D37 PECTINASES AND PROTEASE ENZYME PRODUCTION BY TWO *COLLETOTRICHUM* SPECIES HAVING DIFFERENTIAL DISEASE DEVELOPMENT IN PAPAW FRUIT

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Colletotrichum gloeosporioides, the cause of anthracnose disease in papaw (Carica papaya), develops progressive rotting only when the fruit has reached fully ripened stage (maturity stage 4) whereas C.capsici, another pathogen on papaw, can cause similar, but much larger, lesions 2-3 days before the fruit ripens fully (maturity stage 3). Both fungi are unable to produce lesions in the unripe fruit (maturity stage 1 and 2).

Colletotrichum gloeosporioides grown in liquid cultures incorporated with crude cell walls of unripe papaws produced more polygalacturonate trans-eliminase (PGTE), a trace of polygalacturonase (PG) and protease. Similar cultures of C.capsici contained higher PGTE, moderate PG and protease activity. Pectinase (both PGTE and PG) production by both fungi was greatly stimulated when the cultures were provided with crude cell walls from ripe fruit than unripe.

Pectinase activity of C.capsici culture filtrates was inhibited in vitro by the papaw latex but a similar phenomenon was not observed with Colletotrichum gloeosporioides culture filtrates. Latex content in the fruit was high when unripe and declined during ripening[1] and the inability of C.capsici to produce lesions in the unripe fruit may probably be due to the inhibition of pectinase by the latex. Pectinases may be active in the ripe fruit where the latex content is low.

Resistance of Colletotrichum gloeosporioides to unripe papaws is not related to an inhibition of pectinase, as revealed in these experiments. Also this phenomenon could not be attributed to the antifungal activity of the unripe fruit tissue or to the nutrient differences between unripe and ripe fruit[1].

Reference

1. Dharmasiri, M.A.N., Jayatissa, P.M., and Adikaram, N.K.B. (1985) Proc. Sri Lanka Ass. Adv. Sci. 41 56.