



Keywords: *[Illegible text]*

Introduction

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ischemia-reperfusion injury (IRI) is a major cause of primary graft dysfunction (PGD) after lung transplantation. IRI is a complex process involving multiple mechanisms, including oxidative stress, inflammation, and mitochondrial dysfunction. The reperfusion of the transplanted lung after ischemia leads to the production of reactive oxygen species (ROS) and the activation of various signaling pathways, ultimately resulting in cellular damage and organ dysfunction. Understanding the underlying mechanisms of IRI is crucial for developing effective strategies to prevent and treat this complication.

Procedures to prevent ischemia-reperfusion injury

Several procedures have been developed to minimize IRI during lung transplantation. These include the use of exhaled nitric oxide (ENO), which has been shown to improve oxygenation and reduce pulmonary vascular resistance. Additionally, the use of pulmonary vasodilators like prostaglandin E1 (PGE1) and inhaled nitric oxide (iNO) can help maintain adequate lung perfusion. Other strategies include the use of antioxidants, such as superoxide dismutase and catalase, to neutralize ROS. Furthermore, the use of preconditioning techniques, such as brief periods of ischemia and reperfusion before the final reperfusion, has been shown to reduce the severity of IRI. Finally, the use of cell-based therapies, such as stem cell transplantation, is being explored as a potential strategy to promote lung repair and reduce IRI.