Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN SEOUL, KOREA 2026
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Poster exhibition
Abstract Submission No.: A-0603
Abstract Topic : Basic Research

Mitochondrial Preservation Underlie the Renoprotective Effects of Hypothermia in Kidney Ischemia-Reperfusion Injury

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Objectives : Hypothermia has been reported to attenuate ischemia–reperfusion injury (IRI), yet the molecular mechanisms underlying this protective effect remain incompletely understood. To investigate transcriptomic alterations associated with hypothermic protection, we performed RNA sequencing and network-based analyses in a murine model of renal ischemia–reperfusion injury.
Methods : Male C57BL/6 mice were subjected to bilateral renal ischemia for 20 minutes followed by 24 hours of reperfusion. During the ischemic period, core body temperature was strictly maintained at either 31°C (hypothermic IRI) or 37°C (normothermic IRI). Kidney tissues from sham, hypothermic IRI, and normothermic IRI groups were collected for bulk RNA sequencing. Differentially expressed genes were identified and analyzed using pathway enrichment and protein–protein interaction (PPI) network analyses to uncover functional modules associated with hypothermic protection.
Results : Gene set enrichment analysis revealed significant enrichment of mitochondrial respiratory pathways in hypothermic IRI. In particular, the Reactome pathway "Aerobic respiration and respiratory electron transport" showed strong positive enrichment in hypothermic IRI versus normothermic IRI (NES = 1.92, adjusted $p = 1.36 \times 10^{-9}$). Transcriptomic profiling demonstrated coordinated upregulation of genes encoding components of the mitochondrial electron transport chain, including multiple subunits of complexes I, III, IV, and V. Consistently, PPI network analysis identified a highly interconnected mitochondrial oxidative phosphorylation module comprising NDUF, UQCR, COX, and ATP5 family proteins, suggesting concerted regulation of mitochondrial bioenergetic machinery.
Conclusions : These findings indicate that hypothermic ischemia–reperfusion injury is associated with preservation and coordinated activation of mitochondrial oxidative phosphorylation networks. Maintenance of mitochondrial respiratory function may therefore represent a key mechanism underlying hypothermia-mediated renoprotection. Collectively, our results provide transcriptomic and network-level evidence supporting mitochondrial preservation as a central feature of hypothermic protection in renal ischemia–reperfusion injury and highlight mitochondrial bioenergetic pathways as potential therapeutic targets for acute kidney injury.

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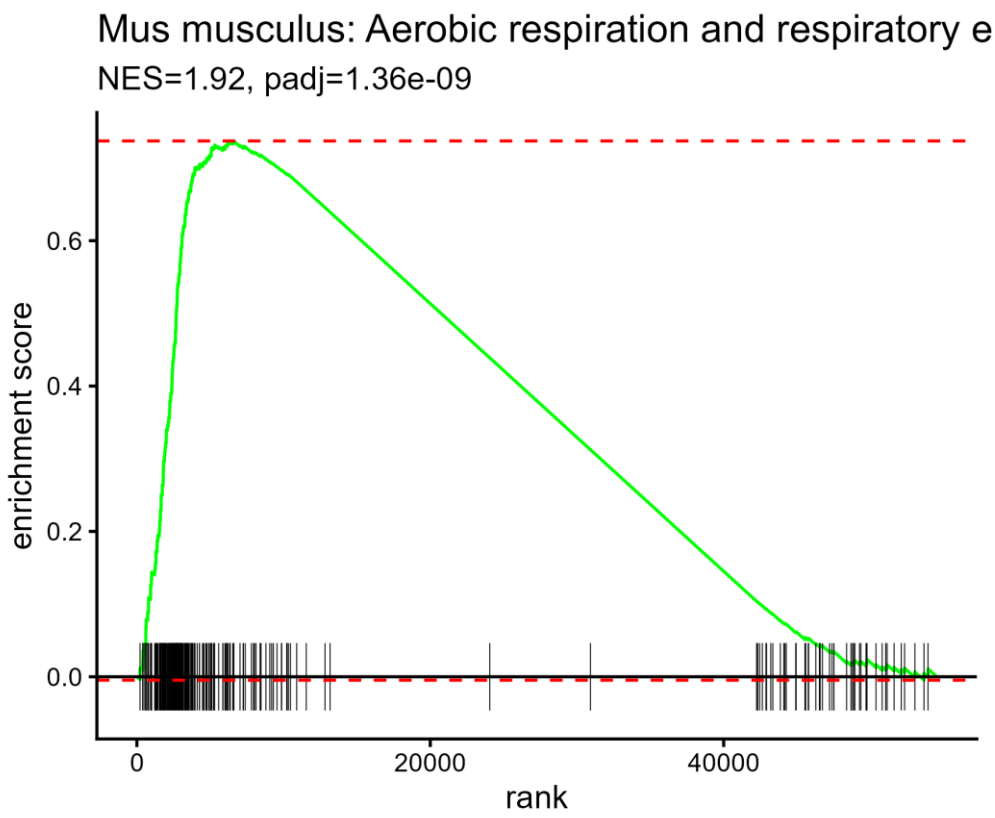

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