The effect of Hsp70 on cytochrome c translocation from mitochondria during programmed cell death in plants.

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Programmed cell death (PCD) is an intricately regulated process with many factors contributing to or preventing the death of a cell. A large group of such factors are the heat shock protein (Hsp) family. Hsps have been shown to inhibit cell death at multiple points in the PCD pathway in animal cells. These points include the inhibition of JNK activation, prevention of cytochrome c release from the mitochondria, disruption of the apoptosome formation by binding to cytochrome c, inhibition of Apaf-1 oligomerization and suppression of procaspase recruitment. It has previously been shown that cytochrome c translocation occurs during plant PCD. Furthermore, a correlation was observed between elevated levels of Hsp70 and decrease levels of PCD in plants. We hypothesize that the mechanism of Hsp70-mediated decrease in PCD is by the intervention of cytochrome c activity. In this work, suspended tobacco cells, treated with a lethal dose of hydrogen peroxide, are prevented from undergoing PCD by receiving a mild heat shock at the onset of treatment. Western blot analysis shows that an increase in induced Hsp70 corresponds to a decreased level of cytochrome c in the cytosolic fraction. Preliminary data indicates that Hsp70 may be involved in the negative regulation of PCD at mitochondrial level in tobacco cells.