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Blast Brain Injury Elevates Catecholamine Biosynthesis in the Nucleus Tractus Solitaries and Oxidative Stress in the Hypothalamus in Rats

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acceleration-deceleration injury with axon damage primarily in the corpus callosum without overt cortical injury. Sham and naïve animals were included as controls and all animals received tamoxifen on 2 and 3 days post-injury. Mice were sacrificed at 3 days, 2 weeks, or 6 weeks post-injury.YFP and cell type markers were detected by immunohistochemistry and quantified using unbiased stereology of cells in the SVZ or manual counts with binning to assess cell localization.

Results
Shh expression, evaluated by immunohistochemistry, is increased at 3d post-injury in astrocytes of the SVZ and corpus callosum in comparison with naïve mice. Tamoxifen administration at 2 and 3 days post-injury resulted in YFP expression among neural stem cells in non-injured mice and following both injury models. This heritable YFP labeling of Shh responsive cells at 2–3 days post-injury identified differences associated with SVZ cells. After CCI, YFP+ cells are increased in the SVZ ipsilaterally at 2wks post-injury and bilaterally at 6wks post-injury, compared to sham and naïve animals. After CBI, YFP cells in the SVZ increase bilaterally between 2 to 6 wks post-injury. In both models, at 2wks and 6wks post-injury, there is an increase in YFP+ cells extending laterally from the SVZ inferior to the corpus callosum. Many of the YFP+ cells colocalize with either a marker of transit amplifying cells (EGFR) or astrocytes and neural stem cells (GFAP).

Conclusions
After CCI and CBI, Shh colocalized with GFAP+ astrocytes in the SVZ and corpus callosum indicating that astrogliosis may account for the increased Shh expression seen at 3 days post-injury. Gli1-CreERT2;R26-YFP mice showed heritable labeling of neural stem cells with tamoxifen administration at 2 and 3 days post-injury when Shh expression was robust. Shh-responsive cells increased in the SVZ and also extended from the dorsal lateral SVZ under the corpus callosum. YFP in cells associated with the SVZ were double labeled with GFAP suggesting that they are neural stems cells or with EGFR suggesting they are in an active state as necessary for migration and repair. Thus, the upregulation of Shh and Shh signaling in the SVZ following TBI may indicate a potential role for Shh in neural stem cells neuroregenerative responses that can be prolonged even after a mild injury in the adult CNS.

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Keywords
traumatic brain injury sonic hedgehog demyelination regeneration stem cells

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BLAST BRAIN INJURY ELEVATES CATECHOLAMINE BIOSYNTHESIS IN THE NUCLEUS TRACTUS SOLITARIES AND OXIDATIVE STRESS IN THE HYPOTHALAMUS IN RATS

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Introduction
Traumatic Brain Injury (TBI) produces major health problems impacting the lives of both military and civilian personnel. TBI disrupts autonomic function but the nature of this disruption is unknown. Following blast brain injury, we assessed selective biochemical markers for autonomic function in adult male Sprague Dawley rats.

Methods
Rats were subjected to head-directed overpressure blast injury (OBI) of 358 kPa magnitude at the target. At the same time for sham controls, rats were anesthetized as the previous group but instead of OBI were exposed just to noise being placed at $\sim$2 m distance from the shock tube nozzle. Sympathetic nervous system activation of nucleus tractus solitaries and in the hypothalamus was evaluated at 6 hours following blast injury by assessing the expression of catecholamine biosynthesizing enzyme, tyrosine hydroxylase (TH) in the nucleus tractus solitaries and NADPH oxidase activity, a marker of oxidative stress,in the hypothalamus.

Results
Following OBI there was a significant elevation in TH protein expression by 49% compared with control ($P < 0.05$). In addition, NADPH oxidase activity was significantly increased by 36% following OBI ($P < 0.05$).

Conclusions
Collectively, the increased catecholamine biosynthesis in nucleus tractus solitaries and oxidative stress in the hypotalamus suggest that OBI results in increased sympathoexcitation in the rat brain. Such effects may be one important factor contributing to autonomic dysfunction following OBI.

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Keywords
blast injury, biomarkers, autonomic dysfunction

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EXPRESSION OF HYPERPHOSHORYLATED TAU PROTEIN FOLLOWING CONTROLLED CORTICAL IMPACT IN IMMATURE (POST NATAL DAY 17) RATS

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Introduction
Traumatic Brain Injury (TBI) remains the leading cause of death and disability in children. As well, upwards of 30% of patients with Alzheimer's Disease (AD) have a history of a single incident TBI. Amyloid beta (Aβ) plaques and neurofibrillary tangles of tau protein (TP) remain as the hallmark postmortem findings.