Petal Blight of Azalea

D. L. Gill

Azalea petal blight, or flower spot, is a striking disease. One day all the flowers on a plant or in a garden may appear normal to the casual observer. The next day they may be blighted as though hot water had been poured over them.

The disease was first noticed near Charleston, S. C., in 1931. In 1932, men who conducted a survey for the Department of Agriculture found the disease in all the gardens they visited within 20 miles of Charleston on both sides of the Ashley and Cooper Rivers. The disease spread rapidly. In 1935 it was discovered in Wilmington, N. C., and in 1936 in Savannah, Ga., Mobile, Ala., and New Orleans.

Freeman Weiss, then of the Department of Agriculture, found the disease in all the gardens they visited within 20 miles of Charleston on both sides of the Ashley and Cooper Rivers. The disease spread rapidly. In 1935 it was discovered in Wilmington, N. C., and in 1936 in Savannah, Ga., Mobile, Ala., and New Orleans.

Moved by the severity of the disease and the great value of the azalea flowers throughout the South, Dr. Weiss began investigations in 1933 at Charleston. He believed at first that the sudden appearance and rapid spread of the disease meant it was spread otherwise than by wind or rain. As insects were considered possible vectors, Floyd F. Smith, an entomologist in the Department, joined Weiss in the investigation in 1935. They continued to work together on the project through 1938.

Azalea petal blight appears first as small white spots on the colored flowers, or as brown spots on white-flowered varieties. The spots enlarge rapidly under favorable conditions until the whole petal or flower softens and collapses. Affected petals fall apart if rubbed gently between the fingers. Flowers injured by insects or weather or otherwise will not do so. The diseased flowers cling to the plant and present an unsightly appearance. Normally healthy flowers of Indian azaleas fall to the ground while still displaying their original color and shape and thus prolong their attractiveness.

Weiss and Smith found the fungus most commonly associated with the diseased flowers to be the cause of it. That point had been hard to prove because the fungus grows slowly and fails to produce spores in artificial culture. Because the fungus was previously unknown, Dr. Weiss named it Ovulinia azaleae.

Hard, black objects—the sclerotia—are produced on the old, diseased flowers while they still cling to the plant or after they fall to the ground. The sclerotia are one-eighth to one-fourth inch long. In that stage the fungus can live through the period when flowers are not present. Their presence confirms diagnosis of the disease.

Sclerotia spread Ovulinia into new areas when some of the surrounding soil is moved with the plants in transplanting. In spring, about the time azaleas bloom, the sclerotia on or just below the soil surface produce on the ends of short stalks small, brown, cup-shaped bodies, known as apothecia. The apothecia are one-sixteenth to one-eighth inch across. Apothecia continue to be produced for 4 or 5 weeks. They flatten as they mature, and
spores (ascospores) of the *Ovulinia* are forcibly discharged from their flat upper surface. A spore may reach a flower by the force of its discharge, or it may be caught in wind currents and carried to a nearby plant. If such a spore reaches a flower when conditions are favorable it germinates, sends a germ tube into the flower, and initiates a new infection. A few days later, as the flower blight progresses, secondary spores (conidia) are produced.

Up to several hundred thousand conidia are produced on a single flower. They spread the disease quickly. Infection thus is produced on successively opening flowers. Spores can produce infection at 40° to 80° F.; infection normally occurs between 50° and 72° and is at a peak at 65°.

High humidity, rain, fog, and dew favor the development of the petal blight disease—conditions frequently present in the South during the azalea season.

Azalea petal blight attacks all varieties of azaleas (*Rhododendron* species). Early-blooming varieties like *Elegans* may largely escape infection because there is not enough inoculum when they flower. The Macrantha azaleas usually bloom so late that conidial inoculum is again very low. Weiss and Smith found the disease on several unidentified hybrid rhododendrons growing interplanted with azaleas. Two true rhododendrons (*R. catawbiense* and *R. carolinianum*) became infected following inoculation.

*Ovulinia* attacks mountain-laurel (*Kalmia latifolia*) when it grows near azaleas. Flowers of highbush blueberry (*Vaccinium corymbosum* and its varieties *fuscatum* and *tenerum*) and of huckleberry (*Gaylussacia baccata*) were experimentally infected. The disease has been of little importance on them. All plants known to be susceptible belong to the family Ericaceae. Research workers could not infect plant parts other than flowers.

Because many insects visit azalea flowers, Weiss and Smith undertook an investigation to determine their relationship to the spread of petal blight. They soon learned that insect injuries were not necessary for the fungus to enter the flower. Early in the study, they thought it probable that spores carried by insects were responsible for the initial infection in the spring. It seemed unlikely that honey bees harbored the fungus in their colony or initiated early infection.

Because primary infection occurred near the ground, soil-inhabiting insects and animals and insects emerging from the soil were investigated, but these workers rarely found that they harbored *Ovulinia*. They could not indict them as carriers of primary inoculum.

