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UNIVERSITÄT  
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SYSTEMS BIOLOGY

## Seminar

### **Control of intercellular innate immune responses by the p38-MK2/3 kinase system in viral infection and LPS-induced inflammation**

**Tuesday, 21.01.2025, 17:00**

Room 027, Building 28 (G28-027)

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Inflammatory cytokines play a central role in intercellular communication between different cells and tissues during the innate immune response to infection or injury. However, complex cytokine responses can have beneficial or detrimental effects. Thus, infections that lead to increased morbidity or mortality are often associated with an exaggerated inflammatory response of the host. It is therefore of great clinical interest to limit harmful inflammatory processes and at the same time ensure the elimination of the pathogen. A potential target in this context is the intracellular p38-MK2/3-mediated signaling pathway, which is involved in the initiation as well as the resolution of inflammatory cytokine synthesis in response to bacterial or viral pathogen-associated molecular patterns (PAMPs). The presented study uncovers the role of the p38-MK2/3 kinase system and involved downstream effector molecules in regulating the composition of a broad cytokine profile in two experimental models of inflammation and highlights the consequences for innate immunity. These models include the LPS-induced acute-phase reaction in the liver and an acute infection with the murine cytomegalovirus (MCMV).

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