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African swine fever virus infection in Classical swine fever subclinically infected wild boars

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Abstract

Background: Recently moderate-virulence classical swine fever virus (CSFV) strains have been proven capable of generating postnatal persistent infection (PI), defined by the maintenance of viremia and the inability to generate CSFV-specific immune responses in animals. These animals also showed a type I interferon blockade in the absence of clinical signs. In this study, we assessed the infection generated in 7-week-old CSFV PI wild boars after infection with the African swine fever virus (ASFV). The wild boars were divided in two groups and were infected with ASFV. Group A comprised boars who were CSFV PI in a subclinical form and Group B comprised pestivirus-free wild boars. Some relevant parameters related to CSFV replication and the immune response of CSFV PI animals were studied. Additionally, serum soluble factors such as IFN-α, TNF-α, IL-6, IL-10, IFN-γ and sCD163 were analysed before and after ASFV infection to assess their role in disease progression.

Results: After ASFV infection, only the CSFV PI wild boars showed progressive acute haemorrhagic disease; however, the survival rates following ASFV infection was similar in both experimental groups. Notwithstanding, the CSFV RNA load of CSFV PI animals remained unaltered over the study; likewise, the ASFV DNA load detected after infection was similar between groups. Interestingly, systemic type I FN-α and IL-10 levels in sera were almost undetectable in CSFV PI animals, yet detectable in Group B, while detectable levels of IFN-γ were found in both groups. Finally, the flow cytometry analysis showed an increase in myelomonocytic cells ($CD172a^+$) and a decrease in $CD4^+$ T cells in the PBMCs from CSFV PI animals after ASFV infection.

Conclusions: Our results showed that the immune response plays a role in the progression of disease in CSFV subclinically infected wild boars after ASFV infection, and the immune response comprised the systemic type I interferon blockade. ASFV does not produce any interference with CSFV replication, or vice versa. ASFV infection could be a trigger factor for the disease progression in CSFV PI animals, as their survival after ASFV was similar to that of the pestivirus-free ASFV-infected group. This fact suggests a high resistance in CSFV PI animals even against a virus like ASFV; this may mean that there are relevant implications for CSF control in endemic countries. The diagnosis of ASFV and CSFV co-infection in endemic countries cannot be ruled out and need to be studied in greater depth.

Keywords: CSFV, CSF postnatal persistent infection, Subclinical CSF, ASFV, Wild boars, Viral load, Innate immune response, Adaptive immune response, Disease

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