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ANALYSIS OF POST-TRAUMATIC STRESS DISORDER GENE EXPRESSION PROFILES IN MOUSE MODELS OF NEUROINFLAMMATION

Background/Aim. Post-traumatic stress disorder (PTSD) affects millions worldwide, and its prevalence is growing dramatically in Ukraine due to the ongoing war. Considering its complex physiological and biochemical nature, the molecular mechanisms underlying PTSD remain poorly understood, highlighting the urgent need for research of the genetic risk markers and the role of immune system dysregulation in its development. **Methods.** PTSD symptoms were induced in a lipopolysaccharide-based (LPS) neuroinflammation mouse model using electric foot shock (EFS), followed by behavioral assessment via open field test (OFT) and elevated plus maze (EPM). The genome-wide gene expression differences were analyzed by RNA sequencing of mouse brain cortex samples on the Illumina platform at the Genomics Core Facility, Masaryk University, Czech Republic. **Results.** Behavioral testing showed increased anxiety-like behavior in the EFS groups, while pre-existing neuroinflammation additionally suppressed locomotor activity. Transcriptomic profiling identified 293 differentially expressed genes (DEGs) in the EFS group, including upregulated *Pdlim2* and *Tmem88b* associated with inflammation, and downregulated *Crhbp* and *Kcnf1* involved in stress response and neuronal excitability. In the LPS group, 763 DEGs were detected, with *Gata3* overexpression driving Toll-like receptor and *Th17* signaling, while *Lzts1* downregulation suppressed neuronal regulatory pathways. Most importantly, the LPS+EFS group revealed 2603 DEGs, which is nearly a tenfold increased compared to EFS alone. Among the most significantly upregulated genes are the *Pdlim2* and *Kcnab3* and downregulated the *mt-Rnr2* and *Alcam*, which may provide evidence about extensive transcriptional reprogramming primarily affecting mitochondrial function and oxidative phosphorylation. **Conclusions.** Our data suggest that prior neuroinflammation substantially amplifies the molecular response to traumatic stress. *Gata3*, a master regulator of immune signaling, and *Pdlim2*, consistently upregulated across all groups, may be considered as key molecular candidates linking immune activation to PTSD development. **Grants/Fundings.** EDUC-WIDE project, funded by the European Union under Grant Agreement No.101136533, is gratefully acknowledged for the financial support of the measurements at the Genomic Core Facility from Masaryk University (Brno, Czech Republic). This work was supported by a grant from the Ministry of Education and Science of Ukraine [grant number 0125U000396].

Keywords: mouse model, inflammation, behavior, post-traumatic stress disorder.

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