DISEASES OF POTENTIAL THREAT TO RUBBER IN INDIA

by
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Natural rubber (*Hevea brasiliensis*) is a native of the tropical rain forests of Amazon basin. Following the discovery of usefulness of rubber, in the nineteenth century attempts were made for commercial cultivation of the tree. Most of the commercial rubber plantation now existing in the world has its origin from a very small collection of seeds made by Henry Wickham in 1876 from the river Tapajoz region in Brazil. Commercial cultivation of rubber was first taken up in South East Asia. The success of these early plantations led to the reintroduction of the cultivated superior selections from Asia into Central and South America and the Carribbean islands. In India commercial rubber cultivation was started in 1902. At present the area under rubber in India is 5.23 lakh hectares.

One of the major constraints of rubber cultivation has been the incidence of diseases, many of them causing considerable crop/tree loss. Cultivated rubber has a very narrow genetic base as it was derived from a very small collection. The lack of wide genetic variability exposes the plant to epidemics. The diseases of rubber tree have been well documented (Petch, 1921; Sharples 1936; Rao 1975). Fungi play the major role as pathogens of rubber tree. Many fungal diseases of rubber bave been reported from India (Rubber Research Institute of India, 1980) of which a few cause epidemics.

Early attempts in rubber disease control in India were made by the UPASI Rubber Experimental Station at Mundakayam from 1916 to 1934. Since the inception of the Rubber Research Institute of India in 1955, concerted efforts were made for rubber disease control by which control strategies for all major diseases bave been developed. However, there are a few diseases of rubber which have not been reported from India. These include South American Leaf Blight (*Microcyclusulei*) white root disease (*Rigidoporus lignosus*) black scab (*Catacoma huberi*) and target spot (*Thanetophorus cucumeris*). The leaf disease caused by *Corynespora cassiicola* which is considered as minor disease in India, needs special attention as it has caused epidemics in our neighbouring countries like Sri Lanka and Indonesia.

1. CORYNESPORA LEAF DISEASE

Epidemics in Sri Lanka

In SriLanka Corynespora leaf disease was first noticed in a polybag nursery in Dartonfield Estate during 1985 on clone RRIC 103. This clone was a very promising progeny of a cross between RRIC 52 and PB 86, developed in 1958 and widely tested for two decades before recommending for large scale planting in Sri Lanka. As the clone fared well in commercial plantings, the extent of area under the clone rose steadily between 1979 and 1985. No disease incidence was reported till 1985. By 1986, Corynespora leaf disease had spread to all the wet districts of Sri Lanka and by 1987 a total area of more than 4000 hectares was affected. (Liyanage *et al.*, 1989).

Corynespora disease in Malaysia

In Malaysia, the disease was detected in the nurseries during 1960, but was considered as a minor disease. The first report on fied planted rubber was in 1975 and subsequently the disease had spread over large areas. A survey conducted in 1990 revealed that the disease incidence in estate sector was 53 per cent and that in small holdings, 67.5 per cent. Thirty five out of sixty three clones observed in the survey were affected by the disease (Tan, 1990).

Epidemics in Indonesia

Corynespora leaf disease was first noticed in Indonesia in 1980 in South Sumatra. Later the disease spread to Java and North Sumatra by 1982 and 1983 and to the whole of the rubber growing tracts of Indonesia by 1988, severely affecting nearly 1200 hectares. Nearly 400 hectares planted with susceptible clones were uprooted which accounted for a loss of more than Rp 200 billion (Sinulingga *et al.*, 1996).

Corynespora leaf disease in India

This disease was first reported in 1958 from RRII experimental station nursery. Later it was reported from other nurseries in Mundakayam, Kanjirapally, Thodupuzha, Trichur and Nagercoil areas (Ramakrishnan and Pillai, 1961). The infection on mature trees was first noticed in 1969 in Kodumon. Infection on mature trees was reported from Chittar in 1970 and Shaliacary, Kaliar and Cheruvally during 1976. (George and Edathil, 1980). Recently, a disease

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outbreak on mature rubber was noticed in RRII Hevea Breeding Substation and KFDC plantations at Nettana in Karnataka.

Symptoms of Corynespora leaf disease

Corynespora infects leaves of all stages. However, light green immature stage of leaf appear to be more susceptible. The symtoms of the disease vary with the clones and the locality. Circular lesions of varying sizes with a papery centre, brown margin and yellow halo are commonly seen.

