Traumatic brain injury (TBI) is associated with acute cerebral metabolic crisis (ACMC). ACMC-related atrophy appears to be prominent in frontal and temporal lobes following moderate-to-severe TBI. This atrophy is correlated with poorer cognitive outcomes in TBI.

A study investigated ability of acute glucose and lactate metabolism to predict long-term recovery of frontal-temporal cognitive function in participants with moderate-to-severe TBI. Cerebral metabolic rate of glucose and lactate were measured by the Kety-Schmidt method on days 0-7 post-injury. Indices of frontal-temporal cognitive processing were calculated for six months post-injury; 12 months post-injury; and recovery (the difference between the six- and 12-month scores). Glucose and lactate metabolism were included in separate regression models, as they were highly intercorrelated. Also, glucose and lactate values were centered and averaged and included in a final regression model. Models for the prediction frontal-temporal cognition at six and 12 months post-injury were not significant. However, average glucose and lactate metabolism predicted recovery of frontal-temporal cognition, accounting for 23% and 22% of the variance, respectively. Also, maximum glucose metabolism, but not maximum lactate metabolism, was an inverse predictor in the recovery of frontal-temporal cognition, accounting for 23% of the variance. Finally, the average of glucose and lactate metabolism predicted frontal-temporal cognitive recovery, accounting for 22% of the variance. These data indicate that acute glucose and lactate metabolism both support cognitive recovery from TBI. Also, our data suggest that control of endogenous fuels and/or supplementation with exogenous fuels may have therapeutic potential for cognitive recovery from TBI [1].

Metabolic crisis occurs frequently after TBI despite adequate resuscitation and controlled ICP, and is a strong independent predictor of poor outcome at 6 months [2].

The initial ionic flux and glutamate release result in significant energy demands and a period of metabolic crisis for the injured brain. These physiological perturbations can now be linked to clinical characteristics of concussion, including migrainous symptoms, vulnerability to repeat injury, and cognitive impairment. Furthermore, advanced neuroimaging now allows a research window to monitor postconcussion pathophysiology in humans noninvasively. There is also increasing concern about the risk for chronic or even progressive neurobehavioral impairment after concussion/mild traumatic brain injury. Critical studies are underway to better link the acute pathobiology of concussion with potential mechanisms of chronic cell death, dysfunction, and neurodegeneration [3].

1)
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