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INTRASPECIFIC VARIATION IN SOLUBLE PROTEIN AND ISOZYME PATTERNS IN *RIGIDOPORUS LIGNOSUS*, THE CAUSATIVE AGENT OF WHITE ROOT DISEASE ON *HEVEA BRASILIENSIS*

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Rigidoporus lignosus, widespread in equatorial and humid tropical zones, is the most dangerous root parasite on rubber tree. Twenty isolates collected from a wide range of geographical origins were studied to evaluate intraspecific variability of this fungus using native polyacrylamide gel electrophoresis technique. Soluble protein banding patterns obtained by isoelectric focusing and isozyme profiles showed a high level of intraspecific polymorphism in *R. Lignosus*. Factor analysis of soluble protein banding patterns unequivocally separated several groups of isolates according to their geographical origins. Enzyme profile comparisons also conformed to biogeographical patterns, with greatest isozyme polymorphism in the Far-East than in Africa. However, geographical distances by themselves were not sufficient to explain all the intraspecific variability.

Key words : *Hevea brasiliensis*, *Rigidoporus lignosus*, White root disease, Isoelectric focusing.

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INTRODUCTION

Rigidoporus lignosus is a widespread white root rot parasite in equatorial and humid tropical zones. This fungus has a wide host range and attacks more than one hundred shrubby species (Nandris *et al.*, 1987a). Although chemical control may be attempted to reduce disease severity (Gohet *et al.*, 1991), *R. lignosus* remains the most dangerous root parasite of rubber tree, *Hevea brasiliensis*, in most of the *Hevea* growing zones (Compagnon, 1986). Woody debris buried in the ground after forest clearing is the primary source of infection in new *Hevea* plantations. The disease spreads mostly from infected trees to healthy ones through lateral root contacts. White root disease expression may vary from one region to another (Peries and Liyanage, 1986; Chee, 1990).

Comparisons of *in vitro* wood degradation ability demonstrated significant variation among African isolates (Nicole *et al.*, 1985). Differences in isolate pathogenicity

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