Shot holes are sometimes formed due to disintegration of the central portion of the lesions. Several lesions may coalese to form large blighted area. Spread of the disease along the veins is evident due to brownish discolouration of the veins. The brownish colour of the veins form a 'railway track' like appearance. Severe infection of the mid rib causes leaf blight. The surrounding tissues turn yellow and later to brown colour. Affected leaves abscise prematurely leading to defoliation of trees. Even a single lesion on the midrib or base of the leaf is sufficient to cause leaf abscission. Severe infection leads to shoot die back. Repeated defoliation can lead to death of infected trees.

Comparison of symptoms of powdery mildew and Corynespora leaf diseases

Corynespora disease incidence is more severe during the refoliation period (January-March) after wintering. Powdery mildew is a more common disease during this period which can be recognised by the appearance of white powdery masses on young leaves. Very young leaves affected by powdery mildew disease crinkle and fall on the ground forming a black carpet. Powdery mildew is more severe when there is dew, mist formation or light showers during refoliation period. Unlike powdery mildew, Corynespora disease occur when the atmosphere is dry. The branches exposed to sunlight are more severely affected. Defoliation by Corynespora is slower than that by powdery mildew. A few partially blighted leaves often remain on the branches. Broom stick like appearance of branches common in powdery mildew affected trees is not usually seen on *Corynespora* infected trees.

Mechanism of infection and disease development

The conidia of the pathogen deposited on susceptible leaves germinate and penetrate the tissues. On young leaves, lesions are noticed within 3 to 4 days. The incubation period is delayed upto 9 days in mature leaves. The tissue damage is attributed to a toxin produced by the fungus. The toxin production has been demonstrated in static cultures of the pathogen on modified alternaria medium *in vitro*. Toxin was detected in 44 hours and maximum production occurred in 10-12 days. The toxin production was maximum at a temperature of 28°C and a pH of 6.0 to 7.0 (Oneirosan *et al.*, 1975).

The symptoms of Corynespora disease could be induced on detached leaves by applying 0.001 ml of crude toxin on pin-prik wounds (Liyange and Liyange, 1986). The toxin affects CO_2 assimilation rate and thereby affect the photosynthetic mechanism of infected leaves (Nugewela *et al.*, 1989). Since a correlation was observed between the size of the lesion caused by toxin and the susceptibility of *Hevea* clones it was suggested that crude toxin can be used for rapid screening of rubber clones.

Clonal susceptibility

The clone noted as most susceptible to Corynespora disease is RRIC 103. Other clones reported as susceptible in Sri Lanka include RRIC 104, RRIM 600, RRIM 725 and Tjir 1. Clones RRIIC 110 and RRIC 133 were found to show susceptibility only in nurseries (Jayasinghe and Silva, 1996).

In Malaysia the most commonly affected clones are RRIM 600 and GT 1. The clones RRIM 701, RRIM 703, RRIM 712, RRIM 725, PBIG, PB 261 and IAN 873 are also reported as susceptible. The disease was observed on clones PB 5/51, PB 217, PB 235, PB 260, PR 107, RRIM 901, RRIM 905 and Tjir1 (Tan, 1990). The clones RRIC 103, Fx 25, RRIM 725, KRS 21, PPN 2058, PPN 2444 and PPN 2447 are also noted as susceptible (Shukor and Hidir, 1996). The clones now rated as resistant viz. RRIM 712, RRIM 902, RRIM 903, RRIM 904, RRIM 915 and RRIM 937 have susceptible parents and so the stability of their resistance is doubtful (Othman *et al.*, 1996).

The clones found highly susceptible in Indonesia are RRIC 103, KRS 21, RRIM 725, PPN 2058, PPN 2444 and PPN 2447. GT 1 and RRIM 600 previously rated as tolerant are now found to be susceptible (Sinuligga *et al.*, 1996). Azwar *et al.*, (1993) analysed the performance of various clones and concluded that only BPM 24 and RRIC 100 showed resistance to *C. cassiicola* in Indonesia.

In India, the clones found susceptible include RRII 105, PCK 2, RRII 118, RRII 300, RRII 305, PCK 1, RRIM 600, PB 86, PB 235, PB 255, PB 260, PB 311, PR 107, GL 1 and Tjir-1. Clones which winter early were found to escape from the disease. The susceptibility

of RRII 105 is of particular concern as large areas are now planted with this clone.

