Investigation of the Effects of Repeated Corticosterone Injections on Glucocorticoid Receptor Expression and Traumatic Memory Formation

Rusiko Ansiani

e-mail: rusiko.ansiani6212@ens.tsu.edu.ge

Department of Biology, Faculty of Exact and Natural Science,
Iv. Javakhishvili Tbilisi State University
13, University Street, Tbilisi, 0186, Georgia

Traumatic memory plays a critical role in the pathogenesis of trauma- and stressor-related disorders, notably **Post-Traumatic Stress Disorder (PTSD)**. From a neurobiological perspective, three brain regions are particularly implicated: the **hippocampus**, which mediates the formation and contextualization of new memories; the **amygdala**, which encodes the emotional salience of experiences; and the **anterior cingulate cortex (ACC)**, which serves as a functional interface between the hippocampus and neocortex, facilitating the consolidation of short-term memories into long-term storage.This study investigated the effects of the stress hormone **corticosterone** on behavioral and molecular outcomes in rodent models of traumatic memory. Specifically, we examined the relationship between behavioral phenotypes and the expression of genes involved in glucocorticoid signaling and memory processing. Our gene targets included **NR3C1** and **NR3C2**, which encode the glucocorticoid receptor (GR) and mineralocorticoid receptor (MR), respectively, as well as **Fkbp5**, which encodes FKBP5—a co-chaperone protein that modulates GR sensitivity and its nuclear translocation. Animal models of trauma were established using varying doses of corticosterone in combination with **contextual fear conditioning (CFC)**. Behavioral assessments were performed using the **Videotrack system** (Viewpoint, France), with animals tested in both CFC and **open field (OF)** paradigms. Key behavioral endpoints included **exploratory activity**, **freezing behavior**, and **center-crossing frequency** in the open field, providing metrics of both fear memory and anxiety-related behavior. Gene expression analysis was conducted using **quantitative real-time PCR (qPCR)**, measuring mRNA levels of the selected targets two hours post-exposure to the traumatic stimulus. Both acute (single) and chronic (repeated) corticosterone administration protocols were evaluated to determine differential effects on gene regulation. Our results revealed statistically significant differences in behavior and gene expression between experimental and control groups. Notably, corticosterone administration induced gene-specific expression changes that correlated with alterations in fear-related behaviors, suggesting modulation of **synaptic plasticity and memory consolidation pathways**. These findings offer novel insights into the molecular underpinnings of traumatic memory and provide a potential mechanistic link between glucocorticoid signaling and the development of PTSD-like phenotypes. This work may inform future strategies for targeted pharmacological intervention in trauma-related disorders.