Kolloquium

Biomedizinische Technik und verwandte Gebiete

Sommersemester 2015

Donnerstag, 09.07.2015, 16:15 - 17:00 Uhr

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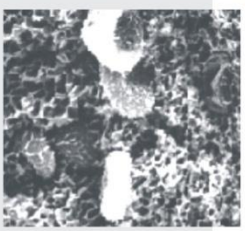
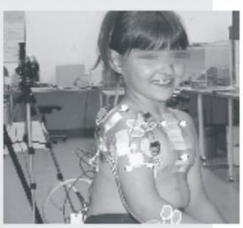
(Moderation: Prof. Dr. Wilhelm Jahnen-Dechent, Institut für Zell- und Molekularbiologie an Grenzflächen (Biointerface) im Helmholtz-Institut für Biomedizinische Technik der RWTH Aachen)





„The Contact System: Novel Perspectives for Interference with Thrombosis and Inflammation“

Abstract:

Combinations of proinflammatory and procoagulant reactions are the unifying principle for a variety of disorders affecting the cardiovascular system. Factor XII (FXII, Hageman factor) is a plasma serine protease that initiates the contact system. This system starts a cascade of procoagulant and proinflammatory reactions via the intrinsic pathway of coagulation, and the bradykinin producing kallikrein-kinin system, respectively. The biochemistry of the contact system in vitro is well understood, however its in vivo functions are just beginning to emerge. This presentation will summarize roles of the FXII-driven contact system in vivo.

Genetically altered mice and large animal models have shown that FXII is essential for thrombus formation while being dispensable for hemostatic processes that terminate blood loss. Challenging the dogma of a coagulation balance, targeting FXII protected from cerebral ischemia and ischemic heart disease without interfering with hemostasis. In contrast, excess FXII activity is associated with a life threatening inflammatory disorder, hereditary angioedema. Platelet polyphosphate (an inorganic polymer), neutrophil extracellular traps (NETs) and mast cell heparin activate FXII with implications on the initiation of thrombosis and edema.

A key aspect of the presentation will be the analysis of common principles, regulation and cross-talk of FXII-driven protease cascades in coagulation and inflammation and its therapeutic implications. Targeting FXII proteolytic activity by the recombinant humanized antibody 3F7 interferes with thrombosis and inflammation in cardiopulmonary bypass system without increasing bleeding risk. Additionally, interference with FXII provides novel perspectives to treat aberrant vascular leakage in Hereditary angioedema and anaphylaxis. Elucidating the FXII-driven contact system offers the exciting opportunity to develop strategies for safe interference with both thrombotic and inflammatory diseases.

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