AZALEA PETAL BLIGHT — ITS LIFE CYCLE AND CONTROL

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Abstract. The occurrence of azalea petal blight caused by the fungus *Ovulinia azaleae* is discussed with a description of the symptoms, host range, and life cycle of the pathogen and suggestions for control of the disease.

Petal blight of azalea, caused by the fungus *Ovulinia azaleae* Weiss, was first recognized in Sydney and in the nearby Blue Mountains in the spring of 1959 (12). The earliest records of the disease are from South Carolina, U.S.A. (13) and Japan (7) in 1931. The disease has also been reported from Great Britain (9) and Switzerland, Belgium and France (11). There is some doubt, however, about the identity of the fungus recorded from Great Britain (6). Since its first appearance in New South Wales it has also been reported from Queensland (17).

Symptoms. The earliest visual symptom of infection is the appearance of small circular spots on the petals. These spots are brown on pale-coloured cultivars and white or cream on dark-coloured flowers. In humid weather the spots enlarge rapidly and expand over the whole corolla or large areas of it. Affected corollas collapse rapidly but remain attached to the parent bush for several months following flowering. This is in contrast to uninfected flowers which are shed soon after they fade. If the weather is continuously humid, or if there are wet periods subsequently, small swellings develop on the diseased corollas and gradually turn black. These are resting bodies of the fungus, called sclerotia.

The flowers are the only part of the plant known to be affected by the disease and attempts to artificially infect other tissues of the plant have failed. Valder (12), however, reports that infection was observed on the calices and flower stalks of the cultivar 'Christmas Pink'. This had occurred where the unopened flower buds were infected and were hanging on collapsed stalks. No spores (conidia) were found except on the corolla itself, but sclerotia developed on affected sepals and flower stalks when they were detached and kept in a moist atmosphere. These infected structures remained on the plant throughout the summer and could be in very close proximity to the next season's flowers as they opened. Being restricted to the flowers the disease does not appear to have any adverse effect on the vegetative development of the host.

Host Range. This disease is known to occur in Australia on Kurume, Indica and other evergreen azaleas, deciduous azaleas,

many other *Rhododendron* species and hybrids, also *Kalmia latifolia*. Petal blight is most severe on evergreen azaleas and rhododendrons. In the U.S.A. it has been shown that other related plants such as *Vaccinium* spp. and *Kalmia* spp. can also be infected (15).

Conditions favouring the disease. Spread of the disease is favoured by mild damp weather. In the United States it has been shown that temperatures between 15 and 20°C are favourable for infection, with 18 to 19° being the optimum (14, 15). These conditions are usually found during late winter and spring in coastal areas in New South Wales. Free moisture is also necessary for infection to become established, but this moisture does not necessarily have to be in the form of rain. Humid weather resulting in dew formation, which could remain on the petals long enough for infection to take place, is sufficient. Shady locations, where drying is delayed and double-flowered plants, which permit films of moisture between petals, also favour disease. When conditions are dry the disease is not active in exposed situations where drying of the plant surface is rapid, but even in such conditions the disease can be observed in shade houses as long as temperatures are favourable during flowering.

Life cycle of the fungus. The disease is spread mainly by asexual spores or conidia. These conidia are typically ovoid, colourless, with an average size of 50 x 28 μm and are formed of two unequal sized cells, the smaller one being regarded as a basal appendage. Each condidium is borne on a hyphal stalk or conidiophore produced from the fungal mycelium with the affected petal tissue. As the conidia enlarge and the conidiophores grow longer, the conidia push through and rupture the cuticle. The conidiophores are produced very close together over the entire surface of the lesion on the petal resulting in a thick mat of conidia. Weiss (14) has observed that as many as 225 conidia can be produced on 1 mm^2 of petal surface. Conidia are produced on both surfaces of the petal but predominantly on the adaxial (inner or upper) surface.

