Pharmacological Modulation of Generalized Inflammatory Response in Hepatocytes by Prolactin.

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The mortality of severe burn injury and sepsis has largely been attributed to the generalized inflammatory response following such injury. In the liver, for example, hepatocytes undergo an acute phase response (APR) in which transcriptional activity switches from that of constitutive proteins to so called acute phase reactants. This response, intended to protect the cell, runs unchecked in burn patients and leads to cell damage and ultimately organ failure. The hypothesis that the hormone Prolactin might ameliorate this APR though the action of a family of proteins termed Suppressor of Cytokine Signaling (SOCS) proteins was tested here. It was found through rtPCR analysis that Prolactin upregulated SOCS3 transcription in HepG2 cells when treated in concert with the inflammatory cytokine IL-6, but did not effect SOCS2 transcription under the same reaction conditions. Also, ELISA performed on cell supernatants showed Prolactin did not effect the HepG2 cell's production of the acute phase reactant LBP. These *in vitro* results lead us to conclude that Prolactin may indeed play a role in ameliorating the APR, and that SOCS3 could be a major mediator of that effect, but further investigation is needed to determine if it modulates APR by downregulating other acute phase reactants.