International Conference on
CARDIOLOGY AND CARDIAC NURSING
July 12-13, 2019 | Zurich, Switzerland

Diabetes, SK channels and coronary endothelial function

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Coronary endothelial dysfunction plays a major role in the progression of cardiovascular disease in diabetic patients. Inactivation of endothelial small conductance calcium-activated potassium channels (SK) in this patient population contributes to coronary endothelial dysfunction. Hypothesis: The altered metabolic signaling in diabetes dysregulates endothelial SK channels of human myocardium and coronary microvasculature. Methods: Atrial tissue and human coronary artery endothelial cells (HCAECS) obtained from diabetic (DM) and non-diabetic (ND) patients undergoing cardiac surgery were used for analyzing metabolic alterations and recording endothelial SK channel currents. Diabetes increases the ratio of NADH/NAD+ in human myocardium. Exogenous administration of NADH into the HCAEC's results in a significant decrease in endothelial K+ current. Pre-treatment the HCAECS with the selective SK inhibitor apamin abolished NADH-induced decrease in K+ currents (Figure). In contrast, inclusion of NAD into the HCAECS results in a significant increase in endothelial SK currents. Diabetes also increased mitochondrial reactive oxygen species (mROS) in HCAECS, and enhanced NADPH oxidase (NOX) and PKC distribution in the human myocardium and coronary microvasculature. Diabetes is associated with metabolic changes in the human myocardium and HCAEC's. Endothelial SK channels are downregulated by the metabolite pyridine nucleotides, NADH and NAD. These data suggest that the altered metabolic signalling may contribute to downregulation of endothelial SK channels and coronary endothelial function in the diabetic patients.

Biography

Jun Feng is currently an associate professor of surgery (research) at Warren Alpert Medical School of Brown University and at Department of Surgery, Cardiovascular Research Center, Rhode Island Hospital. He is also the director and senior research scientist of Cardiothoracic Surgery Research Laboratory at Rhode Island Hospital. He serves as principal investigator on grants funded by National Institute of Health (2 active R01s and 2-NIH-COBRE-pilot projects), American Heart Association (Grant-In-Aid, active), and Rhode Island Foundation. He also serves as co-investigator on a number of grants funded by the National Institute of Health and other research-funding organizations. He has published more than 120 peer-review/editorial articles/book chapters and 140 abstracts as correspondent authors, first authors and co-authors. He has served as an editorial member, editorial commentator and peer reviewer for a number of scientific journals in cardiovascular research and medicine.

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