84th Congress of the European Atherosclerosis Society, 29 May -1 June, Innsbruck, Austria

Opening Ceremony Highlights

The Opening Ceremony is one of the highlights of Congress, and this year proved no exception.

In his welcome address to over 2,000 delegates, EAS President Professor Alberico Catapano (University of Milan, Italy) overviewed the numerous EAS initiatives which help to maintain the momentum of the Society in education, research and advocacy. These include collaboration with other Societies, as with the recently published Sixth Joint Task Force 2016 European Guidelines on Cardiovascular Disease Prevention in Clinical Practice,¹ as well as the recent Consensus Panel with the European Federation of Clinical Chemistry and Laboratory Medicine (EFLM), which recommended that non-fasting blood samples be routinely used for the assessment of plasma lipid profiles.² An eagerly anticipated update to the Joint European Society of Cardiology/EAS Guidelines on Dyslipidaemia Management is expected in the near future.

Furthermore, there are continuing EAS Consensus Panel initiatives led by Professor John Chapman (University of Pierre and Marie Curie and National Institute for Health and Medical Research, Pitié-Salpêtrière Hospital, Paris, France) and Professor Henry Ginsberg (Columbia University, New York, USA), as well as education initiatives from the EAS Academy and Advanced Courses. EAS is also a player in familial hypercholesterolaemia (FH) research, with the FH Studies Collaboration registry studies, led by Professor Kausik Ray (Imperial College London, UK). In his concluding remarks Professor Catapano commented: ‘EAS continues to build on its strengths to deliver excellence in clinical guidance, education and research.’ The work of Professor Steve Humphries (University College London, UK) as Editor-in-Chief of the Society’s journal Atherosclerosis over the last 8 years was acknowledged, with the baton now being taken up by Professor Arnold von Eckardstein (University of Zurich and University Hospital Zurich, Switzerland).

In his address, President of the International Atherosclerosis Society, Professor Yuji Matsuzawa (Sumitomo Hospital, Japan) highlighted the underlying need for both Societies, with cardiovascular disease continuing to be the major cause of death and disability across the globe. ‘We still have an important mission to prevent cardiovascular disease. Given the strengths of EAS resources, continued collaboration between the IAS and EAS is very much welcomed.’

This year’s Congress has something for everyone. As Congress Co-Chair, Prof. Hans Dieplinger (Innsbruck Medical University, Austria) noted: ‘We have put together a potpourri of science, from basic research to clinical studies.’
The Anitschkow Lecture by the Anitschkow Award recipient is the highlight of the Opening Ceremony, and this year was no exception, with an elegant lecture by Professor Peter Carmeliet (Katholieke Universiteit Leuven, Belgium). While mostly remaining quiescent throughout adult life, blood vessels maintain the capacity to rapidly form new vasculature in response to injury or in pathological conditions, as in cancer. Endothelial cells are key players in this process, angiogenesis. Traditional approaches have focused on studying growth factors and other proangiogenic mediators in order to excess growth. In the context of cancer research, although this direction of research has revealed key mechanisms that regulate tissue vascularization, success in the clinic has been hampered due to insufficient efficacy, refractoriness to treatment, and tumour resistance.

Instead, Dr. Carmeliet overviewed recent findings from his research that implicate maladaptation of endothelial cell metabolism as a contributor to endothelial dysfunction, excess angiogenesis and vessel disorganization, suggesting that this may represent a viable target. Studies have shown that endothelial cells rely mainly on glycolysis, one of the major metabolic pathways that convert glucose to pyruvate, for ATP production, and that glycolysis is further enhanced in angiogenic endothelial cells. The key glycolytic regulator PFKFB3 (6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase-3) is an important determinant of angiogenesis influencing the balance of tip versus stalk cells. Pharmacological blockade of PFKFB3 causes a transient, partial reduction in glycolysis, and reduces pathological angiogenesis with minimal systemic harm. ‘These novel findings imply a paradigm shift in anti-angiogenic therapy from targeting angiogenic factors to focusing on vascular metabolism.’

Not only are there potential therapeutic implications for cancer treatment, but the underlying role of angiogenesis in the pathogenesis of atherosclerosis suggests future possibilities for research into novel approaches for management of cardiovascular disease. Whether targeting PFKFB3 is a valuable strategy in atherosclerosis at the moment remains an unanswered question. Research also suggests the possibility for additional approaches focused in endothelial cell metabolism in atherosclerosis research.

Professor Carmeliet will be discussing this in his Tuesday plenary lecture: ‘Angiogenesis and endothelial cell dysfunction in atherosclerosis: a novel target for treatment.’
N. Anitschkow Hall, 10:00-10:30.

References


Joint Publication:
Eur Heart J. 2016 Apr 26. pii: ehw152. [Epub ahead of print]