The effect of LPS isolated from *Ralstonia solanacearum* on Hsp70/Hsc70 accumulation and factors associated with cell death in tobacco (*Nicotiana tabacum*).

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Plants have evolved both constitutive barriers and induced defense mechanisms to protect themselves against pathogen attack. Changes in cytoplasmic Ca^{2+} levels, accumulation of reactive oxygen species (ROS) and nitric oxide (NO) as well as the post-translational activation of mitogen-activated protein kinase (MAPK) cascades amplify the innate immune responses during incompatible host-pathogen interactions and initiate a cell death program commonly referred to as the hypersensitive response (HR). The HR is a complex response following pathogen recognition, characterized by apoptotic (programmed) cell death (PCD), cell wall reinforcement, accumulation of anti-microbial compounds including pathogenesis-related (PR) proteins, and the formation of necrotic lesions to ensure pathogen containment. Lipopolysaccharides (LPS) from Gram-negative bacteria are reportedly strong general elicitors (pathogen-associated molecular patterns, PAMPS) of plant defense responses.

Heat shock proteins (HSP), although not considered classical defense proteins, protect against many lethal stresses. Hsp70, in particular, provides protection against various stresses and appears to prevent the induction of PCD. In plants, increased expression of Hsp70 and an associated decrease in PCD could counteract the plants ability to initiate a protective PCD-based HR with the aim of pathogen containment.

The aim of this study was to determine the effect of LPS (isolated from *R. solanacearum*) on Hsp70/Hsc70 accumulation and on factors associated with cell death in tobacco cells.

Results showed that LPS inhibited Hsp70 accumulation. However, it would appear that LPS does not induce PCD in tobacco. It is possible that the observed decrease in Hsp70 accumulation ensures sustained defense responses associated with early events of PCD.