



VIRAL DISEASES

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CHANGES OF T AND B LYMPHOCYTES IN TISSUES AND BLOOD OF PIGS EXPERIMENTALLY INFECTED WITH AN ITALIAN HIGHLY PATHOGENIC PRRSV-1.1 ISOLATE

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Introduction

Highly pathogenic (HP) PRRSV recently emerged in Europe. The disease is characterized by high fever and respiratory distress in young pigs and high mortality rates and reproductive failure in sows. HP-PRRSV isolates can alter the pathological outcome differently from "conventional" PRRSV, in terms of more severe clinical signs and more intense dysregulation of cellular immunity.

Materials and methods

Nine 70-day-old pigs, treated as follows: 3 intranasally infected 35 days earlier with the HP Italian PRRSV-1.1-PR40 isolate, 3 with the "conventional" Italian PRRSV-1.1-PR11 isolate, 3 kept uninfected, were sampled for lungs, bronchial lymph-nodes and blood to determine T and B lymphocyte tissue distribution and major T and B subsets in blood. Samples were paraffin embedded, EE and ABC stained by immunohistochemistry for CD3 and CD79 α . Blood immune cells were quantified by flow cytometry after staining for CD3, CD4, CD8 α , CD8 α , CD16, TCR $\gamma\delta$ and CD21.

Results

In PR40 pigs, lymph nodes showed severe CD79 α + cell depletion, with positive cells located mainly around secondary follicles, while CD3+ cells were slightly more numerous in the paracortical area. PR11-infected pigs showed a less severe lymph-node depletion, with higher CD3 and CD79 α expression. In lungs, PR40 animals showed absent BALT activation, with scarce CD3+ cells, and very few CD79 α + cells scattered around bronchi.

In blood, CD3+ T cells were decreased in infected groups compared to controls (p<0.05). NKT cells were comparable with controls while TCR $\gamma\delta$ + CD8 α - (p<0.05) and CD8 α + T lymphocytes were lower. A strong decrease was observed also for CD4+CD8 α - T helper lymphocytes and especially for CD21+ B cells (p<0.05). CD4+CD8 α + memory and CD4-CD8 α / β + cytotoxic T lymphocytes were higher in PR40 pigs only (p<0.05).

Discussion and conclusion

PR40 induced more severe immunosuppression than PR11 in target organs, influencing the amount of circulating T, but mostly B lymphocytes, resulting in insufficient activation of cellular immunity.