Methyl palmitate

Methyl palmitate (MP) is a fatty acid methyl ester.

A recent study indicated that adrenergic nerve-dependent functional sympathetic-sensory nerve interactions were abolished by MP in mesenteric arteries. However, the effect of MP on perivascular nerves and cerebral blood flow remains unclear.

In a study by Hsu et al., the increase in basilar arterial blood flow (BABF) after the topical application of nicotinic acetylcholine receptor agonists was measured using laser-Doppler flowmetry in anesthetized rats. The choline (a selective α7- nicotinic acetylcholine receptor agonist)-induced increase in BABF was abolished by tetrodotoxin (a neurotoxin), NG -nitro-L-arginine (a non-selective NO synthase inhibitor), α-bungarotoxin (a selective α7-nicotinic acetylcholine receptor inhibitor), and chronic sympathetic denervation. In addition, the nicotine (a nicotinic acetylcholine receptor agonist)-induced increase in BABF was inhibited by MP in a concentration-dependent manner. The acetylcholine-induced increase in BABF was not affected by MP. The myography results revealed that nicotine-induced vasorelaxation was significantly inhibited by MP, but was reversed by chelerythrine (a protein kinase C inhibitor). MP-induced vasodilation was significantly greater in BA rings without endothelium compared to those with endothelium. Meanwhile, MP did not affect baseline BABF. Our results indicate that MP acts as a neuromodulator in the cerebral circulation where it activates the PKC pathway and causes a diminished nicotine-induced increase in blood flow in the brainstem and that the vasorelaxation effect of MP may play a minor role [1].

1)
Hsu CK, Chang HH, Shang-Jen C, Yang S, Huang KF. Methyl palmitate modulates the nicotine-induced increase in basilar arterial blood flow. Microcirculation. 2021 Feb 17:e12686. doi: 10.1111/micc.12686. Epub ahead of print. PMID: 33595915.