Mitochondrial dysfunction in traumatic brain injury

Mitochondrial dysfunction is an important hallmark of traumatic brain injury and has been thoroughly studied in male rodent models of brain injury, but relatively little is known about these outcomes in females. These studies were designed to examine sex as a biological variable for mitochondria-related outcomes after the severe controlled cortical impact (CCI) mouse model of TBI. Synaptic and non-synaptic mitochondria were isolated from the sham- or CCI-injured cortex, as well as the hippocampus ipsilateral to the craniotomy 3, 12, 24, or 48 h post-surgery, and then bioenergetics, were measured. Subtle variations were observed in the timeline of mitochondrial dysfunction between sexes. Non-synaptic cortical mitochondria from injured females showed early impairment at 12 h post-CCI compared to mitochondria from injured males at 24 h post-CCI. Contrastingly, in the synaptic fraction, mitochondria from injured males showed early impairment at 12 h post-CCI, whereas mitochondria from injured females showed impairment at 24 h post-CCI. Based on bioenergetic impairments at 24 h post-CCI, synaptic and non-synaptic mitochondrial calcium loading was also measured at this time point. Consistent with bioenergetic data at 24 h, non-synaptic mitochondria from injured males had increased calcium loading compared to uninjured control, but this effect was not observed in females. Finally, histological assessment of cortical tissue sparing in each sex was measured at 7 days post-injury. There was a lack of sex-based differences in cortical tissue sparing after severe CCI. Overall, there were some subtle sex differences in mitochondrial outcomes after CCI, but these findings were not statistically significant. This study highlights the importance of utilizing both sexes when measuring mitochondrial function after severe CCI [1)].

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