Neuroscience Lecture Series
Spring 2013

Remyelination: A New Therapeutic Target to Treat Traumatic Brain Injury

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Abstract: There are currently no effective drug treatments for traumatic brain injury (TBI). We have previously shown that the combination of FDA-approved drugs minocycline (MINO) and N-acetylcysteine (NAC) synergistically improved both cognition and memory in an active place avoidance task following either the mild or moderate controlled cortical impact (CCI), a model of TBI (Abdel Baki, et al., 2010). Exactly how these drugs act is unknown, but data suggest that behavioral deficits in the avoidance task may be due to demyelination of a single white matter tract. Two major cell types that modulate remyelination are microglia and oligodendrocytes. Activated microglia are present in demyelinating and remyelinating white matter. MINO plus NAC treatment act synergistically to modulate patterns of microglial activation. This modulation is believed to promote remyelination. Oligodendrocytes are damaged by CCI; protection of oligodendrocytes is another likely action of MINO plus NAC. Taken together, these data suggest that microglial modulation and remyelination induced by MINO plus NAC underlie improvements in cognition and memory following CCI.

Monday, April 1st at 3 p.m.
Morris Library Auditorium

Refreshments served following the lecture
Open to the Public