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• 計畫中文名稱	靜脈麻醉藥物 ketamine 對巨噬細胞所造成的免疫抑制作用和其可能發生的機動研究		
• 計畫英文名稱	--		
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• 中文關鍵字	--		
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• 中文摘要	查無中文摘要		
• 英文摘要	Ketamine is an intravenous anesthetic agent. Clinically, induction of anesthesia with ketamine can cause immunosuppression. Macrophages play important roles in host defense. In this study, we attempted to evaluate the effects of ketamine on macrophage functions and its possible mechanism using mouse macrophage-like Raw 264.7 cells as the experimental model. Exposure of macrophages to 10 and 100 .mu.M ketamine, which correspond to 0.1- and 1-times the clinically relevant concentration, for 1, 6, and 24 h had no effect on cell viability or lactate dehydrogenase release. When the administered concentration reached 1000 .mu.M, ketamine caused a time-dependent release of lactate dehydrogenase and cell death. Ketamine, at 10 and 100 .mu.M, did not affect the chemotactic activity of macrophages. Administration of 1000 .mu.M ketamine in macrophages resulted in a time-dependent decrease in cell migration. Treatment of macrophages with ketamine concentrationand time-dependently reduced phagocytic activities. The oxidative ability of macrophages was suppressed by ketamine in concentration- and time-dependent manners. Treatment with lipopolysaccharide induced TNF-alpha, IL-1beta, and IL-6 mRNA in macrophages. Administration of ketamine alone did not influence TNF-alpha, IL-1beta, or IL-6 mRNA production. Meanwhile, cotreatment with ketamine and lipopolysaccharide significantly inhibited lipopolysaccharide-induced TNF-alpha, IL-1beta, and IL-6 mRNA levels. Exposure to ketamine led to concentration- and time-dependent decreases in the mitochondrial membrane potential. However, the activity of mitochondrial complex I NADH		

dehydrogenase was not affected by ketamine. This study shows that a clinically relevant concentration of ketamine (100 .mu.M) can suppress macrophage function of phagocytosis, its oxidative ability, and inflammatory cytokine production via reduction of the mitochondrial membrane potential instead of direct cellular toxicity.