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“Hypothermic Oxygenated Machine Perfusion Mitigates Ischemia Reperfusion Injury of Liver Grafts and Improves Patient Outcomes Following Liver Transplantation”

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MSB B610
9:30 AM

Join Zoom presentation

<https://rutgers.zoom.us/j/97979105816?pwd=mHWEF0R4IXgLTcXn24bccUZQHC2zPB.1>

Meeting ID: 979 7910 5816
Password: 416425

ABSTRACT

Liver transplantation (LT) is the gold standard treatment for end-stage liver disease, but organ demand continues to exceed supply. Following organ procurement, the cold ischemic preservation period and subsequent organ rewarming and reperfusion are times of graft injury, particularly for extended criteria donors (ECD) livers (i.e., those with advanced age, steatosis [fatty], and donation after cardiac death). Hypothermic Oxygenated Machine Perfusion (HMP-O₂) is a dynamic preservation technique that improves ischemia/reperfusion injury (IRI) compared to the current standard, Static Cold Storage (SCS). The exact molecular pathways affected, particularly in biliary injury, remain undefined. This study integrates transcriptomic, proteomic, biochemical, and histologic analyses to characterize injury pathways in hepatocytes and cholangiocytes, to define how HMP-O₂ mitigates organ injury. HMP-O₂ appears to induce pathways associated with a robust cytoprotective response, characterized by the coordinated activation of chaperone proteins, which decrease cellular stress and inflammatory signaling, and promote hepatocyte and cholangiocyte stability. Markers of organ ischemia/reperfusion stress are detectable in liver effluent and bile fluid, and while HMP-O₂ does not abrogate injury-associated signaling, machine perfusion promotes cellular homeostasis, as early as the peri-preservation period, marked by protective preconditioning. To our knowledge, these data provide the first comprehensive, multi-omic mechanistic insights into injury patterns, comparing HMP-O₂ to SCS. Our findings support the hypothesis that hypothermic oxygenated machine perfusion improves hepatocellular and cholangiocyte resilience to IRI by regulating stress responses, moderating inflammation, and preserving cellular integrity.