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GLOMERELLA CINGULATA ON CAMELLIAS AND THE IMPLICATION FOR PLANT EXPORTS

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INTRODUCTION

In June, 1982, United Kingdom (UK) plant health authorities reported to New Zealand that many camellia plants imported from New Zealand over the previous few weeks were suffering from leaf blotch, leaf drop, stem dieback, and in extreme cases, death.

The causal organism was identified as *Glomerella cingulata* (Stone.) Spauld. and v. Schrenk (con. stat. *Colletotrichum gloeosporioides* Penz.). U.K. authorities contended that a new "camellia strain" of *G. cingulata* had been introduced from New Zealand with camellia plants and that this strain was capable of causing similar effects to that described by Ngo Huy Can, *et al.* (4) in USA.

Glomerella cingulata had not previously been recorded as causing disease of camellias in New Zealand, where it is generally regarded as a ubiquitous secondary pathogen commonly associated with tip dieback of plants (e.g. *Citrus* spp.) especially following winter injury, but important as a fruit rot organism (e.g. causing bitter rot of apples (3).

G. cingulata has been reported as a pathogen of camellia in USA (1) and Australia (2).

PATHOGENICITY TESTS

Field observations in UK had indicated that infection was prevalent on *Camellia* cvs. Donation and Debbie, although it was not confined to these cultivars. For this reason, *Camellia* cv. Donation was selected for use in the pathogenicity tests, which were undertaken as follows:

Experiment 1. Six *G. cingulata* isolates were each tested for pathogenicity on five *Camellia* cv. Donation plants. Each test was undertaken at two temperatures: 15° and 25°C. Each plant was inoculated as follows:

(a) Leaves were wounded by stabbing with a scalpel (six wounds per leaf).

(b) A leaf was removed, leaving an exposed leaf scar.

(c) An unwounded leaf was inoculated.

(d) A stem was cut and a droplet of spore suspension placed in the cut.

Spore suspensions were prepared to provide a spore concentration of $0.5 \pm 0.1 \times 10^6$ spores per ml. All inoculated parts of the plant were covered with a spore suspension of the test isolate, applied with a brush. The following isolates were used:

82/1 — ex U.K. (taken from a N.Z.-grown *Camellia* sp.)
82/2 — ex U.K. (taken from a N.Z.-grown *Camellia* sp.)
82/3 — ex N.Z. *Camellia*
82/4 — ex N.Z. *Citrus*
82/5 — ex N.Z. *Syngonium*
82/6 — ex N.Z. *Macropiper*
82/7 — Control — inoculated with sterile distilled water

Following inoculation the plants were placed in mist cabinets for three days, and were then removed and placed in growth cabinets (12 h day/12 h night) at either 15° or 25°C, as appropriate.

Results are given in Table 1, based on observations after 35 days (9.11.82).

Table 1. Mean from inoculation of both 15° and 25°C treatments.

	Inoculated (leaf lesions)	Wounded leaf (leaf drop)	Unwounded (leaf infection)	Leaf scar (infection)	Wounded (stem infection)
82/1	Tce	−	−	−	−
82/2	+	+	−	+	+
82/3	+	+	−	+	+
82/4	−	−	−	−	−
82/5	+	+	−	+	+
82/6	−	−	−	−	−
82/7	−	−	−	−	−

+ = infection, or positive result
− = no infection, or negative result

No differences in infection occurred between experiments undertaken at 15°C and 25°C, except that infection was more rapid and more severe after 35 days at the higher temperature.

Clear differences in pathogenicity toward camellias was observed among isolates, especially the two U.K. isolates

which had both been isolated from New Zealand camellias in the U.K.

It was of interest to find that the *Syngonium* isolate (82/5) was equally pathogenic to camellia as some of the camellia isolates, although we were unable to demonstrate any pathogenicity toward *Syngonium*.

In no case did the infection on inoculated plants cause disease further than one node below the inoculation point. Later observations showed that plants subsequently produced healthy young growth from below previous wound infections.

Experiment 2. Eight *G. cingulata* isolates were each tested for pathogenicity on four *Camellia* cv. Donation, four *Photinia* cv. Red Robin, and four *Mahonia aquifolium* plants. This experiment was undertaken to test U.S.A. isolates, and to test U.K. concern that the so-called "Camellia strain" of *G. cingulata* was capable of infecting a range of hosts, including *Photinia* cv. Red Robin and *Mahonia aquifolium*, which are exported in significant numbers to the U.K.

Inoculations were undertaken as in Experiment 1, except that following inoculation, plants were maintained only at 25°C.

Isolates used were:

82/1)	82/6)
82/2)	82/7)
82/3)	82/8) ex U.S.A. (from camellia)(Baxter, pers. comm.)
As above	82/9) ex U.S.A. (from camellia)(Baxter, pers. comm.)
82/5)	82/10) ex U.S.A. (from camellia)(Baxter, pers. comm.)

Results are given in Table 2, based on observations after 29 days following inoculation.

Table 2. Mean results from inoculation.

