# Melatonin prevents endotoxin-induced circulatory failure in rats

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### 摘要

#### Abstract

The pineal secretory product melatonin was found to exert protective effects in septic shock. In a host infected by bacterial lipopolysaccharide (LPS), the expression and release of proinflammatory tumor necrosis factor-a (TNF- $\alpha$ ) is rapidly increased, suggesting that TNF- $\alpha$  is associated with the etiology of endotoxic shock. Recent reports show that the expression of NO synthase (NOS) II and the production of superoxide anion (O2.) also contribute to the pathophysiology of septic shock. In the present study we demonstrate that melatonin prevents circulatory failure in rats with endotoxemia and improves survival in mice treated with a lethal dose of LPS. The beneficial hemodynamic effects of melatonin in the endotoxemic animal appear to be associated with the inhibition of (i) the release of TNF- $\alpha$  in plasma, (ii) the expression of NOS II in liver, and (iii) the production of O2. in aortae. In addition, the infiltration of polymorphonuclear neutrophils into the liver from the surviving LPS mice treated with melatonin was reduced. Thus, our results support the clinical use of melatonin in endotoxemia