## P5.06.

# Modulatory effect of a new KYNA derivative on the trigeminal activation

Fejes, A.[1*]; Vámos, E.[2]; Bohár, Z.[1]; Tajti, J.[1]; Fülöp, F.[3]; Vécsei, L.[1]; Párdutz, Á.[1]

*1: Department of Neurology, University of Szeged, Szeged, Hungary*
*2: Department of Physiology, Anatomy and Neuroscience, University of Szeged, Szeged, Hungary*
*3: Department of Pharmaceutical Chemistry, University of Szeged, Szeged, Hungary*

The exact pathomechanism of trigeminal nociception is not fully understood, but the role of glutamate seems crucial. One of the endogenous ionotropic glutamate receptor antagonists is the kynurenic acid (KYNA) formed from L-kynurenine during the tryptophan metabolism. KYNA may modulate nociception, but its therapeutic use is difficult because of its poor penetration properties across the blood brain barrier (BBB). One of the new KYNA derivatives (KYNAd) having better penetrance properties across the BBB could well use in treatment of trigeminal nociception. One of the animal models of somatic trigeminal nociception is the orofacial formalin test, which leads to activation of the trigeminal system. We examined the effect of pretreatment with the new KYNAd on the formalin-induced changes in the CGRP and TRPV1 immunoreactivity in the trigeminal ganglion (TG) and in the c-Fos immunoreactivity in the caudal trigeminal nucleus (TNC). Four hours after the injection of formalin in the right upper lip of rats, the percentage of CGRP-immunoreactive (IR) neurones in the TG was decreased, while that of TRPV1-IR neurones was increased, as was the number of c-Fos-IR neurones in the TNC. The pretreatment with the new KYNAd significantly modulated the formalin-induced changes in the TG and in the TNC. The new KYNAd can modulate the activation of neurones in the trigeminal system acting probably on the glutamate receptors, thus it can be new candidate in the treatment of trigeminal nociception.