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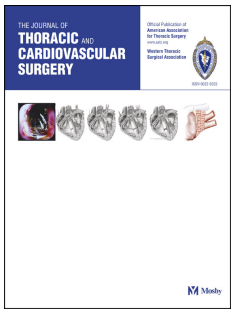

The saturation of novel means to alleviate ischemia-reperfusion injury?

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The saturation of novel means to alleviate ischemia-reperfusion injury?

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Ischemia-reperfusion induced lung injury (IRI) is a complex clinical scenario including increased vascular permeability, lung edema and neutrophil sequestration, all of which determine the outcome of the patient after lung surgery. IRI has extensively been studied, and several molecular pathways are presented as among the most significant target of choice to alleviate detrimental outcome due to IRI. The clinician is drowned by pharmacological novelties, but occasionally, consistent experimental results may light-up a vanishing horizon of optimism in adopting a pragmatic solution to act against IRI.

In their manuscript ”Inhibition of Na-K-Cl co-transporter isoform 1 reduces ischemia-reperfusion induced lung injury”, Lan et al endorses yet another novel means to alleviate IRI based on an in-vitro but in-situ model using the lungs of the mouse (1). The epithelial activity of sodium-potassium-chloride co-transporter (NKCC) regulates lung tissue fluid trafficking and its role during IRI is investigated using transgenic mice with increased (WNK4) or reduced (SPAK -/-) NKCC activity. For control, the lungs of the wild type mouse and bumetanide, an NKCC inhibitor, were studied during IRI. The authors found that the NKCC-pathway participates in aggravating IRI. IRI