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# TAPPING PANEL DRYNESS SYNDROME IN *HEVEA* INCREASES DARK RESPIRATION BUT NOT ATP STATUS

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Tapping panel dryness (TPD) affects the ability of *Hevea* trees to synthesise rubber (cis-poly isoprene) and thus decreases the yield. The present study conducted in *Hevea* clones RRIM 600 and RRII 105 showed that concomitant with an increase in the total sugars and starch contents in the bark, respiration rate also increased but the ATP concentration in the cytosol markedly decreased in TPD affected bark compared to healthy bark from normal trees. This appears to be due to an increase in the non-phosphorylating cyanide resistant alternative respiration in the TPD affected trees.

Key words : *Hevea*, Respiratory pathways, Rubber biosynthesis, TPD.

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## INTRODUCTION

Tapping panel dryness (TPD) is generally considered as a physiological disorder commonly noticed in high yielding *Hevea* clones. It is generally believed that when the level of exploitation exceeds the physiological capacity of the tree to generate latex, the tree succumbs to TPD (Chrestin, 1989). The incidence of TPD increases with high tapping frequency and/or excessive yield stimulation (Commere *et al.*, 1989).

Although many studies have been made to describe the development of TPD, the exact cause of the syndrome is not clear. Cytological disorders associated with TPD development were reported by de Fay and Jacob (1981) and Gomez (1990). Studies on viruses and viroids were inconclusive (Peries and Brojier, 1965; Lim, 1973). Dian *et al.* (1995) analysed the changes in the latex protein pattern during the development of this syndrome. Recently Nataraja *et al.* (1998) studied the stress-induced heat stable protein content in the bark tissues of healthy and TPD affected *Hevea* trees. TPD affected bark was observed to have higher levels of sugars, phenols, soluble proteins, peroxidase activity and HMG-CoA reductase activity than normal healthy bark in the *Hevea* clone RRII 105 (Krishnakumar *et al.*, 1999). Chrestin (1985) proposed a biochemical explanation involving laticiferous senescence through activation of oxidative stress leading to dysfunction of the