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# Photobiomodulation Preconditioning Prevents Hypoxia-ischemia Induced Dyscinesia in a Neonatal Rat Model

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**Objective** Neonatal hypoxia-ischemia (HI) injury caused by oxygen deprivation is the most common cause of severe neurologic deficits and dyscinesia in neonates. The work was designed to evaluate the preventative effect of photobiomodulation (PBM) preconditioning on HI-induced Dyscinesia in a Neonatal Rat Model, and its underlying mechanism of PBM action on brain damage in a HI model in neonatal rats.
**Methods** 10-day-old neonatal Sprague-Dawley rats were randomly divided into 3 groups: (a) control group (animals without ligation); (b) HI group (HI animal with PBM pretreatment); (c) PBM group (HI animal with PBM pretreatment). The hanging wire test and cylinder test were conducted to evaluate the the strength and asymmetry of left (contralateral) paw usage, respectively. The volume shrinkage of the brain was analyzed on postnatal day 29. The neuronal loss, mitochondrial dynamics, mitochondrial fragmentation, cytochrome c release, neuronal apoptosis, dendritic and synaptic injury in hippocampus were tested using the brain collected on postnatal day 16.
**Results** PBM preconditioning significantly attenuated motor function impairment, volume shrinkage, neuron loss, dendritic and synaptic injury after HI. Further mechanistic investigation showed that PBM preconditioning effectively restore HI-induced mitochondrial dynamic changes and inhibit mitochondrial fragmentation, accompanied by a robust suppression of cytochrome c release, and prevention of neuronal apoptosis by inhibition of caspase activation.
**Conclusions** PBM preconditioning can prevent HI induced dyscinesia and brain injury by maintaining mitochondrial dynamics and inhibiting mitochondrial apoptotic pathway.