The influence of prior heat shock protein induction on programmed cell death in tobacco (*Nicotiana tabacum*) during a challenge with *Ralstonia solanacearum*

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In plants and animals there is a complex host-pathogen defence regime, potentially modulating the expression of heat shock proteins (HSP). Host HSP induced during bacterial infection may preserve protein integrity in susceptible interactions and facilitate maturation of newly synthesised defence proteins in resistance responses. Altering host HSP expression may influence this outcome by effecting host programmed cell death.

We hypothesized that a prior induction of HSP, as measured by Hsp70/Hsc70 expression, could positively affect programmed cell death (PCD) - specifically apoptosis, of tobacco during subsequent infection with various strains of the pathogen *Ralstonia solanacearum*.

The Objective of this study included the investigation of a prior induction of Hsp70/Hsc70, followed by the determination of apoptosis parameters such as the flow cytometric detection of Annexin-V PI, TUNEL, mitochondrial membrane potential, Bcl-2/Bax and luminometric detection of ATP and finally determining the effect of a prior HSP induction on the cell death (apoptosis/necrosis) of tobacco cells during a virulent and avirulent pathogen challenge.

Preliminary results indicate that HSP are differentially influenced during bacterial infection, and that a prior induction of host HSP could protect the host against severe bacterial infection most likely by influencing PCD. In conclusion the possible protective nature of HSP induced with or prior to infection may in general be protective but may also counteract apoptosis and influence host survival.