• 系統編號	RN9408-0043
• 計畫中文名稱	麻醉藥物之免疫調控作用研究---探討靜脈麻醉藥物 2,6-雙異丙烷酚調控巨噬細胞功能之機制
• 計畫英文名稱	Modulatory Effects of Anesthetic Agents---Atudy of Suppressive Mechanism of 2,6-Diisopropylphenol on Macrophage Functions
• 主管機關	行政院國家科學委員會 • 計畫編號 NSC91-2314-B038-026
• 執行機構	台北醫學院麻醉科
• 本期期間	9108 ~ 9207
• 報告頁數	11 頁 • 使用語言 英文
• 研究人員	戴裕庭;陳瑞明 Tai, Yu-Tint; Chen, Ruei-Ming
• 中文關鍵字	麻醉藥; 2,6-雙異丙烷酚; 巨噬細胞; 免疫調控; 脂多醣; 細胞激素; 趨化性; 吞噬作用
• 英文關鍵字	Anesthetic; 2,6-Diisopropylphenol; Macrophage; Immune regulation; Lipopolysaccharide; Cytokines; Chemotaxis; Phagocytosis
• 中文摘要	查無中文摘要
• 英文摘要	Background: Propofol is an intravenous anesthetic agent that may impair host defense system. This study was aimed to evaluate the effects of propofol on macrophage functions and its possible mechanism. Methods: Mouse macrophage-like Raw 264.7 cells were exposed to propofol, at 3, 30 (a clinically relevant concentration), and 300 .mu.M. Cell viability, lactate dehydrogenase and cell cycle were analyzed to determine the cellular toxicity of propofol to macrophages. After administration of propofol, chemotaxis, phagocytosis, oxidative ability and interferon-.gamma. mRNA production were carried out to validate the potential effects of propofol on macrophage functions. Mitochondrial membrane potential and cellular adenosine triphosphate levels were also analyzed to evaluate the role of mitochondria in propofol-induced macrophage dysfunction. Results: Exposure of macrophages to 3 and 30 .mu.M propofol did not affect cell viability. When the administered concentration reached 300 .mu.M, propofol would increase lactate dehydrogenase release, cause arrest of cell cycle in G1/S phase and lead to cell death. In the 1 hour-treated macrophages, propofol significantly reduced macrophage functions of chemotaxis and oxidative ability in a concentration-dependent manner. However, the suppressive effects were partially or completely reversed after 6 and 24 hours. Propofol could reduce phagocytotic activities of macrophages in concentration- and time-dependent manners. Exposure of macrophages to lipopolysaccharide induced the mRNA of interferon-.gamma., but the induction was significantly blocked by propofol. Propofol concentration-dependently decreased the membrane potential of mitochondria of macrophages, but the effects were descended with time. The levels of cellular adenosine

triphosphate in macrophages were also reduced by propofol. Conclusions: A clinically relevant concentration of propofol can suppress macrophage functions through inhibiting their mitochondrial membrane potential and adenosine triphosphate synthesis instead of direct cellular toxicity.