Once the conidia appear above the surface of the petal they are readily detached and blown about by air currents and carried to the petals of other flowers or even of the same flower. Weiss and Smith (15) have reported that insects may also help in disseminating the conidia. When a conidium falls on to the surface of a petal it begins to germinate if sufficient moisture is present and the air temperature is between 15 and 20°C. [Portier (11) however, quotes the optimum temperature for conidial germination as 20-24°C.] A thin tube-like structure or germ tube is produced from the larger cell of the conidium which grows for a short time and then forms a swelling at the tip (appressorium) from which a hypha develops which penetrates the cuticle and continues to

grow and branch in between the cells of the petal. Ultimately the cells of the petal become free and lose their structure and the fungus grows in the fluid cell contents. The petal tissue continues to retain its general shape supported only by the cuticle, vascular tissue and infecting fungal mycelium. Soon conidia are produced from the mycelium and successive crops of conidia from infections such as this cause the disease to spread very rapidly in favourable weather conditions.

On the collapsed tissues of the petals a second type of spore develops. On the surface of the lesion, tufts of short spindle-shaped hyphae are produced which bear on their ends chains of spherical unicellular spores 3 to 3-5 μm in diameter. The exact function of these microspores is still unclear.

At the same time blister-like swellings develop within the collapsed tissue. They are translucent at first and then gradually darken and become black. These are sclerotia, composed of dense masses of fungal hyphae, which are meant to serve as resting bodies of the fungus. Sclerotia vary in shape but are usually distinctly cupped, the convex side corresponding to the abaxial (outer) surface of the petal. Minute spines are present on the convex surface of the sclerotium, while the concave surface is smooth. The exact function of these spines is not known although there is speculation (14) that they are receptive organs for the microspores acting as spermatia.

The sclerotia eventually fall to the ground under the infected plant. In climates with cold winters, stalk-like structures, singly or in groups of 2 or 3, are formed on the margins of the sclerotia in the spring when temperatures reach 10 to 14°C (14). At the top of each stalk a cup-shaped organ (apothecium) is formed which contains a number of cylindrical asci intermingled with sterile hairs or paraphyses. Each ascus contains eight ellipsoid, single-celled ascospores. When mature the ascospores are forcibly ejected and carried by air currents on to susceptible azalea flowers on which they germinate if moisture is present and temperatures are between 10 and 14°C. These infections cause lesions (primary infections) which subsequently produce conidia which in turn give rise to further infections.

Although careful observations have been made over a number of years, no further development of the sclerotium has been observed in this country. It seems probably that our winters are not severe enough to stimulate apothecial formation, although artificial cold treatment has also failed to germinate sclerotia.

It is possible that the fungus survives in our generally mild climate in the form of conidia. Portier (11) in France, found that conidia may remain viable for about 9 months at 6°C, for 2 months at 16°C and for less than 1 month at 26°C. In addition, in Au-

stralia, various cultivars of azaleas and rhododendrons help to maintain a succession of blooms almost throughout the year. In coastal areas around Sydney some cultivars bloom intermittently from February throughout the winter until the main flowering season in spring providing the fungus with suitable substrata to exist without the need for a dormant stage as in the colder climates.

Control. Protective sprays with fungicides such as zineb and thiram were the first recommended treatments for the control of this disease. In the U.S.A. nabam and maneb were also recommended. Although these fungicides are effective against the fungus it is impractical to maintain a protective coat of fungicide continuously over the surfaces of petals of newly opened blooms. Even with spray applications spaced at 2 to 3 day intervals only partial control is obtained. Removal of infected flowers does help to eliminate a considerable amount of inoculum, but even in combination with protective fungicides is insufficient to give reasonable control in weather favouring the disease.

Benomyl used as regular weekly or fortnightly sprays at the rates of 0.0125 per cent and 0.025 per cent respectively of active constituent, gave good control of the disease (3). The systemic nature of benomyl compensates for incomplete cover of the plant with fungicide and it functions both as a protectant and an eradicant. Peterson and Davis (10) in the U.S.A. used only two sprays at a 7-day interval and found that benomyl reduced disease by 37 per cent but chlorothalonil (Daconil 2787R) reduced disease by 57 per cent. In further experiments they found that benomyl and chlorothalonil applied at weekly intervals gave similar disease control (50 per cent) with benomyl also completely suppressing sclerotium formation. The best results were obtained with a combination of benomyl and chlorothalonil (58 per cent control). Holcomb (8) in a trial incorporating only two spray applications, found chlorothalonil, maneb and folpet superior to benomyl but made no mention of sclerotium formation. Chlorothalonil was inferior to benomyl in trials in New South Wales (1).

