

## STRATEGIES FOR MITIGATING OXIDATIVE STRESS IN TRANSPLANTED CELLS

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**Introduction.** Transplantation is a vital treatment for organ failure, degenerative diseases and severe tissue injuries. Challenge being, the poor function, survival of transplanted cells caused by oxidative stress. During the transplantation, ischemia and reperfusion injury generates reactive oxygen species (ROS). These highly reactive molecules damage lipids, proteins, and nucleic acids, leading to mitochondrial dysfunction and apoptosis. Oxidative stress leads to graft failure. This study explores strategies to mitigate oxidative stress for improving the survival of transplanted cells.

**Materials and Methods.** A literature review was synthesized from Google scholar, PubMed, Scopus, and Web of Science, focusing on oxidation and antioxidant-based protective strategies from articles between 2010 and 2024. Research regarding transplantation and organ preservation was included. Data on pharmacological treatments and cellular preconditioning were collected and compared. Descriptive analysis was applied to summarize the protective effects reported.

**Results.** Pharmacological antioxidants like N-acetylcysteine serve as vital glutathione (GSH) precursors, replenishing the intracellular thiol pool to facilitate peroxide detoxification, while vitamin C and vitamin E scavenge ROS and protect cells. Activation of the nuclear factor erythroid 2-related factor 2 (Nrf2), a master transcription factor regulating cellular defense against oxidative and electrophilic stress, controls over 200 genes, including antioxidant enzymes like superoxide dismutase and catalase, increasing expression and preventing oxidative damage in transplanted tissues. Cellular preconditioning and antioxidant supplementation before transplantation improve mitochondrial membrane potential and reduce apoptosis. Advanced biomaterial scaffolds and preservation solutions enriched with mitochondria-targeted antioxidants also reduce oxidative injury during the procedure. Specifically, these combined interventions not only preserve structural cellular integrity but also significantly enhance early engraftment rates. By maintaining mitochondrial homeostasis and preventing lipid peroxidation, targeted therapies effectively suppress ROS-induced inflammatory signaling. This synergistic approach -bolsters intrinsic defenses while providing extrinsic ROS scavenging- minimizes acute rejection risks and promotes robust, long-term functional recovery of the graft.

**Conclusions.** Mitigating oxidative stress improves the survival of transplanted cells. Strategies such as antioxidant therapies and the activation of endogenous protective pathways show promising results in reducing graft injury. Future research on targeted antioxidant approaches can enhance long-term graft outcomes.

**Keywords:** Oxidative stress; reactive oxygen species; antioxidant therapy; Nrf2; transplantation; graft survival.