

Differences in replication kinetics and cell tropism for genotype I and II ASFv strains

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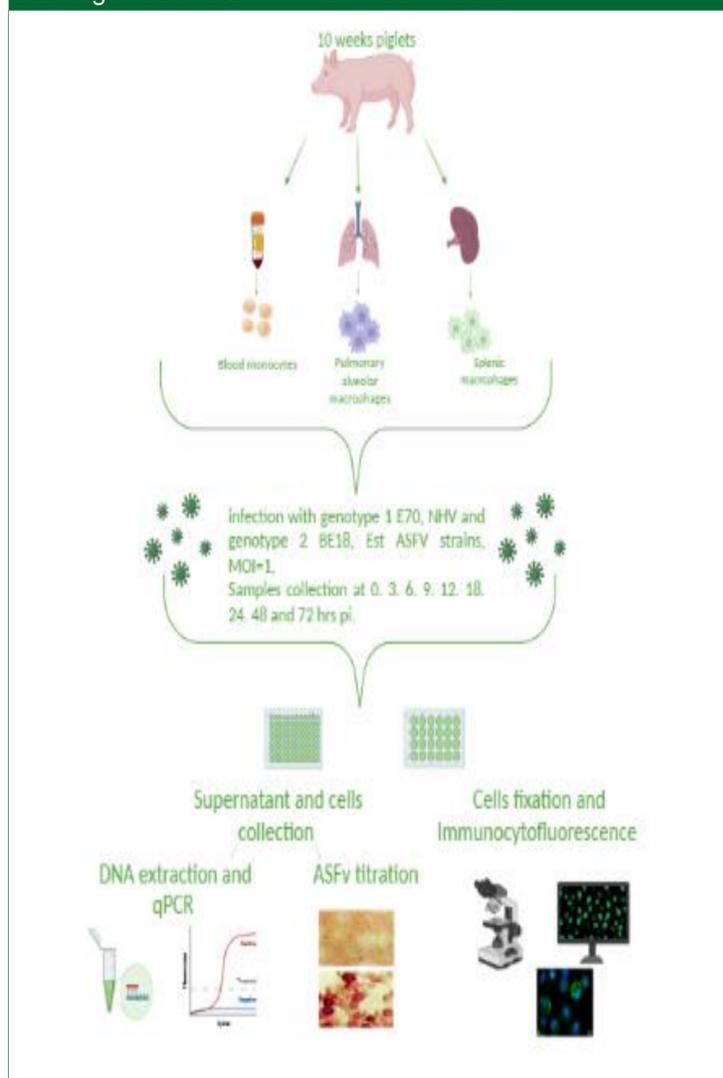
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African swine fever presents a threat for animal health and pig industry worldwide. The causative agent, African Swine Fever Virus (ASFv), is a large and complex DNA virus. The clinical signs of domestic pigs (*Sus scrofa domesticus*) after infection vary from subclinical disease to severe haemorrhagic disease with up to 100% mortality. Different virus strains are an important influencing factor determining the clinical patterns. ASFv has a tropism for macrophages, which seems to play a crucial role in disease pathogenesis and viral dissemination (Razzuoli et al., 2020).

Differences in the kinetics and cell tropism between genotype 1 and 2 strains and/or attenuated and virulent strains may explain some differences in pathological outcomes observed *in vivo* between strains.

Methods

Collection of monocyte/macrophage subpopulations of 3 different tissues
 → infection of subpopulations with 4 different virus strains Belgium 2018 (BE18), E70, Estonia (EST), NHV at MOI=1
 → qPCR/titration by IPMA on supernatants and cells, IF staining to estimate the % of infected cells