A significant finding in New South Wales was that plants sprayed with benomyl could still show symptoms of flower blighting, but careful observation revealed that the fungus *Botrytis cinerea* was the only pathogen present (3). Although normally *B. cinerea* is susceptible to benomyl, isolates from flowers previously sprayed with benomyl were able to grow in culture media containing up to 10 μg/ml of benomyl, whereas isolates from unsprayed flowers did not grow in media containing 2.5 μg/ml of benomyl (Bertus, unpublished data). Bollen and Scholten (5) found an isolate of *B. cinerea* on cyclamen sprayed with 0.25 per cent benomyl, that could grow on a medium containing 1000 μg/ml benomyl.

It is important, therefore, in assessing petal blight to determine accurately whether the associated fungus is *O. azaleae* or *B. cinerea*. It has been observed in the Sydney area that some blighted azalea flowers are only infested with *Botrytis* which can also colonize normally fading blooms. Some of the reports of failure of benomyl to control petal blight adequately could be due to failure to recognize *Botrytis* blighting, especially as the symptoms of this blighting closely resemble *Ovulinia* blight, with the difference that the typical *Ovulinia* sclerotia are not formed.

Experiments with systemic fungicides for the control of fungal die-back of camellia have shown that these compounds are more effective when applied to the root system as soil drenches rather than as foliar sprays (2, 4). This has yet to be proved with azalea petal blight, but if a similar effect is observed soil drenching will have application in container-grown plants.

At the present time, however, the chemical control programme recommended is the regular spraying of bushes alternately with benomyl and thiram commencing with the appearance of colour in the flower buds. These two chemicals should control both *Ovulinia* and *Botrytis*.

Another method of reducing the ravages of the disease would be to exploit any natural resistance in the genus *Rhododendron*. Hemmi and Akai (7) reported variability in susceptibility in a number of rhododendron species, when artificially infected with ascospores, conidia or mycelium. *R. mucronatum* was particularly susceptible and *R. indicum* most resistant (Table 1). The possibility exists, therefore, of breeding azalea cultivars resistant to the disease. Before this can be done it will be necessary to screen a large range of species and cultivars for resistance to petal blight under Australian conditions. The fungus has unfortunately proved difficult to grow in artificial culture in Australia although overseas workers have encountered no such problem. Should this drawback be overcome it will be possible to commence a breeding programme aimed at producing petal blight-resistant hybrids. Such a programme could possibly include resistance to other diseases such as *Phytophthora* root rot.

Table 1. Results of Artificial Inoculation of Flowers of *Rhododendron* spp. with *Ovulinia azaleae*. After Hemmi and Akai (7).

	Percent disease		
	Ascospore Inoculation	Conidial Inoculation	Mycelial Inoculation
R. japonicum	—	100.0	—
R. indicum	35.6	0	20.0
R. obtusum f. *japonicum* (*R. kiusianum*)	25.6	—	—

Table 1 (cont.)

	Percent disease		
	Ascospore Inoculation	Conidial Inoculation	Mycelial Inoculation
R. indicum (*R. lateritium*)	—	77.1	27.5
R. linearifolium f. *macrosepalum* (*R. macrosepalum* f. *linearifolium*)	66.7	—	—
R. mucronatum	100.0	100.0	61.6
R. obtusum	78.9	—	—
R. obtusum var. *kaempferi* (*R. kaempferi*)	78.3	—	55.6
R. pulchrum var. *calycinum* (*R. oomurasaki*)	97.8	—	0
R. reticulatum	—	—	0
R. reticulatum f. *pentamerum* (*R. dilatatum*)	64.4	—	—

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