

Paul Martini Prize 2008 awarded

New approach for atherosclerosis therapy

Wiesbaden, March 31, 2008 (PMS). Findings regarding how vascular cells and leukocytes communicate with each other guide the way to a novel treatment of atherosclerosis and therefore prevention of heart attack and stroke. Today, Prof. Dr. med. Christian Weber and Prof. Dr. rer. nat. Jürgen Bernhagen of the University Clinic of RWTH Aachen were honoured with the Paul Martini Prize for their work in this field. The award celebration took place during the annual meeting of the German Society for Internal Medicine (DGIM) in Wiesbaden. The prize worth EUR 25,000 is awarded every year by the Paul Martini Foundation, Berlin, for outstanding achievements in clinical-therapeutic pharmaceutical research.

Professor Weber is the director of the Institute for Molecular Cardiovascular Research of the University Clinic Aachen and spokesman of the DFG Research Group 809 on "Chemokines and Adhesion Molecules in Cardiovascular Pathogenesis." At the same clinic, Professor Bernhagen is department chair for Biochemistry and Molecular Cell Biology. Together with their working groups, they have been researching the biochemical and cellular processes of atherosclerosis for years.

According to today's understanding, atherosclerosis starts with endothelial dysfunction, induced by metabolic and other influence factors. This is followed by an infiltration of the space between the endothelium and the smooth vascular muscles by various leukocytes. This, in turn, initiates the formation of atherosclerotic plaques and accompanies their pathophysiological progression.

In searching for the causes of the leukocyte infiltration, Weber and Bernhagen realized the key significance of the cytokine MIF (*macrophage migration inhibitory factor*).¹ Among others, it is formed during atherosclerosis-promoting processes of endothelial and smooth muscle cells. The scientists were able to show that MIF effects an adhesion to the endothelium and a chemotactic migration in monocytes and T-cells. This is mediated by

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¹ Bernhagen et al. (2007). MIF is a noncognate ligand of CXC chemokine receptors in inflammatory and atherogenic cell recruitment. *Nature Medicine* 13 (5), 587-595.

the leukocyte receptors CXCR2 and CXCR4, which were newly discovered as signal-transmitting receptors for MIF with high affinity. As a result, MIF is one of the key factors for the leukocyte infiltration.

Furthermore, Weber and Bernhagen were able to show in an animal model that plaques can be reduced in size again and that the inflammatory activity in them will decrease when MIF is neutralized through antibodies. If this could be translated to patients, advanced atherosclerotic lesions could be stabilized and their regression could be initiated.

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However, to realize this novel therapeutic approach, the concept needs to be translated into the development of suitable antagonists for pharmaceutical therapy. The corresponding active ingredient candidates are currently being prepared and will be investigated in collaboration with a company specializing in this area.

The Paul Martini Foundation

This Berlin-based non-profit foundation supports the advancement of pharmaceutical research as well as the research of drug therapy and works to expand the scientific dialog about pharmaceutical research and development between medical scientists at universities, hospitals, the research-based pharmaceutical industry, other research institutions and government agencies. The foundation is sponsored by the German Association of Research-based Pharmaceutical Companies (VFA), Berlin, with its 44 member companies.

The foundation was named after the outstanding scientist and physician from Bonn, Professor Paul Martini (1889 - 1964), in honor of his special achievements and service with regard to the advancement and continued development of clinical-therapeutic research, which he impacted significantly for decades with his "Methods of Therapeutic Examination" published in 1932. The prize awarded annually by the foundation for outstanding clinical research is also named in his honour.