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Ketamine Inhibits Phagocytosis of Canine Peripheral Blood Polymorphonuclear Cells

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Ketamine, a non-competitive *N*-methyl-**D**-aspartate receptor antagonist, is widely used an intravenous anaesthetic agent in human and veterinary medicine. Previously, it was shown that the oxidative burst activity (OBA) of canine peripheral blood leukocytes is inhibited by the culture supernatant from ketamine-treated canine peripheral blood mononuclear cells (PBMCs). In the present study, we examined whether *in vitro* treatment with ketamine increases PGE_2 production of canine peripheral blood mononuclear cells (PBMCs) and, if so, whether the ketamine-induced production of PGE_2 from PBMC inhibits phagocytic capacity and oxidative burst activity (OBA) of canine polymorphonuclear cells (PMNs). The phagocytic capacity and OBA were measured simultaneously by a flow cytometry. The amount of PGE_2 in the ketamine-treated PBMCs culture supernatant was measured by enzyme immunoassay. Ketamine increased PGE_2 production by canine PBMCs. The phagocytic capacity and OBA of canine PMNs were inhibited by either culture supernatant from PBMCs treated with ketamine or recombinant PGE_2. AH-6809, an E-prostanoid $(EP)_2$ antagonist, restored the phagocytic capacity and OBA of canine PMNs, which had been decreased by the ketamine-treated PBMCs culture supernatant and recombinant PGE_2. These results suggest that ketamine has an inhibitory effect on canine PMNs phagocytosis, which is mediated by PGE_2 from canine PBMCs.

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