# VARIABILITY AND DISTRIBUTION OF TAPPING PANEL DRYNESS IN *HEVEA BRASILIENSIS*

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***Abstract*** *A large-scale evaluation trial of 21 clones of Hevea brasiliensis was studied with respect to yield, girth and the incidence of tapping panel dryness (TPD) over nine years of exploitation. Tapping panel dryness was confirmed to be a distinct clonal characteristic with high heritability and low genetic advance. A significantly positive correlation of TPD with girth and girth increment over nine years of tapping was observed. The distribution of TPD affected trees in the field was not random in most of the clones studied. Non- additive gene action in the inheritance of TPD as indicated by the genetic parameters and its implications on Hevea breeding are discussed.*

**Key words**: *Hevea*, clones; large scale trial; tapping panel dryness; distribution; heritability; genetic advance

## INTRODUCTION

Brown bast or tapping panel dryness (TPD), a syndrome plaguing rubber plantations, is characterised by spontaneous drying up of the tapping panel resulting in abnormally low yield or stoppage of latex production. The disease was first reported in Brazil in 1887 from the Amazon forest and at the beginning of the century in Asia (Rutgers and Dammerman, 1914).

The features, causes and possible treatment of tapping panel dryness have been the subject of much research (Sethuraj, 1977; Chrestin, 1989; Pakianathan *et al.*, 1992; Premakumari *et al.*, 1996). Clonal sensitivity to tapping panel dryness was observed by many researchers (Bangham and d' Agremond, 1939; Ostendorf, 1941, Dijkman, 1951; Vijayakumar *et al.*, 1990). TPD is described (IRRDB, 1992) as an abnormal physiological phenomenon induced by tapping. When the level of exploitation of the tree exceeds the physiological capability of the tree to regenerate latex the tree succumbs to TPD. Recent thinking (Sethuraj, 1989) is focused on the question why only a certain percentage of trees in a monoclonal population get affected. The involvement of the genetics of root stocks has been implicated.

The present study was taken up in an attempt to understand the nature of variation of tapping panel dryness through an analysis of the components of variance and to determine the pattern of occurrence of the syndrome in the field.

## MATERIALS AND METHODS

A field trial of 21 clones (Table 1) planted at Kodumon estate in Central Kerala, in a

randomised block design with four replications, and 80 to 100 trees per plot was examined with respect to yield, girth and the incidence of tapping panel dryness (TPD) over nine years of exploitation. The data on yield and girth are cited from Nair and Marattukalam, 1981.

The analysis of variance was made followed by estimation of genetic parameters viz. phenotypic and genotypic coefficients of variation (PCV and GCV respectively), broad sense heritability ($H^2$) and expected genetic advance under selection (GA). The $\sqrt{x+1}$ transformation was applied to data on the percentage incidence of tapping panel dryness. Correlations among the traits were also worked out. The pattern of occurrence of the syndrome in the field was traced through a run test (Siegel, 1956) applied to plot-wise data on the 21 clones, based on the null hypothesis that the occurrence is random.

## RESULTS AND DISCUSSION

Highly significant clonal variation was evident for yield, girth and tapping panel dryness (Table 2). The incidence of TPD ranged from zero (RRIM 604 and RRIM 602) to 17.07 per cent (RRIM 609) with the clones in general showing 2.5 percent of affected trees. The variance ratio of 9.52 was the highest among the traits studied, indicating TPD is a distinct clonal characteristic in confirmation of earlier reports (Sivakumaran and Haridas, 1989).

The clone RRIM 501 showed the highest initial yield (36.68 g/tree/tapping) and mean yield over nine years of tapping (46.83 g/tree/tapping) as reported earlier (Nair and Marattukalam, 1981). Girth increment rate under tapping ranged from 1.44 cm/year in the case of RRIM 617 to 4.09 cm/year in the case of RRIM 612. Yield during summer was comparatively high for clones RRIM 609 and RRIM 603 (Nair and Marattukalam, 1981).

The correlation estimates (Table 3) show significantly positive association of tapping panel dryness with girth and girth increment rate under tapping. Pushpadas (1995) also reported higher incidence of TPD in trees with relatively larger girth, which is attributed to the disparity in availability of inputs like water, nutrients, etc. and the output of latex from trees of varying girth.

The association of TPD with yield, though positive, was not significant. However, it is to be noted that the clones RRIM 609, RRIM 621, RRIM 608, RRIM 603 and RRIM 526 showing high incidence of TPD are among the better yielders of the clones studied. This lends support to earlier reports (IRRDB, 1997) that TPD is a genetic characteristic and is more prevalent among high yielding clones. The syndrome is reported to be an outcome of stress imposed on the rubber tree due to excessive extraction of latex (Paardekooper, 1989). Supporting evidence for this theory is the comparatively higher incidence of tapping panel dryness in clones RRIM 609 and RRIM 603 which did not show any appreciable drop in yield even in the stressed summer months of February-May.

