Propofol specifically inhibits mitochondrial membrane potential but not complex I NADH dehydrogenase activity, thus reducing cellular ATP biosynthesis and migration of macrophages.

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Abstract

Propofol is a widely used intravenous anesthetic agent. Our previous study showed that a therapeutic concentration of propofol can modulate macrophage functions. Mitochondria play critical roles in the maintenance of macrophage activities. This study attempted to evaluate further the effects of mitochondria on the propofol-induced suppression of macrophage functions using mouse macrophage-like Raw 264.7 cells as the experimental model. Macrophages were exposed to a clinically relevant concentration of propofol for 1, 6, and 24 h. Analysis by the Trypan blue exclusion method revealed that propofol was not cytotoxic to macrophages. Exposure of macrophages to propofol did not affect mitochondrial NADH dehydrogenase activity of complex I. However, analysis of flow cytometry showed that propofol significantly decreased the mitochondrial membrane potential of macrophages. Cellular levels of ATP in macrophages were significantly reduced after propofol administration. In parallel with the dysfunction of mitochondria, the chemotactic analysis showed that exposure to propofol significantly inhibited the migration of macrophages. This study shows that a therapeutic concentration of propofol can specifically reduce the mitochondrial membrane potential, but there is no such effect on complex I NADH dehydrogenase activity. Modulation of the mitochondrial membrane potential may decrease the biosynthesis of cellular ATP and thus reduce the chemotactic activity of macrophages. This study provides in vitro data to validate mitochondrial dysfunction as a possible critical cause for propofol-induced immunosuppression of macrophage functions.