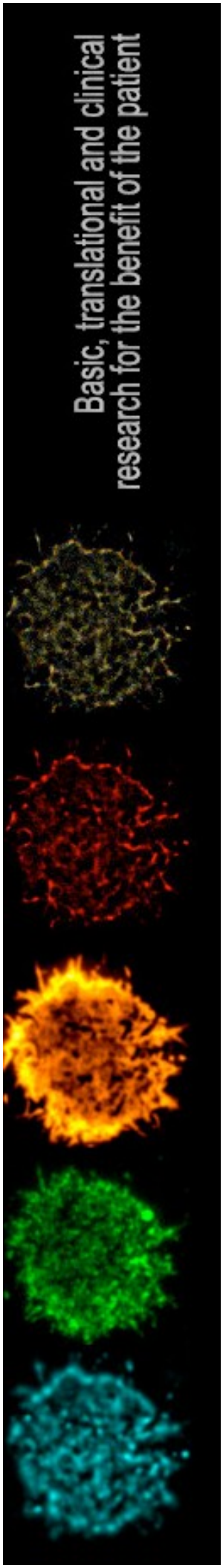


IMMUNOLOGY RESEARCH CLUSTER
MEDICAL UNIVERSITY OF VIENNA

IRC SEMINAR

"Lung epithelia under attack: influenza, interferons and pollutants"

Andreas WACK, PhD

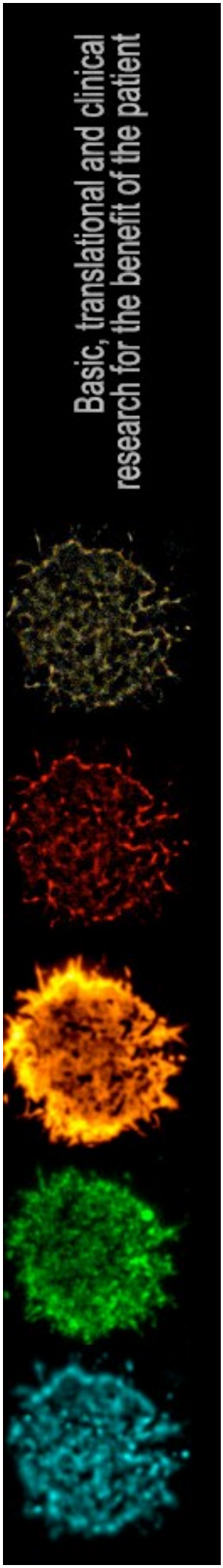
(The Francis Crick Institute, London, UK)

Host: Sylvia Knapp

Tuesday, 24th October 2017
14:00 Uhr

Vienna Competence Center,
Seminar Room, 1st Floor,
Lazarettgasse 19, 1090 Vienna

cluster.meduniwien.ac.at/irc



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Biosketch

After his PhD on thymocyte development at the MRC National Institute for Medical Research (NIMR) in London, Andreas Wack moved to the research institute of Novartis Vaccines in Siena, Italy, where he worked on the modulation of human T and NK cell function by the hepatitis C virus, human dendritic cell subsets and their crosstalk, the mechanism of action of vaccine adjuvants, and next generation influenza vaccines.

Since 2009 he is back at the NIMR, now Francis Crick Institute, where his group studies the responses of airway epithelia and innate immune cells to influenza infection and to influenza-bacterial co-infection. His lab aims to identify determinants of immunopathology and protection and has assessed unique and redundant roles of type I and type III interferons in influenza. A second focus of his group is airway epithelial cell differentiation, a process that has to be efficient and balanced to guarantee timely repair of lung tissue damage during infection.

Selected recent publications

- Villa M, Crotta S, Dingwell K.S, Hirst E.M.A, Gialitakis M, Ahlfors H, Smith J.C, Stockinger B, Wack A.: The aryl hydrocarbon receptor controls cyclin O to promote epithelial multiciliogenesis. *Nature Communications* 7 (2016), 12652.
- Davidson S*, McCabe T*, Crotta S, Gad H.H, Hessel E.M, Beinke S, Hartmann R, Wack A.: IFNλ is a potent anti-influenza therapeutic without the inflammatory side effects of IFNα treatment. *EMBO Mol. Med* 8 (2016), 1099-1112. *equal contribution.
- Ellis G, Davidson S, Crotta S, Wack A.: TRAIL+ monocytes and monocyte-related cells cause lung damage and thereby increase susceptibility to influenza-Streptococcus pneumoniae coinfection. *EMBO Reports* 16 (2015), 1203-18.
- Davidson S, Crotta S, McCabe T, Wack A.: Pathogenic potential of type I interferon in influenza infection. *Nature Communications* 5 (2014), 3864.
- Crotta S, Davidson S, Desmet C.J, Buckwalter M.R, Albert M.L, Staeheli P, Wack A.: Type I and type III interferons drive redundant amplification loops to induce a transcriptional signature in influenza-infected airway epithelia. *PLOS Pathogens* (2013), e1003773.