Harry Goldblatt Award
1997

The Harry Goldblatt Award is presented each year to the author(s) of the paper(s) from last year’s meeting judged by the Publication Committee of the Council for High Blood Pressure Research to represent the most significant new contribution to the understanding of the causes and/or consequences of hypertension. The award is named for the eminent hypertension researcher Dr. Harry Goldblatt and is supported by a generous donation to the council from his family. Included with the award is a $1,000 honorarium and a commemorative plaque.

The 1997 award was presented, in part, to Dr. Kristof Graf from the Department of Medicine/Cardiology, Virchow Klinikum, Humboldt University Berlin and German Heart Institute. Dr. Graf is a fellow of the Council for High Blood Pressure Research and has made a number of contributions to the field of vascular research and hypertension in recent years. Dr. Graf has conducted a number of studies investigating the expression and regulation of neutral endopeptidase and the role of bradykinin in endothelial cells. He has made an important contribution to the understanding of the role of matrix-cell interaction and, in particular, the role of the adhesion protein osteopontin in renin-dependent models of left ventricular hypertrophy. He is currently investigating these mechanisms in human cardiac fibroblasts and myocardial biopsies from patients with cardiac hypertrophy. In addition, he has also done work pertaining to the role of matrix-cell interaction and signal transduction of vascular smooth muscle cells. He has studied the role of angiotensin II and other growth factors on the MAP-kinase pathway and the regulation of vascular smooth muscle functions such as migration, adhesion and proliferation.

The paper for which Dr. Graf and his collaborators are recognized is entitled “MAP Kinase Activation is Involved in PDGF-Directed Migration by Vascular Smooth Muscle Cells.” This paper provided the first evidence that activation of the MAP kinase signaling pathway is essential for vascular smooth muscle cell migration toward PDGF gradients using pharmacological inhibitor of the MAP kinase and an approach with antisense oligodeoxynucleotides against MAP kinase. This work is an important contribution to the field of research investigating the signal transduction pathway of vascular smooth muscle cell migration, which is an underlying mechanism in restenosis and atherosclerosis. Furthermore, this research underscores the potential role of MAP kinase for pharmacological interventions in vascular remodeling processes.
The 1997 award was presented in part to Dr Sandra Pfister, Assistant Professor for the Department of Pharmacology and Toxicology at the Medical College of Wisconsin. Dr Pfister is a fellow of the Council for High Blood Pressure Research and has made a number of significant contributions to the field of hypertension. She has conducted a number of studies specifically concerned with the role of thromboxane (TX) A₂ in the regulation of vascular tone and additional studies identifying and characterizing TXA₂ as a mediator of endothelium-dependent contractions of pulmonary arteries. TXA₂ actions are mediated via a membrane-bound receptor and include platelet aggregation and vasoconstriction. An increased synthesis of TXA₂ is associated with a number of cardiovascular diseases including pulmonary hypertension, unstable angina, and myocardial ischemia.

Dr Pfister’s work was the first to identify a subgroup of rabbits that are deficient in vascular but not platelet TXA₂ receptors. This has important implications to the study of hypertension because one limitation to studying the role of TXA₂ in cardiovascular disease has been the inability to differentiate the contributions of platelet and the vascular smooth muscle to the observed hemodynamic response.

The paper for which Dr Pfister and her collaborators are recognized is entitled “Vascular Smooth Muscle Thromboxane A₂ Receptors Mediate Arachidonic Acid-Induced Sudden Death in Rabbits.” This paper utilized the TXA₂-receptor deficient rabbits as a model to assess the relative contribution of the platelet and vascular TXA₂ receptor to the observed hemodynamic responses associated with arachidonic acid-induced sudden death. The study provided the first evidence that TXA₂-receptor deficient rabbits are protected from arachidonic acid-induced sudden death and supported the hypothesis that vascular TXA₂ receptors mediate the response.
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