J Clin Toxicol 2015 2015, 5:3
http://dx.doi.org/10.4172/2161-0495.S1.016

OMICS International
conferenceseries.com
4th Global Summit on
Toxicology
August 24-26, 2015 Philadelphia, USA

Guanosineimproves learning/memory and biochemical impairments in rats submitted a TBI: Possible effect on purinergic system

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Traumatic brain injury (TBI) is caused by a blow to the head or a penetrating injury that disrupts the normal function of the brain. TBI patients demonstrate a number of complications such as memory loss, anxiety and depression. Guanosine (GUO) has been implicated in neuroprotection through the modulation of glutamatergic system. The objective was to evaluate whether treatment with GUO was able to avoid the behavioral and biochemical alterations caused by TBI. The animals were anesthetized, the cannula was placed over the craniotomy with dental cement and the TBI were realized. After 1hof GUO treatment (7.5 mg/Kg intraperitoneal) was started and continued daily until 20 days. To evaluate the potential target of guanosine action, adenosinergic antagonists were tested (SCH 582610.05 mg/kg and 1 mg DPCPX/ kgip, 15 min after TBI). Locomotor activity was measured in the open field test starting on day 6 after the TBI until the day 9. In a day, 14 anxieties were evaluated, in days 15 and 16 the step-down passive avoidance task was used to study non-spatial short and long term memory and in days 19 and 20, the novel object recognition test was applied. After the behavioral evaluation of animals, the brains were dissected and the expression of some proteins related to synaptic plasticity (CREB, BDNF, synaptophysin and GAP-43) were evaluated in the hippocampus. The locomotor activity was not altered in any of the groups. TBI group demonstrated a significant increase in anxiety behavior and a significant decrease of nonspatial short and long term memory. However, the GUO treatment shows an improvement in behavioral outcomes. DPCPX administration blocked the effect of GUO in the behavioral tests. Moreover, TBI group shows a decrease of synpatic protein expression (Creb, BDNF and GAP-43) and GUO treatment shows an improvement of this protein expression. Corroborate of previous results, DPCPX blocked the effect of GUO. GUO was able to avoid the behavioral and biochemical changes caused by TBI, and this effect seems to be related to Purinergic system.

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Toxicogenomic analysis of the ability of brominated flame retardants TBBPA and BDE-209 to disrupt thyroid hormone signaling in neural cells

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Brominated flame retardants are suspected to act as disruptors of thyroid hormone (T3) signaling. As T3 is well-known to be required for proper brain development, this raises the concern that brominated flame retardants might affect children's cognitive functions. We performed an in vitro analysis of the ability of the most common compounds, tetrabromobisphenol A (TBBPA) and BDE-209, to alter thyroid hormone response based on a model neural cell line and genome-wide analysis of gene expression (RNAseq). We observed a modest but specific alteration of T3 signaling with both compounds. This study mainly illustrates the statistical power provided by genome wide transcriptome analysis in this cellular system to detect modest disruption in T3 signaling and isolate it from other signaling components. Our current effort to transpose this strategy to living mice will be presented.

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