Table 4 shows the estimates of genetic parameters for yield during various years along with those for tapping panel dryness. Yield during the first year and mean yield and girth over nine years of tapping showed moderate to high values of G.C.V., broad sense heritability and genetic advance indicating the existence of additive gene effects, while the estimates of G.C.V., $H^2$ and G.A. for girth increment rate and tapping panel dryness indicated the predominance of

non-additive gene effects. Genetic parameters for yield, girth and girth increment corroborate earlier reports (Mydin *et al.*, 1993) while tapping panel dryness has not been examined in such a perspective before.

Tapping panel dryness emerges as a highly heritable trait inherited through non-additive gene action which results in non-fixable variance. The high heritability value obtained could be due to favourable influence of environment (in this case, the root stock and soil factors) rather than genotype alone and selection for or against such a trait may not be rewarding. Such non-fixable gene action is not immediately fixable by selection as in the case of additive gene action. It gets fixed only if the process of recombination proceeds.

The fact that TPD, though prevalent among high yielders, is not fixable in the first generation holds promise for the *Hevea* breeder as far as early breeding efforts are concerned, since clonal selection follows hybridization in crop improvement programmes. Estimates of broad sense heritability are reliable in the case of a vegetatively propagated species like *Hevea* where all the desirable genetic variation can be fixed by bud grafting. However, in the long run, during the process of generation-wise assortative mating, the need to exercise utmost care in the choice of parents is to be emphasised. Selection against the disorder in the subsequent generation could yield positive results for a TPD free genotype.

Out of the 21 clones studied, 19 showed incidence of TPD (Fig.1), the occurrence in the field being non-random in all the four replications in eight cases, in three out of the four replications in five cases and in two out of the four replications in four cases. Only two clones showed random distribution of the disorder in three out of the four replications.

These observations corroborate earlier reports of non-random distribution of brown bast affected trees (de Souza *et al.*, 1983). According to Paardekooper (1989), TPD affected trees tend to come in clusters and such a non-random distribution is puzzling. He opines that it is unlikely that the disorder would spread through the vessels via root grafts, to neighbouring trees and suggests the involvement of some environmental factors. Murong *et al.* (1994) also reported that TPD affected trees are not distributed randomly in the stand and that the disease is caused by pathogens like RLOs, but this is yet to be proved.

The triggering factor may or may not be an outside agent, but the inherent genetic makeup of the cultivar is what makes the trees susceptible or resistant to TPD. Sensitivity to TPD is a genetic (clonal) characteristic and the most susceptible clones are reported to be the precocious high yielding clones.

As per the taxonomy of the syndrome a division into acute and chronic forms is made (IRRDB 1997). The former is generally irreversible and may be associated with a pathogen as there is evidence that the syndrome affects groups of trees. The latter may be reversible and appears to occur at random although certain clones are more susceptible. In either way, stress is said to be the fundamental reason for the syndrome as also evidenced by the higher incidence among the clones which did not show much yield reduction under stress.

## CONCLUSIONS

The non-random occurrence of TPD in majority of the clones studied needs to be explored

further, supported by pathological investigations. The non additive nature of inheritance of TPD as indicated by the genetic parameters suggests the need to either avoid TPD prone parent clones in advanced generation crosses or to select TPD free genotypes from among the hybrid progeny prior to clonal selection in each cycle of the breeding process.

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***Table 1. Clones studied.***

| Sl.No. | Clone | Parentage |
|---|---|---|
| 1 | RRIM 501 | Pil A 44 x Lun N |
| 2 | *RRIM 526* | Pil B 84 x Pil D 65 |
| 3 | RRIM 601 | Tjir 1 x GT 1 |
| 4 | RRIM 602 | Tjir 1 x GT 1 |
| 5 | RRIM 603 | PB 86 x Pil B 84 |
| 6 | RRIM 604 | Tjir 1 x PB 49 |
| 7 | RRIM 605 | Tjir x PB 49 |
| 8 | RRIM 607 | Tjir 1 x PB 49 |
| 9 | RRIM 608 | AVROS 33 x Tjir 1 |
| 10 | RRIM 609 | AVROS 157 x BD 5 |
| 11 | RRIM 610 | RRIM 504 x Tjir 1 |
| 12 | RRIM 611 | RRIM 504 x Tjir 1 |
| 13 | RRIM 612 | AVROS 157 x PB 49 |
| 14 | RRIM 615 | RRIM 511 x Tjir 1 |
| 15 | RRIM 617 | BR 2 x RRIM 500 |
| 16 | RRIM 618 | Lun N x RRIM 501 |
| 17 | RRIM 620 | RRIM 501 x RRIM 511 |
| 18 | RRIM 621 | RRIM 504 x Tjir 1 |
| 19 | RRIM 622 | Tjir 1 x Pil B 84 |
| 20 | RRIM 623 | PB 49 x Pil B 84 |
| 21 | Tjir 1 | Primary clone |