	Camellia cv Donation						
	Wounded leaf						
	(Leaf lesions)	(Leaf drop)	Unwounded (leaf infection)	Leaf scar (infection)	Wounded (stem infection)	*Photinia* 'Red Robin'	*Mahonia aquifolium*
82/1	–	–	–	–	–	–	–
82/2	+	+	–	+	+	–	Tce
82/3	+	+	–	+	+	–	–
82/5	+	+	–	+	+	–	Tce
82/6	–	–	–	–	–	–	–
82/7	–	–	–	–	–	–	–
82/8	+	+	–	+	+	–	–
82/9	+	+	–	+	+	–	Tce
82/10	Tce	–	–	Tce	+	–	–

+ = infection, or positive result
– = no infection, or negative result

There was no evidence of pathogenicity of *G. cingulata* islates ex camellias shown to *Photinia* 'Red Robin' or *Mahonia aquifolium*.

The U.S.A. isolates were also shown to be more severe in infection than one U.K. isolate (82/2), or the New Zealand isolate (82/3); one U.S.A. isolate (82/10) produced less infection than isolates 82/2 and 82/3.

DISCUSSION

On the basis of the above results it is our view that a specific "Camellia strain" of *G. cingulata* does not exist. We consider that in any given range of isolates of *G. cingulata* from camellias and other hosts, some will cause disease in camellias under conditions of wounding or following physiological stress to plants such as occurs with handling, transplanting, and acclimatation of exported plants; others will not. Although some isolates of *G. cingulata* were capable of infecting camellias through wounds in these tests, there was no evidence to suggest that any of them could be regarded as a virulent pathogen, as inferred by Bertus (2), and Baxter and Plakidas (1).

FUNGICIDE TESTING AND RECOMMENDATIONS

A range of fungicides were tested for effect on germination and growth of three *G. cingulata* isolates in culture in order to develop recommendations for a spray and dip programme for control of the fungus.

Of the fungicides tested, prochloraz (Sportak 50 WP) showed little effect on depressing germination of conidia, but effectively suppressed mycelial growth of the test isolates at concentrations of 10 ppm and higher. Captafol (Difolatan) and dichlofluanid (Euparen), on the other hand, were effective in inhibiting conidial germination at concentrations of 10 ppm and higher. Chlorothalonil (Bravo) suppressed germination of conidia at all concentrations tested (1, 10 and 100 ppm).

Prochloraz has not yet been fully registered in New Zealand for use on ornamentals. Therefore, the spray and dip programme which was developed took into account the assumption that if prochloraz could be obtained, it would only be available in limited quantities for experimental purposes.

The spray/dip programme developed also took into consideration fungicides known to be effective against *Glomerella* on other hosts, and *in vitro* work (unpublished), on control of *Monochaetia karstenii*, another secondary fungus common on camellias.

On this basis, the following spray/dip programme was recommended for camellias and other known or likely hosts being grown for export:

From four months prior to export: (Spray every 10 days alternating with):

1. Benzimidazole e.g. benomyl (Benlate) plus dichlorfluanid, both at full recommended rates.
2. Captafol at full recommended rates.
   (Prochloraz may be substituted in this programme for either 1 or 2, if available).

(As captafol can cause irritation to eyes, nose, throat, and skin of sensitive people handling the fungicide or sprayed plants, captafol sprays should be discontinued at least six weeks prior to export, and benomyl plus dichlorfluanid — alternating with prochloraz if available — continued at 10 to 14 day intervals).

The final dip is to be undertaken in either:

1. Benzimidazole, plus dichlofluanid, both at full recommended rates.
   OR
2. Prochloraz, at 25 g.a.i. per 100 l water.

Exporters were also advised to exercise care in handling plants when removing from growing medium, dipping, and packing to avoid injury.

IMPLICATIONS FOR EXPORT

It has become obvious that on occasions a ubiquitous fungus of little or no consequence as a pathogen in one country, may become of concern on exported plants in the country of destination. This situation may be the result of plants being weakened by transplanting, shipment, and becoming re-acclimatised in the country of destination, especially if this is in the opposite hemisphere. In this respect the physiology of the plants may also be affected, as observations by exporters indicate the air-freighted plants appear to be more suscepitble to *Glomerella* infection than sea-freighted plants retained in cool containers.

New Zealand must be prepared to undertake research to develop control measures where required to ensure that exported plants are of the highest quality and are free from pests and diseases in order to maintain continued acceptance of plant products in overseas markets.

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THE INTRODUCTION TO NEW ZEALAND OF ELMS RESISTANT TO DUTCH ELM DISEASE

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Elms (*Ulmus* spp.) are hardy deciduous trees common throughout the northern hemisphere. They are used both for timber and amenity plantings and for centuries they have been predominant in the European countryside in hedgerows, fields, and wooded areas. Their use most pertinent to New Zealanders is in the urban environment where they are used in street plantings and parks and are frequently seen in the larger home garden. This, however, could change in New Zealand as it has in Europe, the United States, and Canada with the advent of Dutch elm disease (DED), if this dreaded fungus disease ever reaches this country.

HISTORICAL BACKGROUND

There has been two major outbreaks of DED in the northern hemisphere. The disease was first identified in western Europe in 1918 by Dutch scientists — hence the name Dutch Elm Disease. In 1927 it was found in southern England where it caused the deaths of many elms. The epidemic reached its peak about 1936 and then declined with fewer trees being infected and the symptoms becoming less severe. Europe was not alone with its problems, as in 1930 the disease was also identified in the U.S.A. where it was reducing the American elm population.

In Europe, DED appeared to be controllable until the late 1960s when it became obvious that there was another epidemic in England and that the causal fungus was far more virulent than that which had previously infected the elm tree population. Research showed that the second epidemic had been caused by a more aggressive strain of the original fungus and