Anti-inflammatory and antioxidative effects of propofol on lipopolysaccharide-activated macrophages.

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Abstract

Sepsis is a serious and life-threatening syndrome that often occurs in intensive care unit (ICU) patients. During sepsis, inflammatory cytokines and nitric oxide (NO) can be overproduced, causing tissue and cell injury. Propofol is an intravenous agent used for sedation of ICU patients. Our previous study showed that propofol has immunosuppressive effects on macrophage functions. This study was designed to evaluate the anti-inflammatory and antioxidative effects of propofol on the biosyntheses of tumor necrosis factor a (TNF-α), interleukin 1β (IL-1β), IL-6, and NO in lipopolysaccharide (LPS)-activated macrophages. Exposure to a therapeutic concentration of propofol (50 μM), LPS (1 ng/mL), or a combination of these two drugs for 1,6, and 24 h was not cytotoxic to the macrophages. ELISA revealed that LPS increased macrophage TNF-α, IL-1β, and IL-6 protein levels in a time-dependent manner, whereas propofol significantly reduced the levels of LPS-enhanced TNF-α, IL-1β, and IL-6 proteins. Data from RT-PCR showed that LPS induced TNF-α, IL-1β, and IL-6 mRNA, but propofol inhibited these effects. LPS also increased NO production and inducible nitric oxide synthase (iNOS) expression in macrophages. Exposure of macrophages to propofol significantly inhibited the LPS-induced NO biosynthesis. The present study shows that propofol, at a therapeutic concentration, has anti-inflammatory and antioxidative effects on the biosyntheses of TNF-α, IL-1β, IL-6, and NO in LPS-activated macro-phages and that the suppressive effects are exerted at the pretranslational level.