

SYCAMORE ANTHRACNOSE

by Dan Neely

Sycamore anthracnose has a long history. A report on this disease was published in England in 1815. This report is a three-paragraph description of the disease as it occurred in 1810. The disease was not named but the symptoms were described. There is little doubt that a serious infestation of sycamore anthracnose occurred in England in that year. In the 1880's a statement was made that *Platanus occidentalis*, the American sycamore, was no longer present in England. The severity of sycamore anthracnose rendered the species unsuitable for planting in that country.

From 1880 to 1905 numerous reports were published of epidemics of the disease in England, France, Germany, and the U.S. and of attempts to associate disease occurrence with certain weather conditions. The asexual stages of the causal fungus were found and described. The sexual stage of the fungus was found and proved pathogenic by Klebahn, a German, in 1905. He named the fungus *Gnomonia veneta*. In 1914 he found that he had goofed, that he had given the fungus a name someone else had previously used for a different fungus. He subsequently changed the name to *Gnomonia platani*. He published the change of name in a relatively obscure journal, however, and to this day, in the literature the causal fungus is often called *Gnomonia veneta*. E.B. Himelick and I called attention to this confusion in the literature in 1965. Now, some authors are using *Gnomonia platani* as the correct name for the sycamore anthracnose fungus.

From 1905 through 1925 additional studies were published on the causal fungus in pure culture, i.e., studies describing the growth of the fungus on different agar culture media and in different environmental conditions. Epidemics of sycamore anthracnose were related to cool, wet springs. Also, a disease similar to sycamore anthracnose was found on white oak and maple. Because the fungus in culture looked similar to

Gnomonia platani, it was assumed that *Gnomonia platani* caused diseases on several host plants. Today, in the host lists of many texts you will find that several tree species in different genera are affected by *Gnomonia veneta* (*Gnomonia platani*). This I believe to be incorrect.

The range of sycamore anthracnose in the United States is about the same as the range of the American sycamore, the eastern half of the country with the exception of Florida and northern New England. Sycamore anthracnose is more serious in the northern half of the sycamore range than it is in the southern half. Serious epidemics of sycamore anthracnose in the United States have been reported from Kansas, Iowa, Illinois, Michigan, Indiana, Ohio, West Virginia, Pennsylvania, New Jersey, Delaware, New York, Connecticut, and Massachusetts. In Europe, the disease is most frequently found in England, France, Germany, the northern part of Italy, the Balkan States, and southern Russia. It has also been found in Australia. The disease is well distributed over the earth.

Usually sycamore anthracnose does not kill trees; on occasion a nursery tree will die. Even though seriously defoliated during May and June, trees live through the disease. A succeeding flush of leaves will appear during the summer. Dieback does occur, however, and the horticultural value of the tree is decreased. Since the aesthetic value of the shade trees is great, anthracnose is considered a serious disease by many homeowners and plantsmen. Currently I consider it the most important of the canker diseases on deciduous shade trees in the Midwest.

When you look at a sycamore (*Platanus occidentalis*) during the winter, often you will see many twigs originating from a given point on the branch. This is not a true witches'-broom, but it approaches a witches'-broom in appearance. Such a tree has had a long history of sycamore anthracnose.

Later, in the spring when foliation has occurred, foliage on many trees may appear thin. Such a tree may be in full leaf only at the tips of the branches or in the top of the tree. When this situation occurs in May, the tree likely has sycamore anthracnose. A closer look at affected branches will reveal that some leaves are of full size and green, some leaves are small and brown and remain attached to the twig, and some twigs are bare of leaves.

Plant pathologists divide sycamore anthracnose disease symptoms into four phases. The phases can be distinguished partially by the time of year when they occur or the time of year when the fungus damages the tree. The first phase is called bud blight. The fungus has overwintered in the twig. During the early spring a brown area appears around the bud. The fungus girdles the vascular tissue leading to the bud before the bud opens. The fungus is present in both the bark and the wood and can readily be isolated by using laboratory procedures. The brownish discoloration in the wood may extend varying distances from the bud. It often is from $\frac{1}{2}$ inch to $1\frac{1}{2}$ inches up the twig and $\frac{1}{2}$ or 1 inch down the twig.

