NeurAegis Identifies a New Link between Traumatic Brain Injury and Alzheimer's Disease

NeurAegis published a new study, which identifies a new link between calpain-2 activation and tau phosphorylation, and a new potential biomarker.

POMONA, CALIFORNIA, UNITED STATES, September 18, 2017 /EINPresswire.com/ -- NeurAegis published the results of a series of experiments, which could explain why Traumatic brain injury (TBI) could lead to the development of Alzheimer's disease (AD) pathology. The manuscript entitled "The tyrosine phosphatase PTPN13/FAP-1 links calpain-2, TBI and tau tyrosine phosphorylation" was published online in Scientific Reports, September 18, 2017.
TBI increases the risk of dementia, such as AD. Activation of the protease calpain and tau hyperphosphorylation have been implicated in both TBI and AD. However, the link between calpain and tau phosphorylation has not been fully identified. In the published studies, the tyrosine phosphatase PTPN13, which suppresses tau phosphorylation, was identified as a key binding partner of calpain-2. PTPN13 is cleaved by calpain-2, which inactivates its phosphatase activity and generates stable breakdown products (P13BPs). Following TBI, calpain-2 activation not only cleaved PTPN13, but also activated tyrosine kinase c-Abl, which triggered tau phosphorylation; both events enhance tau tyrosine phosphorylation. Post-TBI injection of a calpain-2 selective inhibitor prevented c-Abl activation and tau oligomer accumulation. Thus, the calpain-2-PTPN13-c-Abl pathway provides a direct link between calpain-2 activation and abnormal tau aggregation, which may promote tangle formation and accelerate the development of AD pathology after repeated concussions or TBI. This study also suggests that P13BPs could be potential biomarkers to diagnose mild TBI or AD. These results provide further support for the development of selective calpain-2 inhibitors for the treatment of TBI, which could limit the risk for Alzheimer's disease.

About NeurAegis
NeurAegis has been founded to translate fundamental discoveries on the mechanisms of synaptic plasticity and neuronal survival/cell death into clinical applications. These discoveries are the results of over 30 years of research by the scientific co-founders, Michel Baudry, PhD, Dean of the Graduate College of Biomedical Sciences (Western University of Health Sciences, Pomona, CA), and graduate from Ecole Polytechnique, Paris, France (X68), and Xiaoning Bi, MD, PhD, Professor, COMP (Western University of Health Sciences, Pomona, CA), directed at understanding the roles of selective biochemical cascades in both synaptic plasticity and neuroprotection/neurodegeneration. NeurAegis has identified neuroprotection as a key focus for research and development, because of the high unmet needs and tremendous research potential in this therapeutic area. Much work has been conducted to identify the mechanisms underlying neuronal death and significant progress has been made over the last 10-20 years. Nevertheless, there is still no drug on the market that provides any significant degree of neuroprotection, especially within the crucial minutes to maximum of several hours following any brain insult that results in neuronal loss.
Contact information:
Michel Baudry, CEO, NeurAegis: m.baudry@neuraegis.com

Michel Baudry
NeurAegis
9494687310

email us here
