

PRODUCTION OF CYANIDE BY SEEDS OF *HEVEA BRASILIENSIS* CULTURED *IN VITRO*

J. El Hadrami and J. d'Auzac

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Fragments of internal tegument from immature seeds of *Hevea brasiliensis* cultured *in vitro* produced substantial quantities of hydrogen cyanide (HCN). Release of HCN was considerable during the initial hours and probably resulted from intracellular decompartmentation caused by the slicing of the tissues in culture preparation. Cyanogenic potential depends on the age of seed and appears to be variable from one clone to another. When culture was carried out under confined atmosphere, HCN was present for the first 20 days. Cellulose caps enhanced gas exchanges and led to better callogenesis under the conditions of the experiment.

Key words: *Hevea* seeds, cyanide, *in vitro* culture, callogenesis, *Hevea brasiliensis*.

J. El Hadrami and J. d'Auzac (for correspondence), Laboratory of Applied Plant Physiology, University of Montpellier 2, F-34095 Montpellier Cedex 2, France.

INTRODUCTION

Like many Euphorbiaceae, *Hevea brasiliensis* is cyanogenic. *Hevea* seeds contain cyanogenic glucosides (linamarin, linustatin), over 90 per cent of which being found in the endosperm (Lieberei *et al.*, 1988). The cyanogenic potential of the seeds decreases by 85 per cent during the first 19 days of growth of plantlets. Indeed, Selmar *et al.* (1987) showed that monoglycosylated linamarin is converted to diglycosylated linustatin which is translocated to young leaves where it leads to asparagine, an important nitrogen source for plantlets.

In addition to this recently discovered role as nitrogen reserve, cyanogenic compounds are thought to play a role in defence against predators and pathogens (Schonbeck and Schlosser, 1976). However, it has been shown that release of HCN in *Hevea* during attack by *Microcyclus ulei* enhances

the attack than it protects the host plant (Lieberei, 1984; Lieberei *et al.*, 1983; Lieberei, 1986), which indicates an inverse relation between cyanogenic activity and resistance to the pathogen. The same authors showed that inhibition of active defence in *Hevea* was in particular the result of phytoalexin production by HCN (Lieberei *et al.*, 1989).

Tissue lesions in cyanogenic plants result in decompartmentation and contact between vacuolar or cytoplasmic compounds, i.e. contact between cyanogenic glycosides and the enzymes responsible for HCN release (B-glucosidase and hydroxynitrile-lyase). In addition, HCN, which chelates heavy metals, may inhibit functioning of the respiratory chain and certain metal oxidases such as peroxidases, polyphenol oxidases (Lieberei, 1986), catalase and superoxide dismutase.