Synaptoprotection in Perinatal Asphyxia: An Experimental Approach

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Abstract
Perinatal asphyxia (PA) is an obstetric complication occurring when the oxygen supply to the newborn is temporally interrupted. This health problem is associated with high morbimortality in term and preterm neonates. It severely affects the brain structure and function, involving cortical, hippocampal, and striatal loss of neurons. Nearly 25% of PA survivor newborns develop several neurodevelopmental disabilities. Behavioral alterations, as well as the morphological and biochemical pathways involved in PA pathophysiology, have been studied using an animal model that resembles intrauterine asphyxia. Experimental evidence shows that PA induces synaptic derangement. Then, synaptic dysfunction embodies a putative target for neuroprotective strategies. Over the last years, therapeutic hypothermia (TH), the only treatment available, has shown positive results in the clinic. Several pharmacological agents are being tested in experimental or clinical trial studies to prevent synaptopathy. Preservation of the synaptic structure and function, i.e., "synaptoprotection," makes up a promising challenge for reducing incidental neurodevelopmental disorders associated with PA. Accordingly, here, we summarize and review the findings obtained from the referred experimental model and propose a renewed overview in the field.

Author keywords
animal model
neuroprotective strategies
perinatal asphyxia
synaptopathy
synaptoprotection