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REVIEW THE ROLE OF REACTIVE OXYGEN SPECIES AND ANTIOXIDANT DEFENSE MECHANISMS IN THE PATHOGENESIS OF CONGENITAL HEART DEFECTS: INSIGHTS INTO GENETIC SUSCEPTIBILITY AND ENVIRONMENTAL FACTORS

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Actuality: Congenital anomalies are a significant cause of mortality and morbidity in infancy and childhood. The occurrence of congenital heart defects (CHDs) ranges from 4 to 50 per 1000 live births. Although progress has been made in understanding the genetic factors contributing to heart defects, genetic causes only account for 15% of CHD cases. The remaining cases arise from a complex interplay between genetic susceptibility and environmental factors, including maternal substance abuse, cigarette smoking, exposure to industrial chemicals, viral infections, and others, which can have embryotoxic effects.

Aim: to investigate the role of oxidative stress and antioxidant capacity in the development of congenital heart defects (CHDs) in children.

Discussion: While the role of ROS in cardiovascular diseases (CVDs) is well-documented, limited research has focused on the involvement of ROS in children with CHDs. However, a study by Ercan et al. examined the relationship between oxidative stress and congenital heart diseases in children with cyanotic and acyanotic CHDs. The study found that cyanotic patients had significantly higher levels of oxidants and antioxidants compared to both the acyanotic group and the control group. The authors speculated that the underlying anatomical defect leads to hypoxia, resulting in increased production of free oxygen radicals as well as compensatory elevation of antioxidant substances. Elevated oxidative stress and compromised antioxidant capacity may contribute to the development of CHDs through ROS-mediated mechanisms affecting various intra- and intercellular signaling pathways. Further investigation is necessary to fully unravel the intricate connections between oxidative stress, antioxidant defense systems, and the pathogenesis of CHDs in children. Understanding these mechanisms could potentially pave the way for novel therapeutic strategies targeting oxidative stress and antioxidants for the prevention and management of CHDs.

Conclusion: increased oxidative stress and impaired antioxidant capacity may play a role in the development of CHDs through ROS production, affecting various intra- and intercellular signaling pathways. Further research is needed to fully elucidate the mechanisms underlying the association between oxidative stress, antioxidant defense mechanisms, and the pathogenesis of CHDs in children. Understanding these processes may pave the way for potential therapeutic interventions targeting oxidative stress and antioxidants in the prevention and management of CHDs.