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The homeostatic intracellular repair response (HIR2) and autophagy during cardiac surgery

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We recently reported that the homeostatic intracellular repair response (HIR2) is activated in the human heart during cardiac surgery [Jahania SM, Sengstock D, Vaitkevicius P, Andres A, Ito BR, Gottlieb RA, Mentzer RM Jr.Activation of the Homeostatic Intracellular Repair Response During Cardiac Surgery.J Am Coll Surg. 2013 Feb 12.]. HIR2 is a beneficial stress response to ischemia, hypoxia, and nutritional depletion. The underlying mechanism is adaptive autophagy, a process that eliminates damaged mitochondria and dysfunctional proteins. To confirm our recent findings, we measured the cardiac autophagy proteins Beclin-1, Atg5-12, and p62 in a second cohort of 19 patients undergoing heart surgery with cardiopulmonary bypass (CPB). Right atrial tissuewas obtained prior to initiating CPB and cardioplegia and after weaning the patient from CPB. Autophagy proteins were analyzed by immunoblotting. In this second cohort of patients, we again observed rapid depletion of autophagy proteins in the heart from beginning to end of CPB. These changes in Beclin-1, Atg5-12, and p62 are consistent with accelerated autophagic flux in response to ischemic stress and confirm our original observation. Strategies designed to amplify this salutary response could lead to new therapeutic approaches designed to prevent ischemia/reperfusion injury in patients undergoing cardiac surgeryor PCI for acute myocardial infarction.

Biography

Mentzer is a cardiothoracic transplant surgeon known for his expertise in the areas of ischemia-reperfusion injury, myocardial protection, and organ preservation. He has held numerous administrative positions including Chairman of the Department of Surgery at the University of Kentucky and Dean and Senior Advisor to the President at the Wayne State University School of Medicine. He is currently Professor of Cardiothoracic Surgery and Physiology at Wayne State University School of Medicine and is a member of the WSU Cardiovascular Research Institute in Detroit, MI. He also holds an adjunct appointment in the Donald P. Shiley BioScience Center at San Diego State University where he is a Research Professor in the Department of Biology and the Director of Translational Research. His current research investigates endogenous mechanisms and novel compounds that protect the heart from ischemia/reperfusion injury, even when given after ischemia or at the time of reperfusion.He has 28 years of continuous peer-reviewed research NIH funding, served on numerous NIH and American Heart Association study sections, and conducted multi-institutional and multi-national industry-sponsored clinical research trials.

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