FENS Regional Meeting

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ДНС SNS Друштво за неуронауке Србије Serbian Neuroscience Society

National Neuroscience Society of Romania

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Abstract Book

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Thursday, July 11, 2019

13:45-15:00

Room Atlantic 1
Room Atlantic 2

POSTER SESSION 2

P267

NTPDASE1/CD39 EXPRESSION INCREASED DURING EAE IN ASSOCIATION WITH NUMBER AND ACTIVATION STATE OF MICROGLIA/MACROPHAGES

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Considering neuroinflammatory paradigm, increased extracellular levels of ATP have adverse effects, while adenosine is predominantly anti-inflammatory. In the CNS, NTPDase1/CD39 is the main enzyme that initiates the degradation pathway of extracellular ATP to adenosine.

The aim of the study was to explore the activation state of the cells that express NTPDase1/CD39 – microglia and macrophages, during experimental autoimmune encephalomyelitis (EAE).

Acute monophasic EAE was induced in female Dark Agouti rats. Animals were sacrificed at the disease onset (Eo), peak (Ep) and end (Ee). The lumbosacral parts of spinal cords were isolated for gene (qRT-PCR and in situ hybridization) and protein expression analysis (Western Blot, immunofluorescence and flow cytometry). Activation state of microglia/macrophages was assessed by colocalization analysis of NTPDase1/Iba1 and NTPDase1/CD68 with iNOS or Arg1 as specific markers of pro- and antiinflammatory state, respectively.

During EAE, NTPDase1/CD39 was significantly increased both at mRNA and protein level at Ep. Immunofluorescence combined with flow cytometry showed that reactive microglia and mononuclear infiltrates accounted for the most of the observed increase. Both Iba1 and CD68 microglia/macrophage markers showed higher co-occurrence with iNOS at Eo and Arg1 at Ep, suggesting prevalence of M1-like at Eo and M2-like at Ep. In addition, NTPDase1 showed about three-times higher colocalization with Arg1 compared to iNOS at Ep, suggesting its higher association with M2-like activation state of microglia/macrophages.

Together, our data suggest an association between NTPDase1 up-regulation by reactive microglia and infiltrated macrophages and their transition toward anti-inflammatory phenotype in EAE.