*Table 2. Tapping panel dryness, yield and girth in clones.*

| Clone | Tapping Panel Dryness (%) | Yield (g/tree/tapping) First year | Yield (g/tree/tapping) Mean of 9 years | Summer yield (% of mean yield) | Girth increment (cm year$^{-1}$) |
|---|---|---|---|---|---|
| Tjir 1 | 0.33 | 19.78 | 34.25 | 58.3 | 2.04 |
| RRIM 609 | 17.07 | 25.80 | 37.70 | 76.9 | 3.09 |
| RRIM 501 | 0.85 | 36.68 | 46.83 | 71.2 | 1.63 |
| RRIM 605 | 0.32 | 32.55 | 42.83 | 68.5 | 1.82 |
| RRIM 615 | 0.38 | 22.53 | 34.35 | 60.8 | 1.70 |
| RRIM 620 | 0.76 | 29.45 | 41.43 | 66.4 | 2.09 |
| RRIM 621 | 8.82 | 30.63 | 38.55 | 61.9 | 1.85 |
| RRIM 610 | 1.73 | 30.90 | 34.10 | 61.3 | 1.66 |
| RRIM 607 | 1.79 | 27.38 | 38.73 | 68.7 | 2.27 |
| RRIM 611 | 0.37 | 18.03 | 28.00 | 68.7 | 1.93 |
| RRIM 623 | 1.67 | 28.00 | 37.45 | 58.9 | 2.20 |
| RRIM 612 | 0.75 | 23.63 | 35.35 | 65.8 | 4.09 |
| RRIM 618 | 0.98 | 26.00 | 35.18 | 68.8 | 1.54 |
| RRIM 601 | 1.89 | 24.18 | 35.20 | 65.6 | 1.50 |
| RRIM 603 | 4.47 | 25.60 | 38.53 | 75.8 | 3.01 |
| RRIM 526 | 1.95 | 24.20 | 37.00 | 64.6 | 3.00 |
| RRIM 608 | 5.21 | 24.70 | 41.00 | 65.9 | 2.04 |
| RRIM 617 | 1.54 | 24.83 | 31.43 | 56.4 | 1.44 |
| RRIM 622 | 1.87 | 26.35 | 35.38 | 57.3 | 2.56 |
| RRIM 604 | 0.00 | 24.80 | 35.43 | 61.0 | 1.80 |
| RRIM 602 | 0.00 | 22.55 | 37.55 | 66.8 | 2.16 |
| General Mean | 2.51 | 26.12 | 36.96 | 65.40 | 2.16 |
| Variance ratio (clones) | 9.52** | 7.51** | 6.36** | | 7.87** |
| C.D. (0.05) | 0.002 | 4.40 | 4.52 | | 0.66 |

***Table 3. Correlation of tapping panel dryness with yield and growth.***

| Character | Phenotypic correlation coefficient | Genotypic correlation coefficient |
|---|---|---|
| Yield - first year | 0.1118 | 0.1009 |
| Yield - ninth year | 0.1307 | 0.3095 |
| Yield - mean over nine years | 0.1223 | 0.1521 |
| Girth - at ninth year of tapping | 0.2650* | 0.3342 |
| Girth increment rate | 0.2589* | 0.3150 |

Significant at P=0.05

***Table 4. Estimates of genetic parameters for yield, girth and TPD.***

| Character | G.C.V. | P.C.V. | $H^2$ (%) | G.A. |
|---|---|---|---|---|
| Yield - first year | 60.26 | 97.26 | 61.96 | 6.43 |
| Yield - ninth year | 100.39 | 259.87 | 38.63 | 7.61 |
| Yield - mean over nine years | 36.95 | 64.56 | 57.24 | 5.76 |
| Girth at opening | 9.48 | 16.62 | 57.08 | 3.56 |
| Girth at ninth year of tapping | 37.97 | 59.77 | 63.52 | 8.86 |
| Girth increment rate | 17.56 | 27.78 | 63.21 | 1.01 |
| Incidence of TPD | 0.032 | 0.047 | 68.04 | 0.03 |