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Keywords: *Cardiology, Heart failure, Signal transduction, Innate immunity*

Yasuchika Takeishi is Professor and Chairman, Department of Cardiovascular Medicine at Fukushima Medical University and is responsible for advancing patient care, research, and education at Fukushima Medical University. A 1987 graduate of Yamagata University School of Medicine, he later earned positions at the University of Cincinnati and Case Western Reserve University (1997-2000), and was trained through the NIH-sponsored Specialized Centers of Research in Heart Failure program, which pioneered the use of genetically engineered mice to elucidate mechanisms of cardiac hypertrophy and heart failure. Since 2008, Dr. Takeishi has been Professor and Chair of the Department of Cardiovascular Medicine at Fukushima Medical University.

The global burden of heart failure has motivated Takeishi to focus on how aberrant signal transduction pathways can alter cardiac size and function. Seeing the important role of protein kinase C in the development of cardiac hypertrophy and its progression to heart failure, he found *in vitro* evidence that controlling cellular levels of diacylglycerol (DAG) and its downstream signalling cascade by DAG kinase was responsible for cardiomyocyte hypertrophy. Using genetically engineered mice, DAG kinase blocked cardiac remodelling after pressure overload and myocardial infarction *in vivo.* DAG kinase also prevented progressive heart failure and lethal arrhythmias in mice overexpressing constitutively active Gαq.

Pioneers in cellular and humoral innate immunity inspired Takeishi to investigate the roles of Toll-like receptor (TLR) and pentraxin 3 in heart failure. He found that TLR-2 modulated ventricular remodelling after myocardial infarction and doxorubicin-induced cardio-toxicity through production of inflammatory cytokines. Pentraxin 3 produced from bone marrow-derived cells, rather than cardiac resident cells, played a crucial role in cardiac protection against myocardial ischemia/reperfusion injury by attenuating infiltration of neutrophils, generation of reactive oxygen species and inflammatory cytokines.

The changing landscape of healthcare delivery has motivated Takeishi to investigate clinical biomarkers to stratify the risk of heart failure patients; some novel candidate markers have emerged from this research. Additionally, he has performed molecular imaging with positron emission tomography to characterize metabolic and inflammatory status of the failing heart. Recently, he has investigated the role of clonal hematopoiesis, a novel link between aging and cardiovascular diseases. He found that clonal hematopoiesis with JAK2V617F causally lead to development of pulmonary hypertension and aortic aneurysm.

Productive collaboration with mentors, colleagues, and trainees has yielded more than 500 papers in Nature Communications, Journal of Clinical Investigation, Circulation, Circulation Research, Journal of the American College of Cardiology, Cardiovascular Research, Journal of Molecular and Cellular Cardiology, and others. Takeishi has served as the editor-in-chief of Journal of Cardiology and associate editor of Circulation Journal (Japanese Circulation Society) and worked as an editorial board member of various others.