Insects, they learned, can carry *Ovulinia* conidia from flower to flower but are not effective carriers until infection becomes general and large numbers of conidia are present. Some of the spore-bearing insects were observed to travel 5 miles in 8 days. Inoculation tests showed that they sometimes released spores gradually for several days. Insects therefore may be responsible for the introduction of the disease into nearby uninfected azalea gardens. Consequently any eradication program must be on an area basis if it is to succeed.

Some combined insecticidal-fungicidal dusts had no repelling effect on bees lighting on the flowers. The combination, or fungicidal dusts alone, did reduce the number of spores produced on the flowers and so reduced the number carried by the insects. Dusts made of stomach poisons had no effect on the insects carrying the fungus. Dusts that contained a contact insecticide, derris, pyrethrum, or nicotine sulfate killed adults of most species in 1 to 4 days. The insects were not attracted to poisoned sugar spray applied to the flowers.

The entomological investigations were closed with the conclusion that disease control, rather than insect control, appeared the logical method of attacking the problem. The studies
showed that insects were involved in the secondary spread of the disease, but that their role was less important than it had been thought to be.

Weiss and Smith pointed out that since the *Ovulinia* persists in the sclerotial stage from one flowering season to another, the chief control efforts should be prevention of sclerotial formation, removal and destruction of sclerotia that may form and reach the ground, and prevention of apothecial development by sclerotia that escape.

In accord with those recommendations, large numbers of diseased flowers were picked from the plants and destroyed. The suggestion was made that in isolated plantings the entire bloom for 1 year be destroyed as a means of eliminating the disease—but that was of no value, because some sclerotia were found to live at least 2 years. Because picking off all infected flowers proved to be an almost impossible task, the mulch around the plants was removed and destroyed after the old flowers had fallen. Many sclerotia were destroyed. A heavier mulch replacing the old then tended to prevent apothecia from pushing through to a point from which they could eject spores onto the flowers.

Those practices effected little or no control—at least a few apothecia were always produced to initiate infection, and (except in isolated plantings) secondary infections could be initiated by conidia from outside the treated area. Besides, old sclerotia sometimes remained hanging on plants a year after they were produced, and some sclerotia produced apothecia in the second flowering season.

Drenches applied to the soil about the plants were also tested. The shallow root system of azaleas makes injury from such applications a hazard. Surface applications of sulfur and copper, commonly used as dormant sprays, were ineffective. Acetic acid (1:1,000 to 1:600) did not injure azaleas even with repeated applications and was toxic to the *Ovulinia*.

Fungicidal sprays were applied to the plants before the flowers opened. Because *Ovulinia azaleae* infects only flowers after they show color, the sprays were valueless.

Spraying the open flowers with fungicides was also tried. Effective control by this method was considered unlikely by Weiss and Smith because the waxy surface of the blossoms prevented wetting, there was difficulty in obtaining coverage in the dense masses of blooms, and frequent spraying was necessary because of continuous opening of the flowers over several weeks. Lime-sulfur and copper sprays gave no control but produced no injury in azaleas except flower discoloration. Acetic acid (80 percent diluted to 1 part to 600 parts water) offered some promise when sprayed on open flowers and on the ground, but—because of its solubility in water—rains, dews, and fogs removed it and left the flowers exposed to infection.

A copper-clay or copper-lime dust containing 6.5 percent copper was effective in laboratory tests. Dusting azaleas in the open failed to control the disease if secondary infection was severe. The dusts left an objectionable residue on the flowers. Weiss and Smith concluded that overwintering sources of *Ovulinia* must be largely eliminated if spraying or dusting was to be effective.

Philip Bricrley, of the Department of Agriculture, followed Weiss and Smith at Charleston, working through the 1941 season. His approach was to attempt suppression of apothecial formation by application of chemicals to the soil just before the apothecia developed.

Other workers had found cyanamide (calcium cyanamide) effective against apothecia of the fungus causing brown rot of stone fruits and Elgetol (sodium dinitro cresylate) toxic to perithecia of the fungus causing apple scab. Cyanamide applied to plants containing large numbers of sclerotia at the rates of 200, 400, and 800 pounds an acre prevented apothecial formation. No
apparent damage was produced in azalea plants when 400 pounds an acre of cyanamide were applied around them. Elgctol was applied as a 1-percent solution at 450, 900, and 1,800 gallons an acre. It also prevented formation of apothecia. Similar results were obtained later with cyanamide at Spring Hill, Ala., and Baton Rouge, La.

I treated an entire isolated planting at St. Francisville, La., with 400 pounds of cyanamide an acre in 1942. Petal blight appeared later and failed to become as severe as in previous years. I could not determine whether infection originated from apothecia that escaped the treatment or from conidia from another planting. I observed no injury in azaleas and no lasting injury in other plants.

Dr. Brierley pointed out that two factors weigh against the practical use of a material suppressing apothecial formation: The danger of injury to azaleas or other plants grown in association with them and the need for application on a community-wide basis to accomplish significant reduction of the disease.

