

Apoptosis Induced by MERS-Coronavirus and Discovery of T-Cell MERS-Coronavirus Infection

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Like SARS-CoV infections, Middle-East-Respiratory-Syndrome coronavirus (MERS-CoV) infections occur more frequently in immunocompromised persons, and patients who survive MERS-CoV infections usually have better immune responses than those who die. The association between human immunity and MERS-CoV infection has been well concluded. MERS-CoV is associated with the highest mortality rate among the 6 human known coronavirus. There are substantial abnormal hematological findings, including lymphopenia, thrombocytopenia, coagulopathy and elevated leukocyte numbers, indicating virus infiltration of lymphoid cells and circulating blood. Invasion of the human immune system is indicated by dysregulation of chemokines and cytokines. Markedly decreasing interferon levels in primary human lower respiratory tract cell lines and bronchial epithelium is also demonstrated. Although clinical manifestations as well as in *ex vivo* and *vitro* studies indicates the potential virus dissemination upon MERS-CoV infection, the extra pulmonary involvement has not been confirmed. MERS patients usually show lower type I interferons than those who survive. MERS-CoV can productively replicate in macrophages and dendritic cells that result in dysregulation in antigen-presentation pathway and cytokine. MERS-CoV could persistently induce the expression of proinflammatory cytokines which unassociated with chemotaxis and activation of neutrophils associated with peripheral damage to the surrounding or distant uninfected tissues. MERS-CoV elicits attenuated innate responses with delayed proinflammatory cytokines induction in *in vivo* and cells, which could lead to dysregulated immune responses. Thus, MERS-CoV could dysregulate human innate immunosurveillance on multiple levels. These similar findings have been reported for SARS patients.

MERS-CoV can induce cytokines in T cells, both the intrinsic and extrinsic apoptosis pathways. The unusual capacity of MERS-CoV to infect T cells and induce massive apoptosis might partly lead to its high pathogenicity. MERS-CoV frequently infects T cells from the peripheral blood and from human lymphoid organs, including spleen and tonsil. MERS-CoV can induce cytokines in T cells, both the intrinsic and extrinsic apoptosis pathways. Ineffectual B-cell and T-cell responses with prolonged cytokine expression have been detected in severe cases, whereas a more rapid shut off the innate immune response and potent anti-SARS-CoV antibody response was reported in recovery patients. The anti-SARS-CoV antibody response waned until it was undetectable by 6 years after infection whereas T-cell response could still be detected. Therefore, MERS-CoV vaccines must result in long-term protection against MERS-CoV.

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