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Abstract

HIF-1α as a Potential Target for Pharmacologic Correction after Prenatal Hypoxia †

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Chronic prenatal hypoxia (CPH) is one of the most common causes of neonatal mortality and postnatal disorders of the nervous system and the mental development of infants. The search for new methods to treat the effects of CPH is a high-priority problem of modern pharmacology. HIF-1α in hypoxic conditions activates mechanisms of endogenous neuroprotection and can be used as a target for pharmacological correction.

Aim of the work: To study the possibilities of the HIF-1α-modulating effects of different pharmacological agents in the model of CPH.

CPH was modeled by administering sodium nitrite (50 mg/kg) to pregnant female white rats from day 16 of gestation. The offspring were treated with the following drugs: piracetam, thiotriazoline, nikomex, tamoxifen, cerebrocurin, angiolin, glutoredoxin, l-arginine, and HSF-1 during the first 30 days of life. The concentration of HIF-1α in the plasma and brain homogenate of rats at 30 and 60 days of life was determined using the enzyme-linked immunosorbent assay.

It was found that CPH leads to a decrease in the concentration of HIF-1α in the nervous tissue of the brain and blood plasma. Drug administration results in a significant increase in the content of HIF-1α in the studied objects immediately at the end of treatment with continued positive effects on the 60th day of life. The maximum effect was demonstrated on day 30 by cerebrocurin, angiolin, and HSF1, and on day 60 by cerebrocurin, angiolin, and thiatriazolin.

Our research provides evidence that the medications being tested are an effective way to modulate HIF-1α and may be a new alternative for the treatment and prevention of nervous system disorders in children caused by CPH.

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