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Viral triggers of tuberculosis disease: a case for cytomegalovirus?

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Objectives
Human cytomegalovirus (HCMV) infection induces profound alterations in immune cell populations crucially involved in the control of Mycobacterium tuberculosis (Mtb) infection. Recent epidemiological studies have suggested a link between HCMV seropositivity and Tuberculosis (TB) disease. We in the Schneider group hypothesize that CMV infection will drive TB disease progression. Therefore, we have established a coinfection mouse model using C57BL/6j mice, and started to analyse the outcome of Mtb infection and Mtb-specific immune responses in the context of an acute or latent murine CMV (MCMV) coinfection.

Methods
C57BL/6j were infected intranasally with MCMV and via the aerosol route with Mtb H37Rv. The outcome of coinfection was monitored by determining bacterial and viral load and clinical signs of disease. Cytokine profiles were analysed to get an impression on changes in the local inflammatory environment during coinfection with multiplex bead array assays. To investigate the impact of MCMV coinfection on pulmonary pathology, one lung lobe was processed for histological analysis in groups of Mtb only, MCMV only and coinfected mice. An in vitro part using bone marrow derived macrophages will be established to analyse possible changes in macrophage phenotype as response to the coinfection.

Results
In contrast to our hypothesis, MCMV coinfection did not interfere with on long-term control of Mtb infection. Rather, virus control seemed to be improved in Mtb infected animals.

Conclusions
We have established an in vivo model which can be used to study immunomodulation during MCMV – Mtb coinfection. The results will improve our understanding of how coinfections interact with and modulate the immune system of the coinfected host.