Thioredoxin 1 Plays a Protective Role in Retinas Exposed to Perinatal Hypoxia?Ischemia

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Thioredoxin family proteins are key modulators of cellular redox regulation and have been linked to several physiological functions, including the cellular response to hypoxia?ischemia. During perinatal hypoxia?ischemia (PHI), the central nervous system is subjected to a fast decrease in O2 and nutrients with a subsequent reoxygenation that ultimately leads to the production of reactive species impairing physiological redox signaling. Particularly, the retina is one of the most affected tissues, due to its high oxygen consumption and exposure to light. One of the main consequences of PHI is retinopathy of prematurity, comprising changes in retinal neural and vascular development, with further compensatory mechanisms that can ultimately lead to retinal detachment and blindness. In this study, we have analyzed long-term changes that occur in the retina using two well established in vivo rat PHI models (perinatal asphyxia and carotid ligation model), as well as the ARPE-19 cell line that was exposed to hypoxia and reoxygenation. We observed significant changes in the protein levels of the cytosolic oxidoreductase thioredoxin 1 (Trx1) in both animal models and a cell model. Knock-down of Trx1 in ARPE-19 cells affected cell morphology, proliferation and the levels of specific differentiation markers. Administration of recombinant Trx1 decreased astrogliosis and improved delayed neurodevelopment in animals exposed to PHI. Taken together, our results suggest therapeutical implications for Trx1 in retinal damage induced by hypoxia?ischemia during birth. © 2019 IBRO

perinatal hypoxia?ischemia

reoxygenation

retina

RPE cells

thioredoxin 1

cell marker

green fluorescent protein

neurogenic differentiation factor

recombinant protein

thioredoxin 1

animal experiment

animal model

animal tissue

ARPE-19 cell line

Article

astrocytosis

carotid artery ligation

cell differentiation

cell proliferation

cell structure

controlled study

female

in vivo study

ischemia

nonhuman

perinatal asphyxia

perinatal hypoxia ischemia

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protein expression

protein function

rat

reoxygenation

retina