ABSTRACT

Tapping panel dryness (TPD) syndrome is a serious malady of rubber (*Hevea brasiliensis*) occurring in all rubber growing areas resulting in partial to complete drying of tapping panel. A detailed study was conducted to understand different symptoms of TPD and its prevalence in the rubber clone RRII 105. The possibility of association of viroid with TPD syndrome was also investigated.

It was observed that TPD is initiated in two ways. In the first case, extreme fluidity of latex with abnormally low DRC and the latex flow only from the inner most layers of latex vessels was observed. In the other, very viscous latex with abnormally high DRC and partial dryness of varying degrees and vessels devoid of latex even in the inner layer was seen. When dryness was observed in the virgin (BO 1) panel, the cracks were found to be extended to the opposite panel. Cracks and necrosis started from the tapping panel in some trees while in others it originated near the bud union and later extended towards the tapping cut. The present study revealed that cracking and bulging of bark increased with the age of trees and with progression in period of tapping. Nearly 40% reduction in total latex volume was observed for trees in the category of less than 50% TPD and nearly 90% reduction was observed when the trees were in the category of more than 75% TPD when tapped in BO 1 panel. Contrary to total latex volume, an increase in DRC was noted as the intensity of TPD increased. Dryness of root system was observed along with necrosis, cracking and bulging (as observed on the trunk) in TPD affected trees. The roots corresponding to the dry portion of scion showed dryness although those roots originated from the root stock.

It was observed that the number TPD affected trees increased as the year of tapping progressed at all the locations under observation both in small holdings and in large estates. Out of the 18900 trees observed at Adoor region 3508 trees (18.56%) showed more than 50% TPD, while it was 2425 out of 13700 (17.70%) at Nedumangad, 2153 out of 12700 (16.95%) at Taliparampa, 2821 out of 17300 (16.30%) at Mannarkad, 3106 out of 20200 (15.37%) at Pala and 2273 out of 15100 (15.05%) at Kanjirappally. The percentage of trees in the category of very high TPD intensity (>75%) showed a clear trend of increase from the first year to the last year of tapping at all the locations. The number of TPD trees in the other categories (low, medium and high) did not show such a remarkable trend of increase from BO 1 to BI 1 (renewed) panels. The scale of increase in TPD was more in older trees than in trees at the initial stages of tapping.

Percentage of trees showing TPD symptoms was less when the panel was changed but it increased even to higher values a year after such change. Reversion of TPD symptoms was observed only at a young age. Evidence for natural transmission of TPD from one tree to the other was observed in the present field studies as the number of TPD trees in clusters showed a significant increase with progressive tapping. Only 23.8% of the small holders adopted tapping rest when TPD was observed. When the TPD affected trees were continued to tap in the upward system, more than 50 per cent of the trees showed dryness after four months.

An infectious LMW RNA was detected from different samples drawn from varying ages of trees from different locations. The consistency in the presence of LMW RNA over three years of observation in TPD affected trees and its absence in the healthy ones points to the association of the LMW RNA with TPD. About 30 per cent of the apparently healthy trees which may be symptomless carriers also showed presence of LMW RNA. Such trees later showed TPD symptoms. Presence of bands in apparently healthy samples indicates that the biotic agent can be detected in the tree much before the TPD symptoms are visible. The PCR of the cDNA using viroid specific primers consistently amplified products in the range of viroids in R-PAGE positive TPD affected samples but was absent in R-PAGE –ve apparently healthy samples. The amplified LMW RNA showed 98% sequence homology to Potato Spindle Tuber Viroid (PSTVd) on BLAST analysis.

The R-PAGE test of bud grafted plants under transmission studies showed that all the plants tested from the group in which both stock and scion were viroid +ve, maintained the viroid bands. Viroid was observed to be transmitted from viroid +ve stock to viroid –ve scion. Test tapping showed TPD in both group of plants, namely plants budded with scion from TPD affected trees as well as plants with scion from healthy trees. This shows that root stock also plays a role in the development of TPD. Epinasty symptom development on tomato plants inoculated with total RNA isolated from TPD affected trees showed that the viroid present in rubber can be transmitted to the indicator host indirectly satisfying the Koch's postulates. The viroid was reisolated and it showed LMW RNA band in R-PAGE. The sequence homology of the RT-PCR product obtained from the inoculated tomato with that of Potato Spindle Tuber Viroid proved its viroid relationship.

Key words: *Hevea*, RRII 105, TPD, symptoms, prevalence, viroid, transmission