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Pathogenesis of active MERS-CoV infection in human macrophages

Zheng B J, Zhou J, Chu H, Li C and Yuen K Y
The University of Hong Kong, Hong Kong

Middle East respiratory syndrome coronavirus (MERS-CoV) caused severe pneumonia, multi-organ dysfunction and a higher fatality rate (50% versus 10%) than severe acute respiratory syndrome coronavirus (SARS-CoV). To understand the pathogenesis, we studied viral replication, cytokine/chemokine response and antigen presentation in MERS-CoV-infected human monocyte-derived macrophages (MDMs) versus SARS-CoV-infected-MDMs. Only MERS-CoV can replicate in MDMs. Both viruses were unable to significantly stimulate the expression of antiviral cytokines (IFN- α , IFN- β) but induced comparable levels of TNF- α and IL-6. Notably, MERS-CoV induced significantly higher expression levels of IL-12, IFN- γ and chemokines (IP-10/CXCL-10, MCP-1/CCL-2, MIP-1 α /CCL-3, RANTES/CCL-5, IL-8) than SARS-CoV. The expression of MHC class I and co-stimulatory molecules were significantly higher in MERS-CoV-infected-MDMs than in SARS-CoV-infected-cells. MERS-CoV replication was validated by immunostaining of infected MDMs and ex-vivo lung tissue. We conclusively showed that MERS-CoV can establish a productive infection in human macrophages. The aberrant induction of inflammatory cytokines/chemokines could be important in the disease pathogenesis.