POSTHARVEST LOSSES AND INDUCTION OF RESISTANCE IN AUBERGINE (*SOLANUM MELONGENA*) AGAINST ANTHRACNOSE CAUSED BY *COLLETOTRICHUM CAPSICI*, USING A WEAK PATHOGEN, *FUSARIUM SOLANI*

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A market survey conducted in Kandy area in the central province of Sri Lanka, revealed that approximately 19% losses occur in aubergines at the market and several factors including improper handling practices, fungal diseases, insect damage and physiological disorders contribute to the loss. Anthracnose caused by *Colletotrichum capsici* was identified as a major postharvest disease in aubergine and *Phomophsis* rot and *Fusarium* rot were also encountered during the survey. Investigations were conducted to examine the possibility of inducing resistance in aubergine against anthracnose disease using a relatively weaker pathogen, *Fusarium solani*, and establish the mechanism involved in induction of resistance. Anthracnose disease is currently controlled by application of fungicides. Enhancement or induction of natural resistance mechanisms could have a potential for controlling postharvest diseases in fruits and serve as an alternative approach for fungicides.

C.capsici develops anthracnose lesions in both wounded and unwounded aubergines and the lesion development was observed to be faster in the wounded fruit. *F. solani,* however, caused lesions only when the fruits were inoculated after wounding and not in intact fruits and it shows that *C.capsici* is a more aggressive pathogen in aubergine than *F.solani*. Treatment of unwounded sites with conidia of *F.solani*, at least two days prior to inoculation with *C.capsici* delayed anthracnose development by six days, compared to controls, treated with sterile distilled water. Co-inoculation with conidia of both fungi did not slow down *C.capsici* rotting.

The growth of neither *C.capsici* nor *F.solani* was affected on agar medium. Also *in vitro* germination studies on a mixed preparation of conidia of the two fungi indicated that germination of conidia or appressoria formation of *C.capsici* was not affected by the presence of conidia of *F.solani*. These observations confirm that *F.solani* has no antagonistic effect on *C.capsici.*

Ethyl acetate extracts of peel tissue obtained from aubergines two days after inoculation with conidia of *C.capsici* or *F.solani* when bioassayed on TLC plates with either *C.capsici* or *Cladosporium cladosporioides* showed one prominent antifungal zone at Rf 0.70 and not in healthy tissue showing that the aubergine tissue accumulates phytoalexin/s in response to inoculation by either pathogen. Inoculation of aubergines with either pathogen after wounding resulted in accumulation of more phytoalexin than the fruit inoculated without wounding. In both, the amount of phytoalexin increased progressively with the increase of incubation period after inoculation. The amount of phytoalexin accumulated in fruit tissue inoculated with *F.solani* was significantly greater at all incubation periods than in the tissue obtained from fruits inoculated with *C.capsici.* It shows that the pre-inoculation of aubergines with *F.solani* results in greater phytoalexin accumulation, which is sufficient to prevent the lesion development by *C.capsici. F.solani* appears to be a more effective elicitor of host natural resistance than *C.capsici* and it appears to be associated with phytoalexin accumulation rather than the other mechanisms involved in the induced resistance responses in plants.

To purify phytoalexins, 50g of tissue inoculated with *F.solani* were extracted and the extract was fractionated by flash chromatography. Two compounds with antifungal properties were obtained in pure form. The major compound that accounts for the most antifungal activity was identified as lubimin.

It can be concluded that natural resistance in aubergines against *C.capsici* could be induced using a weaker pathogen, *F.solani* and phytoalexin accumulation appears to be one of the mechanisms of induced resistance.