During the azalea seasons of 1944 and 1945, Cynthia Westcott represented the Department of Agriculture at Spring Hill, Ala. She assembled a number of new and old fungicides. These she tested in the laboratory and garden as protectants against conidial infection. Two offered good control of petal blight with a minimum of discoloration or injury to the flowers. They were: (1) Dithane D-14 (nabam), 1½ quarts; zinc sulfate, 1 pound; lime, one-half pound; and Triton B-1956, 1 fluid ounce to 100 gallons of water; and (2) Phygon (2,3-dichloro-1,4-napthoquinone), 1 pound to 100 gallons of water.

Applications were made three times a week. Satisfactory control resulted on sprayed flowers even when only half a plant was sprayed and infection was severe on the other half.

Those preparations were used in 1946 in a number of parks, estates, and gardens. All reported excellent control, but some found objectionable injury to the flowers. I followed Dr. Westcott at Spring Hill in 1946. I tested other fungicides, wetting agents, concentrations, and frequencies of application, and studied the nature of the flower injury.

Nabam (Dithane D-14 and Parzate Liquid) and zineb (Dithane Z-78 and Parzate) have been outstanding fungicides against petal blight in comparison with other materials. Dithane D-14 and Parzate Liquid sprays were prepared at the strength Dr. Westcott used. Dithane Z-78 and Parzate were used at the rate of 1.4 pounds to 100 gallons. A reduction in the concentration was considered desirable to reduce spray residue on the flowers and possibility of injury. Dithane D-14, 0.9 quart; zinc sulfate, 0.67 pound; lime, 0.34 pound; and Triton B-1956, 1 fluid ounce to 100 gallons, was found satisfactory. The quantity of zineb (Parzate and Dithane Z-78) was successfully reduced to 0.97 pound to 100 gallons.

Santomerse S (2 fluid ounces in 100 gallons) or Dreft (4 ounces in 100 gallons) were found to be satisfactory wetting agents to replace Triton B-1956 in making the Dithane D-14 spray. The wetting agents cause the spray to spread over the flowers in a film instead of forming drops that roll off. Lime is not essential in the spray.

Tests were conducted for 4 years with Dithane D-14 and for 2 years with Phygon, using one, two, and three applications a week. Spraying three times a week gave consistently better results than spraying twice, although many gardeners would have been satisfied with the control obtained with the latter. One application a week was unsatisfactory.

Dr. Westcott found some injury to azalea flowers by Phygon, but did not consider it objectionable in view of the control obtained. In later tests and in the hands of some gardeners, the
injury proved objectionable, particularly after three or more applications of the material. Injury consisted of bleaching and burning of the flowers from the margin inward. It was present to a lesser extent when the concentration was reduced beyond that affording satisfactory control. Some other quinone compounds produced similar injury. We no longer recommended Phygon.

In preparing the Dithane D–14 spray, Dr. Westcott used zinc sulfate containing 25 percent metallic zinc. Some growers who reported injury following the Dithane D–14 spray recommendations were using the same amount of 36 percent zinc sulfate. Comparisons of sprays prepared with equal quantities of the 25- and 36-percent material showed that injury resulted from the latter spray under conditions which tended to dry out the flowers. A reduction in the amount of 36 percent zinc sulfate reduced the injury. If the flowers were deficient in water for a longer period, injury was produced by Dithane D–14 sprays containing any concentration of zinc sulfate. A slightly greater water deficiency resulted in injury to unsprayed flowers. Spray injury consisted of burning and drying of the petals from the margins inward. Because large amounts of water are lost through the flowers and injury is severe with low water content, azaleas in flower should be watered during dry periods. Varying the zinc sulfate contents of the sprays had no effect on the control of petal blight. If no zinc sulfate was added, injury also resulted although control was obtained with three applications a week.

Use of dusts was investigated because of the ease of application, particularly in small gardens. Six-percent dusts of zineb (Parzate and Dithane Z–78) gave satisfactory control but were not as effective as the sprays. Use of dusts is recommended for small gardens not equipped to spray.

Applications of a protectant fungicide should begin at the first evidence of the disease—usually about the time early varieties (such as Elegans) are in full bloom or when midseason varieties (such as Formosa or Pride of Mobile) are beginning to bloom. Applications may be less frequent when all the flowers have opened and are covered with a fungicide. Spraying should be in the form of a fine mist directly into the flowers, but should not be continued until there is runoff of the material. In a number of show gardens, parks, parkways, estates, and home gardens, one of these control methods is used as a regular procedure.

D. L. Gill is associate pathologist in the Bureau of Plant Industry, Soils, and Agricultural Engineering. He studied at Louisiana State University and Cornell University. From 1935 to 1941 Dr. Gill taught ornamental horticulture at Louisiana State University. He has been with the Department of Agriculture since 1941, conducting research on diseases of azaleas, camellias, and other ornamental plants in Southeastern States. He is stationed at the Georgia Coastal Plain Experiment Station at Tifton, Ga.