The purpose of this award is to recognize an outstanding scientist who (i) is making major and independent contributions to the advancement of cardiovascular science, and (ii) is leading a growing research program likely to play a major role in the future. The main criteria for selecting awardees are scientific excellence, independence, and potential for future research contributions. While the Peter Harris Award recognizes lifelong accomplishments and the Richard Bing Award recognizes young investigators, the Outstanding Investigator Award (presented annually) is targeted at established investigators who are in the intermediate phase of their academic career.

In non-Congress years, the Outstanding Investigator Award is presented at the meeting of the ISHR Section to which the winner belongs. The winner presents a major lecture and receives a $5,000 honorarium and a plaque. An announcement of this Award is published in Heart News and Views, and posted in the ISHR website. The winner receives free registration and reimbursement for travel expenses (up to a maximum or $1500 when the recipient delivers the lecture at his/her local Section meeting, and $3,000 when inter-continental travel is required).

Nominations for the Outstanding Investigator Award are sought by the Secretary General from members of the International Council, members of the Editorial Board of the Journal of Molecular and Cellular Cardiology, and the Councils of ISHR Sections. In addition, the Secretary General publishes an open invitation in the ISHR Website for members to submit nominations.

Award Winner

Dr. Jeffery Molkentin

“Thrombospondin4 is a novel regulator of ER stress adaptation and cardioprotection”
Dr. Jeffery D. Molkentin received his Ph.D. in physiology from the Medical College of Wisconsin in 1994, after which he completed postdoctoral training under Dr. Eric Olson at the University of Texas Southwest Medical Center in Dallas. In 1997, he joined Cincinnati Children's Hospital Medical Center of the University of Cincinnati as a faculty member, where he rose through the academic ranks and was promoted to full professor before turning 40. He has won numerous awards, including selection as a Pew Scholar and the Katz Prize in cardiovascular research from the American Heart Association. Dr. Molkentin was an Established Investigator of the American Heart Association from 2003-2007 and is a full investigator of the Howard Hughes Medical Institute.

His publication on cyclophilin-D null mice was one of the most highly cited papers in Nature in 2005. Understanding of the molecular mechanisms controlling cell death is of critical importance for virtually all types of human disease. For example, his laboratory showed that mitochondrial-regulated necrosis underlies muscular dystrophy in a 2008 Nature Medicine paper.

His work on cardiac signal transduction and the control of cell death by necrosis has established him as an international leader in cardiovascular sciences. Dr. Molkentin is widely respected for the depth, breadth and creativity of his work, as well as his collegiality and desire to foster the careers of young investigators.

Dr. Molkentin serves as a full-time member of the National Institutes of Health (NIH) grant awarding study section CCHF (2006-2010), along with many other international review committees. He serves on a number of editorial boards, including The Journal of Biological Chemistry, The Journal of Clinical Investigation, Circulation Research, JMCC, and Physiological Reviews.

Dr. Molkentin has published over 220 original articles, including several landmark papers. His publications on calcineurin-NFAT signaling in the heart helped propel the field of signal transduction forward, and his original paper on the subject stands as the mostly highly cited research paper in Cell in 1998, with over 1,500 citations. His succeeding Science paper showed that calcineurin inhibition with cyclosporin A could prevent pathological cardiac growth in response to pressure overload, further defining the potential medical relevance of his initial discovery. His work in this area also spawned additional investigation into MAP kinase signaling and the calcium regulatory pathways that underlie pathological cardiac growth, two other areas where he is a leading figure.

Dr. Molkentin made major contributions to the understanding of regulatory proteins that control calcium-dependent cardiac contractility and hypertrophy. His 2004 Nature Medicine paper on PKCα defined a new calcium-dependent signaling paradigm that controls contractility at the level of the sarcoplasmic reticulum. This work was extended in a series of publications revealing a novel treatment for heart failure through PKCα inhibition.

More recently, Dr. Molkentin's laboratory has taken a creative and leadership role in defining the relatively unexplored area of cellular necrosis in the heart. He made a key observation that explains how the mitochondrial permeability transition pore functions as a key regulated step in the induction of cellular necrosis.