Q. Jl. Rubb. Res. Inst. Ceylon (1971) 48, 92—99.

BLACK ROOT DISEASE OF *HEVEA* CAUSED BY *XYLARIA THWAITESII*

BY

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INTRODUCTION

The Black Root disease of *Hevea* caused by the Ascomycete fungus *Xylaria thwaitesii* (Cooke), is a root disease of the rubber tree, new to our generation of rubber growers. The taxonomy of the fungus has been, and in our own opinion is still debatable, but for the purpose of this paper we shall use established nomenclature. The fungus was first recorded by Cooke in 1883, on unspecified wood in Ceylon and recorded as a cause of a root disease of *Hevea* by Petch in 1910. The next record came a decade later in 1921 - 22 and the disease symptoms were described by Petch in brief in 1923. No further records of the occurrence of the disease are available for the next 45 years until Mr. H. C. P. Kalpage noticed the unusual fructifications in the Kempitiya Division of Hatbawe Estate, Rambukkana, in 1968. Petch's records are also from the Kegalle area, but unfortunately he does not give a specific location. Once we learnt to recognise the pathogen, the symptoms and the disease, it was observed to be fairly widespread in 5 to 20-year old trees on both divisions of Hatbawe Estate.

SYMPTOMS

The first recognisable above ground symptom is the appearance of clusters of fructifications which arise from dead lateral roots, or more generally, at the collar of the tree (Fig. 1). The young fructifications arise as white knob-like structures, 0·5 – 1·0 cm in diameter, developing in about 20 – 30 days into clusters of erect clubs 2 – 6 cm high and 0·5 – 2 cm in diameter (Fig. 2). They may arise often from flat mycelial mats or from definite stroma or basal masses. The basal mass, which always remains subterrenean may arise from a mycelial stalk growing on small laterals originating from an infected root. Each cluster may have 2 – 30 fructifications and are variable in shape. They are mostly club-shaped but may also be conical, cylindric, ovoid and rarely laterally flattened. In transverse section they are white internally and brownish grey to black in its thin peripheral perithesia-bearing layer. Perithesia occur profusely on the basal masses and the lower parts of the fructification but are less dense and sterile as the upper portions are approached.

On lateral roots the fungal colony advances roughly fan-wise. The advancing edge is white externally but rapidly turns grey to black in colour. With age, the mycelium aggregates into bands or streaks which later form a closely knit network forming a continuous or patchy thin, smooth, black skin on the root surface (Fig. 3). Encrustation of soil particles by the mycelium occurs rarely, and in any case not to the extent characteristic of Brown Root disease.

Internally the cortex becomes discoloured from its normal creamy or off-white colour to a dull brown colour, usually 1 – 2 cm in advance of the fungus. Later the cortex rapidly becomes necrotic and deep brown when fully colonised internally (Figs. 4 and 5).

At the early stages of infection the wood of lateral roots shows a light brown discolouration 2 – 5 mm deep, immediately below the cortex. Later the discolouration extends well into the inner wood which becomes more discoloured, but remains