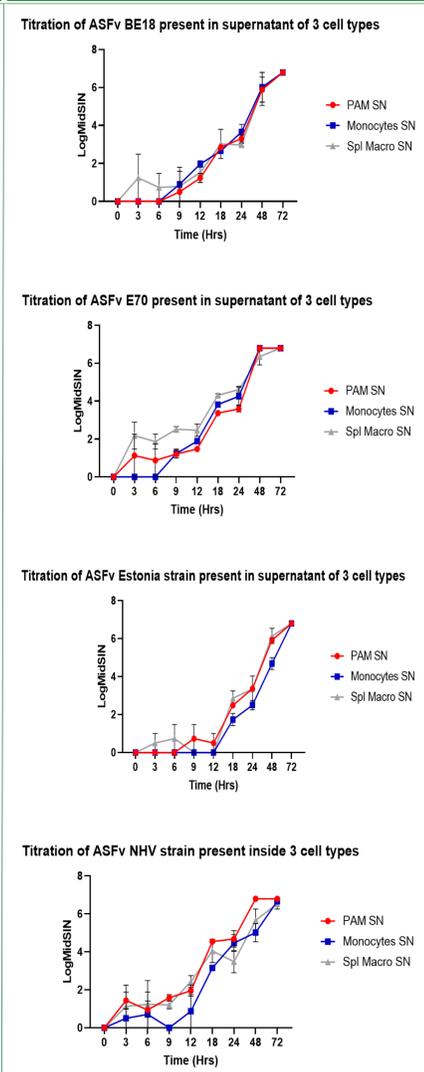
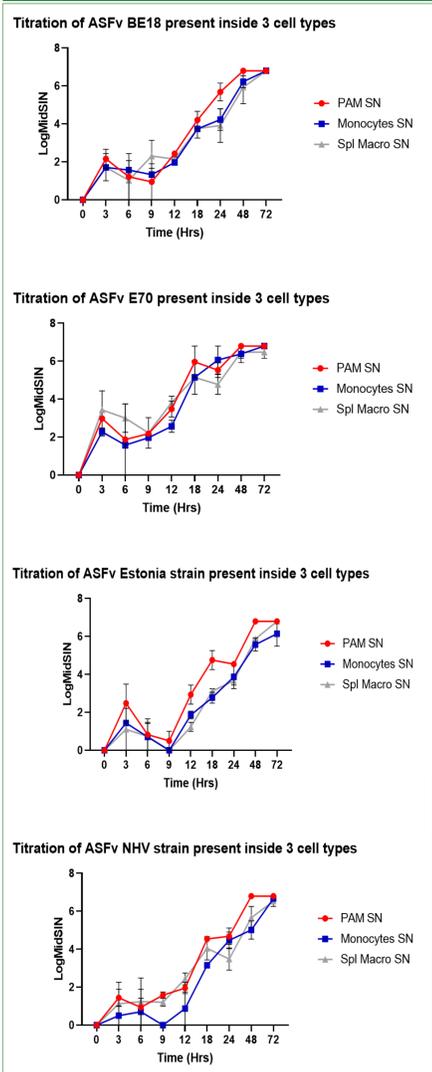


Results

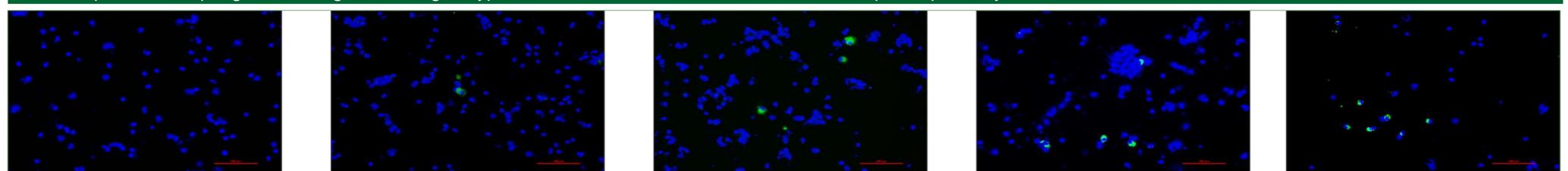
(Intra-) cellular virus titration: Virulent strains have tendency to present higher titers in first timepoints post-infection (p.i.) compared to the attenuated strains

Extracellular virus titration: viral titers in monocytes tend to rise earlier in the viral strains compared to the attenuated strains

Percentage of infected cells: virulent strains infect a higher percentage of monocytes in later timepoints compared with the attenuated strains. BE18, E70 and EST infect a higher percentage of splenic macrophages compared with NHV



Infected splenic macrophages with Belgium 2018, genotype 2 virulent strain at 9,12,18,24 and 48 hours p.i. respectively



Conclusion

Virulent strains tend to have higher titers in early timepoints p.i. compared to the attenuated strains in the (intra-) cellular environment.

In monocytes, titers of the virulent strains rise faster in the extracellular environment compared to the attenuated strains. Virulent strains infect a broader number of monocytes in the late timepoints compared with the attenuated strains. This large number of infected cells explain high viremia and dissemination potential described *in vivo* in more virulent strains.

Reference

Razzuoli et al., "Modulation of Type I Interferon System by African Swine Fever Virus."

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