The canker that causes bud blight does not encircle the twig. If it did, the distal portion of the twig would be killed. When this happens, the second phase of the disease, twig blight, is present. Here the girdling of the vascular tissue occurs around the twig before any of the buds on the twig open. This happens early in the spring or possibly late in the previous fall.

A third phase of the disease is called shoot blight. Shoot blight results from either the girdling of a bud or the girdling of a twig after the leaves have emerged from the bud. Girdling of the vascular tissues at this time results in the death of the young leaves. Twig blight and bud blight are caused in exactly the same manner as shoot blight except that shoot blight occurs later in the year. Shoot blight is the phase that most people see and become concerned about. Shoot blight is the stage we most often attempt to control.

The fourth phase is leaf blight. This is common during the summer. Leaf blades have brownish, discolored areas with necrosis primarily along the

veins. Late spring infections may even occur on the petiole. When petiole infections occur, the leaf drops prematurely without the blades appearing damaged. Fruiting bodies of the fungus appear as black spots on the necrotic leaf blade, veins, or petiole tissue.

Plant pathologists are not certain how the leaf blight phase originates. The lace wing, a common insect pest on sycamore, injures sycamore leaves. Gene Himelick and I have frequently isolated the sycamore-anthracnose-causing fungus from the yellow spots that result from lace wing injury. It is possible that this insect is a carrier of the fungus. Also, since the disease is almost always found along the leaf veins, it is possible that the fungus is translocated inside the xylem of the tree and that the leaf infection is a result of an internal transporting of the fungus rather than an external inoculation.

We do know that the fungus overwinters inside the twig. In one Illinois study we tagged sycamore buds that were beneath leaves extensively invaded with the leaf blight phase of sycamore anthracnose. We put cellophane tubes over the twigs after defoliation to prevent infection of buds, shoots, or twigs from external sources. We wanted to find out if the fungus had moved out of the leaf, down through the petiole, and into the twig before the leaf fell off. We attempted to isolate the fungus from covered buds during the fall, winter, and spring. Forty-six buds were plated in September, and from 16 of them we recovered the fungus. The fungus had moved out of the leaf into the twig before defoliation. In December, 11 of 24 attempts to isolate the fungus were successful; in March only two of 16 attempts were successful, but we had established that the fungus can remain alive in the twig over the winter. We now know where the fungus is during that portion of the year.

Bud, twig, and shoot blight occurred within the cellophane casings to the same extent that it occurred in twigs exposed to external agents. Forty percent of the buds had bud, twig, or shoot blight when covered; 32 percent when uncovered. When covered and artificially inoculated, 50 percent of the buds were diseased. This additional evidence shows that the fungus is overwintering

in the twig and that external sources of the fungus need not be present to cause severe epiphytotic of sycamore anthracnose in the spring.

Sycamore anthracnose can look extremely serious in May and early June, but usually trees will not be killed by the disease. Trees with serious infections recover by July 15. Even though many buds and many shoots have been killed, secondary growth in June and July masks the disease symptoms and many people will not recognize that the trees were infected earlier in the season.

The disease cycle is as follows. The fungus overwinters within the one-year-old twigs of the tree. It can cause damage in the fall or spring by girdling and killing buds and twigs. In the late spring it girdles and kills shoots emerging from the buds. On the dying twigs, the fungus produces spores which can infect the leaves. The pimple-like protrusions of the bark on dying twigs are the result of fruiting body formation and sporulation. The spores may or may not be carried to leaves by lace wing insects, but they do arrive on the leaf surface. The leaves become diseased, as evidenced by the typical necrotic areas along the veins. As the necrotic area enlarges, the fungus grows down the veins, out of the leaf blade, into and through the petiole, and into the twig before the leaf falls. The fungus is then in position for the disease cycle of the following year.

My associates and I have attempted to determine which stages of the life cycle of the fungus are involved in causing sycamore anthracnose. *Gnomonia platani* has one sexual stage and several asexual stages. The sexual stage is rarely seen in nature. To obtain the sexual stage artificially, we placed sections of diseased leaf tissue on water agar in a petri dish in August. The petri dishes were incubated at 40 degrees F for several months. By December 15 the perithecia were present. We have obtained sufficient sexual-stage material in this manner for measurement studies, cultural studies, and spore germination studies.

