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Title: Remifentanil attenuates LPS-induced neutrophil activation

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Body: Surgical trauma and anesthesia are associated with a complex dysregulation of the immune system with neutrophil activation involving activation of both pro-inflammatory and anti-inflammatory cytokines. Several studies demonstrate that opioids modulate the immune response via opioid receptors expressed directly on the immune cells themselves. Neutrophils play a pivotal role in the coordination and regulation of immune responses. However, the ability of opioid directly participating in LPS-induced neutrophil activation has not been fully examined. In the present experiments, the effects of various opioids including remifentanil, sufentanil, alfentanil and fentanyl were investigated. Remifentanil only could attenuate activation of neutrophils exposed to LPS. In particular, remifentanil decreased LPS-induced activation of intracellular signaling pathways, including p38 mitogen-activated protein kinase (MAPK) and ERK1/2, and expression of pro-inflammatory cytokines, including TNF-a, IL- 6 and IL- 8. There was no effect of remifentanil on LPS-induced activation of c-Jun N-terminal kinase (JNK) in neutrophils. These results demonstrate that remifentanil can attenuate LPS-induced neutrophil responses and also suggest that such effects are sufficiently important in vivo to play a major contributory role in neutrophil-mediated inflammatory responses by surgical and anesthetic trauma.