

P014 Control of the NF- κ B inhibitor I κ B α in pathogen-infection
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Infection with the human microbial pathogen *Helicobacter pylori* trigger the innate and inflammatory responses in the host by the activation of NF- κ B regulated immune response genes. Chronic infection with *H. pylori* could lead to the development of an invasive gastric cancer type. The *H. pylori*-induced signaling leading to NF- κ B activation comprises the activity of the I κ B kinases (IKK) complex. Upstream of the IKK complex *H. pylori* induces p21-activated kinase 1 (PAK1), NF- κ B-inducing kinase (NIK) and Rip-like interacting caspase-like apoptosis-regulatory protein kinase (RICK). Ubiquitin-dependent degradation of the NF- κ B inhibitor I κ B α leads to nuclear translocation of NF- κ B. We identified a novel mechanism of NF- κ B control in pathogen-infected cells at which *de novo* synthesized I κ B α is rescued from degradation supporting its replenishment.