Host Range

Corynespora cassiicola has a wide host range. Liyanage *et al.*, (1986) listed eighty eight common plants as hosts of *C. cassiicola*. The list includes plants like *Cacao thobrmae, 'Coffea arabica, Eucalyptus grandis, Eugenia caryophylata and Piper nigrum*. This indicate that the pathogen can survive on most of the common plants found in areas near to rubber plantations.

Control measures

The strategies adopted for control of Corynespora leaf disease in Sri Lanka included

1. base budding of susceptible clones (upto 24 months old) with tolerant clones.
2. crown budding of susceptible clones (2½ to 3½ years old) with tolerant crown clones.
3. Uprooting susceptible clones and replanting with tolerant clones.

Among these methods, only uprooting and replanting became popular among planters as the other two methods demanded careful maintenance of the new bud. Another attraction was the relief payment offered by the Government to the small holders who opted for uprooting and replanting. A total relief payment of 53.9 million rupees was made. The entire infected area of nearly 4000 hectares were either replanted or overbudded within a period of 3 years (1987 to 1990). (Liyanage *et al.*, 1989.

Chemical control of Corynespora is not recommended in Sri Lanka except for nursery plants. In the nurseries, spraying with benomyl, mancozeb, captan or propineb is recommended (Jaysinghe and Silva, 1996).

Experiments conducted in Malaysia indicated that spraying benomyl at the rate of 500 g/ha for 5 to 6 rounds immediately following wintering gave good control of disease in mature areas. Artificial defoliation to induce early wintering was not effective in reducing disease incidence (Hashim, 1994). Supplementation of N fertilizers or micronutrients did not show any influence on disease severity or canopy density (Hashim *et al.*, 1996).

Four to five rounds of spraying with Tridemorph (Calixin 0.61/ha) or mancozeb (Dithane M-45 1.5 to 3 kg/ha) are recommended for control of Corynespora leaf disease in nurseries and young plantations in Indonesia (Soepena *et al.*, 1996).

In India, Bordeaux mixture (1 per cent) or Dithane Z 78 (0.24 per cent) were earlier recommended for Corynespora disease control (Ramakrishnan and Pillai, 1961). Later 0.1 per cent spray of carbendazim (Bavistin) was also found effective (Rajalekshmy *et al.*, 1980) for disease control in nursery. Higher doses of N fertiliser application favoured the disease incidence (Rajalekshmy *et al.*, 1979). Recent experiments conducted in Karnataka indicate that high volume spraying of mancozeb (Dithane M 45) 0.2 per cent, carbendazim (Bavistin) 0.05 per cent of Bordeaux mixture 1 per cent at an interval of 2 to 3 weeks during refoliation period is effective in the control of Corynespora leaf disease on young trees (5 to 7 years old).

2. SOUTH AMERICAN LEAF BLIGHT

South American leaf Blight (SALB) is considered as the most dangerous disease of rubber. It is very destructive, rapidly spreading and very expensive to control. Repeated defoliation of rubber plants by SALB, besides retarding growth can lead to die back of shoots and their eventual death.

SALB was present in the tropical rain forests of South America as a minor disease of the wild rubber tree. When the selections of rubber developed in Asia were reintroduced to South and Central America for commercial cultivation from 1895 onwards, this minor disease of wild rubber tree caused epidemics in the new plantings.

There has been many reports on severe infestation of SALB in South America. In 1927 Ford Mortor company's rubber plantations in Fordlandia, Brazil (3200 ha) had to be abandoned due to SALB epidemics. Similarly Belterra plantations in Brazil planted in 1934 was adandoned in 1943 due to severe SALB incidence. Good year company's Speed way Estate (1000 ha) in Costa Rica and All weather Estate in Panama were also similarly abandoned (Hilton, 1955). Another attempt to recultivate rubber in the SALB escape areas also failed due to emergence of new races of the pathogen which devastated nearly 1 lack hectares out of the 1.5 lakh hectares planted upto 1986 (Liberei *et al.*, 1989).

Symptoms of SALB

The symptoms of SALB on the leaves depend on the leaves at the time of infection. If the infection is at the brown (very young) stage of the

leaves, the leaflets crinkle due to necrosis and fall off leaving the petioles on the branches for a few more days. When the infection occur in the light green stage of the leaves, greyish powdery masses of conidia are seen on the lower surface. The lesions have as angular outline. On the upper surface of the leaf, corresponding to the lesion on lower side, a translucent area is formed. As the leaf matures, the powdery appearance is lost, the lesion becomes brown in colour and its centre rots away (Chee and Holliday, 1986).

