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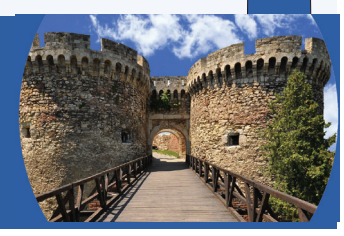

P262

L-TYPE CALCIUM CHANNELS INVOLVEMENT IN THE REGULATION OF NEUROINFLAMMATION AND NEUROREGENERATION AFTER BRAIN INJURY

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Aims: Traumatic brain injury (TBI) causes disruption in homeostasis of calcium ions (Ca2+), important second messenger considered as the major culprit of secondary injury and TBI-induced neuronal damage and death. Ca2+ entry into the cells occurs via various types of voltage-dependent calcium channels (VDCCs). The aim of this study was to evaluate the involvement of Ca2+ entry via L-type CaV1.2 VDCCs in the processes of neuroinflammation and regeneration after brain injury.

Methods: TBI was performed on male Wistar rats by sensorimotor cortex ablation (SCA) at the following coordinates: 2 mm anterior and 4 mm posterior to bregma, and 4 mm lateral from the midline. Temporal and cellular pattern of CaV1.2 expression was followed at different time points post-injury (2, 7, 14, 30 dpi) using double immunofluorescence staining with specific markers.

Results: Upregulation of CaV1.2 expression was detected on reactive astrocytes and astrocytic processes that form glial scar around the lesion site, on subset of proinflammatory microglia/macrophages and neutrophils surrounding the lesion cavity. Interestingly, presence of CaV1.2+ cells was detected in the migratory pathway, consisted of DCX+ progenitors, extending from subventricular zone up to the lesion site. Furthermore, CaV1.2+/DCX+ newborn neurons were detected in subgranular layer of hippocampal dentate gyrus.

Conclusions: We concluded that L-type CaV1.2 calcium channel has an important role in the regulation of processes of neuroinflammation, neuroregeneration and neurogenesis, pointing to the complexity of intercellular regulation of Ca2+ homeostasis after brain injury. Consequently, modulation of CaV1.2 channels expression may be potential target for the treatment of brain injury.