

The possible role of heat shock factor-1 in the negative regulation of heme oxygenase-1

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Abstract

We examined a possible role for heat shock factor-1 (HSF-1) in the negative regulation of HO-1 gene expression in human Hep3B hepatoma cells responding to stimulation with 15-deoxy-Delta^{12,14}-prostaglandin J₂ (15d-PGJ₂) and arsenite. Overexpression of HSF-1 and heat-shock experiments indicated that HSF-1 repressed the 15d-PGJ₂-and arsenite-induced HO-1 gene expression through directly binding to the consensus heat shock element (HSE) of the HO-1 gene promoter. In addition, point mutations at specific HSE sequences of the HO-1 promoter-driven luciferase plasmid (pGL2/hHO3.2-Luc) abolished the heat shock- and HSF-1-mediated repression of reporter activity. Overall, it is possible that HSF-1 negatively regulates HO-1 gene expression, and that the HSE present in the -389 to -362 region mediates HSF-1-induced repression of human HO-1 gene expression.