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## Basic: Acute Pancreatitis

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### Heparin Exacerbates Lung Inflammation in Experimental Acute Pancreatitis through Mobilisation of Xanthine Oxidase

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**Objective:** To evaluate the effect of low molecular weight heparin in xanthine oxidase concentration in plasma during acute pancreatitis and its effect in the inflammatory process in the lung.

**Design:** Randomized, controlled trial.

**Setting:** Experimental laboratory.

**Subjects:** Male Wistar rats.

**Interventions:** Acute pancreatitis was induced by intraductal administration of 5% sodium taurocholate. Low molecular weight heparin (0, 30, 90 or 300 U/kg) was administered immediately after induction of pancreatitis.

**Measurements and Main Results:** Three hours after induction, plasma concentrations of lipase and xanthine oxidase were measured. Myeloperoxidase activity was measured in lung and liver as a marker of neutrophil infiltration. Expression of P-selectin mRNA was measured in lung. During pancreatitis there are an increase in plasma concentration of xanthine oxidase. Administration of heparin also induces an increase in xanthine oxidase concentration both in control and pancreatitis animals. Neutrophil infiltration in the lung showed increased levels during pancreatitis, and heparin increased myeloperoxidase activity in a dose dependent manner. In control animals, heparin administration had no effect on the myeloperoxidase activity. By contrast, in the liver there are no changes as a consequence of pancreatitis or heparin treatment. Pancreatitis induces the expression of P-selectin into the lung. This increase was more relevant when heparin was administered. By contrast, in control animals there is no expression of P-selectin.

**Conclusion:** During acute pancreatitis, heparin administration could mobilize xanthine oxidase attached to endothelial cells, generating a systemic free radical-generating system that could trigger an inflammatory reaction in the lungs.