The next stage of the disease is development of pycnidia of the fungus. Pycnidia are formed on the upper surface of hardening leaves along the fringes of the lesion. The pycnospores are formed in the pycnidia. Pycnospores are not capable of infection and are thought to be spermatia of the fungus.

When the leaf is fully mature, the stroma become massive and ascocarp is formed in errumpent scattered masses around the lesions or the shot holes formed due to death of cells in the lesion. These dark spherical structures are the perithecia of the fungus which bear the ascospores.

Infection of Mulei can also occur on petioles green stem, inflorescence and young fruits. Since the infection occur on a restricted part of actively growing tissue, it leads to tissue distortion. Stem petioles or inflorescence when infected become curled, twisted or spirally rolled with the infected area on the concave surface.

Distribution of SALB

At present SALB is confined to South and Central America and the islands in this region. The disease is reported from Brazil, Bolivia, Ecuador, Guyana, Peru, Surinam and Venezuela. The attempts to cultivate rubber for commercial production in Trinidad and Haiti resulted in spread of SALB to these areas. The disease has been reported from Panama, Costa Rica, Guatimala and Honduras.

Casual organism

The pathogen causing SALB, Microcyclus ulei was first collected and identified as *Dothidella ulei* by Ule in 1905. The pycnidial state of the fungus was observed and named as *Aposphaeria ulei* and the conidial state of the fungus as *Fusicladium macrosporum*. Muller and von Arx transferred the fungus to the genus Microcyclus and established that the different names represent only three stages of the same pathogen.

Host range

Microcycluls ulei infects only the genus Hevea. No other plants are so far known as a host. Of the ten species of *Hevea* SALB is reported only on four species. viz. *H. brasiliensis, H. benthamiana H. guianensis and H. spruceana*. The other six species namely *H. camporum, H. microphylla, H. nitida H. pauciflora, H camargoana* and *H. rigidifolia* are free from infection. (Chee 1976, Holliday, 1970).

Life cycle of *M. ulei*

The fungus over winter in the perithecial stage on mature rubber leaves which remain on the tree or fall on the ground. During cool weather, the ascospores are released. The ascospores germinate and infect the young leaves forming disease lesions on the lower surface. Conidia are produced in large numbers in these lesions. Conidia are responsible for rapid spread of the disease.

The conidia of *M.ulei* are one celled or more commonly two celled and club shaped. The proximal cell of the two celled conidia have a pronounced single twist. The conidia infect and cause disease on brown or pale green young leaves. On mature leaves, conidial infection result in small spots only. On the infected leaves conidia germinate, penetrate the epidermis and the mycelium spreads within the tissue. Sporulation occur in about five days after infection.

After about a month, the stroma develops on upper surface of infected leaves. Initially pycnidia are formed which produce dumb bell shaped pynospores. The stroma become more prominent when ascocarps are formed. The perithecia produce asci each bearing eight aspcospores. Ascospores are two celled. They infect young leaves in the next season.

Physiological races

Efforts to evolve resistant *Hevea* clones by crossing *H. brasiliensis* with *H. benthamiana* failed due to the evolution of physiological races of the fungus. Most of the early crosses were made between high yielding selections of *H. brasiliensis* and clone F 4542 a *H. benthamiana* selection which was showing high resistance. By 1960 it became clear that the resistance derived from F 4542 was not stable. Several offsprings of the crosses made which were inititally classified as resistant, became susceptible.

Chee *et al.* (1986) identified nine physiological races of *M. ulei* based on the behaviour of the pathogen on several differential clones. Hashim and Almeida (1987) confirmed six of these races based on thier infection on five differential clones viz. IAN 717, LAN 3925. LAN 710, Fx 2261 and Fx 985.

Physiology of resistance to infection

The part played by phenols and oxidative enzymes in the pathogenesis of SALB has been studied. Blazquez and Owen (1957) reported presence of tannins in the yellow brown region surrounding the disease lesions. Figari (1965) observed greater inhibition of conidial germination in leaf extracts of resistant clones than of susceptible and suggested that the toxic substance is a flavanol. This was identified as kaemferol 3 rhamnodiglucoside (Martins *et al.*, 1970). Hashim *et al.*, (1980) observed that the toxic substance is querectin. They also observed that clones resistant to SALB had lower peroxidase and IAA oxidase activity. Presence of growth substance like IAA, Kinetin, GA, and β Naphthoxy acetic acid reduced the lesion size on detached leaf discs.

