower canopy than in the upper, exterior canopy.

The amount and type of precipitation have been demonstrated to affect the intensity of dogwood anthracnose epidemics. Periods of drought have preceded severe dogwood anthracnose epidemics in the eastern United States, leading to the hypothesis that drought may predispose trees to greater vulnerability to the disease. However, moisture also plays a key role in facilitating epidemics by fostering infection. In the Northeast, leaf symptoms begin to appear following the first extended rainy period after leaf expansion (27,44). Infection of dogwood foliage can occur anytime during the summer if sufficient rainfall is present (22). In one study in North Carolina, the correlation of disease severity with rainfall improved after 1 June (6). Anderson et al. (3) exposed dogwood seedlings to simulated rain treatments varying in pH from 2.5 to 5.5 on 10 dates over a 42-day period prior to inoculation of the seedlings with conidia of *D. destructiva*. They found that the percentage of leaves infected and the severity of symptoms on infected leaves increased with decreasing rain pH. The increase in tree susceptibility may have been due to destruction of leaf cuticle by the acid treatments (12) or to changes in soil nutrient status (K. O. Britton, *personal communication*).

Topography of dogwood habitat may also affect disease incidence. Data collected in the southern Appalachians has been used as the basis for a disease prediction model (16,22). Dogwoods growing on north-facing slopes had higher levels of disease than did trees growing on east-, south-, or west-facing slopes (Fig. 18) (51). Evaporative demand (an index of drying potential) was measured in the plots and found to be lowest at sites with north-facing slopes. Aspect also influenced the level of disease development on individual trees (16). Lesions increased more rapidly in size on foliage on the north side of trees than on foliage on the east, west, or south sides of the same trees.

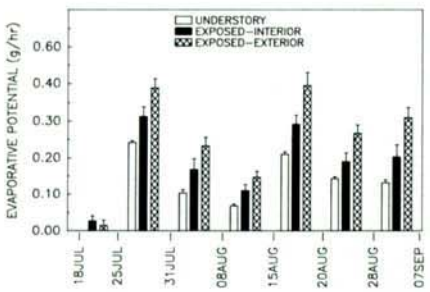

Fig. 17. Relationship between evaporative potential and disease severity under different dogwood canopy conditions. (A) Evaporative potential in interior and exterior canopy of exposed dogwoods, and of entire canopy of understory dogwoods. (B) Disease incidence and severity in interior and exterior canopy of exposed dogwoods, and in the entire canopy of understory dogwoods. Reprinted, with permission, from the *Canadian Journal of Botany* (Can. J. Bot. 70:1093-1096).

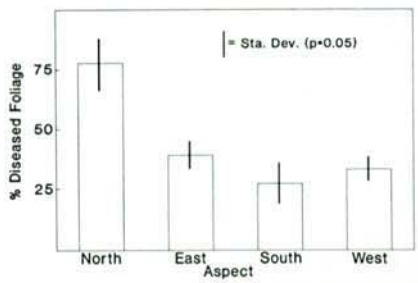

Fig. 18. Effect of plot aspect (slope) on dogwood anthracnose disease severity in plots (10 dogwood trees per plot) located in either north, south, east, or west aspects (four plots per aspect) in Great Smoky Mountain National Park, TN.

