TWINCORE

TWINCORE – Seminar

Friday March 27th, 2015, 1 p.m.
TWINCORE Lecture Hall

"PAI-1/*SERPINE1* Shapes the Extracellular Environment to Prevent Influenza A Virus Maturation"

Dr. Meike Dittmann

Interferon-stimulated genes (ISGs) act in concert to provide a tight barrier against viruses. Recent studies have shed light on the contribution of individual ISG effectors, but most have examined those acting on early, intracellular stages of the viral life cycle. We applied an image-based screen to identify ISGs inhibiting late stages of influenza A virus (IAV) infection. We unraveled a directly antiviral function for the gene *SERPINE1*, encoding plasminogen activator inhibitor 1 (PAI-1). PAI-1 inhibits IAV glycoprotein cleavage, thereby reducing infectivity of progeny viruses. This was relevant for IAV restriction in vivo. Further, partial PAI-1 deficiency, attributable to a polymorphism in human *SERPINE1*, conferred increased susceptibility to IAV in vitro. Our findings reveal that manipulating the extracellular environment to inhibit the last step in a virus life cycle is an important mechanism of the antiviral response.

Who is Meike Dittmann?

Meike Dittmann received her PhD in Molecular Medicine from Ulm University, Germany, in 2010. During her doctoral studies she investigated antiviral resistance mechanisms of human cytomegalovirus (HCMV). Since joining the Center for the Study of Hepatitis C in May 2011, her research has focused on the role of interferon-stimulated genes (ISGs) as effectors of the innate immune system. She was awarded the Rockefeller Women in Science Fellowship and a postdoctoral fellowship by the German Research Foundation.

Contact: Prof. Dr. Thomas Pietschmann
TWINCORE, Centre for Experimental and Clinical Infection Research, Feodor-Lynen-Str. 7, 30625 Hannover, Phone: 0511 220027 130