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CORYNESPORA LEAF FALL: THE MOST CHALLENGING RUBBER DISEASE IN ASIAN AND AFRICAN CONTINENTS

C K Jayasinghe

ABSTRACT

Corynespora leaf fall caused by *Corynespora cassiicola* is currently considered as the most destructive leaf disease of *Hevea* rubber in Asian and African continents. Several outstanding clones in the world namely RRIM 600, GT1, RRII 105, RRIC 103 and RRIC 110 have already succumbed to the disease creating a concern on the future development of the world natural rubber industry. The pathogen affects the young and old leaves on immature and mature rubber causing leaf fall throughout the year. Though the characteristic symptom is the appearance of lesions resembling railway track, a great deal of variation in symptoms has been reported recently depending on the type of clone, locality planted and stage of maturity. This ubiquitous fungus has more than 80 plant species as its hosts and has shown to be having an intra-specific

variation within the isolates. Recently it was discovered that pectolytic and cellulolytic enzymes are also involved in pathogenesis in addition to the toxic metabolites. Under the present economy the only way of managing the disease is the use of genetic resistance. However, it was revealed that dynamic changes of the pathogen might interfere with the long lasting resistance of this perennial host posing a grave threat to the outstanding rubber clones in the world.

Key words: *Corynespora cassiicola*, diagnosis, epidemiology, management, pathogenesis

Corynespora leaf fall (CLF), a disease affecting rubber with relatively a recent origin has now become a grave threat to the natural rubber industry attacking several outstanding clones in South and South East Asia and Central Africa. Recent disease outbreaks in Indonesia (Sinulingga *et al.*, 1996), Malaysia (Shamsul & Shamsuri, 1996), Sri Lanka (Jayasinghe & Silva, 1996), Thailand (Rodesuchit & Kajornchaiyakul, 1996), Cameroon, Gabon and Ivory Coast (Wahounou *et al.*, 1996) resulted in a loss of thousands of hectares cultivated with high yielding clones creating a new concern on the future development of the rubber plantation in Africa and South East Asia.

CLF was first reported on *Hevea* in India in 1958 (Ramakrishnan & Pillay, 1961) and two years later in Malaysia (Newsam, 1960). By the year 1985 the occurrence of the disease has been reported from two more countries namely Nigeria (Awoderu, 1969) and Indonesia (Situmorang & Budiman, 1984). Upto that time this parasite was recognised as a weak pathogen of *Hevea* rubber capable of attacking only the budwood nurseries and deficient seedling nursery plants (Newsam, 1960; Chee, 1987b). The first disease outbreak of CLF occurred in mid 1980's severely affecting several rubber clones particularly RRIC 103, RRIC 52, RRIM 725, KRS 21, PPN 2058 and PPN 2044 in Indonesia, Malaysia, Sri Lanka and Thailand (Liyanage *et al.*, 1986; Chee, 1987b; Pongthep, 1987). The most recent record of the disease comes from Vietnam and it was stated that the outstanding clone RRIC 110 is severely affected with CLF (Dung & Hoan, 1999). As no economical management system was available, the disease affected all susceptible clones causing defoliation and die-back. The respective governments had no other alternative rather than forcing growers to uproot their affected clearings and replant with tolerant clones available during that time.

However, the disease became more serious over the years and by the year 1996, nearly a decade after the first epidemic several clones considered as tolerant during the first epidemic had succumbed to the disease in both Asian and African continents. Among these affected clones; RRIM 600, RRII 105, GT 1 and RRIC 110 were the most outstanding clones in the world and other widely grown clones include IAN 873, PB 260, PB 28/59, PB 235, PB 280, BPM 1, BPM 24, PR 261 and RRIM 701.

Diagnosis

Symptoms appear on both immature and mature leaves. The most characteristic diagnostic feature is described as railway track appearance or herring-bone pattern as a result of browning or blackening of the veins adjacent to the lesions. The area around the lesions gradually becomes chlorotic due to the distruction of chloroplasts. This was the common symptom described in Indonesia (Situmorang & Budiman, 1984), Malaysia (Chee, 1987b), Sri Lanka (Liyanage *et al.*, 1986) and Thailand (Pongthep, 1987) during the first outbreak of CLF.

However, during the last decade a great deal of variation in symptoms was noticed depending either on the maturity of the plant or type of the clone and this subject was renewed with colour illustrations recently (Jayasinghe *et al.*, 1998b).

There were few instances where different symptoms were observed depending on the locality also. The ideal example for this is the CLF disease symptom in India, the country where CLF disease was first reported on *Hevea* rubber. The most common symptom observed in India is the presence of circular or irregular amphigenous spots which measure 1-10 mm in diameter (Ramakrishnan & Pillay, 1961; Rajalakshmy & Kothandaraman, 1996). It has been shown that these spots sometimes may coalesce to form enlarged lesions with brown or white centres. On the lesions typical dark concentric rings also appear.

The typical symptom (herring-bone appearance) which was described previously is unique for the *Hevea* clones; RRIC 103, RRIC 52, RRIM 600, IAN 873, RRIM 725 and seedlings in nurseries. The symptoms produced on the leaves of the clone RRIC 110 is somewhat different from the typical lesions and often mistaken with old *Oidium* patches by the field staff. These lesions are either irregular or polyhedral and surrounded by extended yellow halos when leaflet is viewed against the light . Silvery white papery appearance develops in the centre and sometimes shot holes may also develop due to the disintegration of the centre tissue of the lesion. Another characteristic feature with this clone is the blackening of the portions of secondary veins associated with the polyhedral lesions. Appearance of the blackish linear lesions on midrib of leaflets is the common symptom on the clone RRIC 133 in budwood nurseries. Lesions produced by *C. cassiicola* on the clone RRIC 132 is more or less similar to the lesions of Bird's eye spot disease caused by *Drechslera heveae*. But here the linear lesions are very common and they are brownish black in colour.

