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Estrogen administration modulates Parvalbumin expression in the hippocampus of trimethyltin-treated rats

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Trimethyltin (TMT)-induced hippocampal injury represents a suitable instrument not only to study neuronal and glial responses during neurogeneration and intracellular signaling pathways associated with neuronal damage, but also to validate new strategies to clarify brain repair mechanisms [1-3].

Rats exposed to TMT show severe loss of pyramidal neurons in the CA1 and CA3 hippocampal subfields, showing a subacute pattern developing over three weeks, associated with astroglial and microglial activation, enhanced neurogenesis, seizures and cognitive impairment [1-3]. Since it is known that 17 beta-estradiol (E2) plays a role in neuroprotection and influences GABAergic transmission, also by modulating parvalbumin (PV) expression [4, 5], in the present study we explored, in the hippocampus of TMT-treated ovariectomised rats, the effects of E2 administration.

After TMT or saline treatment, animals (n=6/group) received two i.p. E2 doses (100microg/rat) or vehicle, and were sacrificed on post-treatment day 7. Unbiased stereological analysis of Fluoro Jade-, GAD67- and PV-stained hippocampal sections evidenced that, while E2 administration does not significantly influence the extent of neuronal death, as well as the increase in the number of GAD67 expressing interneurons caused by the toxicant, it induces a significant increase in the number of PV positive hippocampal neurons ($p<0.05$). No significant changes in the expression of Aggrecan, marker of the perineuronal net, were detectable among the experimental groups. qPCR analysis of hippocampal samples confirmed the upregulation of PV mRNA starting 48h after treatment. It is well known that PV interneurons give a crucial contribute to cognitive processes and their dysfunction severely impairs brain functions. They are also responsible for a peculiar form of experience-related plasticity, which modulates memory and learning, thus representing a potential target for neuroprotective strategies [6].

Our results add information concerning the role of in vivo E2 administration on mechanisms involved in cellular plasticity in the adult brain.

References

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Keywords

Trimethytlin, estrogen, hippocampus, neuronal death, parvalbumin.