

Murid herpesvirus 4 infection protects mice from the development of an anti-pneumovirus vaccine-induced TH2 immunopathology

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Gammaherpesviruses are highly prevalent pathogens that establish lifelong latency. However, little is known about how these viruses imprint the immune system of their host. Here we used Murid herpesvirus 4 (MuHV-4) to investigate the impact of gammaherpesvirus infections on the development of an anti-pneumovirus vaccine-induced Th2-skewed immunopathology. Briefly, this respiratory hypersensitivity was induced in mice by a subcutaneous vaccination with formalin-inactivated antigens of pneumonia virus of mice (FI PVM) followed by an intranasal infection with wild-type PVM. We have observed that MuHV-4 infection, either before or after the FI PVM vaccination, prevented the development of the PVM-induced immunopathology while the protection against PVM infection was unaffected. This protective impact against the immunopathology was maintained over time and required pulmonary MuHV-4 replication. Altogether, these results open perspectives for vaccination against pneumoviruses and highlight that some so-called pathogens could be revealed in the end as beneficial for their host.