During the juvenile stage of the plant, specially in polybag nurseries the most characteristic symptom is the production of circular or irregular lesions of varying sizes delimited by a wavy border. Sometimes lesions coalesce to result in irregular papery lesions giving a scorched appearance. If wet weather persists, the leaves shrivel and fall off. Disease on polybag nurseries could be seen on any clone leading to die-back including highly resistant clones in the field such as RRIC 100, RRIC 102 and RRIC 121.

Pathogen

Corynespora cassiicola (Berk & Curt.) Wei, is a ubiquitous fungus that causes many types of symptoms on more than 80 host plants under diverse environmental conditions. Typical colonies on potato dextrose agar are grey to brown. Conidiophores are mostly simple but occassionally branched, septate and pale to light brown in colour in colour. Conidia vary in shape; typically obclavate to cylindrical, straight or curved and multi septate (2-16) with shades of brown. Spores from cultures are mostly needle shape while basal part of the field spores are wide at the hilum with a slight rim and the distal end is narrow. However, isolates collected even within the same agroclimatic zone show a significant diversity in culture morphology, colony colour, growth, pathogenicitiy and spore production (Chee, 1987b; Soekirman & Purwantara, 1987; Liyanage *et al.*, 1988; Breton *et al.*, 1996; Darmono *et al.*, 1996; Jayasinghe *et al.*, 1996).

Epidemiology and pathogenesis

The conidia are wind dispersed and spore release exhibit a diurnal rhythm. It is negligible during the night and commences early morning continuing to rise sharply reaching a peak around 0930 in Sri Lanka (Liyanage, 1987) and mid day in Malaysia (Chee, 1987b; Radziah *et al.*, 1996). Relative humidities close to dew point or free water is necessary for spore germination (Liyanage *et al.*, 1988; Situmorang *et al.*, 1996). According to Malaysian observations, spore liberation occurs mainly in the dry months and Radziah and others (1996) showed that leaf fall also reaches the peak during dry season but infections are likely occur when the leaf surface is wet.

Corynespora cassiicola is a fungus capable of producing a toxin in culture and in infected tissue (Onesirosan *et al.*, 1975). The toxin secretion by the rubber isolate was investigated by several workers (Liyanage & Liyanage, 1986; Situmorang *et al.*, 1996; Breton *et al.*, 1997a) and they have shown that there is a positive correlation between sensitivity to the crude toxin *in vitro* and field susceptibility of the clone. Toxin was identified as a low molecular weight protein of 21KDa with an isoelectric point near to 3.18, named as "Cassiicoline" and pointed out that virulence of different *Corynespora* isolates seemed to be directly linked to their toxin production (Breton *et al.*, 1997a; Breton & d'Auzac, 1999). The resistance of some rubber clones to *Corynespora* infections may depend on their ability to neutralize the toxin or due to the fact that the toxin is poorly or not recognised by its specific receptors (Breton & d'Auzac, 1999). However, exact mechanism of the resistance is not yet established (Breton, *et al.*, 1997b). Jayasinghe and others (1998a) showed that the principal enzyme involved in pathogenesis of the rubber isolate of *C. cassiicola* is pectin lyase and in the latter stages cellulolytic enzymes may also play a significant role.

Genetic variability of the fungus has been investigated (Silva, *et al.*, 1995; Darmano *et al.*, 1996) and shown that there is an intra-specific variation within the *C. cassiicola* isolates and this may be the reason for sudden changes in the host preference of the fungus.

Management

Chemical control of CLF is practiced only in nurseries and an effective control is achieved by spraying benomyl, mancozeb, orthocide and propineb in Sri Lanka (Liyanage *et al.*, 1988); Bordeaux mixture and zineb in India (Ramakrishnan & Pillay, 1961); Bordeaux mixture in Thailand (Kajornchaiyakul, 1987) and benomyl, chlorothalonil, triadimefon and tridemorph in Malaysia (Chee, 1987). Nevertheless, frequent spraying of fungicides to polybag nurseries is essential during the overcast rainy weather as all rubber clones including highly resistant ones in the field are extremely susceptible to CLF during juvenile stage.

Though extensive research has been performed in management of CLF under field conditions (Liyanage *et al.*, 1991; Chee, 1987b; Ismail Hashim *et al.*, 1996; Soepena *et al.*, 1996; Jayasinghe *et al.*, 1997b) no chemical control is recommended in any part of the world. Continuous spraying of benomyl and mancozeb at five day intervals commencing at refoliation prevented the establishment of disease on highly susceptible clone RRIC 103 in Sri Lanka (Liyanage *et al.*, 1991). Soepena and others (1996) showed that application of tridemorph and mancozeb provide a very good control of the disease provided that chemicals are sprayed at weekly intervals. This frequent spraying is not feasible at all, under the present economy of the rubber sector. Further, chemical spraying has become a challenge to the *Hevea* Pathologists as disease occurs through out the year on leaves of all ages unlike in any of the other rubber diseases. Achievement of an adequate coverage on the rubber canopies in undulating lands where most of rubber plantations have been established is also a critical factor. Under the light of this situation the only hope is the planting of resistant clones assuming that their tolerance will remain atleast for several decades. Further every effort should be made to breed clones with horizontal resistance by exposing experimental clones adequately to genetic variability of the pathogen.

Today *Hevea* Pathologists are highly concerned about the rate at which the disease tolerance is breaking down in the clones considered as resistant previously due to the virulence of new races. Hence there is an uncertainly with regard to the disease tolerance of almost all the clones grown in South and SE Asia and Central Africa.

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