Perithecia are the flask-like containers of the sexual spores of the fungus. The spores, called ascospores, occur in groups of eight enclosed in an ascus. The ascus of this fungus is quite dis-

tinative. It has a bright refractive ring at one end. Each spore is hyaline and unequally two-celled, a small cell on one end and a large one on the other end. Germination or growth from the spore occurs only from the larger cell.

My co-workers and I have been able to establish differences between the oak anthracnose fungus and the sycamore anthracnose fungus by using artificial inoculation techniques. We were not able to get the sycamore fungus to infect the oak, nor were we able to get the oak fungus to infect the sycamore. Since both cultural studies and inoculation studies show differences between the two fungi, we believe that they should be called by different names. The fungus that attacks *Platanus* (sycamore) should be called *Gnomonia platani* Kleb., and the one that attacks *Quercus* (oak), *Gnomonia quercina* Kleb. These names are properly published in the scientific literature but have not been used in most of the popular literature.

The shoot blight phase of sycamore anthracnose is often confused with frost injury. In frost-injured trees there is no evidence of a canker or discoloration of the wood beneath the bud. Also with frost injury often only a portion of the leaves on a shoot will be killed. The shoot will often survive and produce a twig even though the tip portion has been frosted. The entire shoot will be killed when sycamore anthracnose occurs.

Within the genus *Platanus*, differences occur in susceptibility to sycamore anthracnose. Individual plants within the species *P. occidentalis* differ appreciably. Also, there is a great difference in the susceptibility of *Platanus occidentalis* (American sycamore) and *P. acerifolia* (London plane). London plane often exhibits leaf symptoms but twig, bud, and shoot symptoms on this hybrid are rare.

Another of our studies has been to determine the conditions under which infections of sycamore anthracnose are severe. E.B. Himelick and I once toured ten states in the Midwest. On this tour, we rated trees according to the percentage of shoots affected by sycamore anthracnose. In the Southern states sycamore anthracnose incidence was zero percent. In one spot in Ohio, 80 percent of the sycamore shoots were affected. Intermediate ratings between these ex-

tremes were numerous. We were able to correlate these data with temperature data for that year and to reach these conclusions: when the mean daily temperature for a 2-week period immediately following the first leaf emergence was in the lower 50's, shoot blight severity was quite high. As the mean temperature increased from 56 to 60 degrees, shoot blight severity decreased. When the temperature averaged above 60 degrees for this period, no shoot blight symptoms of anthracnose appeared.

Illinois data on sycamore anthracnose incidence have been collected by Drs. J.C. Carter, E.B. Himelick, and me since 1944. During this time the severity of occurrence of this disease varied from none to light to severe. These data substantiated the stated relationship of temperature and anthracnose severity.

Using both the Illinois data and the tour data, we were able to correlate sycamore anthracnose severity with temperature but not with rainfall. Also, based on our understanding of the disease cycle, we see no reason why rainfall should be associated with the shoot blight stage of the disease. Since the fungus is already inside the twig, it does not need moisture to infect the tree. This is not to say that rainfall is not required for infection to occur from a source outside the tree which may result in the less damaging leaf blight stage.

Another facet of the disease that we have studied in detail has been the production and release of spores from the twig. These spores serve as inoculum to infect the leaves. These pustules are produced at precisely the same time that leaves are emerging from the buds. We have observed the development of the pustules microscopically after bark portions of twigs were imbedded, sectioned, and stained. The sycamore anthracnose fungus first formed a compact group of cells at the bark cambium. A pushing up of the bark occurred as the fungus mass increased in size. We call this fungus tissue a pressure cushion. For some unexplained reason, in sycamore anthracnose approximately 50 percent of the pressure cushions form near lenticels. The pressure cushion continues to grow until it ruptures the bark.

Immediately below the pressure cushion, the fungus forms a fertile, spore-producing area. The fertile area also increases in size. It either compresses the pressure cushion or pushes it out through the ruptured bark. Finally, the spores are released through the broken bark. These are the spores that can infect leaves.

