

Stress Responses in Digestive Diseases

eGastroenterology Topic Collection: Call for Papers

The goal of this topic collection is to publish top-quality research and review articles that explore the pathophysiological functions and mechanistic basis of intracellular stress signalling from organelles like the endoplasmic reticulum (ER), mitochondria, and lysosome, as they relate to the development of digestive diseases associated with the liver, gut, or pancreas.

The journal also welcomes research on other types of cellular stress responses, including those associated with inflammatory pathways, inflammasome, extracellular vesicles (EV), proteostasis, nutrient homeostasis, and cell death.

Through this collection of papers, the journal hopes to deepen understanding of the mechanistic basis of stress response in various digestive diseases, such as fatty liver diseases, alcohol-associated tissue damage, pancreatitis, intestinal inflammation, and microbial dysbiosis. These scientific findings and discussions may be beneficial and applicable to clinicians in their clinical practice.

Keywords: ER stress, mitochondria, lysosome, organelle contacts, alcohol-associated liver disease, fatty liver, pancreatitis, intestine, inflammation

Submission Information

Please submit your manuscript before **1 June, 2024** via the journal's [submission system](#). Please select the topic collection name '**Stress Responses in Digestive Diseases**' when submitting your manuscript.

Preliminary enquiries may be sent in the first instance to the Journal Editorial Office (egastro@jlu.edu.cn).

To submit, please consult *eGastroenterology*'s [Author Guidelines](#) for more information about the journal, manuscript types, and instructions for manuscript preparation.

Authors whose papers are accepted for the topic collection following peer review will not be required to pay an APC.

Guest Editors

Wen-Xing Ding, PhD

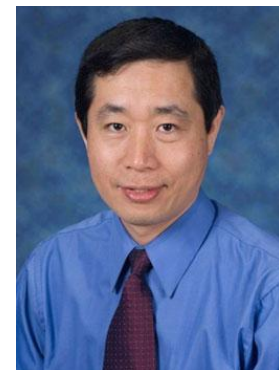
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Dr. Wen-Xing Ding is Professor in the Department of Pharmacology, Toxicology and Therapeutics at the University of Kansas Medical Center. The Ding laboratory has been working on the role of autophagy in alcohol and drug-induced liver injury since 2009. Dr. Ding has published more than 190 papers in peer-reviewed journals and has an h-index of 69. His research work is currently supported by National Institute on Alcohol Abuse and Alcoholism, National Institute of Diabetes and Digestive and Kidney Diseases and National Institute on Aging. Dr. Ding has been a program committee member of American Society of Investigative Pathology (ASIP) since 2014. He is Chair of the Hepatotoxicity Small Interest Group (SIG) of the American Association for the Study of Liver Diseases (AASLD). He also served as a co-chair for the Gordon Research Conference for alcohol-induced end organ injury in 2019. Dr. Ding currently serves as an Associate Editor and Section Editor for *Autophagy*, associate editor for *APSB*, and *Cell & Bioscience* as well as editorial boards for several journals including *Hepatology*, *Scientific Report* and *American Journal of Pathology*. Dr. Ding has been serving as an ad hoc reviewer for several NIH study sections, including HBPP, XNDA and AA1, and a standing member for the XNDA study section.

Kezhong Zhang

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Dr. Kezhong Zhang is a Professor of Molecular Medicine and Genetics and of Biochemistry, Microbiology, and Immunology. Dr. Zhang has so far published more than 160 papers in peer-reviewed journals. The Zhang laboratory is focused on intracellular stress responses originating from the endoplasmic reticulum (ER) and/or mitochondria that modulate inflammation and metabolism that are associated with non-alcoholic fatty liver disease, metabolic syndrome, autoimmune disease, and cancer. Dr. Zhang serves on the editorial boards for 7 peer-reviewed scientific journals. He also serves on multiple NIH study sections and on expert review panels for prestigious national and international funding agencies and academic institutions. Research projects in the Zhang laboratory include: 1) regulation of hepatic energy metabolism by ER or mitochondrial stress sensors; 2) Pathophysiological roles and molecular mechanisms for ER stress sensors in metabolic disorder, inflammatory disease, and neurodegenerative disease; 3) airborne particulate matter (PM_{2.5})-induced cellular stress responses and their effects on non-alcoholic steatohepatitis (NASH) and type-2 diabetes; and 4) roles of ER lipid-raft proteins and UPR transducers in breast cancer malignancy maintenance and therapy resistance.