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The neuroprotective effect of estrogen pre-treatment in a model of neonatal hippocampal injury induced by trimethyltin

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Due to the relevance of hippocampal dysfunction in neurodevelopmental disorders, which affect memory, cognitive abilities and behaviour, developmental studies may represent an important tool for the understanding of cellular and molecular phenomena underlying early hippocampal damage, as well as to study possible therapeutic interventions, which may modify the progression of neuronal death. Since many findings support the neuroprotective effects of 17β-estradiol (E2) administration in different neurodevelopmental models of brain injury [1, 2], the present study investigates the effects of E2-pre-treatment in a model of neonatal hippocampal injury obtained by Trimethyltin (TMT) administration (6,5 mg/kg), characterized by neuronal loss in CA1 and CA3 subfields, associated with astroglial and microglial activation [3, 4]. At P5 and P6 animals received two E2 doses (0.2 mg/kg i.p.) or vehicle. At P7 they received a single dose of TMT (6,5 mg/kg i.p.) and were euthanised 7 days after treatment. Our data indicates that E2 administration significantly improves neuronal death in CA1, reduces the extent of microglial activation and restores TMT-induced reduction of both parvalbumin- and neuropeptide Y-expressing interneurons in the same hippocampal region. Our results add information on the role of in vivo E2 administration on mechanisms involved in neuroprotection and cellular plasticity in the developing brain.

References

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Keywords
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