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The role of decay accelerating factor in the immunopathogenesis of cytomegalovirus infection

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Abstract: A wide variety of the host immune elements play an influential role in the defence against cytomegalovirus (CMV) infection. However, the role of complement in the clearance of CMV infection is less well studied. Decay accelerating factor (DAF/CD55) is a membrane-bound complement regulatory protein that inhibits the formation and accelerates the decay of C3-convertase. Here we hypothesize that murine CMV (MCMV) utilizes DAF as an immunoevasive strategy through down-regulation of host adaptive responses against the virus. To test our hypothesis, DAF knock-out (DAF KO) C57BL/6 mice and wild-type (WT) littermates were infected with a sublethal dose of MCMV, and their immune responses were compared. WT mice lost 7?8% of their initial weight within the first 4 days after infection and quickly began to recover. This is in contrast to the DAF KO mice, that lost a total of 19?4% of their initial weight and did not start recovery until 6 days post-infection. Flow cytometric analysis of lung digests revealed that infected DAF KO mice had a significantly increased infiltration of inflammatory cells, the majority being CD8+ T lymphocytes. Serum levels of tumour necrosis factor (TNF)-? and interferon (IFN)-? were also increased markedly in the DAF KO mice compared to the infected WT mice. More interestingly, increased viral genome copies (DNA) in the splenocytes of DAF KO mice was accompanied with mRNA transcripts in the DAF KO mice, an indication of active viral replication. These data suggest an intriguing effect of reduced DAF expression on host responses following in vivo MCMV infection.