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## Hepatoprotective effects of S-adenosylmethionine and silybin on canine hepatocytes in vitro.

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#### Abstract

Inflammation and oxidative stress are associated with liver injury and development of liver disease. The transcription factors nuclear factor-kappa beta (NF-κB) and nuclear factor erythroid 2-related factor 2 (Nrf2) play critical roles in modulating liver injury and damage. Activation of NF-κB induces production of pro-inflammatory molecules including prostaglandin E2 (PGE2), interleukin-8 (IL-8) and macrophage chemotactic protein-1 (MCP-1). Nrf2 regulates genes controlling antioxidants. Our laboratory previously showed that hepatocytes, the primary functional cell type comprising liver tissue, respond to the cytokine interleukin-1 beta (IL-1β) by increased production of PGE2, IL-8 and MCP-1. This increase is associated with nuclear translocation of NF-κB. In this study, we evaluated whether primary canine hepatocytes pre-treated with the combination of S-adenosylmethionine (SAME; 30 and 2000 ng/ml) and silybin (SB; 298 ng/ml), agents with known anti-inflammatory and antioxidant properties, could attenuate IL-1β-induced inflammation and oxidative stress. The SAME and SB combination reduced cytokine-induced PGE2, IL-8 and MCP-1 production while also inhibiting NF-κB nuclear translocation. These changes were accompanied by increased antioxidant enzyme-reduced glutathione (GSH) comparable to control levels. The study shows for the first time that the SAME and SB combination inhibits both inflammation and oxidative stress through two separate signalling pathways.

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