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# Neonatal Brain Injury and their Neuroprotective Strategies

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## Introduction

Neonatal brain damage is a serious illness that causes significant morbidity and mortality in newborns. Learning problems, delayed motor development, seizure disorders, mental retardation, and cerebral palsy are all possible outcomes of perinatal hypoxia-ischemia, which affects a considerable proportion of neonates born in the United States. Free radical generation, inflammatory mediator activity, and excitotoxicity are among the mechanisms behind this injury, which are complicated and numerous. Hypoxic-Ischemic Encephalopathy (HIE) is a term used to describe brain injury caused by acute or subacute asphyxia in an infant. Critical cellular processes are disrupted as a result of this imbalance. However, white matter damage is more common in premature infants. Although there is no cure for HIE, there are treatments that can help prevent or delay progression. A newborn brain injury may alter the development of important structural and functional connection networks, resulting in neurodevelopmental disability in impacted children. Structure (through diffusion MRI) and functional (by Vifa resting state-functional MRI) neuroimaging approaches can be used to describe these networks [1].

Neonatal Hypoxic-Ischemic (HI) brain injury is one of the major drawbacks of mortality and causes significant short/long-term neurological dysfunction in newborn infants worldwide. To date, due to multifunctional complex mechanisms of brain injury, there is no well-established effective strategy to completely provide neuroprotection. Although therapeutic hypothermia is the proven treatment for Hypoxic-Ischemic Encephalopathy (HIE), it does not completely chang outcomes in severe forms of HIE. Therefore, there is a critical need for reviewing the effective therapeutic strategies to explore the protective agents and methods. In recent years, it is widely believed that there are neuroprotective possibilities of natural compounds extracted from plants against HIE. These natural agents with the anti-inflammatory, anti-oxidative, anti-apoptotic, and neurofunctional regulatory properties exhibit preventive or therapeutic effects against experimental neonatal HI brain damage. In this study, it was aimed to review the literature in scientific databases that investigate the neuroprotective effects of plant extracts/plant-derived compounds in experimental animal models of neonatal HI brain damage and their possible underlying molecular mechanisms of action [2, 3].

Hypoxia-Ischemia (HI) is regarded as one of the major drawbacks of brain damage in the neonates and infants. Neonatal Hypoxic-Ischemic Brain Damage (HIBD) (synonymous with Hypoxic-Ischemic Encephalopathy (HIE)) is the main drawback of newborn deaths and irretrievable and long-lasting neurodevelopmental disabilities in the sufferers. HIE complications estimate 23% of infant deaths all over the world, and have impacted 0.7-1.2 million newborns per annum. Cerebral palsy, epilepsy, mental retardation, motor and cognitive deficits, learning and behavioral disabilities, and other severe neurological disorders were regularly observed in the sufferers based on brain injury grade. In infants with mild HIE, the death probability is 10% and the risk of neurodevelopmental disorders is 30%, while 60% of infants with severe HIE are at risk of death and all sufferers risk exposure to remarkable disabilities [4].

The newborn brain has various barriers that are protective in nature but make medication targeting difficult. The Blood-Brain Barrier, the Blood-Cerebro Spinal Fluid (CSF) barrier, and the CSF-brain barrier are all examples of these barriers. To be effective, most proposed medicines must overcome these barriers. Indomethacin, a medicine used in neonates, may help to improve the blood-brain barrier, resulting in less intraventricular hemorrhage. The blood-brain barrier is one of the neonatal brain's protective mechanisms for preventing dangerous elements from entering the brain. Despite the use of this drug, there is still a high rate of newborn brain injury. The Blood-CSF barrier is the next line of defence. Unlike the blood-brain barrier, this barrier is not impenetrable. The choroid plexus's capillaries allow molecules to flow freely between endothelial cells [5-8].

Tight junctions exist in these cells, forcing molecules to use the complex transport system at their disposal. Active, assisted, and simple diffusion are the modes of transport in endothelial cells. There is currently no evidence that targeting this specific system could provide neuroprotection. The CSF brain barrier, which is made up of ependymal cells that line the ventricles, is another line of defence in the central nervous system. Toxins cannot physically reach the brain because these cells produce glutathione, which acts as a biochemical barrier. Hypoxia can damage the ependymal lining, causing the defence system to fail. Even short exposure to hypoxia-ischemia destroyed the ependymal lining in animal tests. This vulnerability could expose the brain parenchyma to poisons that have made their way into the CSF. Neural stem cells are frequently found around the ventricles [9].

Preoligodendrocytes, in particular, are known to be sensitive to oxidative stress. This cell group should be targeted for neuroprotective measures, and drugs that improve their viability should be identified. Protecting neural stem cells is one focus for prenatal neuroprotection in neonates experiencing severe hypoxia episodes. Increasing cell survival by upregulating biological processes is an appealing strategy. Increasing glutathione production and consequently cell survival is one neuroprotective technique. Over the last 5-7 years, System Xc has gotten a lot of interest as a potential cellular defence approach. 1 mole of cystine is exchanged for 1 mole of glutamate in System Xc [10].

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