University of Victoria | Graduate Studies

Notice of the Final Oral Examination for the Degree of Master of Science

of

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BSc Hons (University of Victoria, 2011)

"Mild Traumatic Brain Injury Produces More Immediate and Prolonged Synaptic Plasticity Deficits in the Juvenile Female Hippocampus"

Division of Medical Sciences

Thursday April 16, 2015
12:30 P.M.
Hickman Building
120

Supervisory Committee:
Dr. Brian Christie, Division of Medical Sciences, University of Victoria (Supervisor)
Dr. Craig Brown, Division of Medical Sciences, UVic (Member)
Dr. Robert Chow, Department of Biology, UVic (Outside Member)

External Examiner:
Dr. Paul Zehr, School of Exercise, Science, Physical and Health Education, UVic

Chair of Oral Examination:
Dr. Dimopoulos, Department of Electrical Engineering, UVic

Dr. David Capson, Dean, Faculty of Graduate Studies

Abstract

Traumatic brain injury (TBI) is the leading cause of disability in individuals under 45 years of age, with mild TBI (mTBI) accounting for the majority of cases. The juvenile brain is in a period of robust synaptic reorganization and myelination, making adolescence a particularly vulnerable time to incur a TBI. Learning and memory deficits that involve the hippocampal formation are often observed following mTBI in adults. To examine this issue in the juvenile brain, we examined changes in hippocampal synaptic plasticity following closed-head mTBI in male and female Long-Evans rats (25-28 days of age). Synaptic plasticity of field excitatory post-synaptic potentials (fEPSPs) was assessed using *in vitro* electrophysiology at either one hour, one day, seven days, or 28 days following mTBI in either the dentate gyrus (DG) or *cornu ammonis* area 1 (CA1) regions of the hippocampus. In female rats, the CA1 region ipsilateral to the impact showed a significant reduction in long-term potentiation (LTP) as early as one hour following mTBI. Similar LTP deficits were apparent at one day in the DG, and persisted to 28 days following injury. In male rats, a deficit in both DG- and CA1-LTP was maximal in the ipsilateral hemisphere by seven days following injury, but these deficits did not persist until 28 days post-injury. These data suggest that the juvenile brain is susceptible to mTBI-induced impairments in plasticity, and sex and regional differences are apparent in the expression and recovery of synaptic plasticity following mTBI.