■ PAST LECTURERS

This honorary lecture was established in 1977 in honor of Arthur Curtis Corcoran.

Dr. Corcoran's major achievement was his early application of clearance methods in both hypertensive patients and animals. The Corcoran Lecture is presented annually at the AHA Hypertension Scientific Sessions by a distinguished honoree from the field of hypertension.

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1978 J. Ian S. Robertson, MD
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1980 Frederic Bartter, MD
1981 Bengt Samuelsson, MD
1982 Pierre L. Corvol, MD
1983 Michael S. Brown, MD
Joseph L. Goldstein, MD
1984 Wai Yiu Cheung, PhD
1985 Robert C. Tarazi, MD
1986 Robert J. Lefkowitz, MD
1987 Edgar Haber, MD
1988 Harriet P. Dustan, MD
1989 Aram V. Chobanian, MD
1990 Hugh E. deWardener, MD
1991 Detlev Ganten, MD
1992 Stevo Julius, MD
1993 Haralambos Gavras, MD
1994 Salvador Moncada, MD, PhD
1995 Masashi Yanagisawa, MD, PhD
1996 Edward D. Frohlich, MD
1997 Alberto Nasjletti, MD
1998 Suzanne Oparil, MD
1999 Norman K. Hollenberg, MD
2000 Friedrich Luft, MD
2001 L. Gabriel Navar, PhD
2002 Gordon Williams, MD
2003 Willa A. Hsueh, MD
2004 Anna F. Dominczak, MD
2005 Carlos Ferrario, MD
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2007 Curt D. Sigmund, PhD
2008 Gregory Fink, PhD
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2010 Timothy L. Reudelhuber, PhD
2011 Jan Danser, PhD
2012 Robin Davisson, PhD
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2019 Arthur C.Corcoran Memorial Lecture

Patrick J. Pagano, PhD, FAHA
Professor, Department of Pharmacology & Chemical Biology
and Principal Investigator, Vascular Medicine Institute
University of Pittsburgh
Pittsburgh, Pennsylvania

■ ARTHUR C. CORCORAN MEMORIAL LECTURE

Dr. Patrick J. Pagano is a tenured professor in the Department of Pharmacology & Chemical Biology and Principal Investigator of the Vascular Medicine Institute since its founding at the University of Pittsburgh, School of Medicine. He is also director of the Molecular Pharmacology Graduate Program,Vice Chair for Graduate Education in Pharmacology and a Distinguished Mentor at the University. Dr. Pagano has been continually funded by the NIH and AHA for his research and recognized for his successes with leading the pharmacology training program as a top-notch program of the highest caliber.

Dr. Pagano received his PhD from New York Medical College and completed his postdoctoral training within the Vascular Biology Unit at Boston University Medical Center focused on the study of endothelial cell biology and vascular dysfunction. Dr. Pagano began his independent career studying reactive oxygen species in the vasculature as an Assistant Professor of Medicine at Boston University Medical Center.

In 1998, Dr. Pagano joined the Hypertension and Vascular Research Division in the Department of Medicine at the Henry Ford Hospital in Detroit as Senior Staff Investigator and held academic positions at Case Western University and Wayne State University. He later became the Division's director of Vascular Biology Research until his move to Pittsburgh in 2008.

Dr. Pagano's research program focuses on NADPH oxidase (NOX) family of proteins and their role in vascular function under both physiological and pathophysiological conditions. Dr. Pagano's laboratory was among the first to identify a non-phagocytic NOX in the vascular wall.

His laboratory is broadly recognized for pioneering work examining the role of adventitia-derived reactive oxygen species (ROS), in particular, superoxide anion (O2.-) and hydrogen peroxide (H2O2), in the modulation of vascular tone, remodeling and inflammation, as well as for the development of the first isoform-specific NOX inhibitor of any kind (Nox2ds-tat).

In recent years, Dr. Pagano and his group have made significant strides in the development of Nox therapeutics. These include the development of a potent and highly selective inhibitor of NOX1 that is being aerosolized to in vivo models for the treatment of right ventricular failure in pulmonary hypertension. Moreover, the Pagano laboratory has developed small molecule leads that are specific for NOX2, which is implicated in myriad diseases from cardiopulmonary disease to neurodegenerative disorders and cancer.

Currently the laboratory is focused on novel NOX agonists and on elucidating mechanistic insights into effector pathways upstream and downstream of the enzyme class as they contribute to pulmonary hypertension, right heart failure, multiple hyperproliferative disorders and aging. on the molecular genetics of cardiovascular diseases produced 17 patents and more than 250 scientific publications.

The Enigmatic Vascular NOX: From Artifact to Double Agent of Change

Patrick J. Pagano, PhD, FAHA

Once considered a possible artifact of bioassays in the destruction of the potent vasodilator nitric oxide, reactive oxygen and nitrogen species have emerged as vital signaling agents as well as agents of oxidative stress.

In 1994, the vascular NOX was first described in endothelial cells, smooth muscle cells and adventitial fibroblasts. Since the advent of the discovery of the non-phagocytic NOX as major parenchymal sources of reactive oxygen species (ROS), many investigators have been sharply focused on the understanding of what controls the NOX in its numerous isoforms. Intensive research has unveiled modulators and scaffolding proteins and delineated mechanistic insights into NOX-dependent signaling pathways and diseases including pulmonary hypertension, atherosclerosis and more generally hyperproliferative disorders.

Among the Pagano lab's original pioneering discoveries was the genetic and biochemical identification of the adventitial NOX. In order to address the physiological relevance of and causality of the NOX in cellular signaling, a wide array of agents from viral modalities to the pharmacological were sought. Toward this goal, the group has made seminal discoveries in the development of first-in-class, NOX-selective inhibitors including the first NOX-selective inhibitor, gp91ds-tat (a.k.a. Nox2ds-tat), and the development of a novel and potent inhibitor of NOX1 and two small molecule leads that are highly-selective for NOX2. Indeed, by adenoviral delivery of a peptide inhibitor to the vascular adventitia, the team was able to show for the first time a potent paracrine effect of NOX-derived ROS on medial smooth muscle growth. Since then innumerable studies have employed these inhibitors to implicate NOXs in myriad signaling processes and diseases from cardiopulmonary disease to neurodegenerative disorders and cancer.

The group's attention has shifted in recent years to the role of NOXs 1 & 2 in pulmonary hypertension-related vascular remodeling. Paradoxically, we have learned that the temporal expression and interplay among the isoforms in various cell types appear to give way to conflicting phenotypes in the vasculature, e.g., proliferative self-renewal and senescence. Recent findings suggest a highly orchestrated modulation of NOXs that both promote and restrain cell differentiation and cell death.