Association of a Species of *Itersonilia* with Parsnip Canker in Great Britain

A SPECIES of *Itersonilia* Derx was isolated by Wilkinson[1] in 1952 from cankers on parsnip roots in the United States. He did not publish a species identification, nor did Sowell[2] in a later note on the fungus; but cultures were afterwards lodged by Sowell at the Centraalbureau voor Schimmelcultures, Baarn, under the name *I. perplexans*. Wilkinson demonstrated the pathogenicity of the fungus and showed that it was also responsible for lesions on the leaves. I have made similar observations in Britain.

The fungus was first obtained, with five others, in the autumn of 1954, from tissue taken from the inner margins of parsnip canker lesions. Isolations were made by surface sterilizing, washing and planting small pieces of infected tissue on prune agar. During the winter of 1955–56, several isolates of each of these fungi were tested in the laboratory for pathogenicity to parsnip roots. Of the six fungi tested, only one was found to infect both wounded and unwounded parsnip tissue and this fungus was identified as a species of *Itersonilia* Derx. The Commonwealth Mycological Institute confirmed this identification and reported that in many respects the fungus resembled *I. perplexans*.

During the winter and spring of 1955–56, further isolations were made from cankerous lesions by the spore-dropping technique used by Wilkinson[1]. Pieces of diseased tissue were attached to the lid of a Petri dish with 'Bostik' and suspended over a plate of prune or Waksman's egg albumen agar[3]. When incubated at 21° C., colonies of *Itersonilia* frequently developed on the agar from ballistospores ejected from the pieces of diseased material. Inoculation of parsnip roots in the laboratory with several of these cultures resulted in rotting, similar to that produced by the 1954 isolates, and re-isolation by the spore-dropping technique from the artificial lesions established the pathogenicity of the fungus.

Itersonilia has been obtained from two types of lesion both of which are quite common in the field. The first type was dark brown to black in colour and extended 2–3 mm. into the tissue of the shoulder. Where the surface tissues were ruptured, the underlying parenchyma usually appeared slightly purplish. In the second type the surface of the root was invariably ruptured. The exposed parenchymatous tissue was light brown to orange-brown in colour, but frequently included areas which were darker brown and rotted. When inoculated into parsnips, the fungus produced lesions only of the first type. Thus although it was clearly the cause of this type of lesion, the significance of its association with the other type remains to be determined.

The fungus has been obtained from parsnips grown at the National Vegetable Research Station and at two farms in Buckinghamshire and one in Kent.

Leaf infection of young plants has been demonstrated by single or successive sprayings of a macerated mycelial suspension on to the foliage of potted parsnips in the glasshouse. The lesions developed within six days and appeared as silvery green patches approximately 1 mm. diameter or as larger patches, each consisting of a necrotic centre surrounded by a light-green halo. The re-isolation of *Itersonilia* from the lesions by the spore-dropping technique confirmed that the lesions were caused by the fungus.

The present work fully supports that of Wilkinson and indicates that a fungal pathogen hitherto unreported in Great Britain is associated with parsnip cankers in Britain. Its occurrence is apparently widespread; but further work will be necessary before it can be determined whether *Itersonilia* is responsible for most, or only part, of the somewhat variable symptoms on parsnips at present grouped under the term 'canker'.

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[1] Wilkinson, R. E., *Phytopath.*, **42**, 23 (1952).
[2] Sowell, G., *Phytopath.*, **43**, 485 (1953).
[3] Waksman, S. A., *Soil Sci.*, **8**, 71 (1919).

Control of Coffee Berry Disease in Kenya

A DISEASE of coffee berries, attributed to a form of *Colletotrichum coffeanum* Noack, was first described from West Rift areas of Kenya in 1922[1]. It caused extensive losses and the abandonment of coffee cultivation on many estates, and although by 1939 selections of coffee which showed some degree of resistance had been made, direct attack with a considerable range of fungicides had led to little, if any, control of the disease. A detailed account of the disease and of investigations up to 1950 has been published[2].

Previous work[1] had shown that disease symptoms could be reproduced by the inoculation of the pathogen into wounded or detached berries; but the degree of pathogenicity was not established until symptoms were produced using spore inoculum without wounding[3].

In 1951, the disease appeared for the first time in the East Rift coffee area, the higher-altitude zones being affected. As this area produces the greater proportion of the Kenya coffee crop, investigations on the disease received a considerable impetus. Further fungicide trials were carried out using 'Perenox', phenyl mercury, 'Fixtan', 'Tulisan' and calcium sulphamate; but no degree of control was observed.

Early in 1955, the effects of a wide range of fungicides were tested on an estate in the East Rift area. Fifteen treatments were compared using six replications of a balanced, incomplete block design with three plots per block. Each block consisted of one head of three multiple-stem trees, the trees selected having three equal heads each. In this way, individual tree variations in susceptibility and yield could be statistically controlled. The following fungicides were applied at monthly intervals during the long rains (March–May) and the short rains (November–December), giving a total of five applications; 'Perenox', 'Karathane', 'Verdasan', 'Fermate', 'Zerlate', 'Dithane', 'Actidione', 'Captan', 'Glyoxalidine' and calcium sulphamate. In addition, 'Perenox' was applied at fourteen-day intervals throughout the season.

Only 'Perenox', applied at fortnightly intervals, and 'Verdasan' gave an effective control of the disease. No control could be detected when 'Perenox' was applied at the same frequency as 'Verdasan'. As