Fig. 1. Clusters of fructifications arising from the root

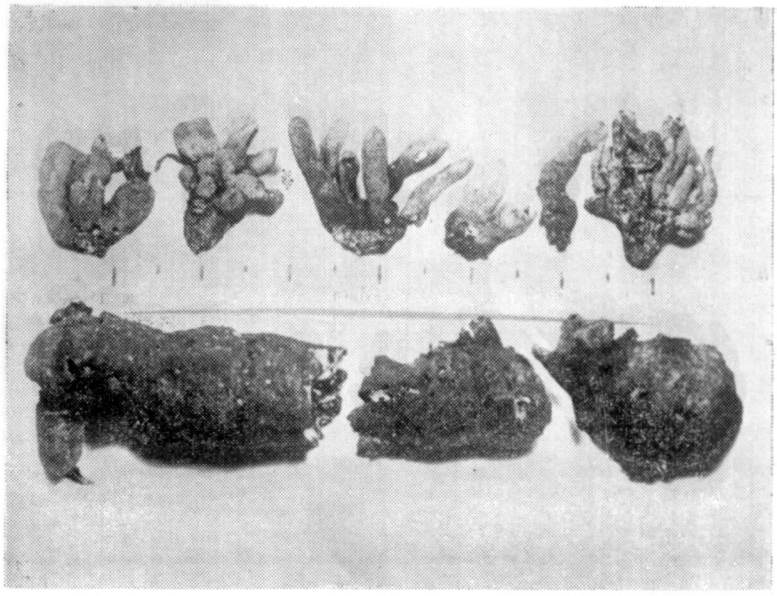

Fig. 2. Various types of fructifications observed in the field

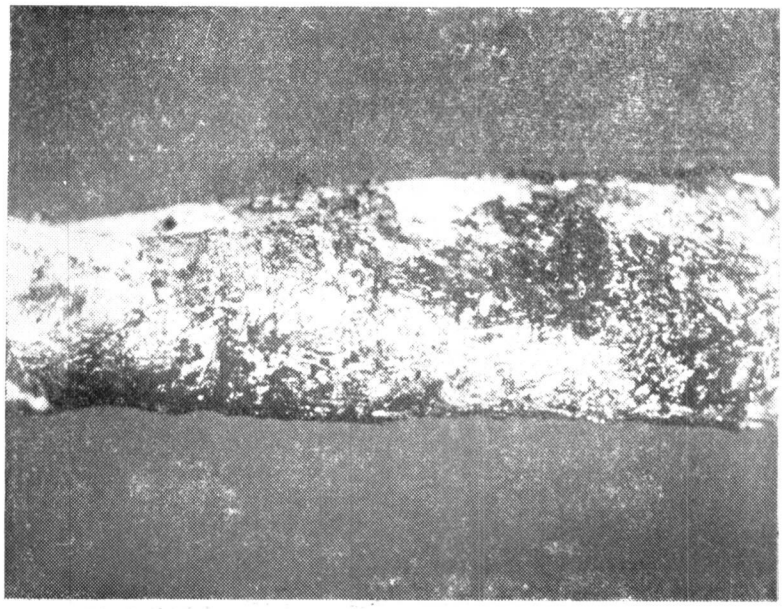

Fig. 3. External symptoms on infected root

Fig. 4. Longitudinal section of infected root showing affected wood

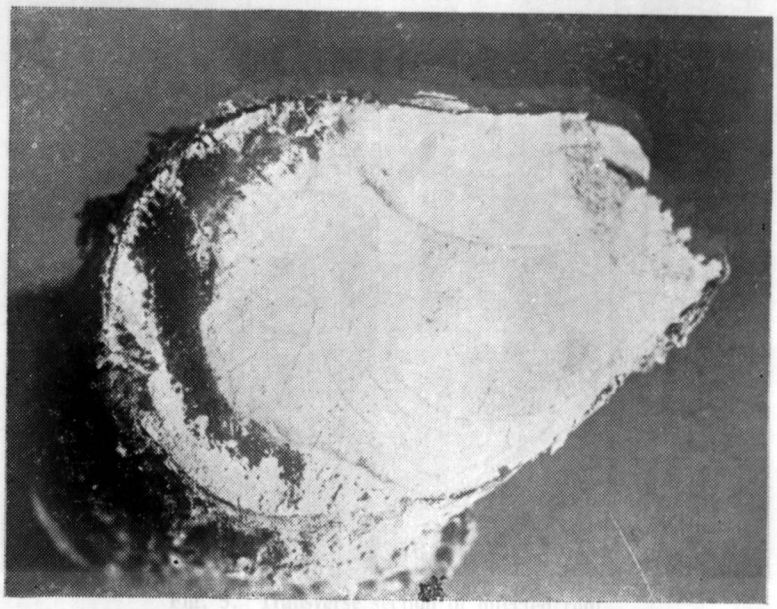

Fig. 5. Transverse section of infected root

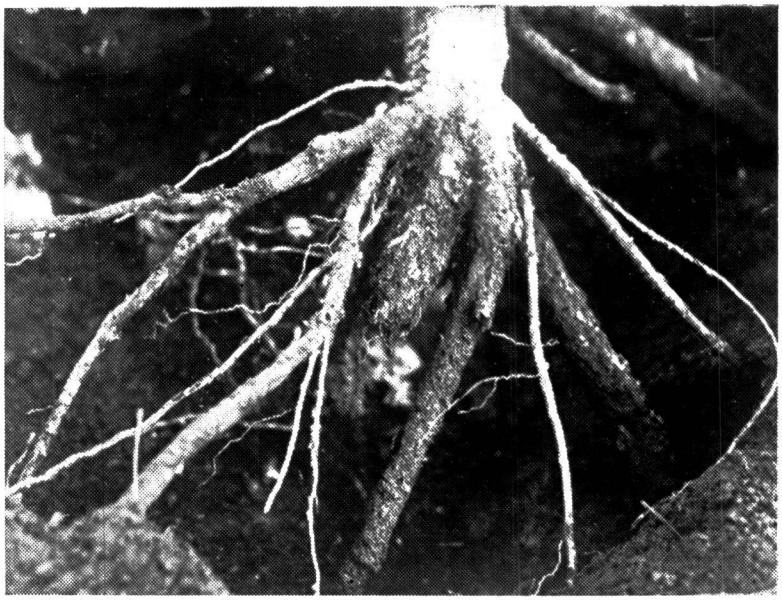

Fig. 6. Exposed root system of young tree showing progress of infection

hard. A characteristic feature is the wetness of this region which remains quite hard, until the final stages of decay. On splitting the root, minute droplets of moisture may be made to exude by pressing the point of a pen-knife into the wood. The rot extends into the inner wood which is gradually transformed into a soft pulpy and extremely wet condition. Early descriptions state that, on breaking the root water will often spurt into the face, but we have not found anything as dramatic as this. Latex exudation from infected roots is not uncommon.

The junction between affected and healthy wood is often sharply demarcated by a brownish or black line. In large laterals short black segregated lines or black zonation lines are not uncommon. The lesion in the wood progresses more slowly than in the cortex.

The fungus having established itself on a small or large lateral root, progresses towards the tap root, and if unchecked girdles it killing the tap root cortex and from there proceeds to infect the other large laterals and secondary roots (Fig. 6). The symptoms on the tap root are similar to those already described, except that latex exudation may be more pronounced. The growth of the fungus and lesion formation is slow compared to the White Root disease. It has been observed that on a root of 3 – 5 cm diameter the progress of the lesion is about 1 m in eight months. On larger roots the rate is still slower. Yet it is evident that once infection sets in, the rate of spread is little affected by wet or comparatively dry conditions. The disease is a slow but sure killer.

It is quite clear both from observation of natural growth and experimentation that the advance of the fungus on the root surface is inhibited on exposure to light, and with the inhibition of growth on the root surface, lesion formation in the cortex and wood also ceases. The entire activity of the fungus must therefore be subterranean. The application of fungicidal chemicals to root surfaces also achieves the same result.

Regeneration of new roots ahead of the advancing lesion is common but these too eventually succumb. Infection also occurs on small roots with woody xylem but not on feeder roots.

A peculiar phenomenon is the absence of any marked symptoms on the foliage, even when most large laterals and the collar region of the tap root are completely killed. All that is noticeable is a reduction in refoliation vigour and consequent slight reduction in canopy density. This however was found to be unreliable for diagnosis. When most of the laterals are rotted the tree is more often than not uprooted by wind. A killed and wilted erect tree is of rare occurrence.

