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. The 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.