Professor Erik S.G. Stroes, MD, PhD

BIOGRAPHY

Professor Erik Stroes received his medical degree from the University of Rotterdam in 1991, and subsequently was trained in internal medicine at the University Medical Center of Utrecht. He subsequently held several clinical and research fellowships/grants. In 2001 he completed his Vascular Medicine specialization and continued his career as staff member of the Vascular Medicine department at the Academic Medical Center in Amsterdam, currently serving as a tertiary referral center for over 1500 patients each year. He is currently professor of vascular. Professor Stroes has a particular interest in lipid disorders in relation to atherogenesis and has participated in numerous lipid lowering trials.

Professor Stroes is a member of the Council for Basic Science, American Heart Association, the International Atherosclerosis Society and chair of the Dutch Society of Atherosclerosis. He is the author or co-author of more than 300 scientific publications associated with his research interests.

ABSTRACT

Promise of epigenetics in atherosclerotic patients

The success of lipid-lowering treatment has uncovered a lipid-refractory residual risk in patients on maximally tolerated lipid-lowering therapy. The search for additional therapeutic moieties to address this residual risk has led to the exciting inflammatory pathways which are active on multiple levels in patients at increased cardiovascular risk. In the atherosclerotic lesions, the presence of monocytes and macrophages is key in determining the proneness of the plaque to rupture. More recently, data have emerged to show that circulating immune cells are already primed within the plasma compartment to a hyperresponsive, pro-adhesive phenotype among other by various circulating lipoprotein fractions (LDLc, Lp(a), remnant cholesterol). Finally, also the bone marrow precursor cells have now been shown to contribute to the systemic inflammatory state, as attested by myeloid skewing of pro-adhesive precursor cells. The positive data from the CANTOS study indicate that ‘intelligent’ application of anti-inflammatory interventions holds the promise of lowering the residual cardiovascular risk; however, it is a careful balance between the benefit of risk lowering and the potential adverse effects of immune suppression. With the insight that innate immune cells can be reprogrammed towards hyperactive phenotypes by epigenetic mechanisms, a new target can be envisioned to decreased the hyperactivity of cellular inflammatory responses by epigenetic remodeling, without increasing the risk of immune suppression. Ongoing studies are currently addressing this novel and exciting concept.

Presentations will be available at www.pace-cme.org
MANAGING DIABETES & CVD: IS EPIGENETICS A NEW WAY FORWARD?

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