The pattern of infection sites, on replantings on which the percentage infection was highest, indicated a more or less even distribution over the whole area. Drawing upon the observations and experiences of old hands who worked the seedling trees of the previous stand of rubber, it was not unreasonable to suspect that the fungus had been endemic in the area in the seedling era. This led us to look for the fungus as an inhabitant on old root debris. Soil excavation around infected trees revealed first that infected roots were in the direction of sites of large decomposing root debris some of which was yet under attack by dead wood termites. Many vacant burrows left by completely decomposed roots were also evident. The first positive evidence was the detection of fructifications of the fungus growing from old debris lying buried in the soil but with portions near the soil surface. These showed the typical black mycelium and it was later found that such pieces of debris could be induced to sprout fructifications when buried near the soil surface and periodically watered. Direct and convincing evidence of infection by root contact with inoculum carrying old root debris was found in the younger five-to seven-year old replantings.

Infection was successfully reproduced by placing pieces of naturally occurring inoculum against healthy roots underground, infection being quite apparent in three months. The role of the food base as the cause of field infection is, in the light of our present knowledge, established. Attempts at spore germination and the inoculation of pieces of sterilized and unsterilized roots with spores of varying ages proved unsuccessful. The role of the spore is therefore yet unknown.

CONTROL

From the foregoing observations, it became clear that the following criteria could be exploited in devising control measures.

1. The slow growth rate of the fungus on the root surface and subsequent internal infection ;

2. The dependence of lesion formation, in the cortex and xylem, on the uninterrupted growth of mycelium on root surface ;

3. The dependence of initial infection on the presence of naturally occurring inoculum on old root debris.

It will of course be immediately obvious that these are more or less the same criteria on which the present method of control of the White Root disease is based. A pilot project on the control of the Black Root disease was therefore designed to test the efficacy of methods of root disease control now used in estate practice.

Eighteen trees were available for treatment in December 1968 and taken at random were treated as follows :—

A Excavation of root system, excision of diseased roots, removal of food base, if any, and refilling with same soil ;

B Same as A plus treatment of wounds only, with 10 - 15% PCNB in a grease base (Fomac) ;

C Control — excavation of root system and refilling with same soil only.

The extent of damage on each root system was recorded for comparison. It is not possible yet to represent accurately and quantitatively the observations on the treated root systems and the controls, neither does it lend to statistical analysis. Yet we may draw some conclusions for future guidance. Both treatments A and B were successful in stopping the advance of the fungus and root regeneration occurred satisfactorily, compared with the controls. The treatment of controls with PCNB grease appeared to enhance root regeneration appreciably.

In December 1969 a large scale field trial on the same lines was carried out involving 103 trees. Preliminary observations after eight months have confirmed our observations on the pilot project. According to our present knowledge, therefore, the excavation of the root system, excision of diseased roots and diseased parts of the tap root and the removal of the food base, if any of it is still in the soil, with or without chemical treatment of wounds, constitute adequate control.

OUTLOOK FOR THE FUTURE

In considering the outlook for the future two questions need be answered: (*a*) What is the severity of the disease in its present location ? (*b*) What chances are there for its spread to other locations bearing in mind that there is positive evidence of the fungus being viable on old roots in the soil for at least 13 – 15 years ?

At Hatbawe Estate in 1968 we were able to detect 20 trees with typical symptoms. Today, 1½ years later, over 200 trees have been found, a third of these in younger replantings. There is little doubt that on Hatbawe Estate it is indeed a problem worth tackling, not so much from the point of view of reduction of the present stand per acre, but as a replanting problem for the future. If left unchecked a build-up of inoculum is bound to occur.

The disease so far has been found only on Hatbawe Estate, on two divisions three to four miles apart. It is hard to believe that it is non-existent elsewhere in the district. Yet if this is so, then strict quarantine measures, even with a view to eradication of the fungus on Hatbawe Estate, is perhaps justifiable. The answer to this, those who are rubber growers, in the Kegalle District, must provide.