PRELIMINARY NOTE ON YELLOW-LEAF DISEASE OF PHORMIUM

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Attention was first drawn to yellow-leaf disease of Phormium by outbreaks in the Manawatu *Phormium* areas some forty years ago, following extensive exploitation of indigenous induced stands for the *Phormium* fibre industry. The disease is characterised by abnormal yellowing of leaves, stunting of growth, and premature flowering; other symptoms are discolouration of vascular bundles in the rhizome, root death and rhizome rotting, leading to wilting and collapse of the leaves, the invasion of all parts by secondary organisms, and finally disintegration and death. From the appearance of the first symptoms to the final collapse the course of the disease may take from a few months to several years.

Surveys have shown that the disease is present in both the North and South Islands of New Zealand, but it is much more prevalent in the North Island; its incidence is higher in natural stands of *Phormium tenax* Forst., than in those of *P. colensoi* Hook.f., though both species are affected. A long standing and well substantiated observation is that outbreaks of the disease have followed extensive draining and protection from flooding of *Phormium* stands.

Accurate assessments of economic losses due to the disease are unobtainable; it is known, however, that the inroads of yellow-leaf were responsible in part for the deterioration and eventual disappearance of many thousands of acres of induced *Phormium* in the Manawatu district. At the present time it threatens the production of half the remaining *Phormium* producing areas in the North Island.

Equally serious have been the limits set by the disease to efforts to domesticate the plant, in the course of which attempts to improve fibre production by selection, breeding, and improved cultural methods, have been nullified by the disease. Particularly has this been so on the Moutoa *Phormium* area, Foxton, where since 1940 the New Zealand Government has linked development and research to maintain a source of fibre.

Since its first appearance, numerous investigations into the nature of the disease have been carried out; in these, fungal, bacterial and virus pathogens have been postulated and physiological factors considered, but the actual cause has remained obscure, until recent work, reported briefly here, provided virtual proof of its virus nature.

In all earlier investigations, the main obstacle to progress has been failure to transmit the disease or to induce recovery in diseased plants. The failure of numerous attempts to find a fungal, bacterial, or physiological cause, indicated the possibility of yellow-leaf being a virus disease.

Chamberlain in September, 1947, working with mature *P. tenax* plants, obtained several apparently successful root grafts between diseased and healthy plants. Three healthy plants showed symptoms of disease by March, 1950. This result is not inconsistent with the existence of a latent period of up to three years suspected from field evidence.

Fry, Matthews and Newhook in November, 1949, set up mechanical transmission trials using the carborundum abrasion technique on eighteen months old *P. tenax* seedlings. A high percentage of transmission was obtained, symptoms showing in some plants after four to six months and in others after sixteen months.

Insect transmission was first obtained by Cumber who, after detecting the presence of an endemic plant hopper, *Oliarus atkinsoni* Myers. (Hem. Cixiidae) in large numbers at Moutoa, initiated transmission experiments on 2 December, 1949. He used adult hoppers collected in the field from diseased plants, and applied them to seven-to-eight months old *P. tenax* seedlings. A high percentage of transmission was obtained; excessive leaf yellowing was apparent in from four to seven weeks, and on final examination (24/5/51) sixteen out of twenty test plants showed typical internal symptoms of yellow-leaf.

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This result was confirmed by Fry, Matthews and Newhook in a trial with eight months old seedlings set up at Auckland on 16 December, 1949, using adult *O. atkinsoni* from the same area at Moutoa as those used by Cumber.

Cumber established further large scale transmission trial in December, 1950, using eighteen months old seedlings. Fry, Matthews and Newhook in January, 1951, set up an extensive trial in which hoppers from an apparently yellow-leaf free area near Kaikohe were used as checks. These experiments confirmed transmission of the disease and showed that the symptoms obtained were not likely to be due to insect feeding toxicity.

Transmission trials with other insects and other possible host plants have so far given negative results. In some such trials set up in Auckland to test transmission by thrips, mealy bugs and aphids, the aberrant nature of the results did, however, indicate the possibilty of seed transmission of the virus. A high incidence of infection developed in some sets of uninoculated check plants obtained from one seed source; check plants from two other seed sources remained healthy. As plants from all three sources had comparable treatment, the most likely explanation for the appearance of yellow leaf in only one of these, is that some seed transmission had occurred.

Strzemienski used dyes and radioactive phosphate to compare translocation in diseased and healthy plants. In diseased plants, roots translocated dyes and phosphates, and an unimpeded movement of dyes was observed in severed leaves. Yet transfer from roots to leaves was interrupted, presumably in the rhizome.

Newhook extended this work with dyes and showed that this interruption to translocation was caused by xylem gummosis associated with phloem necrosis in the rhizome vascular bundles. These symptoms are at first only demonstrable by staining with phloroglucinol and acid, although in later stages the vascular tissue is visibly discoloured pale yellow to dark red. Their occurrence in leaf and root traces in the rhizome, with consequent hindrance of dye uptake, precedes leaf yellowing and wilting, root death and rhizome rot.

The results of transmission experiments can now be taken as virtual proof that yellow-leaf of *Phormium* is caused by a virus.

The role of *Oliarus atkinsoni* in transmitting the disease may explain the long standing observation that outbreaks of this disease have followed drainage and protection of *Phormium tenax* from flooding; in its nymphal stages the insect is located on the roots and leaf bases from March to November; here the nymphs are vulnerable to inundation lasting a few days, whereas it is known that the plant can withstanding flooding to a depth of two to three feet for periods up to four weeks during the winter months. Controlled flooding promises to provide a valuable control measure in some areas.

The part played by cutting operations, and other factors in transmitting the disease by mechanical means has not yet been investigated.

This is the first record in New Zealand both of a virus in an indigenous plant and of an endemic insect vector; examples of endemic viruses in endemic plants are rare; *Phormium* is endemic to New Zealand and Norfolk Island. The evidence to date suggests that the yellow-leaf virus is a new one and should it prove to be endemic, the relationship would be of interest particularly in view of New Zealand's long standing geographical isolation.

Detailed accounts of this work will be published in this Journal.