CHANGES IN pH FOLLOWING FRECKLE INFECTION (PHYLLOSTICTA MUSARUM) AS A DEFENSE RESPONSE AGAINST ANTHRACNOSE

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The host environment pH is found to be a regulatory factor in pathogenesis of certain fungi. Some fungi increase the host pH while others tend to reduce it. Certain *Colletotrichum* spp. have the ability to modulate host pH by secreting ammonia locally into the host tissue, resulting in a pH increase which enables enzymatic secretion and enhance virulence.

Colletotrichum musae causes quiescent infections in unripe green banana fruits which develop into anthracnose lesions during fruit ripening. Freckle disease in banana caused by *Phyllosticta musarum* is associated with a number of defence responses viz. accumulation of phytoalexin and PR proteins, and certain other changes such as lignification and suberization and accumulation of tannins, which in turn reduce the development of anthracnose disease. Six phytoalexins have been isolated from freckle infected banana peel, which contribute to defense against *C. musae*.

The objective of this study was to determine whether P. musarum causes a pH alteration in the banana peel and if so whether this acts as an additional defence response by the fungus against anthracnose.

Freckled and non freckled "*Embul*" fruits at a similar stage of maturity were obtained from the same bunch. A set of freckled fruits and non freckled fruits were inoculated with 50 μ l of *C. musae* spore suspension (10⁵ spores ml⁻¹) per inoculation, while the control sets of freckled and non freckled fruits were treated with sterile distilled water. Lesion diameters and pH of the peel were measured daily for 7 days.

There was a significantly higher anthracnose development in non freckled fruits compared to the freckled fruits. This was observed when fruits were artificially inoculated and also when natural quiescent infections of *C. musae* were allowed to develop. The freckle infected banana peel had a significantly (p=0.05 level) higher pH than the non freckled peel throughout the ripening process. This indicates that *P. musarum* infection increases host pH.

In fruits inoculated with *C. musae*, the peel pH was significantly higher (p = 0.05 level) in the freckled peel compared to the non-freckled peel during initial necrosis. Furthermore, at the onset of tissue maceration pH of the freckled peel was higher than that of the non-freckled peel. However with the expansion of lesions the pH in the non-freckled peel was significantly higher at p=0.05 level, when compared to the freckled peel.

The higher values of pH in the freckled peel during initial necrosis and the onset of tissue maceration could be a contributing factor in limiting anthracnose in bananas. The increase in pH at an advanced stage of lesion development, in non freckled peel, may be due to rapid tissue maceration caused by *C. musae*.

These results indicate that pH increase following P. musarum infection may be an additional defense response in banana fruit.