

**Anatomical study of the degradation of canker wood in tropical trees
and identification of the causal fungi**

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Many studies have reported fungal stem canker diseases associated with wood decay in various species of tropical forest trees, either in plantation forest, e.g. *Acacia mangium* [1] and *Gmelina arborea* [2], or in natural forest and rubber plantations, e.g. *Shorea smithiana* [3] and *Hevea brasiliensis* [4]. Generally, the trees exhibit localized dead areas in the bark, cracks and ruptures on the surfaces of the bark near the canker zone, and apparently decayed exposed sapwood.

Microscopic observations have been conducted to reveal the anatomical features of the xylem of meranti trees [light red meranti (*Shorea smithiana*) and yellow meranti (*Shorea gibbosa*)] and rubberwood (*Hevea brasiliensis*) infected with stem canker [5,6]. Although microscopic observation techniques are not directly applicable to field use, they can provide valuable information for understanding the altered properties of infected xylem. By microscopic observation, the decay pattern of the infected xylem also can be categorized easily and a preliminary identification made of the causal microorganism.

In *S. smithiana* and *S. gibbosa* canker, intensive degradation of xylem cells in the canker margin was conspicuous (Fig. 1). In the degraded xylem, patterns of simultaneous white-rot fungi, such as thinning of cell walls, bore hole formation, rounded pit erosion and eroded channel opening, as well as large voids resulting from the coalition of completely removed xylem cells, could be well recognized. However, the extent of cell damage in *S. smithiana* xylem was higher than that in *S. gibbosa*. In degraded xylem of *S. smithiana*, erosion troughs and numerous conspicuous holes in cell walls were more obvious, and the large voids resulting from the coalition of completely removed cells were also severely enlarged and extended.

The isolation of decay fungi and their identification in affected xylem of cankerous trees is a useful method of detecting their presence in decayed xylem. By phylogenetic analysis based on the sequences of the internal transcribed spacer region of rDNA, Basidiomycete fungi isolated from the decayed xylem of *S. smithiana* and *S. gibbosa* canker were identified as *Schizophyllum commune* and *Phlebia breviospora*, respectively. This is the first report of successful isolation of decay fungi from *S. smithiana* and *S. gibbosa* canker. Therefore, the results of this study could be a useful reference for further pathological studies of tropical trees.

The anatomical changes caused by the isolated decay fungi in the laboratory could provide the basis for diagnosing and evaluating decay by *S. commune* and *P. breviospora* on *S. smithiana* and *S. gibbosa* wood, respectively. In laboratory conditions, simultaneous white rot of *S. smithiana* and *S. gibbosa* wood caused by *S. commune* and *P. breviospora*, respectively, was well characterized. Their decay patterns were similar to those of the decayed xylem of *S. smithiana* and *S. gibbosa* cankerous trees in field conditions. In *S. smithiana* wood, slight erosion of wood cell walls over 12 weeks' incubation with *S. commune* resulted in a small weight loss and could be classified as the early stage of simultaneous decay. These results showed that *S. commune* was a serious destroyer of wood under natural conditions, especially in tropical regions, but caused little wood decay *in vitro*. Meanwhile, in *S. gibbosa* infected with *P. breviospora*, the intermediate decay features of numerous, conspicuous holes as well as erosion troughs in cell walls were found after 8 weeks' incubation. Furthermore, complete degradation of wood cell components, defined as the advanced stage of decay, was found in some areas of wood after 12 weeks' incubation *in vitro*.

In *H. brasiliensis* canker, typical characteristics of infected xylem were confirmed by the presence of abnormal xylem formation in the vicinity of enclosed bark in the stem as well as xylem decay in the canker margin. The abnormal xylem contained fewer shorter vessels of smaller diameter, and significantly wider rays compared with normal xylem, and around the wide growth zones of the canker, axial cells were disoriented and warped towards the canker zones. The decayed xylem exhibited separation among cells and rounded pit erosion, similar to that seen in decay caused by white-rot fungi. I considered that even though the bark was tapped repeatedly, while the trees were still actively producing latex canker did not occur on *H. brasiliensis* stems. Apparently, the latex acts as an antifungal compound and plays an important role in restricting fungal infection of the wounded tissues, so that the fully developed surface callus could

completely close the bark wound when wound cambium had formed. Canker disease on *H. brasiliensis* was assumed to be initiated by unhampered pathogen infection of the open wound of the tapping bark, when the trees were not producing latex or when their latex yield was extremely low.

To increase our understanding of canker wood in tropical trees, the progress of infection by pathogenic fungi should be studied in different types of plantation forest using various kinds of trees and under different physiological conditions.

Plantation forests are increasing in tropical countries. In East Kalimantan, Borneo, plantations of rubberwood and meranti species are increasing. Because high quality timber from these plantation forests is in strong demand, cankerous wood should be reduced by introducing cultural methods to prevent infection by pathogenic fungi.

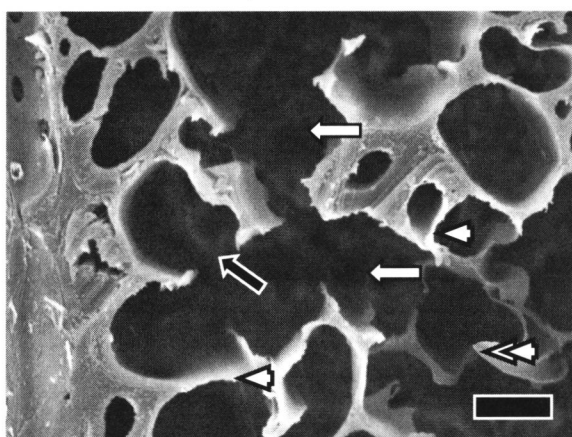


Fig. 1. Decay features of xylem in the canker margin of *S. smithiana*. Note thinning (single head arrows) and collapse (double head arrow) of the cell walls, erosion channel (black arrow), and complete removal of cells (white arrows), which simultaneously occurred in one region of decay. Bar 10 μ

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