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INHIBITORS OF HDACS AND DNMTS AFFECT PAIN SENSITIVITY IN THE INFLAMMATORY PAIN MODEL

Elene Abzianidze[1, 2], E Kvaratskhelia[1], M Zarandia[1], K Dzagoevi[1] and M Tsagareli[1, 2]

[1]Tbilisi State Medical University, Georgia
[2]Ivane Beritashvili Center of Experimental Biomedicine, Georgia

Chronic pain is associated with abnormal cortical structure and function especially in brain regions involved in pain modulation. Recent studies have focused that the epigenetic modulators such as histone deacetylases (HDACs) and DNA-methyltransferases (DNMTs) are key molecules to be related to chronic inflammatory pain. The aim of the present study was to determine whether global DNA methylation and histone modifications in anterior cingulate cortex (ACC), hippocampus and rostral-ventromedial medulla (RVM) significantly correlate with the severity of pain sensitivity in the rat model of inflammatory pain. We investigated the possibility of epigenetic agents to reverse the pain sensitivity observed following formalin test. Adult rats receiving intraplantar formalin were tested for antinociception follows injection of non-steroid anti inflammatory drugs (NSAIDs) in combination with inhibitors of DNA-methyltransferases and HDACs (5-azacitidine (5-AzaC) and SAHA, respectively) using thermal and mechanical paw withdrawal tests. Levels of global DNA methylation, DNMT1, DNMT3a/b and HDACs were evaluated in nuclear extracts of ACC, hippocampus and RVM neurons. Received results demonstrate that treatment with 5-AzaC and SAHA in combination with NSAIDs reduce pain sensitivity which is accompanied with altered levels of HDAC, DNMT3b and global DNA methylation in ACC, hippocampus and RVM in chronic pain experimental model. These data support that changes in brain structure and cortical function that are associated with chronic pain conditions may be mediated by epigenetic mechanisms such as DNA methylation and histone acetylation.

eleneabzianidze@gmail.com
eabzianidze@tsmu.edu