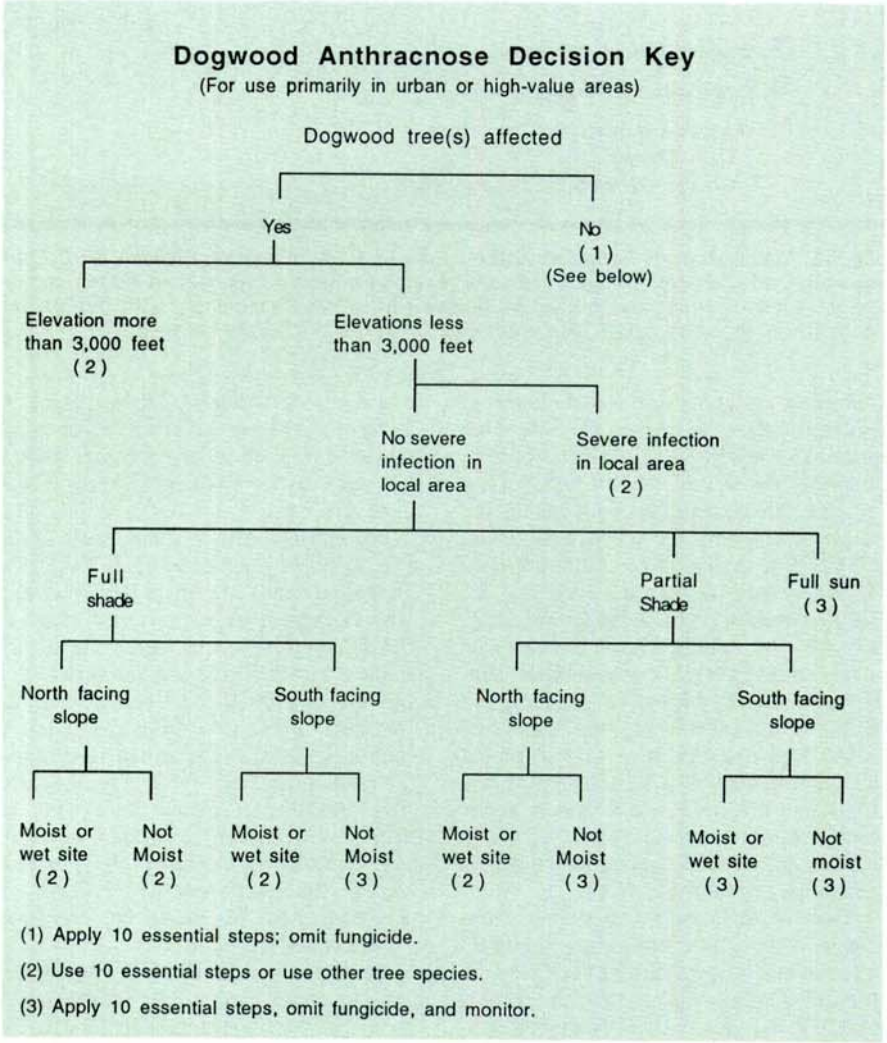

Fig. 19. A decision key for control actions to protect dogwoods against dogwood anthracnose, reprinted from Anderson et al. (4). The "10 essential steps" are cultural management techniques described in detail in Bailey and Brown (5).

Foliage on the east side of trees had the least increase in lesion size (51).

In the southern Appalachians, proximity of trees to streams and higher elevation have been associated with greater dogwood anthracnose severity (2,16,29). Trees located within 20 m of water declined more rapidly from dogwood anthracnose than did trees located further from streams over a 5-year period (30). By 1992, many trees located within 50 m of streams had died; whereas trees located more than 50 m from a stream suffered much less from dogwood anthracnose. At Lookout Mountain, TN, disease incidence was positively correlated with elevation in both urban and forest habitats in 1989. In 1990, however, due to high inoculum pressure and high precipitation levels, disease severity was high at all elevation sites, and the relationship with elevation was no longer evident (29,50).

Disease Management

A 10-point control program for dogwoods has been developed (5), which advises monitoring for disease using cultural management strategies and treating with fungicide when necessary. This program received the general concurrence of the Dogwood Anthracnose Work Group (DAWG), a group of 50 or more researchers, foresters, nurserymen, and regulatory personnel, which has met almost annually since 1988. These control measures have been refined based on site hazard ratings into a decision tree (Fig. 19) (4). Many of the findings reported at meetings of the DAWG are included in a recent review article on dogwood anthracnose (22).

Cultural conditions that favor the growth of dogwood, such as optimum fertilization, trickle irrigation, and at least 30% full sun, have been recommended (5,25). Adequate sunlight appears to be a very important factor in disease management, because disease increases more rapidly in understory conditions than in exposed locations (24,27), and surviving dogwoods are most often those with relatively high sun exposure. Mulching dogwoods improves aeration, increases moisture retention, reduces competition, and reduces trunk injury from lawn maintenance operations. Irrigation that does not wet the foliage may reduce the impact of anthracnose by preventing drought stress (24). Since short-distance spread is probably accomplished by rain splash onto conidiomata on dead twigs, pruning out branches bearing conidiomata may be of some value, although practical benefit has not been demonstrated (1,52).

Fungicides are recommended if disease pressure is high. Protectant fungicides labeled for anthracnose control on dogwoods, which have chlorothalonil, mancozeb, or thiophanate methyl as their active ingredients, must be applied every 7 to 14 days, beginning with leaf emergence and continuing throughout the period of leaf expansion and early growth. The systemic fungicide propiconazole need only be applied every 28 days. Later sprays may be needed if the summer is very wet (8,9). Systemic fungicides applied by trunk injections or as soil drenches have shown little promise in controlling anthracnose (2).

In forest environments, disease control efforts are limited by economic considerations. Stand management history can impact disease severity. Britton et al. (10) found anthracnose was less severe in stands of *C. florida* where the timber had been clear-cut 30 years previously than in stands where timber had been partially harvested. Dogwoods were also more numerous in clear-cut and control stands than in partially harvested stands. Since these plots were not established for the purpose of studying dogwood, damage during harvest to residual trees may have reduced the benefits of the release from competition. We do not know the effects of harvesting stands after anthracnose is present.

The greatest hope for long-term management of anthracnose is the search for resistant host material. *C. florida* and *C. nuttallii* are the only native *Cornus* spp. that have been observed to be naturally infected by *D. destructiva*. *C. florida* seedlings with some resistance have been tentatively identified (K. Britton, *unpublished*; M. Windham, *unpublished*). These trees are currently being propagated for further screening and eventual inclusion in breeding programs.