Likely behaviour of *M. ulei* in Asia

In the SALB endemic areas of tropical America the annual rainfall exceeds 250 cm with no long dry period. Relative humidity is more than 80 per cent during most part of the year. Moderate SALB incidence is noticed even in areas with less than 200 cm rainfall with no long dry period. In areas with long dry period disease incidence is low. But such areas are not suitable for growth of rubber. Chee (1980) observed that epidemics of SALB occur when daily temperature is less than 22°C for more than 13 hours and RH more than 92 per cent for more than 10 hours with rainfall more than 1 mm for a period of 7 days.

Considering the climatic conditions of South East Asia, it can be predicted that if SALB is introduced into this area, it can cause epidemics. The rubber growing areas of South India receive 250-300 cm rainfall annually. The relative humidity also is high (60-95 per cent) particularly during both South West and North East monsoon periods. The high RH observed from September to December coupled with the afternoon showers of North East monsoon which can prolong the period of leaf wetness can cause ideal situation for rapid spread of the disease in South India if susceptible stage of leaves are available. Such susceptible leaves can occur in the nursery plants and on trees which refoliate early. Once infected, ther disease can spread very fast as the entire planting is with susceptible clones.

An argument often made is that since the spores of *M. ulei* are heavy and are dispersed in water droplets, their long distance transport is unlikely. But the spread of the similar pathogen causing coffee rust (*Hemileia vastatrix*) from South Asia to South America in the past Nutman *et al.*, 1960) provide ample evidence for spores carried in rainsplash crossing oceans.

Management of SALB

Attempts to develop SALB resistant clones were initiated by Ford Mortor Company following the failure of their early plantations in Fordlandia and Belterra. The plants which survived SALB epidemics (eg. F 170, F 315 F 351, F 1425, F 4542) were crossed with high yielding selections from Asia (eg. Avros 49, Avros 193, Avros 363, PB 86 Tjir 1 etc.) Later the Brazilian Research Institutes (IPEAN and CNPSD) continued the work by crossing introduced high yielding clones with primary Ford clones and the progenies of earlier Ford crosses. Unfortunately in most of the crosses resistance derived from F 4542 was used and this resistance broke down with the appearance of physiological races of *M. ulei*. Lately, *H. pauciflora* (eg. clone P. 10) is being crossed with *H. brasiliensis* and *H. benthamiana*.

The failure of the early crosses was mainly due to the dependence on vertical resistance. In vertically resistant material infection by *M. ulei* causes hypersensitive reaction leading to cell collapse and prevention of further spread of the fungus. This type of resistance can be overcome by new races of the pathogen. A more desirable type of resistance is horizontal resistance in which there is no host cell collapse, but the rate of fungal spread, lesion development and sporulation of the fungus are affected. Hashim and Periera (1989) indicated that clones GT 711, RRIM 501, CNSAM 7701, SIAL 842 AND SIAL 263 possess such type of resistance.

Crown budding

In the 1940s crown budding was used to salvage some of the surviving trees in Ford plantations. But the lack of resistant crown clones and the depressive effect of crown on growth and yield of trunk clone were the main limitations. But the technique was useful in avoiding SALB. Currently high yielding clones like RRIM 600, GT 1, PB 235 and PB 260 are crown budded with *H. pauciflora* clones PA 31, PX and SIAL 842 in Brazil.

Chemical Control

Chemical control of SALB is very costly as more than six rounds of spraying is necessary to obtain satisfactory disease control. In the nurseries, weekly spraying of triademefon 0.02 per cent or chlorothalonil 0.3 per cent were found to be effective.

In Brazil, spraying of mature trees with fungicides was attempted only since 1970s. Aircrafts, thermal fog generators and tractor mounted airblast sprayers were tried. The fungicides used initially were copper

and mancozeb. Bur newer systemic fungicides like benomyl (0.3 kg/ha), chlorothalonil (1.6 kg/ha) and thiophanate methyl 0.3 kg/ha are now being used. Resistance to benomyl has been noticed in certain *M. ulei* isolates (Hashim, 1988).

Prevention of SALB

The outbreak of SALB in the Asian selections of rubber planted in South America raised concern about the spread of the disease beyond South America. The magnitude of the threat of SALB and preventive measures to be undertaken have been described (Hilton, 1955). Since then attention has been directed to quarantine measures against SALB.

The possible ways by which SALB can accidentally be introduced to Asia and Africa are :

1) importation of infected rubber planting material

2) importation of plants other than rubber contaminated by spores of *M. ulei*.

