

P6.06.

The role of neurotensin and dopamine interaction in conditioned place preference

László, K.¹; Madarassy-Sz., A.¹; Kiss, Á.¹; Tóth, K.¹; Ollmann, T.¹; Péczely, L.²; Kertes, E.²; Lénárd, L.^{1,2}

1: Institute of Physiology, Pécs University Medical School, Pécs, Hungary

2: Neurophysiology Research Group of the HAS2, Pécs University Medical School, Pécs, Hungary

Tridecapeptide Neurotensin (NT) in the central nervous system acts as a neurotransmitter and neuromodulator. The central nucleus of amygdala (CeA), part of the limbic system, plays an important role in learning, memory, anxiety and reinforcing mechanisms. We recently showed that NT microinjected into the CeA plays a role in positive reinforcement. We supposed that these effects might be due to modulations of mesolimbic-mesocortical dopamine (DA) system. The aim of our study was to examine in the CeA the possible effects of NT and dopamine interaction on reinforcement in conditioned place preference test. Male wistar rats were microinjected bilaterally with 100 ng NT or 250 ng NT (Sigma: N 3010, injected in volume of 0.4 µl) or 35 ng NTS1 antagonist SR 48692 (Sanofi-Synthelabo) alone, or NTS1 antagonist 15 min before 100 ng NT treatment or vehicle solution into the CeA. Other animals received 5 µg D2 receptor antagonist (sulpiride: Sigma: S7771, injected in volume of 0.4 µl) alone, or D2 receptor antagonist 15 min before 100 ng NT treatment or vehicle solution into the CeA. Rats that received 100 ng NT or 250 ng NT spent significantly more time in the treatment quadrant during the test session. Prior treatment with the non-peptide NTS1 antagonist, equimolar to NT treatment (60 pM and 60 pM, respectively) blocked the effects of NT. Prior treatment with dopamine D2 receptor antagonist blocked the positive reinforcing effects of NT. Antagonists in themselves did not influence the place preference. Our results show that in the rat CeA NT has positive reinforcing effect via NTS1, because selective NTS1 antagonist can block this action. The rewarding effect of NT may be due to the modulation of DA system, since it could be blocked by DA D2 antagonist pretreatment.

Supported by NKTH-OTKA K68431 and by the HAS.