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RESEARCH & COLLABORATION PROFILE

KEY WORDS Liver Injury, Mitochondrial Physiology, Inflammation, Redox Biology, Vascular Biology



Research Snapshot. Prof. Lee investigates how cellular stress, inflammatory signaling, and metabolic adaptation determine tissue injury and repair, with particular emphasis on liver disease. His research integrates animal physiology, molecular and cellular biology, mitochondrial/redox mechanisms, and immune-cell responses to define mechanisms of drug- and alcohol-induced liver injury. In parallel, he studies ETV2-dependent hematovascular and endothelial programs relevant to vascular regeneration and disease. His work connects PRDX-mediated oxidative stress, CHI3L1-LCN2-dependent leukocyte recruitment, hepatic lipid metabolism, and ETV2-driven endothelial plasticity.

RESEARCH EXPERTISE

- Liver injury & regeneration: APAP-induced acute liver injury, alcohol-related liver disease, steatosis, inflammatory recruitment, and repair responses.
- Mitochondrial & redox physiology: PRDX family proteins, ROS/JNK-MAPK signaling, mitochondrial dysfunction, and metabolic adaptation during tissue stress.
- Inflammation & innate immunity: CHI3L1, LCN2, neutrophil/macrophage responses, cytokine signaling, and inflammatory disease mechanisms.
- ETV2 & vascular biology: Hematovascular lineage specification, endothelial differentiation, vascular regeneration, EndMT, and tumor vascular biology.

RECENT RESEARCH HIGHLIGHTS

- Liver Injury and Inflammation: Identified the role of the CHI3L1–LCN2 axis in neutrophil recruitment and acetaminophen-induced liver injury.
- Alcohol-Associated Liver Disease: Investigated metabolic mechanisms regulating ethanol-induced hepatic steatosis, including AMPK–PPARα signaling.
- Redox Biology: Defined ETV2-dependent mechanisms regulating endothelial differentiation, plasticity, and vascular remodeling.
- Redox Biology: Investigated the roles of peroxiredoxins and oxidative-stress signaling in cellular and tissue injury.

CURRENT RESEARCH DIRECTION

Our current research focuses on the mechanisms that determine the transition from liver injury to recovery, with particular interest in mitochondrial function and immune-cell responses.

POTENTIAL COLLABORATION RESEARCH

Best-fit collaborator areas:

- **1.** Liver Injury and Recovery

Integrating liver injury models with single-cell and multi-omics analysis to identify key regulators of tissue recovery.

- **2.** Liver Cell–Cell Crosstalk

Investigating interactions among hepatocytes, macrophages, neutrophils, and endothelial cells during liver regeneration.

- **3.**. Mitochondrial Regulation

Identifying mitochondrial mechanisms that control inflammation, metabolic recovery, and liver regeneration.

- **4.** Data-Guided Target Discovery

Combining bioinformatics and transcriptomics with experimental validation in cell and animal models.

LONG-TERM RESEARCH VISION

To identify key molecular and physiological mechanisms that determine whether injured tissues progress toward disease or recover through successful repair and regeneration.

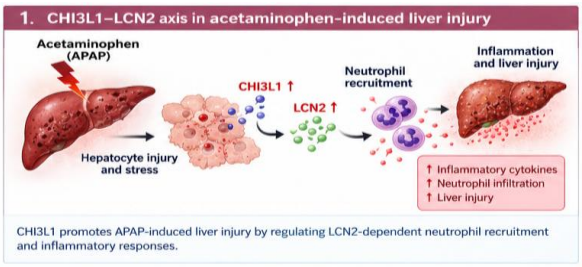
SELECTED RECENT PUBLICATIONS

- Liver injury and inflammation
- Alcohol-associated liver disease
- Mitochondrial and redox biology
- ETV2 and vascular regeneration

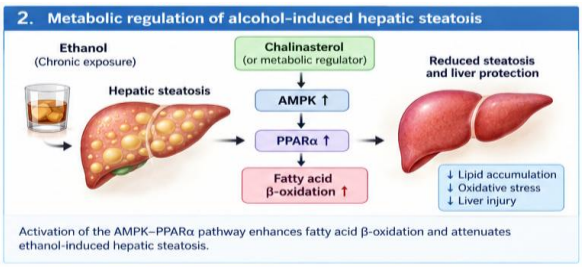
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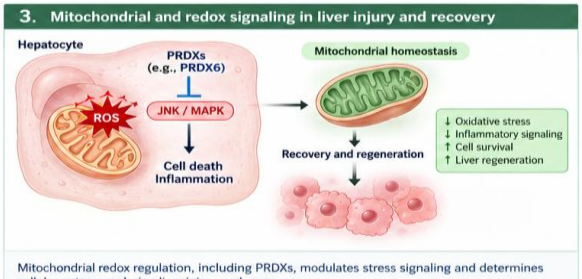
REPRESENTATIVE RESEARCH FIGURES / SCHEMES



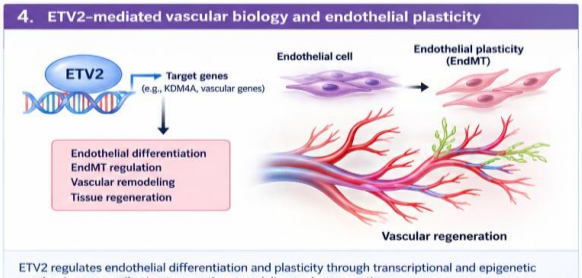
CHI3L1 promotes APAP-induced liver injury by regulating LCN2-dependent neutrophil recruitment and inflammatory responses.



Activation of the AMPK–PPARα pathway enhances fatty acid β-oxidation and attenuates ethanol-induced hepatic steatosis.



Mitochondrial redox regulation, including PRDXs, modulates stress signaling and determines cellular outcomes during liver injury and recovery.



ETV2 regulates endothelial differentiation and plasticity through transcriptional and epigenetic mechanisms, contributing to vascular remodeling and regeneration.