ABSTRACT:

The porcine reproductive and respiratory syndrome virus (PRRSV) causes large economic losses in pork production because of its ability to persist and spread in herds despite vaccination. An essential feature of the infection is a strong but ineffective antibody response with limited immunological memory. The ability of the virus to dysregulate the host immune system is explained by various mechanisms, including antibody-dependent enhancement of infection, polyclonal activation of B cells by the superantigen effect, the presence of immunodominant epitopes, inhibition of nonspecific immunity, or a different role of cytotoxic T cells. Recent evidence suggests that PRRSV affects the nascent T cell repertoire in the thymus and induces tolerance to viral epitopes that are crucial for virus neutralization. Specific T helper cells are absent and cannot initiate an adequate B cell response to produce high-affinity virus-neutralizing antibodies. However, the remaining T helper cells are not affected and help to produce anti-PRRSV antibodies, which are ineffective and cannot eliminate the virus. The cytotoxic T cells needed for the elimination of infected cells are also affected. This bachelor thesis describes the various features of PRRSV infection and compares the known information on the immune response against the virus to show that the proposed mechanism can explain seemingly contradictory observations.

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KEY WORDS:

Porcine reproductive and respiratory syndrome virus (PRRSV), adaptive immune response, T cell, B cell, thymocyte, thymus, antibody, dysregulation.