ABSTRACT

The effect of *Crinipellis perniciosa* (Stahel) Singer infection on cotyledonary abscission in cacao seedlings.

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Cotyledonary abscission in cacao (*Theobroma cacao* L.) seedlings, and its prevention by artificial inoculation with *Crinipellis perniciosa* (Stahel) Singer was examined morphologically, anatomically, histochemically, physiologically and biochemically. It was found that non-senescent cotyledonary stalks did not have a well-defined abscission zone. However, senescence was associated with the formation of a separation layer which constituted the region at which abscission occurred. The onset of senescence was suggested to be signalled by a critical proportion of depleted cells to filled storage cells. Electrophoretic studies revealed that a unique isoperoxidase was associated with separation layer development. Separation occurred adaxially to abaxially, but was centrifugal in tissues containing the vascular tissues and followed an irregular pathway through the cell walls and middle lamellae. Apart from enzymatic factors, mechanical factors e.g stress at the fracture face due to release of pentoses and a consequent reduction in crosslinking; breakage of xylem vessels and external vibrations were also suggested to participate in abscission.

The studies conducted on infected seedlings revealed that:

(a) initiation of the separation layer was prevented;

(b) lignification of stalk cells was not responsible for the non-abscission of cotyledons in non-necrotic seedlings;

(c) a translocatable factor responsible for symptom induction was probably absent;

(d) cotyledonary lipid reserves were utilized at a slower rate than those of healthy seedlings;

(e) cotyledonary sink strength was due to factors other than an increased photosynthetic rate;

(f) phenolic compounds were tightly associated with symptom expression;

(g) cytokinins were probably the main PGRs responsible for symptom expression;

(h) dry matter accumulated in the root and shoot systems at a slower rate but the shoot to root ratio was similar to healthy seedlings;

(i) the rate of total dry matter accumulation was significantly lower than that of healthy seedlings, in spite of axillary shoot production, hypertrophy and callus formation in infected seedlings;

(j) the utilization of cotyledonary reserves was delayed and reduced in comparison to that of healthy seedlings;

(k) the water content of the cotyledons was maintained at a higher level during pathogenesis;

(l) the activity of isoperoxidases common to abscission and maturation were observed and

(m) an additional isoperoxidase and a laccase isozyme were present only in diseased areas.

In order to prevent cotyledonary abscission, the pathogen needed an incubation period of 5-11 days prior to the initiation of abscission in order to reach a critical distance from which it could exert its effects on the cells which would form the separation layer. Phenolic accumulation was viewed as an integral part of the host-pathogen response and was suggested to be responsible for the necrosis and eventual death of cells in infected seedlings. The prevention of cotyledonary abscission in non-necrotic seedlings was presumably due to the maintenance of reserve material above a critical limit of 20% of the initial CDM and the influence of altered auxin and, moreso, cytokinin levels in delaying senescence effects. Eventually, necrosis and death of cells prevented any physiological separation and the cotyledons persisted until mechanical forces detached the cotyledonary stalk from the node. It is recommended that the role of cytokinins, auxins, phenolic compounds and oxidative enzymes in symptom development in infected seedlings be critically assessed.