Resistance genes in other *Cornus* species make them useful as current planting alternatives for high-hazard areas, as well as good candidates for breeding stock or resistance gene donors. The nonnative *C. kousa* is relatively resistant (28,42) and possesses large floral bracts similar to those of *C. florida*. The cultivar *C. kousa* var. *chinensis*, selected for its larger bracts, appears to be less resistant than *C. kousa* (13). Other resistant species native to North America include *C. racemosa* and *C. canadensis* (22), as well as *C. amomum*, *C. alternifolia*, and *C. mas* (13). Hybrids of *C. florida* × *C. kousa*, recently released as the Stellar series by Rutgers University, also produce showy bracts and appear to have some resistance to anthracnose (7,34). Resistance levels within the series vary with the cultivar.

Movement of infected nursery stock has resulted in a number of potential new disease foci (D. Johnson, T. McCay-Buis, *personal communications*). Limiting the interstate movement of infected host material is recommended to reduce the rate of disease spread.

Questions to be Resolved

Since the onset of dogwood anthracnose in the United States in the mid-1970s, we have learned a great deal about the disease, especially concerning epidemiology, identification and characterization of the pathogen, and appropriate management strategies. Several important and interesting questions about this disease remain unresolved.

Where did it originate? No disease with symptoms similar to dogwood anthracnose was reported in pre-1970s surveys of dogwood diseases. Examination of herbarium specimens of various *Cornus* species revealed no fungi conspecific with *D. destructiva* prior to the 1970s (38). The coincidental appearance of the disease near ports of entry (Seattle and New York), its rapid spread, and its lethal nature are indications that *D. destructiva* is an introduced pathogen. The lack of variation in DNA amplification profiles of *D. destructiva* isolates from both coasts (46) provides additional strong evidence that the fungus was a recent introduction to North America. Although the introduction of *D. destructiva* through plants or seed of infected *C. kousa* imported from the Orient has been proposed, we are unaware of any confirmation of the fungus on *Cornus* hosts outside of North America. For now, the origin of dogwood anthracnose remains unknown.

What are the long-term effects on dogwood populations? From our field observations in southeastern New York, the disease impact on the current population of flowering dogwoods in northeastern United States appears to be much less severe than during the 1980s. Many landscape and roadside dogwoods have recovered from earlier infection, and foliar symptoms often are limited to scattered spots and blotches. Epicormic branches and new annual cankers appear less often. In woodlands, it is possible that earlier mortality of the most susceptible dogwoods has left the more resistant trees, but this explanation would not apply to trees in more open landscape sites, where mortality from anthracnose was less common. This apparent trend of decreasing disease severity in one region is puzzling and inconsistent with other introduced diseases of North American forest trees, most notably chestnut blight and Dutch elm disease (20). One explanation is that environmental conditions in the northeast may not have been favorable for severe anthracnose in recent years. Another explanation might be the reduction of the vast inoculum pool present during the height of the initial epidemic. Disease severity is still high at the southern edge of the disease distribution (K. O. Britton, *unpublished*).

Relatively little is known about the long-term effects of anthracnose on native dogwood populations, or on associated wildlife species that utilize the leaves or fruits as a preferred food. In woodland sites in Maryland and New York, where mortality of dogwoods was severe over a

decade ago, there are survivors, including seed-bearing trees, but as yet little regeneration by seedlings (22). These observations prompt several questions: Will such sites gradually become repopulated with *C. florida*? What effects will anthracnose have on this process, and how long will *D. destructiva* remain at a high enough level to initiate new infections?

One line of research is suggested by a unique biocontrol for the chestnut blight disease. European and American hypovirulent (weakened) strains of *Cryphonectria parasitica*, the chestnut blight pathogen, cause a less severe reaction in their woody hosts than do the normal virulent strains. Double-stranded RNA within the fungus has been shown to be the genetic factor responsible for hypovirulence (17). The discovery of dsRNA in isolates of *D. destructiva* has been discussed, and suggests research into possible hypovirulence in this pathogen.

Can we induce genetic improvements in dogwoods susceptible to anthracnose? Conventional breeding methods to produce interspecific hybrids between *C. florida* and *C. kousa* have been successful, but results take a long time (34). Selection for disease resistance in native populations of *C. florida* has been attempted, but the results so far are inconclusive (22,42). It may be possible by new genetic engineering techniques to achieve the direct transfer of genes encoding disease resistance factors from *Cornus* species other than *C. florida, C. kousa,* and *C. nuttallii.* This would have the advantage of quicker results and the transfer of the desirable resistance without compromising the ornamental qualities of dogwoods. A tissue culture system for regenerating *C. florida* from somatic embryos has already been developed (45). This will allow the reliable and efficient production of clones from genetically engineered dogwoods.

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