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KINETICS OF EXPRESSION OF CD163 AND CD107A IN THE LUNG AND TONSILS OF PIGS AFTER INFECTION WITH PRRSV-1 STRAINS OF DIFFERING VIRULENCE

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Introduction

Porcine Reproductive and Respiratory Syndrome (PRRS) is one of the most important diseases of the porcine industry worldwide. Highly pathogenic strains (HP-PRRSV) causing atypical outbreaks have been notified, stressing the marked genetic and antigenic viral variability, which has encouraged the interest to understand the immunobiology of PRRSV strains of differing virulence. Porcine alveolar macrophages (PAMs) are the primary target cells for PRRSV replication, in fact, the scavenger receptor CD163 of PAMs is the main surface receptor of PRRSV, allowing its internalization. On the other hand, it has just been reported the role of CD107a as a marker for activated and degranulated macrophages.

Material & Methods

This study assesses the expression, distribution and kinetics of PRRSV antigen, CD163 and CD107a in lung and tonsil tissues of an experimental infection with three different PRRSV-1 strains: Lelystad (PRRSV-1 prototype); 215-06 (British field strain); and a HP-PRRSV strain (SU1-bel). Animals were euthanized at 3, 7 and 35 days post-infection (dpi). Lung and tonsil samples were processed for histopathological and immunohistochemical studies by using specific antibodies against PRRSV, CD163 and CD107a.

Results

SU1-bel caused the most severe lesions and the widest viral distribution in the lungs as well as in the tonsils. These animals displayed larger number of $CD163^+$ macrophages at 35dpi in both tissues than other strains ($P<0.05$). The molecule CD107a was mainly expressed in the cytoplasm of PAMs and septal macrophages, showing statistically significant differences between infected and control groups at 35 dpi.

Discussion & Conclusion

The virulence of PRRSV-1 strains under study was able to modify the kinetics of expression of CD163, suggesting that SU1-bel may cause a higher predisposition to suffer PRRSV reinfection. In case of CD107a, regardless of the virulence of strains, the number of $CD107a^+$ cells increased, proposing that CD107a might play a key role in macrophages activation.