

INDUCTION OF CHITINASE AND PHENOLICS IN BANANA CV. EMBUL IN RESPONSE TO *PHYLLUSTICTA MUSARUM* INFECTION AND ELICITORS OF INDUCED DEFENCE RESPONSES

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In a previous study, *Phyllosticta musarum*, the causal organism of freckle disease, was shown to induce several defence reactions in the immature banana fruit cv. Embul. These include phytoalexin and PR protein accumulation, lignification and suberization at the infection site. *P. musarum* infected bananas are resistant to anthracnose disease caused by *Colletotrichum musae*. The objectives of the present study were to investigate *P. musarum* - induced defence responses further and search for effective elicitors to induce banana fruit defences.

Total soluble phenolic acids, free phenolic acids, glycoside bound and ester bound phenolic acids were extracted and quantified from both *P. musarum* infected and non-infected fruit peel. Chitinase enzyme (a PR protein) activity in the peel of freckled and non-freckled banana inoculated with *C. musae* or without inoculation was assessed using a gel diffusion assay. Cell wall derived elicitors of *P. musarum* and *C. musae* were extracted and tested for the induction of browning and accumulation of phytoalexins in the host tissue. The chemical nature of the crude cell wall extracts was also tested with general chemical tests. In order to determine efficacy of chemical defence inducers, banana fruits at harvesting maturity were sprayed with different concentrations of Salicylic acid (SA) or Acibenzolar-s-methyl (Bion®). The concentrations tested were 0, 10, 500, 1000 and 2000 mg/l of SA and 0, 20, 200, 400 and 1000 mg/l of Bion®. Treated fruits were inoculated with *C. musae* and anthracnose disease development was recorded.

An increased phenolic synthesis was observed in fruits with freckled peel than those with non-freckled peel. The amount of all the sub-classes of phenolic acids was found to be higher in freckled peel. Chitinase enzyme activity was markedly higher in the unripe freckled peel than that of the non-freckled peel. A marked increase in the level of chitinase was observed with the onset of anthracnose lesion development more prominently in the freckled peel.

The cell wall extracts of *P. musarum* or *C. musae*, applied on to wounded sites elicited rapid browning and accumulation of phytoalexins in banana tissues, inferring that *P. musarum* extract was more effective than the *C. musae* extract in elicitation. The crude elicitor extract of *P. musarum* contained mainly polysaccharides while the extract of *C. musae* contained polysaccharides or/and glycoproteins. Spray treatment of freshly harvested banana with SA or Bion® resulted in a significant (ANOVA, $p = 0.05$) decrease in anthracnose disease development during ripening. SA at 500mg/l and above reduced anthracnose lesion area by 17–28% and Bion® at 200mg/l reduced the lesion area by 50%. However, the reduction was not consistent with increasing SA or Bion® concentration. In conclusion, the present study revealed two new defence responses associated with freckle infection; the induction of chitinase enzyme and phenolic acids. The elicitors of *P. musarum* and *C. musae* are chemically different. Chemical defence activator Bion® is more effective than SA in inducing resistance in banana fruit cv. Embul.