

This conference report was compiled by Dr Sandrine Lecour.
Dr Sandrine Lecour is from the Hatter Heart Research Institute, University of Cape Town.
Laureate of the Louis Vogelpoel travelling Scholarship given by the SA Heart Association, Western Cape.

This meeting was held in Bologna, Italy, a city that houses the oldest University in the world (1088). It was not only the Congress of the World Section, it also included the Annual Meetings of the Australasian, European, Japanese and North American sections, which were hosted in equally prestigious Universities in Italy, namely Padua and Ferrara, from 20 to 22 June.

Almost 2 000 delegates and guests came to learn more about all matters relating to the heart from Cell to Man to Society. A very dense program from 8 am till 9 pm was organized in 5 plenary sessions, 56 Frontier symposia, 4 Nobel Laureates and landmark scientific evening lectures, and more than 700 posters. The South African delegation was represented by 9 researchers (11 posters presentations and 2 talks).

Although South Africa belongs to the European section of the ISHR, I elected to attend the American Section meeting that was held prior to the World Meeting in the old Santa Lucia church in Bologna. At that meeting, I was particularly interested in a session on sphingosine 1 phosphate (S1P) and the heart. S1P is a constituent of the HDL particle and is responsible for part of the nitric oxide-mediated vasodilatory effect of HDL. In this session, Prof J Heller Brown, Prof J Karliner and Dr Levkau discussed the potential protective role of various activated S1P receptors isoforms against ischemia-reperfusion. A rapid elevation of S1P-containing HDL plasma levels may be a future therapeutic approach in patients at high risk of acute myocardial infarction.

At the world meeting, various topics were covered with several full sessions on the metabolic syndrome. Prof Yusuf stressed the fact that metabolic syndrome is not a genetic disease but a man-made disease. A novel protein kinase seems to emerge as an important signalling molecule in the heart. Protein kinase D, previously described as protein kinase C μ has emerging myocardial functions such as the control of

cellular growth and survival and, more recently, its role as a regulator of cardiac gene expression (Dr McKinsey) and contractile function (Prof Avkiran).

Several sessions focused on the protection against myocardial ischemia and reperfusion, with particular emphasis on the recently discovered phenomenon of ischemic postconditioning, whereby small episodes of ischemia-reperfusion, administered immediately upon revascularisation, can protect the heart against reperfusion injury. Understanding the protective pathways involved in this very promising therapy may lead to the development of novel drug therapies to limit cell death in coronary heart disease. Some of the work that we presented at the meeting was related to the critical role of the immune system as a mediator in ischemic postconditioning. Interestingly, we have shown that mice deficient in the cytokine tumor necrosis factor (TNF) failed to be protected with ischemic postconditioning, therefore suggesting that activation of TNF is required for ischemic postconditioning to protect the heart. We also demonstrated that the protective effect of ischemic postconditioning seemed to be dependent on age, as older mice failed to be protected.

Meeting abstracts have been published in *Journal Molecular and Cellular Cardiology*, June 2007, volume 42, Supplement 1 and are available online at <http://www.sciencedirect.com/science/journal/00222828>.

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