

## SEMINAR ANNOUNCEMENT

We would like to invite you to attend this seminar hosted by Prof Vinay Tergaonkar:

Date: Wednesday, 27 November 2019  
Time: 11:00AM – 12:00NN  
Venue: Level 3, IMCB Seminar Room 3-46, Proteos, Biopolis

### Exploitation of deubiquitinylases in *Helicobacter pylori*-induced NF- $\kappa$ B and gastric cancer



**Prof. Dr. Michael  
Naumann, PhD**

*Director, Institute of  
Experimental Internal Medicine  
(IEIM),  
Otto von Guericke University,  
Magdeburg, Germany*

#### Abstract

Continuous inflammation causes chronic gastritis and NF- $\kappa$ B regulated cell survival genes could contribute to the development of neoplasia and gastric adenocarcinoma. Herein, high salt intake, sedentary lifestyle and the microbial pathogen *Helicobacter pylori* represent high risk factors for the development of gastric cancer. Prominently, NF- $\kappa$ B is regulated by covalent conjugated proteins, e.g. ubiquitin. This modification is directed by E3 ligases and counteracted by deubiquitinylases (DUBs), which control the transcriptional activity of NF- $\kappa$ B regulated survival genes. Herein, the DUB molecules USP48, A20 and others promote cell survival in gastric tumour cells. In human gastric biopsies of “*H. pylori* gastritis” and “adenocarcinoma” we observed an immunoreactivity. *H. pylori* induces the canonical (RelA/p50) and the non-canonical (RelB/p52) NF- $\kappa$ B pathways, which are regulated by the ubiquitin editing enzyme A20. Further, we studied the impact of therapeutic lead structures against DUBs on cell survival in preclinical studies. Overall, our studies provide novel insights in the molecular pathogenesis of gastric cancer development, which allowed the definition of DUBs as novel predictive biomarkers and promising therapeutic lead structures eventually in combination with chemotherapeutics.

**ALL ARE WELCOME** (No registration required)