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Special Issue Reprint

Oxidative Stress in the Newborn

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This Special Issue explores the role of oxidative stress (OS) in neonatal disorders and potential treatments to help neonates combat OS-related health problems. Basal OS, determined by normal intrauterine hypoxia, is vital for fetal organ development and growth. This may explain why broad-spectrum antioxidant treatments have not been effective in reducing prematurity-related health issues. Conditions such as chorioamnionitis, placental insufficiency, and maternal exposure to reactive oxidative species (ROS) inducers can raise OS levels, increasing the risk of OS-related illnesses in neonates. The perinatal transition, marked by a sudden increase in oxygen levels, also produces large amounts of ROS. When neonates are born prematurely, their immature antioxidant defenses struggle to cope with the surge in oxidative stress. As a result, excessive oxidative stress can lead to conditions such as bronchopulmonary dysplasia, periventricular–intraventricular hemorrhage, necrotizing enterocolitis, retinopathy of prematurity, or periventricular leukomalacia. Furthermore, OS triggers innate inflammation, which further damages immature organs. The cycle of OS and inflammation creates a harmful loop that traditional single-target, single-drug treatments often find hard to control. Animal models have been used to study OS-induced neonatal health issues, and many promising therapies have been explored. However, the limited antioxidative capacity remains a major challenge for researchers. We are still far from finding a cure for OS-related organ damage in premature neonates. New strategies are urgently needed.

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