

## KEITH REIMER, M.D.

1946-2002

Keith Arnold Reimer, M.D., Ph.D., Professor of Pathology at Duke University Medical School, internationally recognized cardiovascular scientist, pathologist, and teacher, died on March 15, 2002 of metastatic renal cell carcinoma at the age of 56. Keith began his career in experimental pathology studying ischemic injury of the kidney, however he quickly shifted his focus to myocardial ischemic injury, the field in which he went on to make his major scientific contributions. After completing the MD/PhD program at Northwestern University in Chicago, Keith joined the faculty at Duke University in 1975 as Assistant Professor of Pathology. Early in his career, working in collaboration with Dr. Robert B. Jennings, he published landmark studies describing and characterizing the "wavefront phenomenon" of myocardial ischemic cell death. These studies, published in two papers (*Circulation* 56: 786-794, 1977; and *Laboratory Investigation* 40: 633-644, 1979), have been cited more than 1000 times. During the early 1980s, Keith developed methods to measure baseline predictors of infarct size, such as area at risk and collateral flow, that have become the standard for generating reliable and reproducible data to test cardioprotective interventions. The effort to discover cardioprotective interventions led to one of Keith's most notable achievements – the description of one of the strongest and most reproducible interventions for reducing infarct size: ischemic preconditioning. Numerous investigators and laboratories have worked to better understand this remarkably effective intervention, and the ever-expanding number of studies on ischemic preconditioning, in a wide variety of tissues, have consistently confirmed the original observation that brief periods of ischemia and reperfusion are not detrimental, but are actually markedly protective. The original article describing the phenomenon of ischemic preconditioning, "Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium" (*Circulation* 74: 1124-1136, 1986) has been cited more than 1700 times.

Keith was an active member of the ISHR since 1976, and was elected a Councilor of the American Section in 1979, serving until 1985. He was a finalist for the Richard Bing Young Investigator Award of the ISHR in 1980. Keith served as Secretary of the American Section from 1985-1994, and as a member of the Council of the International Society from 1989-1995. In 1997, he became President-Elect of the American Section and was the sitting President of the American Section, as well as a member of the International ISHR Council, when he died.

## About the Award...

Each year, the International Council selects a speaker to deliver the Keith Reimer Distinguished Lecture at the World Congress or at the annual section meeting of one of the three largest ISHR Sections. The purpose of this lecture is to honor the memory of Dr. Reimer and to recognize his contributions to cardiovascular research. The topic of the lecture must be in the field of ischemia, coronary hemodynamics, cardiac metabolism, or contractile mechanisms. The speaker receives a plaque and \$1,000 honorarium in addition to travel expenses.

*This award is funded by a generous contribution from  
Chugai-Pharmaceutical Co.*



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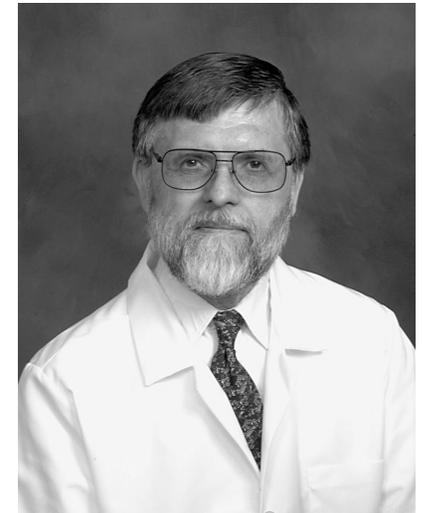
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## THE KEITH REIMER DISTINGUISHED LECTURE 2010



Keith Reimer, M.D. 1946-2002



Honored Speaker

**Dr. Richard L. Moss**

“Phenotypic responses due to induced genetic ablation of cMyBP-C in adult mice”

# RICHARD L. MOSS

2010 HONORED SPEAKER  
KYOTO, JAPAN



Dr. Richard Moss is Director of the Cardiovascular Research Center and Senior Associate Dean for Research in the School of Medicine and Public Health at the University of Wisconsin. He received his Ph.D. in Physiology and Biophysics from

the University of Vermont in 1975, followed by postdoctoral studies with Dr. Fred Julian at the Boston Biomedical Research Institute. In 1979, Dr. Moss was appointed Assistant Professor of Physiology at the University of Wisconsin, where he then served as Chair of Department from 1988 to 2009. During this time he led the founding of the Cardiovascular Research Center and the M.S. in Biotechnology degree program. Dr. Moss was an AHA Established Investigator and currently holds an NHLBI Merit Award. He is the Robert Turell Professor of Physiology and recipient (2007) of an honorary Doctor of Medicine degree from Uppsala University.

Dr. Moss presently serves on the editorial boards of *Circulation Research*, the *Journal of Molecular and Cellular Cardiology*, and the *Journal of General Physiology*. He is a Fellow of both the ISHR and the AHA. He has served as a member of the NIH Physiology

Study Section (1993 to 1997), special emphasis panels at NIH, and several AHA review committees. Dr. Moss was previously a member of the AHA Research Program and Evaluation Committee (1999-2003), the AHA Peer Review Committee (2002-2005), and the Executive Council of the Biophysical Society (1997-1999). He organized the 2002 Annual Meeting of the ISHR North American Section held in Madison, WI and has since served as President of the North American Section (2006-2009). Dr. Moss is currently Past-President of the North American Section, has served on the ISHR-International Council since 2003, and was recently elected Secretary-General of ISHR-International.

Dr. Moss's research focuses on the roles of myofibrillar accessory and regulatory proteins as modulators of myocardial contraction in health and in diseases such as heart failure and heritable hypertrophic cardiomyopathies. He has co-authored more than 150 papers and has supervised more than 20 graduate students and post-doctoral fellows who now hold positions in academic medical centers and research institutions around the world. By biochemically extracting proteins from permeabilized myocardium, Dr. Moss and his collaborators showed that thick filament proteins such as regulatory light chain and myosin binding protein C modulate the extent and rate of force development. Using knock-out, knock-in and transgenic approaches, they further showed that PKA phosphorylation of MyBP-C is principally responsible for the acceleration of myofibrillar contraction kinetics due to  $\beta$ -adrenergic stimulation. An emerging theme is that

regulation of contraction via  $\text{Ca}^{2+}$  binding to troponin or the phosphorylation of accessory proteins such as MyBP-C involves modulation of positive cooperativity in the binding of cross-bridges to actin. Dr. Moss's recent work has focused on mechanisms of contractile dysfunction and  $\text{Ca}^{2+}$ -triggered arrhythmias in hypertrophic cardiomyopathies due to mutations in MyBP-C.

## *Previous Award Winners...*

**Elizabeth Murphy**  
(Sapporo, Japan: 2009)

**David Eisner**  
(Athens, Greece: 2008)

**Eduardo Marbán**  
(Bologna, Italy: 2007)

**Garrett Gross, PhD**  
(Toronto, Canada: 2006)

**Masao Endoh, MD, PhD**  
(Osaka, Japan: 2005)

**R. John Solaro, PhD**  
(Brisbane, Australia: 2004)

**Gerd Heusch, MD, PhD**  
(Strasbourg, France: 2003)

**Roberto Bolli, MD**  
(Madison, Wisconsin: 2002)