3) contamination of body, baggages and belongings of air travellers from South America.

The long viability of *M. ulei* spores may favour its spread through contamination on imported plants and on human bodies and belongings. The conidia of *M. ulei* stored at 24°C on glass slides were viable even after three weeks. Conidia on infected leaves stored at 85-100 per cent RH remained viable for two weeks and when desicated for sixteen weeks (Chee *et al.*, 1986). The ascospores survive for three weeks within the perithecia and for two weeks after their release (Chee, 1980). Zang *et al.*, (1986) showed that conidia of *M. ulei* stored for 7 days on materials like glass, artifical leather, polyethylene cloth and infected leaves gave 26 to 53 per cent germination.

The spores of *M. ulei* can be killed by:

1. Direct exposure to UV light (253 nm) for atleast 15 minutes.
2. Exposure to moist heat (55°C) or dry heat (75°C) for 30 minutes.
3. Exposure to formalin vapour for 15 minutes (35 per cent Formaldehyde at the rate of 1 ml/8 cu cm).
4. Soap solution (40 mg/l).

Detailed schedules for treatment of seeds and budwood of rubber imported from SALB endemic areas have been outlined (Association of Natural Rubber Producing Countries, 1995). This involves treatment of the materials either in one or two intermediary quarantine stations outside SALB endemic area, besides treatment and certification at the point of origin. Strict quarantine treatments are undertaken on arrival in Asia. The imported materials are grown in isolation and under close supervision.

Quarantine agreements

There are two agreements which cover prevention of introduction of SALB into Asia :

1. Asia and Pacific Plant Protection Commission Agreement.
2. ANRPC Agreement on SALB.

Phytosanitary regulations in India

The import of plant materials into India are regulated by :

1. Destructive Insect Pest Act (1914).
2. Plants, fruits and seeds (Regulation of imports into India) order 1984 (amended in 1989).

The conditions laid down through these rules with respect to import of *Hevea* into India are :

1. Import of rubber and all species of Hevea from America and West Indies is prohibited.
2. Import from other countries should accompany an official phytosanitary certificate from the country of origin.
3. The declaration in phytosanitary certificate should include absence of contamination with *M. ulei* and *Sphaerostilbe repens*.

ANRPC Technical Committee on SALB

The ANRPC has set up a Technical Committee on SALB. The terms of reference of the Committee are :

1. Periodically review the progress of SALB and other economically important exotic diseases of *Hevea*.
2. Suggest measures for strengthening phytosanitary measures.
3. Monitor airline movement between South America and NR growing countries.
4. Work out joint emergency eradication programmes.
5. Establish and operate a fund for emergency eradication of SALB and other exotic diseases.
6. Ensure that member countries have technical and administrative machinery for emergency eradication.
7. Encourage research on SALB and other exotic diseases.
8. Co-ordinate activities of SALB country committees.

9. Establish liaison with relevant plant protection councils/ committees in other regions of the world.

Although India is not a signatory to ANRPC agreement on SALB we co-operate with all activities of ANRPC on prevention of SALB. One scientist of RRII has been trained in Brazil on IRRDB Fellowhip and one scientist in Malaysia under ANRPC training programme. We take part in the meetings of ANRPC technical committee on SALB. The twelfth meeting of this committee was held in Brazil during August 1997. We have also displayed posters on SALB at International airports and important offices of the Rubber Board. A booklet on SALB was distributed to all quarantine officers in the country.

How competent are we to do emergency eradication?

In case of an accidental introduction of SALB into India, we are technically competent to eradicate the disease. We have vast experience of over 30 years in aerial spraying of rubber areas. We have also developed very efficient ground sprayers which can throw fungicides to a height of about 25 meters. The fungicides used in control of SALB are available in India. Experiments conducted at RRII has indicated that ethepon at higher doses can be sprayed for defoliation of rubber with minimum harmful effects. However the practical feasibility of using defoliation as a method for emergency eradication in India is doubtful because most of the rubber is cultivated by small holders as homestead garden interspersed with other cultivated crops. So aerial spraying of defoliants if required may demand strong administrative support.

The effectiveness of eradication depends on how quickly we act. It is therefore very important that the incidence of the disease anywhere in the country is brought to the attention of Rubber Board or RRII. Since rubber estates in India are managed under very close supervision we can expect that such disease incidence will be noticed immediately. Let us hope that the quarantine measures now adopted will prevent any accidental introduction and that the disease which has not spread beyond South and Central America over nearly a century will remain so.