Dr. J.C. Carter began fungicide control studies with sycamore anthracnose in the 1940's. He continued until 1958, when Dr. E.B. Himelick and I assumed the anthracnose work. Dr. Carter tested many of the fungicides used prior to the advent of the organic mercuries. He found that only the organic mercury fungicides effectively controlled the shoot blight phase of the disease. Apparently, the organic mercury fungicides have the ability to penetrate into the bark and slow down fungus growth in the bark, thus preventing much of the disease. No other fungicide tested to date has the localized systemic capability to do this. We know that this fungicide will control the disease if applied at the right time or times. We have been able to control the shoot blight stage in most years with one application, but, two may be required occasionally. The organic mercury fungicide should be applied when the sycamore buds are swelling in the spring. Studies conducted in New Jersey confirm that one spray application with an organic mercury fungicide will control the disease.

However, the organic mercuries are being phased out or prohibited from use by the U.S. Government, and we no longer can recommend the organic mercury fungicides for sycamore anthracnose control. At the present time we can recommend no fungicide that will control the disease. We are continuing our search for an effective fungicide in laboratory and field trials.

Sanitation is not a practical control measure. We have cleaned trees thoroughly through pruning operations, and the next year, when the temperature conditions were suitable for sycamore anthracnose to develop, the pruned trees were affected almost to the same extent as were trees that had not been pruned. We also believe that the raking of leaves is of no importance. Although the sexual stage of the fungus occurs on the leaves, the asexual stage fruiting

on the twigs provides an abundance of inoculum for disease dissemination and inoculation.

We do recommend that, if you are in an area where sycamore anthracnose is severe, you plant the London planes instead of the American sycamore. The London plane may not have the same shape or quite the same growth characteristics as the sycamore, but it is closely related horticulturally and is an acceptable substitute. Also, when trees have been severely damaged by anthracnose, we recommend fertilizing and watering to hasten secondary growth and increase tree vigor.

Sycamore anthracnose has been subjected to more experimentation and more observation than

has any other shade tree canker disease, yet we cannot fully describe or explain it. We know little about how the causal fungus initially enters the tree or genetic resistance to it. When the government pulled the organic mercury rug out from under us, we were left without even a control recommendation. At the rate at which we are progressing on sycamore anthracnose, a scientist could concentrate all of his efforts on this one disease and still be guaranteed a lifetime of work, because there's still so much to learn.

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ABSTRACTS

Shigo, A.L. and Alex Shigo. 1974. **Detection of discoloration and decay in living trees and utility poles.** USDA Forest Service Research Paper NE-294. Upper Darby, Pa. 11 p.

Discoloration and decay are major causes of damage to living trees, utility poles, and wood products throughout the world. They are caused by bacteria and fungi that digest wood inside of trees and poles, hidden from view. A method is described for detecting discoloration and decay in living trees and creosoted utility poles. The method and devices have come from research involving many people over a seven-year-period. A probe was inserted into a 3/32-inch diameter hole made by drill bits 8 inches and 12 inches long mounted in a portable, light-weight, battery-operated drill. The probe was attached by a flexible cable to a portable, light-weight, battery-operated meter, a "Shigometer", that delivered a pulsed electric current and measured resistance to it. As the probe was inserted into the hole, the meter measured in ohms the resistance of the wood in contact with the probe tip. As the probe was pushed inward, if the tip contacted only sound tissues, slight changes in resistance were measured. When the probe tip passed from sound wood to discolored or decayed wood there is an abrupt decrease in resistance. The magnitude of the decrease in resistance indicated the degree of discoloration or decay. The depth of the probe when the needle on the meter began to decrease indicated the position of the discolored or decayed wood.

Loomis, R.C. and W.H. Padgett. 1975. **Air pollution and trees in the East.** USDA Forest Service State and Private Forestry, NE and SE Areas. 28 p.

The benefits from trees are not limited to the rural countryside. Their importance in urban areas is recognized today more than ever. Among other benefits, they provide esthetic settings, cooling shade, and protection from wind, dust, and noise. Air pollution levels are high enough in some areas to cause plant injury. This is true not only in and around some of the larger cities, but also in rural areas where large pollution sources are present. The air pollutants discussed in this publication are sulfur dioxide, oxidants, fluorides, ethylene, oxides of nitrogen, ammonia, chlorine and hydrogen chloride, and particulates.